Color Experience in Blindsight?

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Abstract:

Blindsight, the ability to blindly discriminate wavelength and other aspects of stimuli in a blind field, sometimes occurs in people with lesions to striate (V1) cortex. There is currently no consensus on whether qualitative color information of the sort that is normally computed by double opponent cells in striate cortex is indeed computed in blindsight but doesn't reach awareness, perhaps owing to abnormal neuron responsiveness in striate or extra-striate cortical areas, or is not computed at all. The existence of primesight, the experience of colored

afterimages in blindsight, has been taken to suggest that qualitative color information is computed either in pre-striate or striate cortical areas but is not broadcast to working memory. I argue here that a recent study in which color phosphenes were induced in a blindsighter using bilateral transcranial magnetic stimulation indicates that computations necessary for conscious color vision are lost in blindsight. Owing to this loss, the neural responsiveness in extrastriate cortical areas is abnormal and hence is unable to give rise to color awareness. Blindsight is thus degraded vision in which the computations necessary for conscious color vision have been lost.

0. Introduction

Blindsight is a kind of residual vision found in people with lesions to the primary visual cortex. Blindsighters typically report no visual awareness but they are nonetheless able to make above-chance predictions about the shape, location, color and movement of visual stimuli presented to them in their blind find.[1]

The issue of whether blindsight really is blind has been the subject of much controversy. Paul Azzopardi and Alan Cowey (1997) used a sophisticated signal detection analysis experiment to show that blindsight is not like degraded normal vision. A similar argument, based on comparisons between discrimination performance and levels of awareness in sighted and blind fields was made by Larry Weiskrantz (2009) based on the results of Robert Kentridge, Charles Heywood & Weiskrantz (1999). More recently Morten Overgaard et al (2008) and Mark Christensen et al. (2008) have argued that the current methods for reporting visual awareness do not suffice for determining visual awareness. They conducted a series of studies using improved measures for determining visual awareness and argued on the basis of these studies that blindsight is indeed severely degraded conscious vision. However, their method of testing for subjective awareness assumes that reported clarity correlates with visual awareness, an assumption which is highly questionable.

[1] As most cases of blindsight are unilateral, blindsighters tend to have a sighted and a blind field.

Despite the controversy over what counts as visual awareness, researchers do agree that if blindsight is conscious sight, the awareness it gives rise to is remarkably different from the awareness that arises in normal sight. While blindsighters are able to reliably discriminate wavelength in some cases, they do not normally report color experiences, or color qualia.[2]

There are two main explanations of the lack of color qualia in blindsight. One explanation is that qualitative color information is not computed in blightsight owing to the lesions in the primary visual cortex. Another is that qualitative color information is computed elsewhere (e.g. in the LGN, superior colliculus or extrastriate regions) but that feedback, or feed-forward, to the primary visual cortex is needed in order for the qualitative color information to reach awareness.

By 'qualitative color information' I mean primarily the information generated when neurons (in, for example, the primary visual cortex) compare color inputs of parts of a scene to adjacent parts in terms of light intensity and wavelength (double-opponent processes).[3] The color phenomenology of normal color experience most likely arises partially as a result of double opponent cells computing qualitative color information by virtue of the structure of their

[2] Here and throughout I shall use the term 'qualia' in a relatively non-committal way to refer to the phenomenal properties of mental states (what it is like to be in that mental state).

[3] In normal subjects double-opponent cells in the primary visual cortex perform this particular task. Qualitative information processing in blindsight could be mediated by the compromised V1 cortex if sufficiently many neurons remain functional (Stoerig and Cowey 1989). However, the superior colliculus or other sites in the thalamus, hypothalamus or midbrain (via direct retinal projection) could also be performing some of the qualitative functions normally performed by V1 (Danckert and Goodale 2000, Ro 2008). Wavelength discrimination is believed to take place in thalamo-extrastriate neural pathways (involving V4) (Danckert et al 1998).

receptive fields.[4] This information, then, is broadcast through the ventral stream to working memory in pre-frontal cortical areas.[5]

While it is notoriously difficult to offer a proper conceptual characterization of 'qualitative color information', there is strong evidence to suggest that qualitative color information is the output of computational processes that correlate with the (perceived) brightness of a visual stimulus (the subjective counterpart of luminance).[6] Antony Morland et al (1999) made comparisons between stimuli presented in the blind and sighted hemifields and within the blind hemifield alone in blindsight subject G.Y. The studies demonstrate that G.Y. had "conscious access" to motion, luminance and chromatic variations of the drifting gratings but GY was not phenomenally aware of the stimulus, which shows that there was a qualitative difference between the percepts he derived from the normal and blind hemifields. It was concluded that G.Y. was "normal" in terms of his perception of luminance and chromatic color but was unable to see the stimulus presented to him in his blind field because the detection of the stimulus' luminance didn't lead to an appearance of brightness. Apparently the further processing of information from pre-striate brain regions in striate cortical areas is necessary for brightness to be perceived. If this is right, then it would seem that blindsighters are blind to color because they do not compute all the components relevant to color awareness (hue, saturation, and brightness) and hence do not compute color (understood as a product of hue, saturation, and brightness).

Recent studies confirm this hypothesis. Juha Silvanto and his colleagues conducted a series of studies in which colored phosphenes were induced in blindsight subject G.Y. via

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[4] Chromatic probably cannot be experienced without the processing of double-opponent cells, but the absence of color experience in patients retaining V1 but with lesions elsewhere suggests that activation of double-opponent cells in V1 doesn't give rise to chromatic color experience. The double opponent cells send information elsewhere and these receiving areas (or areas receiving signals from them in turn) can't normally produce color experience without an intact double-opponent system. I owe this clarification to the reviewers.

[5] Following Block (2008), I take 'working memory' to mean 'memory that has to be actively maintained'. Working memory contains the information that is directly accessible to us for reporting.

[6] Brightness is the perceived intensity of light reaching the eye, and luminance is the actual intensity of the light.

bilateral transcranial magnetic stimulation (Silvanto et al. 2008). The studies showed that whether a colored phosphene was induced in G.Y.'s blind field depended exclusively on processes in his contralesional hemisphere. The researchers took this to suggest that the lesions to the primary visual cortex compromised normal neural responsiveness in extrastriate areas (Silvanto 2008). I argue here that the empirical data gathered in this study also show that qualitative color information was not computed in G.Y.'s ipsilesional hemisphere, which explains the abnormal neural responsiveness in extrastriate areas. I go on to question the researchers' hypothesis that the findings indicate that the primary visual cortex is not a neural correlate of visual awareness.

1. Blindsight

D.B., the first extensively tested blindsight subject, was found to be able to make above-chance predictions about the location and movement of objects, but without any visual awareness (Weiskrantz 1998). Weiskrantz coined the term 'blindsight' to refer to the unconscious processes responsible for visual performances in forced-choice tasks involving discriminations of simple stimulus properties such as location, contrast, orientation, and color in people with lesions to the primary visual cortex. Because blindsighters are effectively blind despite performing remarkably well in forced-choice paradigms, the blindsight phenomenon offers strong support for the hypothesis that the primary visual cortex is part of the neural correlate of visual awareness. Since the first blindsight studies of D.B. numerous other subjects with blindsight have been tested. Most blindsight subjects tested have been found to have spectral sensitivity to wavelength (Stoerig and Cowey 1989) and the ability to reliably discriminate wavelength of stimuli in their blind field (Stoerig and Cowey 1992). Thus, while there typically is some degeneration of thalamic color-processing mechanisms (LGN) in blindsight patients, neurons which process information about wavelength are apparently still functional.

One of the most recent discoveries in the blindsight arena is that blindsight can be temporarily induced in normal subjects with transcranial magnetic stimulation (TMS) applied over visual cortex (Boyer, Harrison and Ro 2005, Lamme 2006). Transcranial magnetic stimulation has been known to cause phosphenes when applied over the early parts of the

visual system (the occipital lobe), but when TMS (at particular intensities) is applied over the visual cortex right after presenting the visual stimulus, the visual cortex is temporarily disrupted and the information from the retinotopical representation does not reach visual awareness. Subjects in whom blindsight has been induced with TMS have been shown to have quite remarkable abilities to detect color and other aspects of stimuli presented to them in their blind field (Boyer, Harrison and Ro 2005). They report having no visual awareness of the stimulus.

Studies using normal subjects in which blindsight has been magnetically induced have the advantage over traditional studies that they control for the possible rewiring of the brain, a phenomenon known to take place in some blindsight patients (see e.g., Gazzaniga, Fendrich and Wessinger 1994). On the other hand, TMS-induced blindness studies are more difficult to get right, as the possibility that structures in the visual cortex compute the information that allows for above-chance performance on forced-choice tests must be eliminated.

Unexperienced blindsighters are unable to use their vision to navigate the world on their own. They need prompting by an experimenter who tells them when to make a guess about a stimulus. As Ned Block (1995) points out, blindsight differs from normal sight in more than one way. Not only does it not give rise to a visual phenomenology in the presence of a stimulus, but it also requires prompting for the computed information to reach working memory and hence for blindsight to have what Block calls 'access consciousness' to the visual stimulus. As David Milner and Melvyn Goodale (2001) suggest, the prompting is plausibly required because most of the information from the retina in blindsighters ends up in cortical structures such as the parietal lobe and the primary motor cortex, and normal blindsighters haven't learned to act verbally on unconscious cuing from motor programming.

However, Block imagines a case of a super-blindsighter who has acquired the ability to guess when to make a guess about stimuli in his blind field.[7] From a third-person point of view a very good super-blindsighter would be hard to distinguish from a normal subject with bad vision. But there would be an internal difference between the two. The super-blindsighter would have no visual awareness associated with his vision. The subject with bad vision would

[7] A recent debate about the philosophical implications of these kinds of cases can be found in Carruthers (2001) and Siewert (1998, 2001).

have visual awareness, even if his experience would be blurry and not allow him to make all the right predictions about his external environment.

No case of a real super-blindsighter has been found, but there are blindsight subjects with improved abilities. Some of the first blindsight subjects to be tested have since then gone through extensive testing by numerous researchers and have read a lot of the literature on blindsight. Some of these individuals (e.g., G.Y.) have acquired the ability to guess when to make a guess about a stimulus in their blind field in experimental settings (Sahraie et al., 2006), or show other remarkable capacities (in terms of e.g. accuracy) (e.g. D.B.) (Trevethan, Sahraie and Weiskrantz 2007, Kentridge, Heywood and Weiskrantz 2007). Training has been shown to have similar effects in cases of induced blindsight (Schwiedrzik, Singer, and Melloni 2008). In experimental settings trained blindsighters may thus come close to satisfying Block's criterion for being super-blindsighters.

Despite extensive training, however, blindsight subjects do not seem to gain visual awareness over time (Zeki and ffytche 1998, Cowey 2004). They can make the relevant predictions about stimuli in their blind field but their subjective reports suggest that the "unseen" stimuli do not normally give rise to experiences with a distinctly visual phenomenology (Lau and Passingham 2006, Persaud, McLeod and Cowey 2007, Persaud and Cowey 2008, Persaud and Lau 2008, Rees 2008, Ro 2008, Weiskrantz 2009).

2. Feelings but no Qualia

There is, however, a lot of controversy surrounding the hypothesis that blindsight subjects do not have experiences with a distinctly visual phenomenology. Blindsight subjects have been taken to report feelings in some cases in which they are asked to make predictions about stimuli in their blind find ("a feeling of something happening"). But there is no consensus about whether these feelings are indicative of a visual phenomenology or arise through the exercise of the blindsighters' ability to identify visual stimuli, though most researchers lean towards the latter hypothesis (Sahraie et al. 1997, Barbur, Weiskranz, Harlow 1999, Kentridge and Heywood

1999). The phenomenology is cognitive and high-level ("a feeling of something happening") rather than perceptual and low-level.

The fact that some blindsight subjects report having feelings in conjunction with the presence of an "unseen" stimulus has led some researchers to question the methods used for testing for visual awareness. In traditional blindsight studies binary methods were used for testing ("do you see something?"). This was then followed up with questions about the subject's certainty about his subjective report. It has been argued that these tests may not be sufficiently sensitive for the gathering of subjective reports (Morland et al. 1999, Stroerig and Barth 2001, Ro et al. 2007). One problem is that the subject and the researchers may mean different things by 'see'. A second problem is that the subject and the researchers, even if they mean the same thing by 'see', may have different thresholds for what counts as "seeing something". A third problem is that the traditional binary methods ask about whether the subject "sees" anything, not about the phenomenology of his experience (if any) (Lau and Passingham 2006).

New more fine-grained methods have been developed for testing for visual awareness (Overgaard et al. 2008, Christensen et al. 2008). Based on prior testing on normal subjects, one team of researchers developed a four-point scale, the 'Perceptual Awareness Scale' (PAS), for testing visual awareness in blindsight (Overgaard et al. 2008).[8] The (PAS) method tests for clarity of the visual stimulus. On a series of trials on blindsight subject G.R., using the (PAS) method, not nearly as many stimuli were reported as being clear as in the trials on her normal field, but it was found that accuracy correlated with reported visual clarity of the stimulus. The relationship between accuracy and awareness was the same in the intact and the blind fields. Reported awareness thus is predictive of accuracy. When comparing the trials, where standard methods were used, to the trials using the PAS method, it was evident that G.R.'s threshold for reporting awareness was lower when the PAS method was used than when standard methods

[8] (CI) "clear image" ("I know what was shown"), (ACI) "almost clear image" ("I think I know what was shown") (WG) "weak glimpse" ("something was there but I don't know what"), and (NS) "not seen".

were used (Overgaard et al. 2008). The researchers concluded that blindsight is not unconscious vision but severely degraded conscious vision.

However, while these findings are interesting, it is not clear that reported clarity of stimulus is a good measure of visual awareness. A stimulus could be reported as clear in virtue of allowing the subject to blindly detect it. The scale does admittedly say "clear *image*". However, subjects may not have a good sense of the difference between an image and a thought (see Hurlburt and Schwitzgebel 2007: 61). Furthermore, even if a stimulus perhaps does give rise to an experience with a "clear" phenomenology, this does not provide any evidence of visual awareness of color, shape, or location, though PAS could perhaps be adopted to gauge, for example, visual awareness of color.[9]

Other studies have been conducted in which the highly experienced blindsubject G.Y. was asked what it was like for him when a visual stimulus was present in his blind field (Morland et al. 1999, Stroerig and Barth 2001). But these studies have been criticized on the grounds that they presuppose that there is something it is like for G.Y. when a stimulus is present. The studies furthermore used moving stimuli, which G.Y. is known to have been aware of (Persaud and Lau 2008). The first criticism is not quite to the point, for arguably there is something it is like for a visual stimulus to be present in the blind field. Certainly there is something it is like to make a qualified guess about a visual stimulus. But the fact that there is makes methods that ask blindsighters "what it's like" questions less than sufficiently sensitive for testing for visual awareness.

Navindra Persaud and Hakwan Lau recently decided to educate G.Y. on phenomenal consciousness (Persaud and Lau 2008). G.Y. was given definitions of 'qualia' from *The Oxford Companion to the Mind*, *The Stanford Encyclopedia of Philosophy*, *Consciousness Explained* by Daniel Dennett and "Epiphenomenal Qualia" by Frank Jackson. He read them at his leisure. He was then tested for blindsight and was questioned afterwards about whether he understood the notion of qualia, and whether he 'experienced qualia'. G.Y reported that he thought he understood the notion but didn't understand what was meant by 'ineffable' and 'private'. To

[9] Thanks to an anonymous referee here.

the second question he first responded that he never has qualia in his right (affected) hemifield in everyday life and only 'very rarely' has qualia in his right hemifield when performing well in experimental conditions. But the researchers' follow-up questions prompted him to retract the latter claim. They concluded that G.Y. never 'experiences qualia'.

While the research reviewed in this section does not conclusively establish that blindsight subjects do not have visual experiences, it does seem to be an indicator of a lack of visual awareness of color and other aspects of the visual stimuli presented to blindsight subject in their blind fields. Ned Block has famously argued that lack of access to information does not entail lack of phenomenal consciousness (1995, 2007, 2008). Granting Block's distinction, one could argue that subjects with blindsight are phenomenally conscious of colors, shapes, orientation, and so on, but unable to access the information. However, I argue below that qualitative color information, needed to experience color, is most likely not computed in normal cases of blindsight. Hence, in blindsight visual memory will not normally contain the color information needed for phenomenal awareness to arise in the blind field.

3. Qualitative Information Processing

If blindsight subjects who can reliably discriminate wavelength do not normally have color experiences, then we may reasonably ask what explains this lack of color experience (or color qualia). In normal subjects color qualia arise in part because the visual system computes qualitative information on the basis of a retinal input. Blindsight subjects have lesions to the primary visual cortex, which is believed by some to be a neural correlate of visual awareness (Pascual-Leone and Walsh 2001, Silvanto et al. 2005, Lamme 2001). But they can in some cases reliably discriminate wavelength and other aspects of visual stimuli in their blind field. So, other (retinal, midbrain, or extrastriate) regions of the visual system must be involved in processing information about the visual stimulus. The question thus arises whether qualitative color information is computed by other parts of the visual system in the case of blindsight but is not broadcast to working memory or is not computed at all.

Uriah Kriegel (2009: 310-111) has recently argued that the visual system does compute qualitative information in the case of blindsight. His argument runs as follows. Some blindsight patients have primesight (blindsight subject D.B.) (Weiskrantz et al. 2002, Schärli, Harban and Hogben 1999). Primesight is the experience of after-images with contrary (or in some cases: the same) color after long exposure to color patches and luminance changes. But it seems that color experience cannot be grounded in purely quantitative color information (e.g., non-comparative information about wavelength or luminance). So, by an inference to the best explanation, some blindsight subjects have intact visual areas that process qualitative information.

The argument is a good theoretical argument for thinking that qualitative information is processed by V1-exterior regions in blindsight subjects but simply is not broadcast to working memory. Blindsight subjects have the information relevant for visual awareness but cannot access it. Or if you prefer: Blindsight subjects have the information relevant for visual awareness but the information never reaches awareness.

Kriegel's argument needs to be modified slightly, for as we will see below, it is possible that qualitative information computed by the visual areas of the intact hemisphere can give rise to visual awareness in the hemifield corresponding to the damaged hemisphere. So, I suggest that we reformulate Kriegel's argument as follows: (1) The visual system in the damaged hemisphere of some blindsight subjects computes information that gives rise to qualitative after-images in the corresponding blind field. (2) It is unlikely that non-qualitative information computed in the damaged hemisphere can give rise to qualitative after-images in the corresponding blind field. Hence, by an inference to the best explanation, (3) the visual system in the damaged hemisphere of some blindsight subjects computes qualitative information. However, I will now argue, (3) is not the best explanation of (1) and (2).

4. Magnetically Induced Color Phosphenes

In a recent study carried out by Juha Silvanto and colleagues colored phosphenes were induced in blindsight subject G.Y. by transcranial magnetic stimulation (TMS) applied over visual area V5/MT (Silvanto et al. 2007, Silvanto, Cowey and Walsh 2008, Silvanto 2008). TMS is known to induce colorless phosphenes (flashes of light) when applied to the early parts of the visual system (the occipital lobe) of normal subjects. In one study conducted by Silvanto's team unilateral stimulation applied over V5/MT regions in G.Y.'s intact hemisphere induced a moving white phosphene spanning the sighted hemifield, and bilateral stimulation of V5/MT induced a continuous white arc spanning both hemifields (Silvanto et al. 2007). Unilateral stimulation over V5/MT areas in the ipsilesional hemisphere never induced a phosphene. It was determined that it was an increase in TMS intensity over the intact hemisphere that was crucial for inducing phosphenes in G.Y.'s blind field.

In a subsequent study G.Y. and three control subjects were exposed to a colored sheet of paper (either uniformly colored or bicolored) (Silvanto et al. 2008). With visual adaptation to a uniformly colored stimulus, unilateral stimulation of G.Y.'s normal hemisphere or one hemisphere in the control subjects induced a phosphene in the corresponding hemifield that had the same color as the stimulus. Bilateral stimulation, on the other hand, induced a phosphene spanning both hemifields and with the same color as the stimulus in both G. Y. and the control subjects.

The exposure to color was then restricted to one hemifield in the control subjects. Here the component of the induced phosphene overlapping the adapted hemifield was colored and the component overlapping the unadapted hemifield was colorless. In G.Y. the bilateral phosphene appeared uniformly colored when the adaptation was restricted to the normal field. But it appeared colorless when the adaptation was restricted to the blind field.

With a bi-colored stimulus control subjects experienced phosphenes that were bi-colored. For example, if the right hemifield had been adapted to red and the left hemifield had been adapted to green, then the part of the phosphene spanning the right hemifield would be red and the part of the phosphene spanning the left hemifield would be green. In G.Y. the color of the phosphene depended on the color of the stimulus to which his normal field had been

adapted. If the normal field had been adapted to red and the blind field had been adapted to green, then the phosphene would be green. Hence, the studies show that in G.Y., unlike in normal subjects, the color of the phosphene induced in the blind field depended exclusively on the color to which the normal hemifield had been adapted.

Sivanto (2008) takes these findings to counter an earlier hypothesis that feedback to V1 from higher visual brain regions in one of the hemispheres is required for visual awareness in the visual field determined by that hemisphere (Silvanto et al. 2005). This hypothesis has been backed up by studies in the monkey (Azzopardi et al. 2003) and neuroimaging studies (Barbur et al. 1993) which indicate activity in extra-striate areas in blindsight subjects, in the absence of visual awareness. Since there is activity in blindsighters' extra-striate areas but no visual awareness, it was initially thought that blindsighters lacked visual awareness because feed-back to V1 was impeded (Silvanto et al. 2005). Silvanto argues on the basis of his recent studies that V1 is not a neural correlate of visual awareness. He entertains the possibility that contralesional striate cortical areas were a neurological basis for G.Y.'s blind field percepts but rejects this hypothesis on the grounds that V1 does not contain a retinotopic representation of the ipsilateral visual field.

The reason extra-striate activitation fails to give rise to visual awareness in the absence of a functional V1, Silvanto says, is that lesions to V1 result in abnormal neural responsiveness in extra-striate areas. According to him, the role of feedback to V1 in normal subjects is to enhance the nerve signal in extra-striate areas. It is primarily the enhanced signals in extra-striate areas that are responsible for generating visual awareness.

However, I believe another interesting result can be gathered from these findings. G.Y. experienced a colored phosphene in his blind field but this phenomenon was shown to depend exclusively on color information processing in the intact hemisphere. The color of the phosphene never depended on the color to which G.Y.'s blind hemifield had been exposed. So, it seems that the qualitative color information which gave rise to color experience in G.Y. came exclusively from G.Y.'s intact hemisphere. This suggests that G.Y.'s damaged hemisphere was unable to compute qualitative color information on the basis of exposure to colors. Apparently

no part of his damaged hemisphere could take over the role normally played by the double opponent cells in striate cortex. The qualitative color information that gave rise to the colored phosphene in G.Y.'s blind field was computed and supplied by the contralesional hemisphere.

This is not to say, of course, that no color processing took place in G.Y.'s ipsilesional hemisphere. Blindsight subjects can sometimes discriminate wavelength in their blind field (Stoerig and Cowey 1992). However, there are strong arguments for regarding wavelength processing in the retina and thalamus as a precursor to color vision rather than a part of color vision itself (Kentridge, Heywood and Weiskrantz 2007). Color vision, in common with other aspects of vision, produces percepts of distal stimuli. Plenty of visual illusions (e.g., those of Beau Lotto) demonstrate that we do not have access to the proximal stimulus (Purves and Lotto 2003, Corney and Lotto 2007). Wavelength can be viewed as part of the proximal stimulus. The distal stimuli that possess color are objects' surfaces and the colors we see are estimates of the reflectance properties of those stimuli, not the wavelength of light reflected from them (which depends on the nature of the illuminant light).[10] This strongly suggests that the information computed by further processing of wavelength in the retina and the thalamus cannot be viewed as qualitative color information.

Though the data gathered in Silvanto's study are based on a single case (like most other blindsight data), they do suggest that damage to the visual areas of one hemisphere compromises the computation of qualitative color information in that hemisphere. If this is right, then blindsight subjects do not lack color experience because the qualitative color information computed does not reach awareness owing perhaps to the sluggishness of the otherwise qualitatively unaltered neural signals in extrastriate areas. Rather, blindsighters lack color experience because qualitative color information is not computed in the ipsilesional hemisphere in the first place. Hence, the information that reaches the prefrontal cortical areas in blindsighters via extrastriate cortical processing normally lacks a qualitative element. Blindsight thus differs from normal sight in terms of which types of color information are computed. In blindsight, information about absolute wavelength is computed, whereas

[10] Thanks to an anonymous referee here.

qualitative color information (hue, saturation, brightness) normally is not computed. In normal sight, both information about wavelength and qualitative color information are computed.

Silvanto's study does not have any direct implications for primesight. But primesight could arise via a similar kind of information processing mechanism. It is possible that retinotopical representations in primesight are partially processed in the intact hemisphere and that interaction between the damaged and intact hemispheres facilitates the broadcasting of information to working memory in the damaged hemisphere. If this is so, then the qualitative content of the color experience is determined by the blindsighters' intact brain regions, while the state of primesight itself depends on residual and feed-forward processing in the blindsighters' damaged brain regions.

However, it remains to be shown what exactly the underlying mechanism of primesight is. In Silvanto's TMS study unilateral stimulation of either G.Y.'s damaged hemisphere or his intact hemisphere did not give rise to colored phosphenes in the blind field. In order for colored phosphenes to appear in the blind field bilateral stimulation of the two hemispheres was required. So, bilateral stimulation seemed to facilitate the interaction between the hemispheres. There is no direct evidence that this sort of hemispheric interaction occurs in primesight. However, Lawrence Weiskrantz (1992) reports that interactions between the two hemispheres may play a role in generating afterimages. In D. B. afterimages were found to transfer from one eye to the other, something that does not happen in normal subjects (Weiskrantz 1992). Hemispheric interaction is also known from cases of affective blindsight. In a recent affective blindsight study it was found that consciously perceived fearful responses were more easily recognized in conjunction with unconsciously perceived congruent responses in the opposite hemifield. The findings show that consciously and unconsciously perceived fearful responses were integrated across hemispheres, and that integration across hemispheres was sustained by subcortical connections (Tamietto and de Gelder 2008).

It is known independently that split-brain patients have greater subcortical hemispheric integration compared to normal subjects (Corballis 1995). This indicates that in normal subjects

cortical processes inhibit subcortical integration. Given the unconscious and subcortical nature of the neurological basis of blindsight, we should thus expect to find a decreased inhibition of subcortical hemispheric integration in people with blindsight. Primesight is only seen in people with unilateral lesions to striate cortex. Hence, these findings strongly support the hypothesis that primesight depends on input from the intact hemisphere.

If this is right, then we have a straightforward response to Kriegel's argument from primesight to the conclusion that blindsighters compute qualitative color information. Kriegel's argument in its modified form went as follows: (1) The visual system in the damaged hemisphere of some blindsight subjects computes information that gives rise to qualitative after-images in the corresponding blind field. (2) It is unlikely that non-qualitative information computed in the damaged hemisphere can give rise to qualitative after-images in the corresponding blind field. Hence, by an inference to the best explanation, (3) the visual system in the damaged hemisphere of some blindsight subjects computes qualitative information. We can now see where the argument goes wrong. The best explanation of after-images in the blind field is not that blindsight subjects compute qualitative color information but rather that qualitative color information computed in the contralesional hemisphere sometimes reaches awareness in the ipsilesional hemisphere through interhemispheric interaction.

5. Explaining the Lack of Awareness in Blindsight

Our hypothesis that qualitative color information is not normally computed in blindsight offers a partial explanation of why blindsight isn't normally associated with color awareness. It may be thought that this explanation is redundant, as the underlying mechanisms of blindsight themselves offer an explanation of the lack of color awareness in blindsight. That, however, is not the case.

There are good grounds for thinking that blindsight makes use of alternative pathways compared to normal vision. The recent cases in which blindsight was induced in normal subjects by applying TMS over the visual cortex seem to confirm that blindsight does not make

use of the standard visual pathway (from the retina to area V1 in occipital cortex via the lateral geniculate nucleus in the thalamus) for information processing (Ro 2008). Rather, it makes use of a spared pathway directly from the retina to the superior colliculus in the mid-brain. The superior colliculus is thought to be anatomically and functionally equivalent to the optical tectum in non-mammalian vertebrates, in which this retinotectal projection is the main efferent pathway from the retina.

From the superior colliculus there are projections to the parietal cortex, the later part of the dorsal stream, which is involved in the guidance of motion. According to David Milner and Melvyn Goodale's two-pathway hypothesis, information processed by the dorsal stream does not reach conscious awareness but is transformed directly into immediate action (Goodale and Milner 1992, Milner and Goodale 2008). But the superior colliculus also has projections to the lateral geniculate nucleus (LGN), which in turn has projections to V1. In normal individuals, then, the superior colliculus has projections to two brain structures involved in vision, which suggests that the superior colliculus could be a neural basis of blindsighters' above-chance visual performance in forced-choice paradigms

Further evidence for the central role of the superior colliculus in blindsight comes from the fact that at least some color information seem to be computed by the superior colliculus in the midbrain in blindsight (via a direct route from the retina). The superior colliculus is not activated by short-wavelength cones in the retina. These might informally be characterized as being optimally activated by purplish light, and studies have shown that there is a shift of sensitivity towards the red end of the color spectrum in blindsight subjects (Weiskranz 2009).[11]

But processes in the superior culliculus most certainly are not on their own sufficient for visual awareness. So, if the superior culliculus is a neural basis of blindsighters' above-chance visual performance, then it would seem that we have a straightforward explanation of why blindsight is not associated with any distinctly visual awareness. The question of to what extent

[11] Both blindsight subjects and normal subjects show greater pupil responses to red stimuli (Barbur, Weiskranz and Harlow 1999).

blindsighters compute color, then, is irrelevant to the question of why blindsighters are not visually aware of stimuli in their blind field.

However, explanations of the lack of visual awareness in blindsight that make appeal to alternative pathways for visual processing presuppose what we have been arguing here, viz. that blindsighters do not compute colorIf the superior colliculus, the LGN or extrastriate areas really did compute qualitative color information in cases of blindsight, then we should expect blindsight to be associated with visual awareness. So, the fact that blindsight makes use of an alternative pathway that bypasses the primary visual cortex and sends retinal information directly to the superior colliculus and then from here to extrastriate areas does not by itself explain why blindsight doesn't correlate with color awareness. Our hypothesis that qualitative information isn't computed in normal cases of blindsight thus offers a more plausible explanation of the lack of visual awareness: There is no visual awareness in ordinary cases of blindsight, because visual awareness requires that double opponent cells compute qualitative information about the visual stimulus. Yet in blindsight the double opponent cells in the striate areas are not functional and hence cannot compute the relevant information. As no other brain areas can take over the role of the double opponent cells in striate cortex, no qualitative color information is normally computed in blindsight.

6. Is Blindsight Degraded Ordinary Vision?

As noted above, it is sometimes claimed that blindsight is just degraded ordinary vision. As Weiskranz remarks, there are three ways to read this claim. First, it could be read as the claim that the performance in blindsight is less accurate than the performance in normal sight. Second, it could be read as the claim that blindsight and normal sight use the same mechanisms when computing information. Third, it could be read as the claim that blindsight really is a kind of (degraded) conscious vision. Let us look at these three claims in light of the conclusions drawn in previous sections.

As Weiskrantz (2009) points out, there is not much ground for the claim that the performance in blindsight is less accurate than the performance in normal sight. There are blindsight subjects who perform better in their blind field than in the unaffected region of the visual field in experimental settings. Blindsight subjects do not normally rely on their blindsight in everyday life. So, one could understand the claim that blindsight is degraded ordinary vision as a claim about blindsight performances in everyday life. However, the meager role that blindsight plays in everyday life is merely a contingent feature of blindsight. It is not essential to it (recall Block's super-blindsighters). So, blindsight is not degraded ordinary vision in the first sense.

As for the second sense, Silvanto's study does raise the question of to what extent the identification of color in the blind field of ordinary blindsight subjects depends on processing in the contralesional hemisphere. If the contralesional hemisphere were the neural basis of blindsight, then the neural basis would be a fully functioning mechanism, in which case the neural basis of blindsight would not be different from that of ordinary vision. However, it is unlikely that all processing of color stimuli presented to a blindsighter's blind hemifield takes place in contralesional brain regions. In the study conducted by Silvanto's team colored phosphenes were induced in G.Y.'s blind field only when G.Y.'s damaged and intact hemispheres were simultaneously stimulated. This indicates that while areas in G.Y.'s intact hemisphere were involved both in processing the information from the color stimulus and in broadcasting the computed information to working memory, activity in the damaged hemisphere was required for a colored phosphene to appear in the blind field. So, blindsight is not degraded ordinary vision in the second sense.

As for the third sense, blindsight is not a kind of unconscious vision in the sense that there is nothing it is like to experience blindsight. As reported by many blindsighters, feelings of something happening could occur in conjunction with visual stimuli in the blind field. For these blindsighters, there is a sense in which there is something it is like to experience blindsight. However, in the TMS study conducted by Silvanto information processing in G.Y's intact hemisphere was found to be required in order for color experience to occur in the blind

field. Color information processing in the damaged hemisphere never reached awareness on its own (the intact hemisphere was needed to assist with that task). So, generalizing from this study, color information processing in the damaged hemisphere is a process that never reaches awareness, which makes blindsight a form of unconscious vision. So, blindsight is not degraded ordinary vision in the third sense either.

There is, however, a sense in which blindsight is degraded *compared to* ordinary vision. If our hypothesis that qualitative color information is not computed in blindsight is correct, then blindsight is (qualitatively) degraded compared to ordinary vision. There are types of information not normally computed in blindsight which are normally computed in ordinary sight.

7. Neural Correlates of Visual Awareness

One interesting consequence of the proposed interpretation of the findings in Silvanto's study is that it encourages a modification of the original interpretation suggested by the researchers. Cases of blindsight show that neural activation in extra-striate cortical areas does not suffice for visual awareness. A standard interpretation of these findings is that striate cortex is a neural basis of consciousness (Pascual-Leone and Walsh 2001, Silvanto et al. 2005, Lamme 2001). But Silvanto (2008) suggests a different explanation. Lesions to striate cortex not only compromise neuron activity in striate cortex, they also compromise normal neuron activity in extra-striate cortex. In G.Y. neural activity in extra-striate regions in the contralesional hemisphere played a crucial role in generating phosphenes in his blind field, a finding which Silvanto takes to suggest that the primary visual cortex is not a neural correlate of consciousness.

However, as David Chalmers points out, on one common understanding of the concept of a neural correlate of consciousness, a neural correlate of consciousness is a correlate of a content of consciousness. Following Chalmers, we can define the neural correlate of a qualitative content of color awareness as follows:

> A neural correlate of the qualitative contents of color experience is a neural representational system N such that representation of a content in N directly correlates with representation of that content in color experience.

Silvanto's study lends support to the hypothesis that the V4/V8 color complex in G.Y.'s contralesional hemisphere represents chromatic colors (red, blue, green), and that this representation directly correlates with a representation of chromatic colors in color awareness.[12] So, extrastriate areas in G.Y.'s contralesional hemisphere plausibly form a neural correlate of the chromatic color content of color experience (the result of adding hue and saturation to brightness).

However, there is reason to think that the primary visual cortex is a neural correlate of color contrast represented in color experience (brightness and brightness contrast) (Morland et al. 1999), and that the neural responsiveness in extrastriate areas is impaired when this type of information is lacking (Kentridge, Heywood and Weiskrantz 2007). According to Silvanto, the loss of color awareness in normal cases of blindsight is due to the abnormal neural responsiveness in extrastriate cortex. That seems exceedingly plausible. However, the fact that the neural responsiveness in extrastriate areas is abnormal in subjects with blindsight is consistent with striate cortex being a neural correlate of some of the qualitative contents of color experience. The input to extrastriate cortical areas in normal cases and in blindsight cases are of fundamentally different kinds. In normal cases, the input to extrastriate areas is the result of double opponent processes (cone and spatial opponent processes). In blindsight cases, the input to extrastriate areas is the result of wavelength processing and cone opponent processing in the thalamus. In blindsight, the informational input from lower visual areas does not suffice for normal neural responsiveness in extrastriate areas because the normal computation of saturation, hue and hue contrast depends on the informational output of double opponency processing. So, pace Silvanto, lack of awareness in blindsight cannot be

[12] A newly charted area named 'area V8' is now thought to be the "color center". Previously V4 was believed to play this role (Heywood, Kentridge and Cowey 2001, Kentridge, Heywood, and Weiskrantz 2007). Here I take the 'V4/V8 color complex' to refer to the color center regardless of whether its location is in V4, V8 or both.

explained simply by noting that neural activity in extrastriate areas is sluggish owing to V1 lesions that slow down neural activity in higher areas of the cortical stream. The reason the neural activity in extrastriate areas is sluggish is that the neurons in extrastriate areas cannot function normally without the right kind of qualitative input (brightness). This suggests, then, that the primary visual cortex represents luminance and color contrasts that correlate directly with the representation of brightness and brightness constancies in color experience. This makes the primary visual cortex a neural correlate of some of the qualitative content of color experience.

There is independent evidence for the view that the primary visual cortex is a neural correlate of some of the qualitative contents of color experience. As cases of achromatopsia demonstrate, awareness of the individual hues and saturation correlates with activity in the V4/V8 color complex (Heywood, Kentridge and Cowey 2001, Heywood and Kentridge 2003). But while achromatopsics have no awareness of hues and saturation, they do have some awareness of brightness, brightness contrast and the corresponding achromatic colors (black, white and shades of gray). This awareness endows them with the ability to read, recognize contours, watch television even if they don't see the colours in any normal sense and navigate the world. Studies have shown that achromatopsics' processing of luminance is not normal (Kentridge, Heywood and Cowey 2004, Kentridge, Cole and Heywood 2004, Kentridge et al 2004). Robert Kentridge, Charles Heywood and Alan Cowey (2004) show that achromatopsics sometimes can process color contrast and luminance but that they cannot integrate contrast signal in the construction of color. Likewise, Kentridge, Heywood and Weiskrantz (2007) show that a subject with lesions to striate cortex (blindsight subject DB) cannot construct color contrasts but can discriminate wavelengths. In neither case does the ability to perform contrast or wavelength discrimination lead to normal color vision.

However, unlike blindsighters, achromatopsics are able to process color contrast, allowing them to have some, albeit, abnormal (achromatic) color awareness. The abnormal (achromatic) color experience found in achromatopsics represents achromatic color contrasts (brightness and brightness contrast), and the representation of this content in color experience

correlates directly with the representation of color contrast in the primary visual cortex, which is relatively intact in subjects with achromatopsia.

The upshot is that while it is correct that the absence of functional striate cortical areas impedes optimal neural responsiveness in extrastriate areas, the main reason for the impediment is that the information from the retina and the thalamus has not undergone processing by double opponent cells in striate areas to yield information about color contrast locally and across the scene. In cases of blindsight the information entering extrastriate areas is thus not of the right kind to yield optimal neural responsiveness. In G.Y., bilateral stimulation of V5/MT allowed information from contralesional extrastriate areas to travel through colossal pathways to the ipsilesional hemisphere, yielding relatively normal neural responsiveness in ipsilateral extrastriate areas. This then would guarantee the presence of a colored phosphene spanning the blind field.

8. Concluding Remarks

Blindsight subjects are sometimes able to discriminate wavelength in the absence of any experience of color. There are two possible explanations of this disassociation between conscious experience and visual performance. It could be that the visual system computes qualitative color information (e.g., chromatic contrast) but does not broadcast it to working memory. Or it could be that qualitative color information is not computed in the first place. It has been argued that the existence of primesight, which occurs in some blindsight subjects, gives us reasons to think that the visual system does compute qualitative color information but that this information does not reach visual awareness.

I have argued, however, that, under one interpretation of recent empirical data, qualitative color information is not computed at all. A study conducted by Silvanto and

colleagues showed that color phosphenes could be induced in blindsight subject G.Y.'s blind field when both hemispheres were simultaneously activated using transcranial magnetic stimulation and a colored stimulus was presented to his sighted field for an extended period of time. The phosphenes reliably had the same color as the color stimulus to which his contralesional hemisphere had been adapted. I argued that the findings show that only G.Y.'s contralesional hemisphere can compute qualitative color information. This information then travels to the ipsilesional hemisphere via colossal pathways and gives rise to color experience in the blind field. The ipsilesional hemisphere was required in order for this qualitative color information to give rise to a colored phosphene in G.Y.'s blind field but the contralesional striate and extra-striate cortical areas were required in order for this information to be computed in the first place. This seems to show that the mechanisms believed to underlie blindsight do not themselves compute qualitative color information, though it is empirically evident that they compute non-qualitative color information (e.g. information about wavelength). Blindsight thus plausibly is a genuine form of unconscious vision rather than a severely degraded form of normal conscious vision, as some researchers have argued.

The limitations of this interpretation of Silvanto's findings is that even if it shows that blindsighters do not compute qualitative color information, answering the specific question of whether color is computed in blindsight does not tell us anything about the reasons other kinds of visual information do not elicit consciousness in blindsight but can nonetheless be reliably discriminated. Whether the interpretation generalizes will depend on whether Silvanto's study can be repeated for attributes other than color. In a study conducted by Marcel (1998) a flash gun built into a black box with shaped slits (semi-circles or lines) was used to produce after-images in blindsight subjects G.Y. and T.P. The study showed that the ipsilesional hemisphere of the blindsight subjects processed information about shape which gave rise to visual awareness in the blind field when the visual stimulus produced a coherent figure. This study seems to lend itself to an interpretation similar to the one we applied to the data from Silvanto's study. Though a more careful look at the empirical data is needed, I predict that Marcel's data will confirm that the primary visual cortex is crucial also for the conscious representation of contour.

Do our conclusions have any consequences for the nature of color perception? The findings certainly seem to confirm that hypothesis that color perception need not be correlated with distinctly visual awareness. Blindsighters can discriminate wavelength but owing to the lesions to the primary visual cortex, they cannot compute color. They most likely lack the ability to perceive brightness (Morland et al 1999), which seems to require optimal neural responsiveness in the primary visual cortex, and because of the lack of perceived brightness they are unable to produce visible hue and saturation. The wavelength processes that do occur in blindsighters thus proceed below the level of conscious awareness.[13]

References

Azzopardi, P. and Cowey. A. 1997. "Is Blindsight like Normal, Near-Threshold Vision?" *Proc Natl Acad Sci U S A* 94: 14190-4.

Azzopardi, P., Fallah, M., Gross, C. G., and Rodman, H. R. 2003. "Response Latencies of Neurons in Visual Areas MT and MST of Monkeys with Striate Cortex Lesions", *Neuropsychologia* 41: 1738-1756.

Barbur, J. L., Watson, J. D., Frackowiak, R. S., and Zeki, S. 1993. "Conscious Visual Perception without V1", *Brain* 116: 1293-1302.

Barbur J. L., Weiskrantz L., Harlow J. A. 1999. "The Unseen Color Aftereffect of an Unseen Stimulus: Insight from Blindsight into Mechanisms of Color Afterimages", *Proceedings of The National Academy of Sciences* 96: 11637-11641.

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Block, N. 1995. "On a Confusion about a Function of Consciousness", *The Behavioral and Brain Sciences* 18: 227-287.

Block, N. 2007. “Consciousness, Accessibility and the Mesh between Psychology and Neuroscience”, *Behavioral and Brain Sciences* 30: 481-548.

Block, N. 2008. “Consciousness and Cognitive Access”, *Proceedings of the Aristotelian Society*, 108: 289-317.

Boyer, J. L., Harrison, S., Ro, T., 2005. “Unconscious processing of orientation and color without primary visual cortex”, *Proceedings of the National Academy of Science* 102: 16875-16879.

Carruthers, P. 2001. “Who is Blind to Blindsight?”, *Psyche* 7 (4). http://journalpsyche.org/ojs-2.2/index.php/psyche/article/viewFile/2581/2498.

Chalmers, D. J. 2000. “What is a Neural Correlate of Consciousness?”, in Metzinger, T. ed., *Neural Correlates of Consciousness: Empirical and Conceptual Questions*, Cambridge, MA: The MIT Press, 17-39.

Christensen, M. S., Kristiansen, L., Rowe, J. B. and Nielsen, J. B. 2008. “Action-Blindsight in Healthy Subjects after Transcranial Magnetic Stimulation”, *Proceedings of the National Academy of Science* 29; 105: 1353–1357.

Corballis, M. C. 1995. “Visual Integration in the Split Brain”, *Neuropsychologia* 33: 937–959.

Corney, D. and Lotto, R. B. 2007. “What Are Lightness Illusions and Why Do We See Them?”, PLoS Computational Biology 3: e180.

Cowey, A. 2004. “The 30th Sir Frederick Bartlett Lecture: Fact, Artefact, and Myth about Blindsight”, *Quarterly Journal of Experimental Psychology* 57: 577–609.

Danckert, J., Maruff, P., Kinsella, G., De Graaff, S., Currie, J. 1998. “Investigating Form and Colour perception in Blindsight Using an Interference Task”, *NeuroReport* 9: 2919-2925

Danckert, J. and Goodale, M. 2000. “Blindsight: A Conscious Route to Unconscious Vision”, *Current Biology* 10: R64-R67.

Gazzaniga, M. S., Fendrich, R., and Wessinger, C. M. 1994. “Blindsight Reconsidered”, *Current Directions in Psychological Science* 3: 93-96.

Goodale, M. A. and Milner, A. D. 1992. “Separate Visual Pathways for Perception and Action”, *Trends in Neurosciences* 15: 20–25.

Heywood, C.A., Kentridge, R.W. and Cowey, A. 2001. “Colour & the Cortex: Wavelength Processing in Cortical Achromatopsia”, in De Gelder, B.; De Haan, E. & Heywood, C.A. (eds.) *Varieties of Unconscious Processing: New Findings & Models*, Oxford: Oxford University Press: 52-68.

Heywood, C.A. & Kentridge, R.W. 2003. “Achromatopsia, Color vision & Cortex”, *Neurological Clinics of North America* 21: 483-500.

Hurlburt, R. T. and Schwitzgebel, E. 2007. “Describing Inner Experience”, Cambridge, Mass.: MIT Press.

Kentridge, R.W. and Heywood, C.A. 1999. “The Status of Blindsight: Near-Threshold Vision, Islands of Cortex & the Riddoch Phenomenon”, *Journal of Consciousness Studies* 6: 3-11.

Kentridge, R.W., Heywood, C.A. and Weiskrantz, L. 1999. “Effects of Temporal Cueing on Residual Visual Discrimination in Blindsight”, *Neuropsychologia* 37: 479-483.

Kentridge, R.W., Heywood, C.A. & Cowey, A. 2004. “Chromatic Edges, Surfaces & Constancies in Cerebral Achromatopsia”, *Neuropsychologia* 42: 821-830.

Kentridge, R.W., Cole, G.G. and Heywood, C.A. 2004. “The Primacy of Chromatic Edge Processing in Normal & Cerebrally Achromatopsic Subjects”, *Progress in Brain Research* 144: 161-169.

Kentridge R. W., Heywood, C. A., Weiskrantz, L. 2007. “Color Contrast Processing in Human Striate Cortex, *Proceedings of the National Academy of Sciences* 104: 15129-15131.

Kriegel, U. 2009. *Subjective Consciousness. A Self-Representational Theory*, Oxford: Oxford University Press.

Lamme,V.A. 2001. "Blindsight: The role of Feedforward and Feedback Corticocortical Connections", *Acta Psychologica* 107: 209-228.

Lamme, V. A. F. 2006. "Zap! Magnetic Tricks on Conscious and Unconscious Vision", *Trends in Cognitive Sciences* 10: 193-195.

Lau, H. C., Passingham, R. E. 2006. "Relative Blindsight in Normal Observers and the Neural Correlate of Visual Consciousness", *Proceedings of the National Academy of Science* 103: 18763-18768.

Marcel, A. J. 1998. "Blindsight and Shape Perception: Deficit of Visual Consciousness or of Visual function?", *Brain* 121**:** 1565-1588.

Milner, D. and Goodale, M. 1995. *The Visual Brain in Action*, Oxford University Press.

Milner, A. D. and Goodale, M. A. 2008. "Two Visual Systems Re-viewed", *Neuropsychologia*, 46: 774–785.

Morland, A. B., Jones, S. R., Finlay, A. L., Deyzac, D., Le, S., and Kemp, S. 1999. "Visual Perception of Motion, Luminance and Colour in a Human Hemianope", *Brain* 122: 1183–1198.

Overgaard, M. and Fehl, K. and Mouridsen, K. and Bergholt, B. and Cleeremans, A. 2008. "Seeing without Seeing? Degraded Conscious Vision in a Blindsight Patient", *PLoS ONE* 3.

Pascual-Leone, A. and Walsh, V. 2001. "Fast Backprojections from the Motion to the Primary Visual Area Necessary for Visual Awareness", *Science* 292: 510–512.

Persaud, N., McLeod, P. and Cowey, A. 2007. "Post-Decision Wagering Objectively Measures Awareness", *Nature Neuroscience* 10: 257-261.

Persaud, N and Lau, H. 2008. **"**Direct Assessment of Qualia in a Blindsight Participant", *Consciousness and Cognition* 17: 1046-1049.

Persaud, N. and Cowey, A. 2008. "Blindsight is Unlike Normal Conscious Vision: Evidence from an Exclusion Task", *Consciousness and Cognition* 17: 1050-1055.

Purves, D. and Lotto, R.B. 2003. *Why We See What We Do: An Empirical Theory of Vision*, Sunderland, Mass.: Sinaur Associates INC. and London: Macmillan Press.

Rees, G. 2008. “The Anatomy of Blindsight”, *Brain* 131: 1414-1415.

Ro T. 2008. “Unconscious Vision in Action”, *Neuropsychologia* 46: 379-383.

Ro T., Farne, A., Johnson, R.M., Wedeen, V., Chu, Z., Wang, Z.J., Hunter J.V., and Beauchamp, M.S. 2007. “Feeling Sounds after a Thalamic Lesion”, *Annals of Neurology* 62: 433-441.

Sahraie, A., Weiskrantz, L., Burbur, J. L., Simmons, A., Williams, S. C. R. & Brammer, M. J. 1997. “Pattern of Neuronal Activity Associated with Conscious and Unconscious Processing of Visual Signals”, *Proceedings of the National Academy of Science* 94: 9406–9411.

Sahraie, A., Trevethan, C. T., MacLeod, M. J., Murray, A. D., Olson, .J. A., Weiskrantz, L. 2006. “Increased Sensitivity after Repeated Stimulation of Residual Spatial Channels in Blindsight”, *Proceedings of the National Academy of Sciences* 103: 14971–6.

Schärli, H., Harman, A.M. and Hogben, J.H. 1999. “Residual Vision in a Subject with Damaged Visual Cortex”, *Journal of Cognitive Neuroscience* 11: 502-510.

Schwiedrzik, Singer, and Melloni 2008. “Sensitivity and perceptual awareness increase with practice in metacontrast masking”, *Journal of Vision* 9: 1-18.

Siewert, C. 1998. *The significance of consciousness*, Princeton University Press.

Siewert, C. 2001. " Spontaneous Blindsight and Immediate Availability: A Reply to Carruthers", *Psyche* 7(8). http://journalpsyche.org/ojs-2.2/index.php/psyche/article/viewFile/2585/2502.

Silvanto, J., Cowey, A., Lavie, N. and Walsh, V. 2005. "Striate Cortex (V1) Activity Gates Awareness of Motion", *Nature Neuroscience* 8: 143–144.

Silvanto, J., Cowey, A., Lavie, N., Walsh, V. 2007. "Making the Blindsighted See", *Neuropsychologia* 45: 3346–3350.

Silvanto, J., Cowey, A. and Walsh, V. 2008. "Inducing Conscious Perception of Colour in Blindsight", *Current Biology* 18: R950-R951.

Silvanto, J. 2008. "A Re-Evaluation of Blindsight and the Role of Striate Cortex (V1) in Visual Awareness", *Neuropsychologia* 46(12):2869-71.

Stoerig, P. and Cowey, A. 1989. "Wavelength Sensitivity in Blindsight", *Nature* 342: 916-918.

Stoerig, P. and Cowey, A. 1992. "Wavelenth Discrimination in Blindsight", *Brain* 115: 425-444.

Stoerig, P. and Barth, E. 2001. "Low-level Phenomenal Vision Despite Unilateral Destruction of Primary Visual Cortex", *Consciousness and Cognition* 10: 574-587.

Tamietto, M. and de Gelder, B. 2008. “Affective Blindsight in the Intact Brain: Neural Interhemispheric Summation for Unseen Fearful Expressions”, *Neuropsychologia* 46: 820-828.

Trevethan, C. T., Sahraie, A., Weiskrantz, L. 2007. “Can Blindsight be Superior to ‘sighted-sight’?”, *Cognition* 103: 491–501.

Weiskrantz, L., Barbur, J. L., Sahraie, A. 1995. “Factors Affecting Conscious versus Unconscious Visual Discrimination with V1”, *Proceedings of the National Academy of Sciences* 92:6122-6126.

Weiskrantz, L. 1998. *Blindsight: A Case Study and Implications*, Oxford: Oxford University Press.

Weiskrantz, L., Cowey, A., Hodinott-Hill, I. 2002. “Prime-Sight in a Blindsight Subject”, *Nature Neuroscience* 5: 101-2.

Weiskrantz, L. 2009. “Is Blindsight Just Degraded Normal Vision?”, *Experimental Brain Research* 192: 413-416.

Zeki, S. and Ffytche, D. H. 1998. “The Riddoch Syndrome: Insights into the Neurobiology of Conscious Vision”. *Brain* 121: 25-45.