Blindsight

James Danckert¹, Christopher Striemer² and Yves Rossetti^{3, 4}

1. Department of Psychology
University of Waterloo
200 University Avenue West
Waterloo, Ontario, N2L 3G1, CANADA
2. Department of Psychology
MacEwan University
Edmonton, Alberta, T5J 4S1, CANADA
3. Inserm UMR-S 1028, CNRS UMR 5292, ImpAct, Trajectoires, Centre de Recherche en Neurosciences de Lyon, Université Lyon 1, 16 avenue Lépine, 69676 Bron, France
4. Plate-forme “Mouvement et Handicap”, Hôpital Henry-Gabrielle, Hospices Civils de Lyon, 20 route de Vourles, Saint-Genis-Laval, France

Correspondence should be addressed to:

James Danckert

Email: jdanker@uwaterloo.ca

Ph: 1 519 888 4567 ext. 37014

Abstract

For over a century research has demonstrated that damage to primary visual cortex does not eliminate all capacity for visual processing in the brain. From Riddoch's (1917) early demonstration of intact motion processing for blind field stimuli, to the iconic work of Weiskrantz and colleagues (1974) showing reliable spatial localization, it is clear that secondary visual pathways that bypass V1 carry information to the visual brain that in turn influences behavior. In this chapter we briefly outline the history and phenomena associated with blindsight, before discussing the nature of the secondary visual pathways that support residual visual processing in the absence of V1. We finish with some speculation as to the functional characteristics of these secondary pathways.

Key words: blindsight, occipital cortex, extrastriate, residual vision

Introduction

In the early part of the 20th Century, a handful of clinical reports challenged the established view that humans are rendered completely blind following damage to primary visual cortex, by showing that patients with such damage can have some residual sensitivity to light or motion (e.g., Bard 1905; Pick 1905). However, these case reports had insufficient detail, making clear interpretation of them as 'blindsight' cases difficult. George Riddoch (1917) observed a curious dissociation in war veterans who had suffered penetrating head wounds affecting the occipital cortex. In a series of patients he was able to delineate what he called fields for object vision and fields for motion. Many of his patients had regions of visual space in which they were unable to see static objects but could report a sense of movement. The capacity to accurately perceive the motion of objects in the otherwise blind field now bears his name – Riddoch's phenomenon (Zeki & Ffytch, 1998; Zeki, 1995). It is worth noting that even in this early report, Riddoch's patients were not describing 'normal' vision, with one stating that his perception "had neither form nor colour. It gave him the impression of a shadow." (Riddoch, 1917, p. 22).

It was not until much later in the 20th Century that Lawrence Weiskrantz and colleagues coined the oxymoron 'blindsight' to refer to a broader range of residual visual capacities in patients with occipital damage (Weiskrantz 2010; Sanders et al., 1974; Weiskrantz et al., 1974; see Danckert et al., 2019 for a concise definition of the term). In the early 70's a cluster of three independent studies from three European countries pioneered the modern investigation of blindsight. Pöppel et al., (1973) first described the ability to make saccades to the location of targets presented in the blind field following primary cortical damage. Ten months later

Weiskrantz and colleagues (Sanders et al. 1974; Weiskrantz et al. 1974) produced the first case description, of subject DB, reporting that not only the eye but the hand could be directed to unseen visual stimuli. Within a year the first group study of blindsight patients was published by Perenin and Jeannerod (1975), who flashed patterns into the scotomata of patients and compared the pointing performance of patients with post-geniculate lesions against that of patients with pre-geniculate lesions. Only the six patients with post-geniculate lesions showed any significant accuracy in pointing to targets in the blind field (Perenin & Jeannerod, 1975).

Pöppel, Weiskrantz, Jeannerod and their colleagues pioneered the modern approach to studying blindsight, in which stimuli are presented in the blind field and the patient is asked to respond in some manner – via two-alternative forced choices, or by acting towards things they do not report seeing (i.e., pointing or saccading to unseen target locations; Figure 1). Clearly, the patients required some encouragement given they never reported ‘seeing’ the targets themselves. Nevertheless, both eye movements and pointing movements were reliably directed towards the blind field target locations – not as accurately as for sighted field targets, but well above chance levels (particularly for pointing; Figure 1).

A wealth of research since this pioneering work has shown that patients with hemianopic field defects arising from damage to primary occipital cortex can nevertheless respond at above chance levels to stimuli in blind fields across a broad range of visual properties and tasks (for a recent review see Ajina & Bridge, 2016). From localizing targets, to discriminating wavelength, form, orientation, direction of motion and even responding to affective content, it seemed clear that visual information can be processed by neural pathways that bypass V1.

Controversy

Given the unusual and paradoxical phenomena described in blindsight patients, and its impact on many fields including philosophy, it was understandable that the field greeted these initial findings with some skepticism (Campion et al., 1983; Fendrich et al., 2001, 1992; Gazzaniga et al., 1994). One challenge to the idea that residual pathways that bypass V1 are responsible for blindsight came from Gazzaniga and colleagues (Fendrich et al., 1992; Gazzaniga et al., 1994). They suggested that ‘spared islands’ of occipital cortex may in fact support the phenomena in question, particularly since perimetry can only *sample* the visual field and not test its entirety. The proposal was that information could be conveyed along the geniculostriate pathway via these spared islands, albeit in a degraded form – so degraded that conscious perception and reportage were lost. In some sense, this explanation relates to the confidence of patients in their self-reported visual experience. The fact that their responses to visual stimuli are better than chance indicates that visual information is indeed processed in their brain, but not with sufficient fidelity for them to report confidently about them. The ‘spared-islands’ account was discounted by structural and functional neuroimaging (MRI and fMRI). In the first instance, it was evident that some patients who demonstrated blindsight did not in fact have any sparing of V1 on structural imaging (e.g., Barbur et al., 1993). Functional neuroimaging studies in the late 1990’s and early 2000’s showed activation in extrastriate but not striate regions in response to blind field stimuli (Baseler et al., 1999; Goebel et al., 2001; see Cowey, 2010 and Stoerig & Cowey, 2007 for reviews). In addition, patient GY, perhaps the most studied blindsight patient in the literature, has been shown to have above chance performance for a

wide swathe of spatial locations throughout his blind visual field (Kentridge et al., 1997; see also Danckert & Culham, 2010 for fMRI data from GY showing extrastriate activity driven by blind field stimuli). This would not be expected if residual visual capacities were only supported by isolated, scattered islands of occipital cortex.

The spared-islands hypothesis was dealt a further blow by work showing residual visual capacities in hemispherectomy patients (Perenin & Jeannerod 1978; Ptito et al., 2001, 1991, 1987; Ptito & Leh, 2007; see also Georgy et al., 2019 for a recent account of functional reorganization of receptive fields in a hemispherectomized blindsight patient). Residual visual capacities have been shown as well in these patients, who have had all or at least a substantial portion of the cortical mantle removed from one hemisphere, including the occipital cortex, typically as a treatment for medication resistant epilepsy. Perhaps the most prominent of these is known as the *redundant target effect*, which describes reduced reaction times for target detection when multiple instances of the same target type are present (Marzi et al., 1986). This effect is evident in hemispherectomy patients even when the additional targets which drive the effect are located in the blind hemifield (Tomaiuolo, et al., 1997). Clearly, with no cortical tissue in the decorticated hemisphere, this effect must depend on subcortical processing and not on spared islands of striate cortex.

A second challenge to blindsight is more persistent than the spared-islands hypothesis. It is plausible that light scatter from either intraocular or extraocular sources drives residual vision (Campion et al., 1983). That is, depending on stimulus properties (particularly brightness and contrast) light may ‘scatter’ from its source to portions of the retina which project to primary visual cortex in the unaffected hemisphere, enabling above-chance performance. In

addition, light can scatter within the orbit and if not carefully controlled can lead to information projecting to the undamaged hemisphere. In either case, this information would necessarily be degraded relative to information projected directly to those same regions and so, while it may inform behavior, it may be insufficient to inform conscious report (Campion et al., 1983). Even the earliest clinical reports of residual visual capacities mention this possibility and made attempts at controlling for it (Bard, 1905). The potential confound of light scatter is one that researchers must account for in experimental design, either by minimizing the potential for extraocular scatter (e.g., using isoluminant colour differences between targets and backgrounds), or by directly measuring the potential influence of scatter. Extraocular scatter can be tested by simply blocking off the hemiretinas projecting to the damaged cortex (note: this is easily achieved by creating spectacles with the temporal hemiretina of one eye and nasal hemiretina of the other 'blacked out' like a blindfold; see Danckert & Culham, 2010). If above chance performance persists in such a circumstance it must be due to light from the stimulus falling on regions of the retina that project to the unaffected hemisphere (Danckert & Culham, 2010). To test for intraocular light scatter one can place stimuli at the approximate location of the blind spot within the blind field. That is, the blind spots, where the optic nerve joins the retina, tend to be symmetrically located in each eye, allowing experimenters to approximate the location of the blind spot in the blind hemifield. Above-chance performance for a stimulus projected to this location would indicate intraocular scatter. Such controls were important in demonstrating that one patient initially demonstrating unusual blind field performance was in fact using a decision rule based on light scatter. That is, when targets were presented close to the midline of the patient's hemianopic field defect, she reliably guessed locations furthest into

the periphery and vice versa (Danckert & Culham, 2010). Using controls for light scatter revealed that the patient was in fact using an unusual decision rule based on light scatter (Danckert & Culham, 2010).

Many studies of blindsight patients, including some of our own (e.g., Danckert et al., 1998), have not adequately controlled for the concerns raised by light scatter. However, when such controls have been included, it is evident that residual visual capacities remain (Faubert, Diaconu, Ptito, & Ptito, 1999; King, Azzopardi, Cowey, Oxbury, & Oxbury, 1996; Zihl & Werth, 1984; see also Azzopardi & Cowey, 1997 for discussion of near-threshold accounts of blindsight). Even in the earliest work on patient DB, it was shown that discrimination of form was at chance when stimuli were presented to the approximate location of the blindspot in the blind field (Weiskrantz et al., 1974; 1987; see also Mazzi et al., 2016 for further discussion of the influence of different paradigms on the demonstration of blindsight).

There is agreement currently that blindsight is neither an artefact of light scatter, nor dependent on spared islands of visual cortex in the damaged side. The capacity to respond above chance to stimuli in the blind hemifield suggests that there are secondary pathways carrying visual information to extrastriate cortex that do not depend on input from V1.

The taxonomy of blindsight

The variety of behaviours exhibited in blindsight have led to a number of taxonomies intended to distinguish between classes of residual abilities (Table 1). Weiskrantz (1998) proposed a dichotomous distinction driven entirely by the presence or absence of some phenomenological experience reported by the patient. His *Type I blindsight* referred to residual

visual capacities evident in the complete absence of any conscious sensation. *Type II blindsight* referred to residual capacities that are occasionally accompanied by a sense or feeling that something happened, somewhat akin to the “shadow” description given by one of Riddoch’s patients (1917) and two of Bard’s (1905). Indeed, Riddoch’s phenomenon is commonly accompanied by a sense or feeling that is not strictly “seeing” (see Kentridge, 2015 for a detailed discussion of the phenomenology associated with Type II blindsight). This second type of blindsight, although accompanied by a feeling or sense of knowing that some event happened in the blind field, does not reach the threshold of what we would normally describe as a conscious visual experience.

Table 1. Taxonomies of blindsight.

Weiskrantz (1998)	Type I	no conscious percepts
	Type II	a feeling or sense, without “seeing”
Danckert & Rossetti (2005)	Action blindsight	pointing, grasping, saccades
	Attention blindsight	cuing effects, motion discrimination
	Agnosopsia	form and wavelength discrimination
de Gelder et al (1999)	Affective blindsight	non-conscious responses to blind field affective stimuli

More recently, we proposed a three-part distinction that divided residual vision by the type of behaviours elicited and, in a reciprocal sense, the type of tasks used to elicit them (Danckert & Rossetti, 2005). Within this classification, residual behaviours are divided primarily into action blindsight (e.g., pointing, saccading, grasping towards blind field stimuli) and effects that suggest an influence of attentional orienting (e.g., cuing effects, motion discrimination; Table 1). The third category – agnosopsia (Zeki & Ffytche, 1998), refers to more perceptual capacities such as form and wavelength discrimination, capacities typically elicited using forced-choice or implicit processing paradigms and which rarely, if ever are accompanied by the kind of ‘feeling’ of something being there, which is characteristic of Type II blindsight (see Brogaard, 2015 for review). Within this third category, one study has even shown effects of semantic priming from stimuli in the blind hemifield, although to our knowledge this has not been replicated (Marcel, 1998). Finally, Beatrice de Gelder and colleagues (1999) introduced a fourth class of residual abilities, now known as affective blindsight (Table 1). Here the patient shows reliable responses to affective stimuli presented to the blind field. Although this could be considered a type of agnosopsia, we consider it a separate category due to the fact that specific emotions seem to trigger the phenomenon and that it likely relies on a visual pathway that is partially distinct from those that support the other forms of blindsight.

The various phenomena of blindsight

The two classes of residual visual behaviour that have been most commonly demonstrated in blindsight are motion discrimination and action blindsight. Beyond the original papers of Weiskrantz, Jeannerod, and Zeki and their colleagues, the following represent just a

sub-sample of blindsight and motion articles (Alexander & Cowey, 2009; Azzopardi & Hock, 2011; Barton & Sharpe, 1997; Intrilligator et al., 2002). Similarly, for pointing, the most prominent action blindsight behavior, these papers are representative of methods and findings (Perenin & Jeannerod 1975, 1978; Corbetta et al., 1990; Danckert et al., 2003; Smits et al., 2019). A typical observation of these investigations is that both patients with retrogeniculate damage and patients with hemi-decortication are capable of locating targets in blind hemifields with eye and hand movements. Motion discrimination is rather straight-forward and can sometimes be associated with Riddoch's phenomenon or the sense/feeling of an event described in Type II blindsight (Arcaro et al., 2019; Alexander & Cowey, 2009; Azzopardi & Hock, 2011; Intrilligator et al., 2002). Since Weiskrantz and colleagues' (1974) initial study, pointing and making saccades to targets in the blind hemifield has also been relatively straight-forward to demonstrate. Beyond the need to encourage the patient to "guess" at where something they clearly did not see appeared, it is a simple matter to measure accuracy of these movements against actual target locations.

Action blindsight.

Since the original demonstrations of target localization by Weiskrantz and colleagues (1974) a number of other residual visuomotor functions have been demonstrated in blindsight including grasping, hand orientation, obstacle avoidance and, more recently, visuomotor adaptation. An early study by Perenin and Rossetti (1996) showed that a blindsight patient could orient his hand correctly to 'post' a card through a mail slot in his blind hemifield that was tilted at different orientations. In a second experiment the patient appropriately scaled his grip

aperture to match the varying size of objects within the blind field. In contrast, if the patient was asked to perceptually match the orientation or size of objects in his blind field, his verbal responses fell to chance (Perenin & Rossetti, 1996). Also, when the patient attempted to guess the perceptual property of the blind field targets during his action, or when the action was delayed by two seconds, his performance fell to chance (Rossetti 1998; Rossetti et al. 2000). Whitwell and colleagues (2011) replicated and extended these findings by demonstrating intact grip scaling to objects within the blind field regardless of whether visual feedback about the reaching hand was available. Similarly, their patient was unable to determine the size of those same objects using perceptual estimates. This intact grip scaling towards blind field targets was eliminated when a 2-second memory delay was introduced between the presentation of the target and the onset of the patient's reaching which was consistent with earlier suggestions that delayed actions may shift the visual representation of the action from the dorsal to the ventral stream (e.g., Milner and Goodale, 2006; Rossetti, 1998; Rossetti et al, 2000). Furthermore, this suggests that action blindsight needs the image of the object to be present on the retina at the moment the reach is initiated.

Stephen Jackson (1999) devised a clever way of examining grasping movements by placing only portions of the to-be-grasped objects in the blind field. Patient GY was first asked to reach towards objects of different widths presented wholly within his blind field. In this instance, like pointing or making saccades, GY is completely guessing, and for him grip apertures were only minimally related to target width. However, when GY now reached towards an object that crossed the vertical midline, being partially in the sighted field and partly in the blind field, he was able to reliably scale grip aperture, akin to a manual 'perceptual

completion' effect. It is worth noting that GY also oriented his grip to the appropriate axes of objects presented completely within his blind field (Jackson, 1999).

Another approach to examine action blindsight is to examine the ability of patients to avoid unseen obstacles. In daily life, some patients describe some sort of awareness of an obstacle when they are approaching a lamppost while walking on the sidewalk, although this experience is not visual. de Gelder and colleagues (2008) first studied navigation and obstacle avoidance in a patient who was left completely cortically blind following extensive bilateral lesions to early visual cortex. Despite his blindness a video of the patient clearly showed that he was able to avoid obstacles while walking down a hallway. In a later study, Striemer and colleagues (2009) demonstrated obstacle avoidance during a reaching task in a patient (CB) who had a left homonymous hemianopia. Typically, our reach trajectories veer away from obstacles (Chapman & Goodale, 2008, 2010; Mon-Williams et al., 2001; Saling et al., 1998). By placing obstacles for a reaching movement within the blind field, but at varying distances from the reaching hand, it was possible to show that patient CB's reach trajectories veered away from unseen obstacles in his blind field when they were placed closer to the reaching hand (but see Ross et al., 2018 for null evidence of obstacle avoidance and Striemer et al., 2018 for a reply to that work). As with their earlier study, patient CB's reaches were no longer sensitive to the position of unseen obstacles when a 2-second delay was introduced between offset of the obstacles and onset of his reach. Again, this suggests that action blindsight requires the image of objects to be still present on the retina at the moment the action is initiated.

In addition to research demonstrating preserved grip scaling and obstacle avoidance, there has been some evidence that actions directed towards objects in the blind field can be

adjusted in-flight based on target location or orientation (Danckert et al., 2003; Prentiss et al., 2018). For example, in one recent study it was demonstrated that a blindsight patient could adjust her movements in response to errors in order to compensate for a visual perturbation. Specifically, Striemer and colleagues (2019a) demonstrated that a patient who was blind to static stimuli following extensive bilateral lesions in V1 and the ventral stream, was surprisingly still able to adapt to optically displacing prisms (Striemer et al., 2019a). The patient demonstrated Riddoch phenomenon such that she was still able to discriminate direction of motion at above chance levels (see Arcaro et al., 2019). The patient was then presented with reversing checkerboard patterns as targets for pointing movements. When the patient wore either leftward or rightward shifting prisms, her reaching movements were initially deviated in the direction of the prism shift. Interestingly, she was capable of reducing pointing errors over subsequent trials. When the prisms were removed (following 100+ adaptation trials) the patient demonstrated significant after-effects in the direction *opposite* the prism shift, a hallmark indicator of adaptation. These prism after-effects were observed despite the fact the patient was not informed about how the “special glasses” could influence motor responses, and that she was not able to see her hand as it moved toward the target. Striemer and colleagues (2019a) hypothesized that the non-conscious visual inputs that enabled adaptation likely originated in regions such as area MT, the PPC, and the superior colliculus, each of which are known to send visual projections to the cerebellum (via the dorsolateral pons) which is critical for adaptation. In summary, it is clear that many aspects of the guidance of visuomotor behaviours from reaching, to grasping, to visuomotor adaptation, can be carried out without reliance on representations processed within primary visual cortex (e.g., Rossetti 1998).

Attention blindsight.

Beyond visuomotor behaviours, the next most commonly demonstrated blindsight capacities fall under the umbrella of attention blindsight (Table 1). Work from Robert Kentridge and Charles Heywood (1999a, b; Kentridge et al., 2004) was the first to demonstrate cuing effects that improved performance for blind field targets in patient GY. GY was asked to perform a forced-choice task to determine which of two spatial locations a target occupied. Targets could have different contrasts and appear with or without a cue to its onset. Although GY demonstrated blindsight even without these temporal cues, blind field localization was better with them. This improvement was most evident for targets with low or medium contrast and when the cue preceded the target, as one would expect. Intriguingly, there was even some benefit when targets *preceded* the cue, suggesting that GY was able to use attentional orienting to make retrospective decisions regarding the location of blind field targets (Kentridge & Heywood, 1999a). In subsequent work, GY was asked to make orientation discriminations for blind field targets in a traditional Posner cuing paradigm. In this instance the time to make orientation judgements was facilitated by valid cues but the cues did not improve accuracy (Kentridge et al., 2004). Taken together, this work suggests at least two important points. First, attention and conscious awareness should not be conflated. Second, many attentional affects do not rely on processing within primary visual areas in order to unfold in predictable ways.

Beyond the attentional cuing effects just described, research has shown a range of what can be characterized as implicit effects that likely depend to some extent on attention. These show that stimuli in the blind hemifield can influence the processing of targets in the sighted

hemifield. One such effect is interference from incongruent stimuli, which is typically explained in terms of selective attentional functioning (e.g., Danckert et al., 1998). An example is the classic flanker paradigm (Eriksen & Eriksen, 1974). In this effect, an incongruent stimulus at the mirror location in the blind hemifield across the vertical meridian from a visible stimulus inflates reaction times to those visible stimuli, when either form or colour is manipulated to create the incongruence (Danckert et al., 1998). These effects hint at a fairly crude, low-level bottom-up influence of blind field stimuli on target processing.

Another commonly used paradigm that has revealed attention blindsight is the *redundant target effect*. In this paradigm the participant must respond as quickly as possible to the presentation of a target or targets on a screen (Marzi et al., 1986). Typically, participants respond faster to two targets presented simultaneously than to a single target (Marzi et al., 1986). Carlo Marzi and colleagues (1986) were the first to demonstrate an intact *redundant target effect* in a patient with hemianopia, even when one of the two targets was located in their blind field. This effect has been replicated in numerous blindsight studies (e.g., Corbetta et al., 1990; Striener, et al., 2009), and even in hemispherectomy patients (Tomaiuolo et al., 1997, Leh et al., 2006). This work shows that attention can be drawn to blind field stimuli and, in the absence of conscious awareness, still produce the kinds of benefits (e.g., faster reaction times) and costs (e.g., interference) typically seen in experiments with normally sighted individuals, although the magnitude of these effects are smaller in blindsight patients.

Agnosopsia.

Beyond action and attention blindsight are a range of phenomena that we categorized as *agnosopsia*, which translates as “to not know what one sees” (Zeki and Ffytche, 1998). Even though the patient is completely unaware of stimuli within their blind hemifield they may still be able to guess their correct perceptual characteristics at above chance levels. The most commonly observed residual functions of this kind are form and wavelength (i.e., colour) discrimination. In the original investigation of blindsight, Weiskrantz and colleagues (1974) demonstrated with two-alternative forced-choice procedures that patient DB was able to discriminate both form (i.e., X vs. O) and orientation (horizontal vs. vertical) at better than 90% accuracy, despite having no confidence in his judgments. Subsequent studies of DB have demonstrated that he could make accurate same/different judgments about the shapes of pairs of stimuli (i.e., square vs. rectangle or circle vs. oval) in his blind field, and that he was even able to spontaneously name line drawings of objects and identify colour photographs (Trevethan et al., 2007a). In another study, DB was surprisingly able to discriminate Gabor patches presented at a *lower* contrast in his blind field compared to his sighted field (Trevethan, et al., 2007b). A result of this kind raises the possibility that performance in well studied patients such as DB and GY may reflect practice-induced plasticity. Along these lines, numerous studies have shown that repeated stimulation of the blind field can improve visual processing under certain circumstances (for a review see Sahraie, 2007).

Another residual capacity classified as agnosopsia is wavelength discrimination. Weiskrantz and colleagues (1974) also studied colour processing in patient DB and found that he was better than chance at discriminating red from green under a number of different testing

conditions. However, Weiskrantz and colleagues (1974) warned that, “while these scores are in the direction of indicating at least some residual capacity to differentiate red vs. green, in the absence of more systematic and better stimulus control they cannot be taken as being more than suggestive.” (p.720). In subsequent investigations Petra Stoerig and Alan Cowey (1989, 1992) have convincingly shown that blindsight patients can discriminate different wavelengths for stimuli in blind hemifields even when luminance and contrast were tightly controlled. Their studies in three patients suggested that both rod and cone inputs were present in blind field processing, even demonstrating some degree of colour opponency.

Affective blindsight.

One final area of blindsight that appears to be related to agnosopsia, but likely relies on different neural pathways, is affective blindsight, the ability to detect or discriminate affective information in the blind hemifield. de Gelder and colleagues first showed in 1999 that, at least in patient GY, affective information contained in static and dynamic facial expressions could be discriminated above chance for blind field stimuli. GY was able to distinguish most effectively between happy and sad facial expressions, although he was also reliably above chance for discriminating angry from fearful expressions (de Gelder, et al., 1999). Based on these findings, de Gelder and colleagues suggested another residual pathway for visual information to reach the brain without first running through V1, in this instance a pathway that connected the colliculus and/or pulvinar with the limbic system. This hypothesis was partially supported by subsequent neuroimaging studies in both blindsight patients (de Gelder et al., 2005) and healthy adults (e.g., Morris et al., 1999; Whalen et al., 1998) that demonstrated increased

amygdala activation in response to unseen emotional faces (for reviews see, Celeghin et al., 2015; Tamietto & de Gelder, 2010).

In addition, work by Cecere, Bertini, Ladavas and their colleagues (Bertini et al., 2013, 2019; Cecere et al., 2014), has shown that the presentation of fearful faces in the blind field can facilitate the processing of stimuli such as faces or Gabor patches in the sighted field. Although these studies indicate that emotional faces within a blind field can influence performance, they should not necessarily be taken as direct evidence of affective blindsight as none of the patients in these studies could discriminate between emotional faces presented only within their blind field, and none showed evidence of any other type of blindsight.

In perhaps the most dramatic demonstration of affective blindsight to date, Pegna and colleagues (2005) demonstrated affective blindsight in a cortically blind patient with bilateral lesions affecting V1 and early ventral stream areas. Their patient could not discriminate between simple shapes (i.e., a circle vs. a square) or the gender of faces. However, the patient was above chance for discriminating between happy and sad and between happy and fearful faces. An fMRI experiment in the same patient showed increased activity in the right amygdala when the patient was presented with images of emotional faces in their blind field, with fearful faces producing the greatest response (see also Burra et al., 2019; see also van den Stock et al., 2014 and Burra et al., 2013 for discrimination of bodies and eye gaze).

In a recent study (Striemer et al., 2019b) we replicated and extended the findings of Pegna and colleagues (2005) in a patient who was completely blind to static stimuli following bilateral lesions affecting both V1 and early face and object processing regions of the ventral stream. Despite her extensive lesions, the patient was still able to discriminate happy from

fearful and happy from angry faces at above chance levels. This despite the fact she was unable to discriminate the gender of those same faces. It is worth emphasizing that this patient was able to discriminate emotions from faces even though core regions of the face processing network (i.e., the fusiform and occipital face areas; e.g., Fox et al., 2009; for a review see Gobbini & Haxby, 2007) were damaged in both hemispheres.

Dissociations.

One of the more fascinating and frustrating aspects of blindsight, is that no two patients are exactly alike. Patients vary widely in the range of residual blindsight abilities they have, which may reflect the fact that different forms of blindsight are supported by different neural pathways, which will be affected variably by different lesions in different patients. In the early work of Marie-Thérèse Pérenin and Marc Jeannerod (Perenin & Jeannerod, 1975, 1978; Perenin 1978) patients could reach accurately to targets without being able to identify their shape, and similar dissociations were found in some of their hemidecorticated patients (Perenin & Jeannerod, 1978; Perenin, 1978). A related dissociation was also reported between prehension parameters (size and orientation) and corresponding perceptual characteristics (Perenin & Rossetti, 1996). As another example, a study by Maurizio Corbetta and colleagues (1990) examined the redundant target effect and manual localization in four patients with hemianopia. All four patients demonstrated above-chance localization when allowed to look towards and point to the target location in their blind field after the target had disappeared. However, one patient who showed above-chance localization in this 'look and point' task was unable to localize those same targets when he was forced to fixate centrally while pointing.

Furthermore, only one of the four patients showed a redundant target effect for two targets presented simultaneously, one to the sighted and one to the blind hemifield. This study made two important observations. First, different patients will show different patterns of spared abilities in the blind hemifield. Second, not all tasks are created equal: some tasks may be more sensitive to blindsight abilities than others (see Mazzi et al., 2016).

The notion that different patients may demonstrate distinct patterns of spared abilities is highlighted in our own work. For example, in the work on obstacle avoidance, patient CB showed blindsight in the redundant target effect paradigm but not when asked to localize blind field targets (Striemer et al., 2009). In contrast, in the work on grasping (Whitwell et al., 2011), the patient was able to localize targets within their blind field, despite failing to show a redundant target effect. More recent work from Chabanat and colleagues (2019) showed intact motion but not colour discrimination in the blindsight patient they tested, even though the same stimuli were used for both tasks. These differences in patterns of residual visual behavior may reflect differences in the *strength* of secondary pathways, or alternatively, such differences may be the outcome of damage that affects distinct residual pathways, leaving only some secondary routes to cortical visual processing intact while damaging others. By this view, each of the domains of residual behavior in the taxonomy discussed above may be supported by distinct pathways for conducting visual information to the brain that bypass V1 (Danckert & Rossetti, 2005).

Residual visual pathways

Demonstrations of residual visual processing have not been limited to humans (for review see Leopold, 2012). In macaque monkeys research has shown intact threat responses (pupillary dilation and blink responses) to blind field stimuli (Humphrey, 1974). Target localization, form and motion discrimination have all been found in decorticated monkeys (Cowey & Stoerig, 1995; Feinberg et al., 1978; Stoerig & Cowey, 1997). What we gain from work in non-human primates is the capacity to directly examine the neural pathways that might support these residual visual abilities. Such studies have demonstrated direct links from the lateral geniculate nucleus to extrastriate cortical regions, substantiating the notion that residual visual capacities are supported by secondary visual pathways that do not involve striate cortex (Bullier & Kennedy, 1983; Lyon et al., 2010; Nassi et al., 2006; Sincich et al., 2004).

In a more recent study, direct evidence that residual vision depended on the function of the lateral geniculate nucleus was obtained via reversible deactivation of this nucleus using a GABA agonist delivered directly to it (Schmid et al., 2010). This study used fMRI in conjunction with reversible inactivation of the lateral geniculate nucleus in macaque monkeys with chronic V1 lesions (Schmid et al., 2010). Prior to geniculate inactivation, neural activity was observed in a swathe of extrastriate, superior temporal and lateral intraparietal regions despite the absence of any inputs from V1. In other words, fMRI showed evidence of intact residual visual processing in the monkeys. This activity was eliminated when the GABA agonist was directly administered to the lateral geniculate nucleus (Schmid et al., 2010).

A direct pathway from the lateral geniculate nucleus to extrastriate cortical regions is not the only secondary pathway shown to carry visual information (Figure 2). Complex

pathways originating from the brachia of the optic tract, proceeding to the superior colliculus, and from there connecting with the lateral geniculate nucleus and/or the pulvinar have been demonstrated (Bridge et al., 2016; Kaas & Lyon, 2007; Lyon et al., 2010; Warner et al., 2015). From both the pulvinar and lateral geniculate nucleus there are pathways for visual information to reach dorsal and ventral extrastriate cortex that bypass V1 (see Tamietto & de Gelder, 2010 and Kaas & Lyon, 2007 for reviews). Which kind of blindsight is supported by these secondary visual pathways likely depends on the terminus of their projections. As such, both the geniculate and colliculo-pulvinar pathways likely play a role in aspects of both action and attention blindsight through their projections to posterior parietal cortex (Kaas & Lyon, 2007). Lyon and colleagues (2010) demonstrated a disynaptic relay from the superior colliculus to dorsal stream areas V3 and MT, which could support motion discrimination and action blindsight capabilities. In addition, as mentioned previously, Schmid and colleagues (2010) demonstrated that inactivation of the lateral geniculate nucleus eliminated the ability of monkeys to localize targets in their blind field via saccades.

Less research has been conducted on the neural pathways for attention blindsight. The few studies that have been conducted have focused on the redundant target effect. Leh and colleagues (2006) examined this effect in patients with and without blindsight following hemispherectomy. In separate blocks of trials, they used monochromatic (i.e., black/white) stimuli, which can activate the superior colliculus, and isoluminant blue/yellow stimuli, which activate short wavelength S-cones that do not project to the superior colliculus (Sumner et al., 2002). The redundant target effect was only evident for monochromatic but not S-cone stimuli, suggesting that this form of attention blindsight (as least in hemispherectomy patients) may be

mediated by the superior colliculus (see also Leh et al., 2010; Marzi et al., 2009; Tamietto et al., 2010 for supporting behavioural and neuroimaging results).

In the case of affective blindsight, it has been argued that the automatic processing of emotional information is served by a subcortical pathway projecting from the retina to the superior colliculus and pulvinar and from there to the amygdala, bypassing V1 (Figure 2; for review see Diano et al., 2016; Pessoa & Adolphs, 2010; Tamietto & de Gelder, 2010). Others have argued that the anatomical evidence for this pathway is limited (e.g., Pessoa & Adolphs, 2010). In a review of the anatomical literature, Pessoa and Adolphs (2010) pointed out that, while the connections from the superior colliculus to the inferior pulvinar are strongly interconnected with visual areas within the dorsal stream (Kaas & Lyon, 2007), inputs to the amygdala come from the medial pulvinar, which is also connected with a number of different areas, including ventral stream regions (Amaral et al., 2003; Freese & Amaral, 2005). It is undoubtedly the case that the amygdala plays an important role in affective blindsight. However, it may be that the connection between the superior colliculus/pulvinar and amygdala is mediated by input from other cortical regions (Pessoa & Adolphs, 2010). Of note though, our recent report of affective blindsight in a patient with large bilateral lesions implies that input from face processing regions such as fusiform and occipital face areas are *not* required for affective blindsight (Striemer et al., 2019b; see also Tamietto et al., 2012).

The pathways involved in agnosopsia are even less clear. Some speculate that this may be mediated by cells in the koniocellular layers of the lateral geniculate nucleus, which lie between the parvo- and magnocellular layers, and which project to both striate and extrastriate regions (Sincich et al., 2004). In area MT, for example, 10% of the input is directly from the

geniculate nucleus, and of these 63% are from the koniocellular layers (Sinchich et al., 2004). Such extra-striate projections of koniocellular origin may support functions ranging from wavelength and form discrimination, to motion discrimination. However, more work is needed to directly link anatomy with behavior in these secondary pathways.

In humans, diffusion tensor imaging provides some insight into the secondary pathways that may support blindsight (Leh et al., 2006; Ptito & Leh, 2007). In partially hemispherectomised patients, this imaging technique has shown intact projections from the superior colliculus to visual association areas and even to frontal regions involved in the guidance of saccades (i.e., the Frontal Eye Fields; Leh et al., 2006), but only in those patients with evidence of blindsight. In patients with damage restricted to V1 diffusion tensor imaging has also been used to show intact pathways from the lateral geniculate nucleus to motion processing regions (i.e., MT+; Ajina et al., 2015). Again, this pathway was only evident in patients demonstrating blindsight for motion. Of note, their study also did not find support for the involvement of the superior colliculus in motion processing in blindsight. In healthy individuals, the same imaging method has confirmed the existence of secondary pathways that bypass V1 and connect human motion processing regions (area V5/MT) with posterior thalamic nuclei and the superior colliculus (Lanyon et al., 2009; Pedersini et al., 2020; see Tamietto & Morrone, 2016 for an excellent recent review).

Implications

The anatomic implications of blindsight seem fairly obvious to us now; there are multiple pathways for visual information to reach cortical regions and not all need to be relayed

through primary visual cortex to influence behaviour. The fact that such pathways exist and that they can support some residual visual function in the absence of conscious awareness belies another question – what function do these residual pathways serve? One possibility is that secondary pathways provide low resolution representations of visual information that are suitable for rapid responding. In the context of affective blindsight, such a rapid function may be adaptive in generating fight-or-flight behaviours in response to emotionally arousing stimuli. This speculation is perhaps supported by recent evidence that affective blindsight relies on the processing of low spatial frequencies (Burra et al., 2019). In our own work on affective blindsight (Striemer et al., 2019b), the patient's performance fell to chance when she was asked to provide a subjective confidence rating of her discrimination choices after each trial. Perhaps, at least for this patient, the demonstration of affective blindsight relies on patients making fast 'gut' reactions, reactions that were hindered by the requirement to indicate confidence.

The idea that blindsight pathways may serve rapid responses to visual input, separate from the geniculostriate pathway that supports conscious vision, also accords with the frequently demonstrated blindsight response to moving stimuli. There are many circumstances in which a rapid response to movement in the periphery is critical, from obstacle avoidance to intercepting moving objects (e.g., imagine dodging an errant baseball thrown in your direction). These pathways may have had a more prominent role in controlling behavior earlier in our evolutionary history. In a similar vein, these pathways may be more active early in neural development when visual-cognitive capacities are immature and the environment is hazardous (Bridge et al., 2016). Prior to developing the sophisticated visual recognition capacities supported by the geniculostriate and ventral visual pathways, we may rely more heavily on

signals that convey looming objects, obstacles, or movement, in these more rudimentary pathways (Bridge et al., 2016). Such a hypothesis is difficult if not impossible to test in humans. However, recent work in marmosets suggests that V1 lesions acquired in early life is accompanied by preservation of connections from the retina via the pulvinar to motion sensitive area MT (Warner et al., 2015). In contrast, retinal input to the lateral geniculate nucleus deteriorates following these early V1 lesions. Previous work by Payne and colleagues (1996, 1998) in cats and Moore and colleagues (1995, 1996) in monkeys has also shown that residual visual abilities are more robust in animals lesioned early in development. Although speculative, this may explain why blindsight seems to be more prominent in humans whose visual cortical damage was sustained early in life. For example GY, who has demonstrated an extraordinary range of residual visual abilities, suffered occipital damage after a car accident when he was 7 years old (Barbur et al., 1993).

From Bard's, Pick's and Riddoch's early findings in the early part of the 20th Century, to modern tractography and functional neuroimaging studies, the residual visual capacities in the fascinating phenomenon of blindsight demonstrate the sheer complexity of visual processing in the primate brain. Visual processing is highly distributed not only within visual cortex itself, but along distinct subcortico-cortical pathways. We may yet be at the threshold of new discoveries as we start to ask more challenging questions about the functions that are served by these so-called 'secondary' visual pathways. Furthermore, the fact that we can still process visual information outside of awareness forces us to contemplate difficult philosophical questions regarding the nature and function of consciousness (e.g., Block, 1995; Brogaard, 2011, 2015).

Acknowledgements

The authors would like to thank Adam Morrill for help with Figure 2. This work was supported by Natural Sciences and Engineering Research Council Discovery grants to JD and CS and by the Labex/Idex CORTEX ANR-11-LABX-0042, CNRS, INSERM and the Hospices Civils de Lyon grants to YR.

Figure 1. The upper panel shows a schematic of an eye movement trial for blind field looking in the Weiskrantz et al., (1974) paper. This is typical of blindsight work in that stimuli are presented in the blind field and extinguished before a go signal tells the patient to perform the task. The vertical, bidirectional arrow in the target panel indicates that in this study targets for eye movements were jittered up and down within a 4 degree range, perhaps inducing Riddoch's phenomena. The middle panel provides a schematic of a typical setup showing blind field pointing while central fixation is maintained. The lower panels show saccadic (left) and pointing (right) data from patient DB adapted from Figures 2 and 3 in Weiskrantz et al (1974). Perfect performance would fall along the dotted line, bars represent the range of movements made.

Figure 2. A schematic of the known visual pathways in the primate brain. The primary (i.e., geniculostriate) visual pathway relays approximately 80-90% of the information from the retina to the lateral geniculate nucleus (lateral geniculate nucleus) and from there to primary visual cortex (V1). However, even if V1 (or post-geniculate structures) are damaged there are secondary visual pathways (in blue) through which visual information can be relayed to extrastriate regions in the dorsal and ventral visual streams from the lateral geniculate nucleus, or from the superior colliculus (superior colliculus) via the pulvinar (Pulv.) nucleus. These secondary pathways are thought to be responsible for the residual visual functions observed in blindsight. In the case of affective blindsight (pink) an additional connection from the pulvinar to the amygdala (Amyg.) is thought to relay the visual information necessary for affective processing.

References

- Ajina, S., & Bridge, H. (2017). Blindsight and unconscious vision: what they teach us about the human visual system. *The Neuroscientist*, 23, 529–541.
- Ajina, S., Pestilli, F., Rokem, A., Kennard, C., & Bridge, H. (2015). Human blindsight is mediated by an intact geniculo-extrastriate pathway. *Elife*, 4, e08935.
- Amaral, D. G., Behniea, H., & Kelly, J. L. (2003). Topographic organization of projections from the amygdala to the visual cortex in the macaque monkey. *Neuroscience*, 118, 1099–1120.
- Arcaro, M. J., Thaler, L., Quinlan, D. J., Monaco, S., Khan, S., Valyear, K. F., ... & Culham, J. C. (2019). Psychophysical and neuroimaging responses to moving stimuli in a patient with the Riddoch phenomenon due to bilateral visual cortex lesions. *Neuropsychologia*, 128, 150–165.
- Azzopardi, P., & Cowey, A. (1997). Is blindsight like normal, near-threshold vision?. *Proceedings of the National Academy of Sciences*, 94, 14190–14194.
- Barbur, J. L., Watson, J. D. G., Frackowiak, R. S., & Zeki, S. (1993). Conscious visual perception without V1. *Brain*, 116, 1293–1302.
- Bard, L. (1905). De la persistance des sensations lumineuses dans le champ aveugle des hémianopsiques. *La Semaine Médicale*, 22(May), 3–255.
- Barton, J. J., & Sharpe, J. A. (1997). Motion direction discrimination in blind hemifields. *Annals of Neurology*, 41, 255–264.

- Baseler, H. A., Morland, A. B., & Wandell, B. A. (1999). Topographic organization of human visual areas in the absence of input from primary cortex. *Journal of Neuroscience*, *19*, 2619–2627.
- Bertini, C., Cecere, R., Ladavas, E., (2013). I am blind, but I "see" fear. *Cortex*, *49*, 985–993.
- Bertini, C., Cecere, R., & Ladavas, E. (2019). Unseen fearful faces facilitate visual discrimination in the intact field. *Neuropsychologia*, *128*, 58–64.
- Block, N. (1995). On a confusion about a function of consciousness. *Behavioral and brain sciences*, *18*(2), 227-247.
- Bridge, H., Leopold, D. A., & Bourne, J. A. (2016). Adaptive pulvinar circuitry supports visual cognition. *Trends in Cognitive Sciences*, *20*, 146–157.
- Brogaard, B. (2011). Are there unconscious perceptual processes?. *Consciousness and cognition*, *20*(2), 449-463.
- Brogaard, B. (2015). Type 2 blindsight and the nature of visual experience. *Consciousness and Cognition*, *32*, 92–103.
- Bullier, J., & Kennedy, H. (1983). Projection of the lateral geniculate nucleus onto cortical area V2 in the macaque monkey. *Experimental Brain Research*, *53*, 168–172.
- Burra, N., Hervais-Adelman, A., Celeghin, A., De Gelder, B., & Pegna, A. J. (2019). Affective blindsight relies on low spatial frequencies. *Neuropsychologia*, *128*, 44–49.
- Burra, N., Hervais-Adelman, A., Kerzel, D., Tamietto, M., De Gelder, B., & Pegna, A. J. (2013). Amygdala activation for eye contact despite complete cortical blindness. *Journal of Neuroscience*, *33*, 10483–10489.

- Campion, J., Latto, R., & Smith, Y. M. (1983). Is blindsight an effect of scattered light, spared cortex, and near-threshold vision. *Behavioral and Brain Sciences*, 6, 423–486.
- Cecere, R., Bertini, C., Maier, M.E., Ladavas, E., 2014. Unseen fearful faces influence face encoding: evidence from ERPs in hemianopic patients. *Journal of Cognitive Neuroscience*, 26, 2564–2577.
- Celeghin, A., de Gelder, B., & Tamietto, M. (2015). From affective blindsight to emotional consciousness. *Consciousness and Cognition*, 36, 414–425.
- Chabanat, E., Jacquin-Courtois, S., Havé, L., Kihoulou, C., Tilikete, C., Mauguière, F., ... & Rossetti, Y. (2019). Can you guess the colour of this moving object? A dissociation between colour and motion in blindsight. *Neuropsychologia*, 128, 204–208.
- Chapman, C. S., & Goodale, M. A. (2010). Seeing all the obstacles in your way: the effect of visual feedback and visual feedback schedule on obstacle avoidance while reaching. *Experimental Brain Research*, 202, 363–375.
- Chapman, C. S., & Goodale, M. A. (2008). Missing in action: the effect of obstacle position and size on avoidance while reaching. *Experimental Brain Research*, 191, 83–97.
- Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience*, 3, 201–215.
- Corbetta, M., Marzi, C. A., Tassinari, G., & Aglioti, S. (1990). Effectiveness of different task paradigms in revealing blindsight. *Brain*, 113, 603–616.
- Cowey, A. (2010). The blindsight saga. *Experimental Brain Research*, 200, 3–24.
- Cowey, A., & Stoerig, P. (1995). Blindsight in monkeys. *Nature*, 373, 247–249.
- Danckert, J., Tamietto, M., & Rossetti, Y. (2019). Definition: Blindsight. *Cortex*, 119, 569–570.

- Danckert, J., & Culham, J. C. (2010). Reflections on blindsight: Neuroimaging and behavioural explorations clarify a case of reversed localisation in the blind field of a patient with hemianopia. *Canadian Journal of Experimental Psychology*, 64, 86–101.
- Danckert, J., & Rossetti, Y. (2005). Blindsight in action: what can the different sub-types of blindsight tell us about the control of visually guided actions?. *Neuroscience & Biobehavioral Reviews*, 29, 1035–1046.
- Danckert, J., Revol, P., Pisella, L., Krolak-Salmon, P., Vighetto, A., Goodale, M. A., & Rossetti, Y. (2003). Measuring unconscious actions in action-blindsight: exploring the kinematics of pointing movements to targets in the blind field of two patients with cortical hemianopia. *Neuropsychologia*, 41, 1068–1081.
- 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.
- de Gelder, B., Tamietto, M., Van Boxtel, G., Goebel, R., Sahraie, A., Van den Stock, J., ... & Pegna, A. (2008). Intact navigation skills after bilateral loss of striate cortex. *Current Biology*, 18, R1128–R1129.
- de Gelder, B., Morris, J. S., & Dolan, R. J. (2005). Unconscious fear influences emotional awareness of faces and voices. *Proceedings of the National Academy of Sciences*, 102, 18682–18687.
- de Gelder, B., Vroomen, J., Pourtois, G., & Weiskrantz, L. (1999). Non-conscious recognition of affect in the absence of striate cortex. *Neuroreport*, 10, 3759–3763.
- Diano, M., Celeghin, A., Bagnis, A., & Tamietto, M. (2017). Amygdala response to emotional stimuli without awareness: facts and interpretations. *Frontiers in Psychology*, 7, 2029.

- Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & Psychophysics*, 16, 143–149.
- Faubert, J., Diaconu, V., Ptito, M., & Ptito, A. (1999). Residual vision in the blind field of hemidecorticated humans predicted by a diffusion scatter model and selective spectral absorption of the human eye. *Vision Research*, 39, 149–157.
- Feinberg, T. E., Pasik, T., & Pasik, P. (1978). Extrageniculostriate vision in the monkey: VI. Visually guided accurate reaching behavior. *Brain Research*, 152, 422–428.
- Fendrich, R., Wessinger, C. M., & Gazzaniga, M. S. (2001). Speculations on the neural basis of islands of blindsight. *Progress in Brain Research*, 134, 353–366.
- Fendrich, R., Wessinger, C. M., & Gazzaniga, M. S. (1992). Residual vision in a scotoma: implications for blindsight. *Science*, 258, 1489–1491.
- Fox, C. J., Moon, S. Y., Iaria, G., & Barton, J. J. (2009). The correlates of subjective perception of identity and expression in the face network: an fMRI adaptation study. *Neuroimage*, 44, 569–580.
- Freese, J. L., & Amaral, D. G. (2005). The organization of projections from the amygdala to visual cortical areas TE and V1 in the macaque monkey. *Journal of Comparative Neurology*, 486, 295–317.
- Gazzaniga, M. S., Fendrich, R., & Wessinger, C. M. (1994). Blindsight reconsidered. *Current Directions in Psychological Science*, 3, 93–96.
- Georgy, L., Celeghin, A., Marzi, C. A., Tamietto, M., & Ptito, A. (2016). The superior colliculus is sensitive to gestalt-like stimulus configuration in hemispherectomy patients. *Cortex*, 81, 151–161.

- Gobbini, M. I., & Haxby, J. V. (2007). Neural systems for recognition of familiar faces. *Neuropsychologia*, 45(1), 32-41.
- Goebel, R., Muckli, L., Zanella, F. E., Singer, W., & Stoerig, P. (2001). Sustained extrastriate cortical activation without visual awareness revealed by fMRI studies of hemianopic patients. *Vision Research*, 41, 1459–1474.
- Hendry, S. H., & Reid, R. C. (2000). The koniocellular pathway in primate vision. *Annual Review of Neuroscience*, 23, 127–153.
- Humphrey, N. K. (1974). Vision in a monkey without striate cortex: a case study. *Perception*, 3, 241–255.
- Jackson, S. R. (1999). Pathological perceptual completion in hemianopia extends to the control of reach-to-grasp movements. *Neuroreport*, 10, 2461–2466.
- Kaas, J. H., & Lyon, D. C. (2007). Pulvinar contributions to the dorsal and ventral streams of visual processing in primates. *Brain Research Reviews*, 55, 285–296.
- Kentridge, R. W. (2015). What is it like to have type-2 blindsight? Drawing inferences from residual function in type-1 blindsight. *Consciousness and Cognition*, 32, 41–44.
- Kentridge, R. W., Heywood, C. A., & Weiskrantz, L. (2004). Spatial attention speeds discrimination without awareness in blindsight. *Neuropsychologia*, 42, 831–835.
- Kentridge, R. W., Heywood, C. A., & Weiskrantz, L. (1999a). Effects of temporal cueing on residual visual discrimination in blindsight. *Neuropsychologia*, 37, 479–483.
- Kentridge, R. W., Heywood, C. A., & Weiskrantz, L. (1999b). Attention without awareness in blindsight. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 266, 1805–1811.

- Kentridge, R. W., Heywood, C. A., & Weiskrantz, L. (1997). Residual vision in multiple retinal locations within a scotoma: Implications for blindsight. *Journal of Cognitive Neuroscience*, 9, 191–202.
- King, S. M., Azzopardi, P., Cowey, A., Oxbury, J., & Oxbury, S. (1996). The role of light scatter in the residual visual sensitivity of patients with complete cerebral hemispherectomy. *Visual Neuroscience*, 13, 1–13.
- Lanyon, L. J., Giaschi, D., Young, S. A., Fitzpatrick, K., Diao, L., Bjornson, B. H., & Barton, J. J. (2009). Combined functional MRI and diffusion tensor imaging analysis of visual motion pathways. *Journal of Neuro-Ophthalmology*, 29, 96–103.
- Leh, S. E., Ptito, A., Schönwiesner, M., Chakravarty, M. M., & Mullen, K. T. (2010). Blindsight mediated by an S-cone-independent collicular pathway: an fMRI study in hemispherectomized subjects. *Journal of Cognitive Neuroscience*, 22, 670–682.
- Leh, S. E., Mullen, K. T., & Ptito, A. (2006). Absence of S-cone input in human blindsight following hemispherectomy. *European Journal of Neuroscience*, 24, 2954–2960.
- Leopold, D. A. (2012). Primary visual cortex: awareness and blindsight. *Annual Review of Neuroscience*, 35, 91–109.
- Lyon, D. C., Nassi, J. J., & Callaway, E. M. (2010). A disynaptic relay from superior colliculus to dorsal stream visual cortex in macaque monkey. *Neuron*, 65, 270–279.
- Mazzi, C., Bagattini, C., & Savazzi, S. (2016). Blind-sight vs. degraded-sight: different measures tell a different story. *Frontiers in Psychology*, 7, 901.

- Marzi, C. A., Mancini, F., Metitieri, T., & Savazzi, S. (2009). Blindsight following visual cortex deafferentation disappears with purple and red stimuli: A case study. *Neuropsychologia*, 47, 1382–1385.
- Marzi, C. A., Tassinari, G., Aglioti, S., & Lutzemberger, L. (1986). Spatial summation across the vertical meridian in hemianopsics: A test of blindsight. *Neuropsychologia*, 24, 749–758.
- Milner, D., & Goodale, M. (2006). *The visual brain in action* (Vol. 27). Oxford University Press. Oxford.
- Mon-Williams, M., Tresilian, J. R., Coppard, V. L., & Carson, R. G. (2001). The effect of obstacle position on reach-to-grasp movements. *Experimental Brain Research*, 137, 497–501.
- Moore, T., Rodman, H. R., Repp, A. B., Gross, C. G., & Mezrich, R. S. (1996). Greater residual vision in monkeys after striate cortex damage in infancy. *Journal of Neurophysiology*, 76, 3928–3933.
- Moore, T., Rodman, H. R., Repp, A. B., & Gross, C. G. (1995). Localization of visual stimuli after striate cortex damage in monkeys: parallels with human blindsight. *Proceedings of the National Academy of Sciences*, 92, 8215–8218.
- Morris, J. S., Öhman, A., & Dolan, R. J. (1999). A subcortical pathway to the right amygdala mediating “unseen” fear. *Proceedings of the National Academy of Sciences*, 96, 1680–1685.
- Nassi, J. J., Lyon, D. C., & Callaway, E. M. (2006). The parvocellular lateral geniculate nucleus provides a robust disynaptic input to the visual motion area MT. *Neuron*, 50, 319–327.
- Payne, B. R., & Lomber, S. G. (1998). Neuroplasticity in the cat’s visual system Origin, termination, expansion, and increased coupling of the retino-geniculo-middle

- suprasylvian visual pathway following early ablations of areas 17 and 18. *Experimental Brain Research*, 121, 334–349.
- Payne, B. R., Lomber, S. G., Macneil, M. A., & Cornwell, P. (1996). Evidence for greater sight in blindsight following damage of primary visual cortex early in life. *Neuropsychologia*, 34, 741–774.
- Pedersini, C. A., Lingnau, A., Cardobi, N., Sanchez-Lopez, J., Savazzi, S., & Marzi, C. A. (2020). Neural bases of visual processing of moving and stationary stimuli presented to the blind hemifield of hemianopic patients. *Neuropsychologia*, 141, 1–17.
- Pegna, A. J., Khateb, A., Lazeyras, F., & Seghier, M. L. (2005). Discriminating emotional faces without primary visual cortices involves the right amygdala. *Nature Neuroscience*, 8, 24–25.
- Perenin, M-T, & Jeannerod, M. (1975) Residual vision in cortically blind hemifields. *Neuropsychologia*, 13, 1–7.
- Perenin, M-T, & Jeannerod, M. (1978) Visual function within the hemianopic field following early cerebral hemidecortication in man. I. Spatial localization. *Neuropsychologia*. 16, 1–13.
- Perenin, M. T., & Rossetti, Y. (1996). Grasping in an hemianopic field. Another instance of dissociation between perception and action. *Neuroreport*, 7, 793–797.
- Pessoa, L., & Adolphs, R. (2010). Emotion processing and the amygdala: from a 'low road' to 'many roads' of evaluating biological significance. *Nature Reviews Neuroscience*, 11, 773–782.

- Pick, A. (1905). Ueber den Gang der Rückbildung hemianopsischer Störungen nach paralytischen Anfällen. *Deutsche Med Wochenschr* 31, 39.
- Pöppel, E., Held, R., & Frost, D. (1973). Residual visual function after brain wounds involving the central visual pathways in man. *Nature*, 243, 295–296.
- Prentiss, E. K., Schneider, C. L., Williams, Z. R., Sahin, B., & Mahon, B. Z. (2018). Spontaneous in-flight accommodation of hand orientation to unseen grasp targets: a case of action blindsight. *Cognitive Neuropsychology*, 35, 343–351.
- Ptito, A., & Leh, S. E. (2007). Neural substrates of blindsight after hemispherectomy. *The Neuroscientist*, 13, 506–518.
- Ptito, A., Fortin, A., & Ptito, M. (2001). Seeing in the blind hemifield following hemispherectomy. *Progress in Brain Research*, 134, 367–378.
- Ptito, A., Lepore, F., Ptito, M., & Lassonde, M. (1991). Target detection and movement discrimination in the blind field of hemispherectomized patients. *Brain*, 114, 497–512.
- Ptito, A., Lassonde, M., Leporé, F., & Ptito, M. (1987). Visual discrimination in hemispherectomized patients. *Neuropsychologia*, 25, 869–879.
- Riddoch, G. (1917). Dissociation of visual perceptions due to occipital injuries, with especial reference to appreciation of movement. *Brain*, 40, 15–57.
- Ross, A. I., Schenk, T., Billino, J., Macleod, M. J., & Hesse, C. (2018). Avoiding unseen obstacles: Subcortical vision is not sufficient to maintain normal obstacle avoidance behaviour during reaching. *Cortex*, 98, 177–193.
- Rossetti, Y. (1998) Implicit short-lived motor representations of space in brain damaged and healthy subjects. *Conscious & Cognition*, 7, 520–558.

- Rossetti Y., Pisella L., & Pélisson, D. (2000) New insights on eye blindness and hand sight: temporal constraints of visuo-motor networks. *Visual Cognition*, 7, 785–808.
- Sahraie, A. (2007). Induced visual sensitivity changes in chronic hemianopia. *Current Opinion in Neurology*, 20, 661–666.
- Saling, M., Alberts, J., Stelmach, G. E., & Bloedel, J. R. (1998). Reach-to-grasp movements during obstacle avoidance. *Experimental Brain Research*, 118, 251–258.
- Sanders, M. D., Warrington, E., Marshall, J., & Wieskrantz, L. (1974). "Blindsight": Vision in a field defect. *The Lancet*, 303, 707–708.
- Schmid, M. C., Mrowka, S. W., Turchi, J., Saunders, R. C., Wilke, M., Peters, A. J., ... & Leopold, D. A. (2010). Blindsight depends on the lateral geniculate nucleus. *Nature*, 466, 373–377.
- Sincich, L. C., Park, K. F., Wohlgemuth, M. J., & Horton, J. C. (2004). Bypassing V1: a direct geniculate input to area MT. *Nature Neuroscience*, 7, 1123–1128.
- Smits, A. R., Seijdel, N., Scholte, H. S., Heywood, C. A., Kentridge, R. W., & de Haan, E. H. F. (2019). Action blindsight and antipointing in a hemianopic patient. *Neuropsychologia*, 128, 270–275.
- Stoerig, P., & Cowey, A. (2007). Blindsight. *Current Biology*, 17, R822–R824.
- Stoerig, P., & Cowey, A. (1997). Blindsight in man and monkey. *Brain*, 120, 535–559.
- Stoerig, P., & Cowey, A. (1992). Wavelength discrimination in blindsight. *Brain*, 115, 425–444.
- Stoerig, P., & Cowey, A. (1989). Wavelength sensitivity in blindsight. *Nature*, 342, 916–918.
- Striemer, C. L., Enns, J. T., & Whitwell, R. L. (2019a). Visuomotor adaptation in the absence of input from early visual cortex. *Cortex*, 115, 201–215.

- Striemer, C. L., Whitwell, R. L., & Goodale, M. A. (2019b). Affective blindsight in the absence of input from face processing regions in occipital-temporal cortex. *Neuropsychologia*, 128, 50–57.
- Striemer, C. L., Chapman, C. S., & Goodale, M. A. (2018). The role of non-conscious visual processing in obstacle avoidance: A commentary on Ross et al., 2016. *Cortex*, 98, 269–275.
- Striemer, C. L., Chapman, C. S., & Goodale, M. A. (2009). “Real-time” obstacle avoidance in the absence of primary visual cortex. *Proceedings of the National Academy of Sciences*, 106, 15996–16001.
- Sumner, P., Adamjee, T., & Mollon, J. D. (2002). Signals invisible to the collicular and magnocellular pathways can capture visual attention. *Current Biology*, 12, 1312–1316.
- Tamietto, M., & Morrone, M. C. (2016). Visual plasticity: blindsight bridges anatomy and function in the visual system. *Current Biology*, 26, R70–R73.
- Tamietto, M., Pullens, P., de Gelder, B., Weiskrantz, L., & Goebel, R. (2012). Subcortical connections to human amygdala and changes following destruction of the visual cortex. *Current Biology*, 22, 1449–1455.
- Tamietto, M., & De Gelder, B. (2010). Neural bases of the non-conscious perception of emotional signals. *Nature Reviews Neuroscience*, 11, 697–709.
- Tamietto, M., Cauda, F., Corazzini, L. L., Savazzi, S., Marzi, C. A., Goebel, R., ... & de Gelder, B. (2010). Collicular vision guides nonconscious behavior. *Journal of Cognitive Neuroscience*, 22, 888–902.

- Tomaiuolo, F., Ptito, M., Marzi, C. A., Paus, T., & Ptito, A. (1997). Blindsight in hemispherectomized patients as revealed by spatial summation across the vertical meridian. *Brain*, 120, 795–803.
- Trevethan, C. T., Sahraie, A., & Weiskrantz, L. (2007a). Form discrimination in a case of blindsight. *Neuropsychologia*, 45, 2092–2103.
- Trevethan, C. T., Sahraie, A., & Weiskrantz, L. (2007b). Can blindsight be superior to ‘sighted-sight’?. *Cognition*, 103, 491–501.
- van den Stock, J., Tamietto, M., Zhan, M., Heinecke, A., Hervais-Adelman, A. G., Legrand, L., ... & de Gelder, B. (2014). Neural correlates of body and face perception following bilateral destruction of the primary visual cortices. *Frontiers in Behavioral Neuroscience*, 8, 30.
- Warner, C. E., Kwan, W. C., Wright, D., Johnston, L. A., Egan, G. F., & Bourne, J. A. (2015). Preservation of vision by the pulvinar following early-life primary visual cortex lesions. *Current Biology*, 25, 424–434.
- Weiskrantz, L. (2010). Blindsight in Hindsight. *The Psychologist*, 23, 356–358.
- Weiskrantz, L. (1998). Consciousness and commentaries. *International Journal of Psychology*, 33, 227–233.
- Weiskrantz, L. (1987). Residual vision in a scotoma: A follow-up study of ‘form’ discrimination. *Brain*, 110, 77–92.
- Weiskrantz, L., Warrington, E. K., Sanders, M. D., & Marshall, J. (1974). Visual capacity in the hemianopic field following a restricted occipital ablation. *Brain*, 97, 709–728.

- Whalen, P. J., Rauch, S. L., Etcoff, N. L., McInerney, S. C., Lee, M. B., & Jenike, M. A. (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *Journal of Neuroscience*, *18*, 411–418.
- Whitwell, R. L., Striemer, C. L., Nicolle, D. A., & Goodale, M. A. (2011). Grasping the non-conscious: Preserved grip scaling to unseen objects for immediate but not delayed grasping following a unilateral lesion to primary visual cortex. *Vision Research*, *51*, 908–924.
- Zeki, S. (1995). The motion vision of the blind. *Neuroimage*, *2*, 231–235.
- Zeki, S., & Ffytche, D. H. (1998). The Riddoch syndrome: insights into the neurobiology of conscious vision. *Brain*, *121*, 25–45.
- Zihl, J., & Werth, R. (1984). Contributions to the study of “blindsight”—I. Can stray light account for saccadic localization in patients with postgeniculate field defects?. *Neuropsychologia*, *22*, 1–11.