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Why is "blindsight" blind? A new perspective on primary visual cortex, recurrent activity and visual awareness

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ABSTRACT

The neuropsychological phenomenon of blindsight has been taken to suggest that the primary visual cortex (V1) plays a unique role in visual awareness, and that extrastriate activation needs to be fed back to V1 in order for the content of that activation to be consciously perceived. The aim of this review is to evaluate this theoretical framework and to revisit its key tenets. Firstly, is blindsight truly a dissociation of awareness and visual detection? Secondly, is there sufficient evidence to rule out the possibility that the loss of awareness resulting from a V1 lesion simply reflects reduced extrastriate responsiveness, rather than a unique role of V1 in conscious experience? Evaluation of these arguments and the empirical evidence leads to the conclusion that the loss of phenomenal awareness in blindsight may not be due to feedback activity in V1 being the hallmark awareness. On the basis of existing literature, an alternative explanation of blindsight is proposed. In this view, visual awareness is a "global" cognitive function as its hallmark is the availability of information to a large number of perceptual and cognitive systems; this requires inter-areal long-range synchronous oscillatory activity. For these oscillations to arise, a specific temporal profile of neuronal activity is required, which is established through recurrent feedback activity involving V1 and the extrastriate cortex. When V1 is lesioned, the loss of recurrent activity prevents inter-areal networks on the basis of oscillatory activity. However, as limited amount of input can reach extrastriate cortex and some extrastriate neuronal selectivity is preserved, computations involving comparison of neural firing rates within a cortical area remain possible. This enables "local" read-out from specific brain regions, allowing for the detection and discrimination of basic visual attributes. Thus blindsight is blind due to lack of "global" long-range synchrony, and it functions via "local" neural readout from extrastriate areas.

1. Introduction

Investigations into the effect of posterior occipital lesions on human visual perception have concluded that the induced field defects are absolute (Holmes, 1918; Horton & Hoyt, 1991).[1] However, when assessed through forced choice paradigms, some patients with V1 damage are able to detect stimuli presented in their blind field, despite reporting a complete lack of

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[1] Riddoch (1917) argued that awareness of motion information may be selectively preserved. However, this observation may have been due to V1 lesion being incomplete in many of the patients, and motion processing being less susceptible to impairment (Cowey, 2004). In well-known blindsight patients in whom complete absence of V1 has been confirmed, motion awareness is not preserved (e.g. Weiskrantz, 1997; Stoerig & Cowey, 1995).

conscious visual experience. This phenomenon was first reported by Pöppel et al. (1973), whose sample of cortically blind patients was able to localize visual stimuli presented in their blind field with eye movements. Subsequently it was shown that patients with V1 damage can localize unseen stimuli also by pointing, even more accurately so than by eye movements (see Cowey, 2004, 2010 for reviews). This detection and discrimination ability in the absence of visual experience is known as blindsight (Weiskranz, Warrington, Sanders, & Marshall, 1974). Intriguingly, blindsight is not merely a subcortical phenomenon, as extrastriate regions in the damaged hemisphere can be activated by blind field stimuli (e.g. Goebel, Muckli, Zanella, Singer, & Stoerig, 2001; Rodman, Gross, & Albright, 1989a; Schmid, Panagiotaropoulos, Augath, Logothetis, & Smirnakis, 2009), and these activations are functionally significant, as their disruption by transcranial magnetic stimulation (TMS) impairs blindsight performance (Alexander & Cowey, 2009). Blindsight is likely to be mediated by pathways projecting directly to extrastriate cortex, bypassing V1 (Cowey & Stoerig, 1989; Rodman, Gross, & Albright, 1989b; Schmid et al., 2010).

The failure of extrastriate activation to reach awareness when V1 is lesioned, despite the ability of this activation to guide visual detection, appeared to suggest unique role for V1 in visual awareness. Blindsight thus challenged, together with a number of electrophysiological findings, the strictly hierarchical feedforward models of visual processing in which V1 was viewed as merely a source of extrastriate input (e.g. Crick & Koch, 1995). This led to a shift towards models emphasizing the role of recurrent processing in visual awareness, a view which gained substantial support primarily from brain stimulation studies carried out in both blindsight patients as well as in neurologically normal observers. However, the picture has recently been complicated by demonstrations of conscious experience in blindsight, as well as by evidence implicating feedback activity in V1 in *all* visual processing, whether conscious or not.

The aim of the present manuscript is to review these developments. Firstly, I will discuss the origins of the hierarchical feedforward model, its inability to explain various neurophysiological phenomena, and how feedback activity involving V1 took center stage in theories of awareness. Secondly, I will review the key evidence in favor of these views, as well as recent results which have challenged them. Thirdly I will evaluate two key arguments of the feedback model: is blindsight a true dissociation of awareness and visual detection? Are extrastriate responses relatively normal in the absence of V1, ruling out an explanation in terms of impoverished extrastriate responses? Finally, I will offer an explanation of blindsight in terms of "local" computations and "global" inter-areal neural synchrony.

2. Hierarchical and feedback models of visual processing and awareness

2.1. The hierarchical view of visual awareness

Two characteristics of the visual cortex strongly contributed to the conventional view that visual information processing giving rise to conscious perception is fundamentally hierarchical in nature (e.g. Crick & Koch, 1995). One of these is anatomical hierarchy, based on the principle that connections between visual areas are normally reciprocal in nature, and that within a reciprocal pair of connections, there are differences in the laminar distribution of cells of origin and axonal termination (Rockland & Pandya, 1979, 1981; Maunsell & Van Essen, 1983; Felleman & Van Essen, 1991). One type of projection arises primarily from cells in supragranular layers, with a small contribution of 10–15% from infragranular layers, and terminates in the granular layer; this type of pathway was named *feedforward* (or ascending) projection, and it provides the excitatory drive of neuronal activity (e.g. Orban, 1984; Maunsell & Van Essen, 1987; Stone, Dreher, & Leventhal, 1979). A second type of projection arises from cells in both supra-and infragranular layers and terminates most densely in layer I and/or VI. This type of pathway was named the *feedback* or descending projection and it has a modulatory influence on the activity induced by feedforward activity (e.g. Dreher, 1986; Van Essen & Maunsell, 1983). The model of cortical hierarchy was constructed such that each area was assigned just below the highest area to which it provides ascending input; visual areas sharing intermediate projections were placed on the same level of the hierarchy. In this hierarchy (see Fig. 1), V1 is at the bottom, and regions in the parietal, temporal and frontal regions at the top.

The second feature of the visual system contributing to the hierarchical view was functional specialization, which is a key feature of the extrastriate cortex (Zeki, 1974, 1978). For example, nearly all neurons in the upper part of the posterior bank of the superior temporal sulcus, now known as V5 or MT, respond to visual motion but not to other attributes such as wavelength (Zeki, 1974), a finding later confirmed by other studies (Albright, 1984; Van Essen, Maunsell, & Bixby, 1981). The motion selectivity of V5/MT has also been demonstrated in the human visual cortex (Zeki et al., 1991). The existence of a color-selective visual area, V4, has also been proposed (Zeki, 1973, 1977), although the precise location of a "color – center" in both the monkey and human cortex is still a matter of controversy (Heywood, Gadotti, & Cowey, 1992; Tootell, Nelissen, Vanduffel, & Orban, 2004; Hadjikhani et al., 1998). At higher levels of the ventral cortical hierarchy, neurons display an even higher level of specialization, for example, cells in the inferotemporal (IT) cortex are tuned to complex geometrical figures (e.g. Tanaka, 1993) and faces (Perrett et al., 1982). In contrast, regions (such as V1 and V2) at the bottom of the cortical hierarchy act as filters for a range of low-level stimulus attributes that fall within its receptive field (e.g. Van Essen et al., 1992).

Anatomical hierarchy coupled with functional specialization contributed to the view that visual processing proceeds in a feedforward fashion, with the role of lower-level areas such as V1 to merely provide input to regions at the top of the hierarchy where high-level visual perception and awareness arise (Crick & Koch, 1995; Felleman & Van Essen, 1991). This was, to an extent, consistent with Barlow's (1972) view that "perception corresponds to the activity of a small selection from the very numerous high-level neurons, each of which corresponds to a pattern of external events of the order of complexity
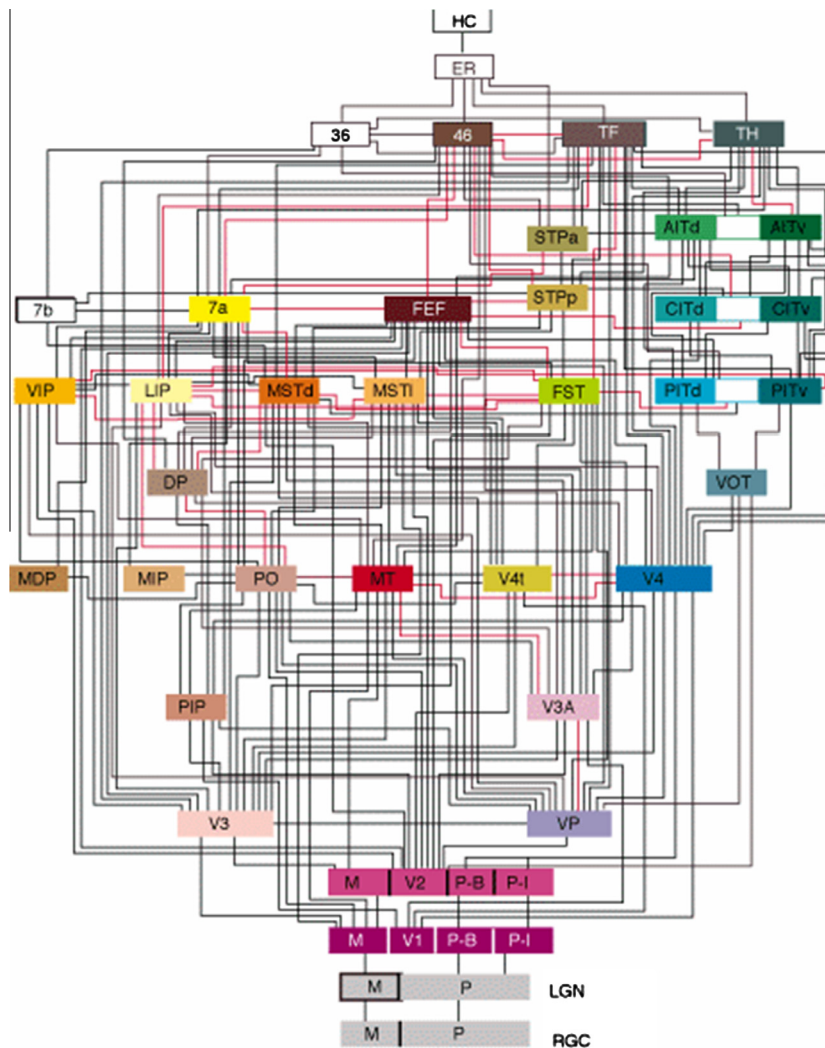
Fig. 1. Hierarchical organization of the monkey visual system (from Felleman & Van Essen, 1991). V1 is the lowest cortical level of the hierarchy, extrastriate areas are at intermediate levels, and temporal, parietal and frontal regions at the highest level. This anatomical hierarchy contributed to the view that visual processing proceeds in a feedforward fashion, with the role of lower-level areas such as V1 to merely provide input to higher-level regions where conscious experience arises.

of the events symbolized by a word". In more moderate versions of this view, perception was proposed to arise from joint activity of populations of high-level neurons in various regions in the visual cortex. For example, activation of neurons in the face-selective regions in the IT cortex would give rise to face perception and motion awareness would result from activation in the motion-selective area V5/MT (see e.g. microconsciousness theory by Zeki & Bartels, 1999).

2.2. Why anatomical hierarchy does not imply functional hierarchy: Problems with the hierarchical view of visual processing

Subsequently it became apparent that a strictly hierarchical model cannot account for a number of neurophysiological phenomena. One prediction of the feedforward model is that response latencies of a given visual area can be predicted from its level in the hierarchy; areas at high levels should have longer latencies than those at lower ones, as a result of the time required for the transfer of information from one level to the next. However, a direct comparison between onset latencies of single-unit responses between various levels of the hierarchy is inconsistent with this (Raiguel, Lagae, Gulyas, & Orban, 1989; Schmolesky et al., 1998). For instance, the frontal eye fields (FEF), placed at level 8 in the cortical hierarchy, exhibit visual latencies comparable to those in level 2 (V2), level 3/4 (V3), level 5 (V5) and level 6 (MST). Furthermore, the latencies in all of these areas were, on average, only 6–9 ms longer than the average V1 response. These results are in conflict with the view that information flow in the visual system proceeds in a strict feedforward fashion.

A second problem relates to the phenomenon that neuronal tuning evolves during the visually evoked response. A hierarchical model would predict that the functional role of a lower-level area is concluded once it has fed information forward

to the higher level. However, this is difficult to reconcile with the findings that "early" and "late" components of evoked responses in V1 can be functionally distinct. In V1, 50 ms after the presentation of a textured figure overlying a textured background, neurons show selectivity for the local orientation of the line segments that make up the figure; at 80 ms, the figure ground boundary selectively evokes a larger response than the rest of the scene, and at 100 ms the elements of the interior of the figure evoke a stronger response than the background elements (Lamme, 1995; Zipser et al., 1996). Importantly, it is this late stage of V1 activity that correlates with the monkey's behavioral report, in terms of figure-ground segregation being reflected in neural responses (Super, Spekreijse, & Lamme, 2001). But in a strict feedforward model, these "late" responses would have no bearing on the information processing at higher levels, as they are not contained in the initial feedforward sweep.

A third, and perhaps the most striking phenomenon inconsistent with hierarchical models, is that normal responsiveness of neurons at the bottom of the cortical hierarchy (V1 and V2) is dependent on feedback from higher-level regions: inactivation of V5/MT leads to a significant decrease in neuronal responses in early visual areas, an effect present already in the earliest stages of the V1 response (Hupe et al., 1998, 2001). Specifically, feedback has been found to facilitate evoked responses to objects within the classical receptive field as well as enhance suppression induced by background stimuli in the surround. Thus feedback connections appear to act as a form of gain control of already active neurons (rather than a driver of otherwise silent neurons). Importantly, the effects were dependent on stimulus conditions, with strongest effects found for low salience targets (Hupe et al., 1998). This result demonstrates that the cortical hierarchy acts in "reverse"; anatomically lower levels are reliant on input from higher levels for normal responsiveness.

2.3. Recurrent models of visual processing

These neurophysiological phenomena challenged the strictly hierarchical conceptualization of information processing in the visual cortex and gave rise to various models emphasizing recurrent activity between visual areas. They appeared in the context of prior theories which proposed recursive or adaptive resonance networks to link the visual system by means of a series of ascending and descending pathways (Miller, Galanter, & Pribram, 1960; Milner, 1974; Grossberg, 1976; Edelman, 1978). The underlying principle of these models is that sensory data activate a feedback process wherein a learned template modulates the sensory data until a consensus is reached between what the data are (provided by bottom-up, feedforward input) and what we "expect" them to be (via top-down, feedback modulation) (Grossberg, 1976). The findings of Bullier's laboratory (Hupe et al., 1998, 2001) gave feedback models a strong neurophysiological foundation.

One of the models motivated by the new neurophysiological evidence was Bullier's (2001) highly influential Integrated Model of Visual Processing, which was not concerned with how conscious perception arises, but rather, attempted to explain how information across the visual scene can be integrated so that "global" properties such as shadows and lighting artifacts can be taken into account when the "local" aspects of the visual image are computed. In theory, this could be achieved with local horizontal connections within a single cortical area. However, a V1 axon can reach a distance of only 0.6 degrees of visual angle and as a result, transmission of information over a distance of one degree visual angle through horizontal connections would take 100 ms. As 90 per cent of a neuron's output is transmitted within the first 100 ms of its response, it is unlikely that the integration takes place through horizontal connections in V1 (Heller, Hertz, Kjaer, & Richmond, 1995). Due to their larger receptive fields, areas higher up in the dorsal and ventral processing streams are more capable of integrating information across long distances in the visual field. However, as higher visual areas are also more selective, this integration can only involve a particular stimulus attribute. In Bullier's (2001) model, the problem of long-distance integration of various stimulus parameters is solved by retroinjecting the global computations carried out by higher-level areas through feedback connections into V1 and V2, where they guide the fine-detail analysis. The convergent nature of feedback connections means that they can carry information from long distances in the visual field and are therefore perfectly suited for guiding the fine-detail analysis in V1 (Angelucci, Levitt, Walton, Hupe, & Bullier, 2002). Furthermore, as input from the magnocellular processing stream reaches the visual cortex approximately 20 ms earlier than that conducted by the parvocellular stream (Nowak, Munk, Girard, & Bullier, 1995; although smaller latency differences of no more than 10 ms have also been observed; e.g. Maunsell et al., 1999), it is ideal for providing the initial global percept that guides parvocellular processing in V1.

2.4. Recurrent models of visual awareness

The first theoretical framework proposing that reentry of information into lower-level visual areas is necessary for visual awareness specifically was put forward by Pollen (1999). Pollen's theory was an attempt to accommodate the findings that while V1 appears to be indispensable to visual awareness, its activation is insufficient to generate a percept if the integrity of regions such as the parietal cortex is disrupted. This in Pollen's (1999) model was accomplished through feedforward and feedback pathways that link visual areas together into recursive loops. All visual areas in the ventral processing stream from V1 through the inferotemporal cortex serve as possible substrates for different aspects of phenomenal visual experience. For conscious perception to arise, a consensus needs to be reached between the visual areas that process the given stimulus; this is achieved through feedforward and feedback pathways linking multiple cortical areas; conflicts of information prevent conscious perception of that stimulus. Synchronous neuronal activity on a short timescale within and across cortical areas may also serve as a marker that a "steady state" (or consensus) has been achieved, and is therefore a correlate of awareness. In Pollen's (1999) model, V1 and V2 provide respectively the fine-grained and medium-grained representations in the
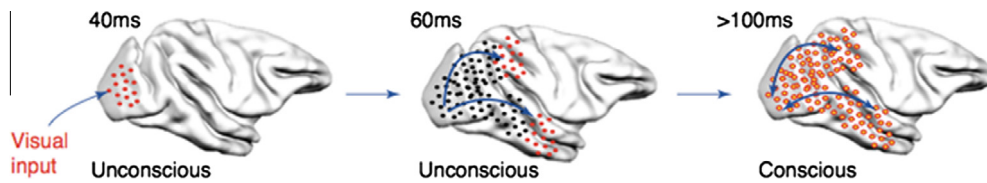
Fig. 2. In Lamme's model (2001), conscious visual experience requires recurrent processing. Visual input reaches the early visual areas (V1) at 40 ms after stimulus onset. Visual information is then rapidly fed forward to the extrastriate areas and parietal and temporal cortex (60 ms). This feedforward sweep of information processing is unconscious. At around 100 ms, early visual areas and higher areas engage in recurrent interactions, which are necessary for visual awareness. Specifically, extrastriate activation is fed back to V1 in order for that activation to be consciously perceived (from Lamme, 2001).

luminance domain. In contrast, phenomenal experience of complex objects and of three-dimensional representations of the visual world depend upon computations in V4 and the temporal lobe. Therefore, in this view the activity of which visual area mostly correlates with a phenomenal experience depends on the features of the stimulus.

2.5. Why blindsight appears to implicate V1 in visual awareness

Blindsight has played a key role in the study of visual awareness because it appears to demonstrate a direct link between V1 and conscious experience of visual qualia. Two lines of reasoning are central to this view. Firstly, the highly accurate unconscious detection and discrimination performance in blindsight has been taken to indicate that V1 lesions selectively impair conscious perception while leaving unconscious visual functions intact. The second argument involves the cortical basis of blindsight. Residual unconscious vision in the absence of V1 could be explained in terms of subcortical regions which continue to process visual information; and as visual awareness is often attributed to cortical activity, the unconscious nature of these visual functions would not be surprising. However, this possibility is ruled out by demonstrations of subcortical pathways which can activate extrastriate areas in the absence of V1 (e.g. Rodman et al., 1989a, 1989b; Cowey & Stoerig, 1989; Goebel et al., 2001; Schmid et al., 2010) and by brain stimulation evidence indicating that these extrastriate activations are necessary for blindsight (Alexander & Cowey, 2009). Thus in the absence of V1, the argument goes, extrastriate regions can be activated and this activation can guide visual functions but not reach conscious experience. These two arguments are paramount: if extrastriate neural responses were severely impoverished by the V1 lesion and only few low-level visual functions survived, it would be more parsimonious to attribute the loss of awareness to reduced functioning of the extrastriate visual cortex.

The dissociation between conscious and unconscious visual processing, together with demonstrations of the cortical basis of blindsight, gave rise to the view that conscious experience of *all* visual attributes relies on V1 and that *all* extrastriate activation needs to be fed back to V1 in order for its content to be consciously perceived. This possibility has been speculated by various researchers (e.g. Stoerig & Cowey, 1995; Weiskrantz, 1997), but it was formalized in Lamme's (2001) influential model linking V1, feedback and visual awareness. Its main premise is that unconscious visuo-motor transformations (as in blindsight) may be executed in an entirely feedforward processing cycle, while visual awareness is critically dependent on feedback connections to V1 (see Fig. 2; see also Block, 2005). As soon as a region has been activated by the feedforward sweep, recurrent interactions between neurons within that area and neurons that have been activated earlier at lower levels can begin. These interactions are mediated by horizontal connections and feedforward/feedback circuits between and within areas. They are expressed in neuronal responses as modulatory influences from beyond the classical, feedforward, receptive field (Albright & Stoner, 2002; Lamme & Spekreijse, 2000).[2]

3. Key evidence on the importance of extrastriate-V1 feedback in visual awareness

3.1. Evidence consistent with the feedback model

Much of the subsequent key evidence on the role of feedback activity in visual awareness has come from studies employing transcranial magnetic stimulation (TMS). TMS has a number of properties that makes it an ideal tool for testing feedback models (see e.g. Sandrini, Umiltà, & Rusconi, 2011; Silvanto, 2013; Van de Ven & Sack, 2013; Walsh & Pascual-Leone, 2003). Firstly, it can assess the necessity of cortical regions in perceptual functions with millisecond precision and a spatial resolution of approximately one centimeter[3] (e.g. Amassian et al., 1989). Secondly, because the neural activity induced by TMS spreads to anatomically connected regions (e.g. Ilmoniemi et al., 1997), corticocortical information flow can be traced. Thirdly, TMS can directly activate regions that are no longer receiving feedforward input due to a lesion lower down in the visual system. In some cases, it may be difficult to determine whether loss of functionality in a given brain area is due to local

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[2] However, why this modulation should be the hallmark of visual awareness has not been explicitly addressed.

[3] In contrast, functional magnetic resonance imaging (fMRI) cannot directly assess feedback activity due to its slow temporal resolution, while the low spatial resolution of electroencephalography precludes strong conclusions regarding the cortical regions involved (see e.g. Tong, 2003 for fMRI evidence on the role of V1 in awareness). This review focuses on TMS studies as they form the backbone of the empirical evidence.
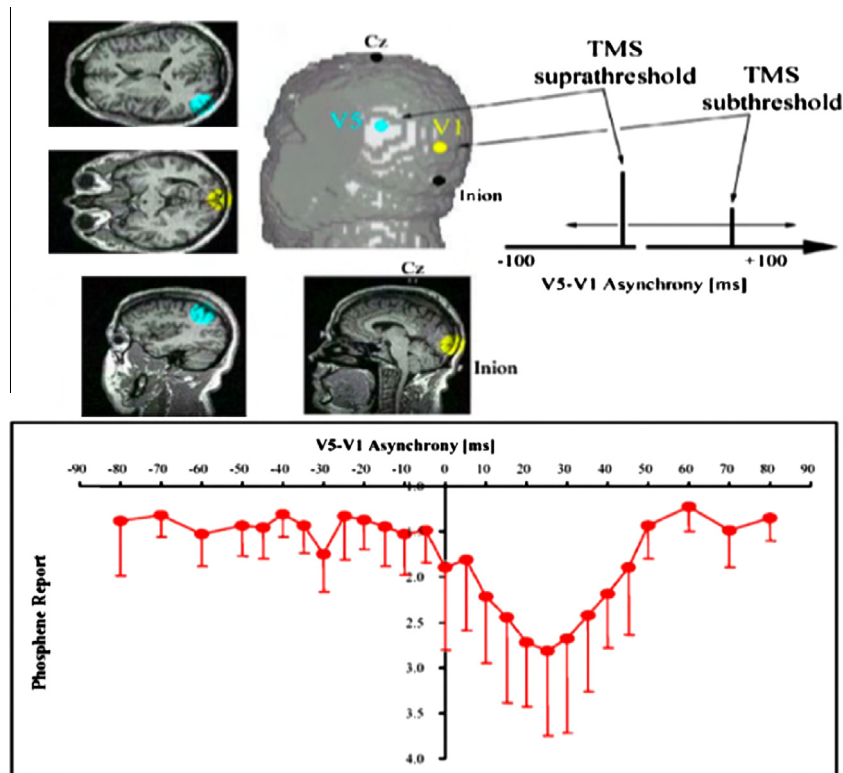
Fig. 3. Demonstration of the importance of extrastriate-V1 feedback in phosphene perception in neurologically normal observers (Pascual-Leone & Walsh, 2001). Subthreshold TMS applied over V1 *before* stimulation of V5/MT has no effect on the percepts induced by V5 TMS. But when the V1 TMS pulse *follows* the V5/MT stimulation, phosphene perception is impaired. The timing of these effects supports the view that feedback to V1 is necessary for the V5/MT activation to be consciously perceived (figure adapted from Juan et al., 2004).

damage or whether it arises from loss of input due to damage to lower levels in the processing stream. In such circumstances, TMS can be used directly to stimulate specific areas to test their functionality. This approach was used by Cowey and Walsh (2000), who applied TMS over presumably intact extrastriate visual areas such as V5/MT in the blindsight subject GY in order to test the ability of these regions to generate phosphenes (perception of flashes of light that can be induced by direct stimulation of the visual cortex) in the absence of V1. GY suffered almost total destruction of his left V1, resulting in complete blindness in the right visual field with the exception of some macular sparing (Azzopardi & Cowey, 2001). He was able to perceive phosphenes induced from the normal, right, hemisphere. But stimulation of intact extrastriate regions in the damaged hemisphere did not generate phosphenes, indicating that these areas cannot generate conscious percepts in the absence of V1 in the same hemisphere. (This conclusion assumes that the extrastriate neurons can be sufficiently activated in the absence of V1; as will be discussed in the last section, this may not be the case.)

Thus the early TMS evidence from blindsight was consistent with the view that activity in the extrastriate cortex needs to be fed back to V1 in order to be perceived consciously. If these feedback signals are disrupted, visual awareness of extrastriate activation cannot arise. Pascual-Leone and Walsh (2001) tested this hypothesis in neurologically normal observers by inducing phosphenes from V5/MT with suprathreshold TMS, and applying another TMS pulse over V1,[4] below phosphene threshold, at various stimulation-onset asynchronies (see Fig. 3). The logic was that, if V5/MT activity needs to be fed back to V1 in order to reach awareness, then the disruption of this feedback by the application of TMS over V1 should prevent the content of this activation from being consciously perceived. The results showed that when the V1 stimulation postdated the V5/MT pulse by 10–40 ms, phosphene motion was degraded. In contrast, when the V1 TMS pulse preceded the V5/MT stimulation, phosphene appearance was unaffected. From the temporal specificity of V1 involvement in the perception of V5/MT phosphenes it was inferred that extrastriate activation reaches awareness via V1.

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[4] While selective stimulation of V1 by TMS is unlikely to be possible as the application of TMS over the occipital pole also affects regions such as V2 and V3 (Salminen-Vaparanta, Koivisto, Noreika, Vanni, & Revonsuo, 2012; Thielscher, Reichenbach, Uğurbil, & Uludağ, 2010), neuropsychological evidence suggest that the suppressive effects of TMS in many visual tasks are most likely to reflect the disruption of V1 (see Pascual-Leone & Walsh, 2001), as lesions to areas such as V2 have more subtle perceptual consequences (e.g. Merigan et al., 1993).

A subsequent study employing a similar paired-pulse paradigm further supported this view by demonstrating that it is the activation level of V1 that determines whether V5/MT activation reaches awareness. Specifically, it was shown that participants perceived features (motion and shape) of their V5/MT phosphene even when V5/MT was stimulated at a subthreshold intensity, if this stimulation was followed 10–50 ms later a by V1 *suprathreshold* pulse (Silvanto, Cowey, Lavie, & Walsh, 2005). In contrast, when both sites were stimulated at subthreshold level, no phosphenes were perceived. Taken together with the findings of Pascual-Leone and Walsh (2001), this result suggested that the activation level of V5/MT does not determine whether motion is perceived; even though V5/MT influences information content in V1 via feedback connections, it is V1 activity level which determines whether that activation reaches awareness.

The importance of V5/MT-V1 feedback in visual awareness was also demonstrated in conscious perception of visually presented motion. In a study by Silvanto, Lavie, and Walsh (2005), TMS was administered over V1/V2 or V5/MT in different time windows during the performance of a motion detection task. The results showed two critical periods of V1 activity, one "early" window approximately 40 ms after stimulus offset and another "late" period at 80 ms after stimulus offset. Interestingly, the critical time window of V5/MT activity fell between the early and late V1/V2 time windows. This pattern of result suggested that V5/MT obtains visual information through V1 feedforward activity (reflected in the V5/MT critical time window postdating the early V1/V2 effect), after which the information is fed back from V5/MT to V1 (reflected in the "late" V1 critical period postdating the V5/MT critical time window). The "late" V1 activity in response to this feedback would underlie visual awareness.

3.2. Feedback to V1: critical for conscious perception per se or visual processing in general?

Although the above as well as numerous subsequent studies (e.g. Camprodon, Zohary, Brodbeck, & Pascual-Leone, 2010; Heinen, Jolij, & Lamme, 2005; Laycock, Crewther, Fitzgerald, & Crewther, 2007) supported the view that feedback activity plays an important role in visual perception, it was not clear whether this activity was unique to conscious perception or rather a general feature of visual cortical information processing. This issue has been examined in a number of studies. Koivisto, Mantyla, and Silvanto (2010) used TMS to investigate the necessity of the "late" V1 activity for both conscious and unconscious visual perception, through the use of both subjective and objective measures of motion perception on each trial. Specifically, participants were asked to provide a rating of their subjective perceptual experience of visually presented motion and in addition to perform a forced-choice direction discrimination task on the same stimuli. If the "late" period of V1 activity is unique to visual awareness, the application of TMS during this stage should weaken subjective experience of motion but leave forced-choice discrimination performance on unaware trials unaffected. In contrast, if the "late" V1 activity reflects a general principle of visual cortical information processing, TMS should disrupt performance in both cases. The results showed that both aware and unaware motion perception were impaired by TMS at the "late" time window: there was a significant reduction in both the strength of subjective experience of motion and the discrimination performance on unaware trials. Thus the "late" period of V1 activity in response to extrastriate feedback appears to be necessary for both aware *and* unaware visual perception.

This result seems inconsistent with the findings that blindsight patients can detect motion, as in such patients feedback to V1 cannot occur. One possible explanation is that in the study of Koivisto et al. (2010), V1 feedback was necessary because the motion was across very small distances; the target stimulus appeared within an imaginary square subtending 0.7×0.7 degrees of visual angle and the dots moved only pixel per frame. With such small-scale motion stimuli, the small V1 receptive fields might be needed for encoding the stimulus, and this could be accomplished by the engagement of V5/MT-V1 feedback connections (Hochstein & Ahissar, 2002). Indeed, as will be discussed in Section 4.1, detection of fine details is absent in blindsight.

Subsequent studies have been consistent with this result (e.g. de Graaf, Cornelsen, Jacobs, & Sack, 2011; Jacobs, de Graaf, Goebel, & Sack, 2012; Koivisto, Railo, Revonsuo, Vanni, & Salminen-Vaparanta, 2011). Jacobs et al. (2012) compared the critical time windows of V1 activity in behavioral priming and visual awareness. The behavioral impact of a prime can be independent from its conscious experience, suggesting two independent neural signatures. However, TMS revealed no time window at which TMS selectively affected visual awareness, indicating that this dissociation is not manifested in the timing of V1 involvement. One study by Koivisto et al. (2011) did find a weak dissociation between the effects of TMS on subjective awareness and forced-choice discrimination in a task involving the detection of the orientation of an arrow. Whereas subjective awareness was impaired by TMS at time windows ranging from 60 ms to 120 ms from stimulus onset, the forced-choice task was not significantly affected beyond 90 ms, indicating that the critical time window of awareness extended beyond that of the forced-choice task. However, the TMS effects on both awareness and accuracy peaked at 90 ms, and the dissociation may have been due to different susceptibilities of the two tasks to TMS-induced disruption. Indeed, the temporal profile of the TMS effect was very similar for the two judgments. Furthermore, the dissociation was found for only one stimulus type, indicating that the effect is not generalizable across stimulus categories.

In addition to V1, its neighboring region V2 has also been implicated in visual awareness (Salminen-Vaparanta, 2012). But as no dissociation between forced-choice performance and subjective awareness ratings was found, this result can also be explained in terms of V2 being involved in low-level, pre-conscious processes, the disruption of which will prevent the target stimulus from reaching phenomenal awareness. With respect to V2, it is also worth noting that its role in awareness is not

strongly supported by lesion evidence, as V2 damage impairs complex stimulus processing but does not have such a detrimental effect on phenomenal awareness as a V1 lesion (Merigan et al., 1993).

3.3. TMS-induced blindsight: evidence of conscious effects in V1 or merely of response bias?

Although both conscious and unconscious performance are impaired by V1 TMS at similar time windows, subjective Yes–No judgments of stimulus presence have been found to be more strongly affected than forced-choice discrimination performance (Boyer, Harrison, & Ro, 2005; Christensen, Kristiansen, Rowe, & Nielsen, 2008; Jolij & Lamme, 2005; Ro, Shelton, Lee, & Chang, 2004). Moreover, forced-choice accuracy has been found to remain above chance on trials in which participants report not having consciously seen the stimulus. This phenomenon is referred to as TMS-induced blindsight, because it appears to mirror the dissociation between objective and subjective measures of performance found in patients with V1 lesions. The problem with these studies is that participants' judgments in the Yes–No detection task depends partly on their bias, independently of sensitivity, to report the stimulus as being present or absent. Participants may have different criteria for deciding target presence, and under- or over-confidence will strongly affect the accuracy of forced-choice performance (e.g. left vs. right discrimination) on trials on which participants responded not having perceived the target in the Yes–No task. This issue can be overcome with the use of signal-detection theory (Green & Swets, 1966) in which the measure of sensitivity, d prime (d'), is independent of bias. A key finding in blindsight subject GY is that his receiver operator characteristic curves (ROCs) qualitatively differ from those of normal participants. Specifically, using SDT analyses, Azzopardi and Cowey (1997) showed that the blindsight subject GY was approximately half as sensitive to targets during the Yes–No task as during the forced-choice task. This difference was not statistically significant in the healthy controls. However, in TMS-induced blindsight, when signal-detection analysis is carried out, there is no longer evidence of unconscious stimulus detection or discrimination (Lloyd, Abrahamyan, & Harris, 2013), with the exception of affective information (Jolij & Lamme, 2005). In short, TMS-induced blindsight does not appear to be a robust phenomenon and therefore does not offer support for the view that V1 is uniquely involved in conscious perception.

In conclusion, while numerous TMS studies are certainly consistent with the view that feedback processes contribute to conscious experience, they fall short of demonstrating the existence of mechanisms unique to visual awareness. Thus while feedback processes are certainly important for visual perception, it does not seem parsimonious to attribute them exclusively to conscious experience (see e.g. Lau, 2008). It is also important to note the electrophysiological evidence indicating that the importance of extrastriate-V1 feedback is stimulus-dependent (Hupe et al., 1998), which would not be the case if they were the hallmark of *all* awareness. Nevertheless, it is important to acknowledge the contribution of TMS to the understanding of feedback processes in the human cortex. Since the pioneering studies of Cowey and Walsh (2000) and Pascual-Leone and Walsh (2001), much has been learnt about the information flow in the visual cortex and cortico-cortical interactions with this technique.

3.4. Conscious perception of visually presented information in the absence of V1

Theories linking V1 feedback with visual awareness rely strongly on the neuropsychological evidence of field defects being absolute. In contradiction with this, there have been claims that blindsight subjects are consciously aware of motion in their blind field (e.g. Barbur, Watson, Frackowiak, & Zeki, 1993; ffytche, Guy, & Zeki, 1996; ffytche & Zeki, 2011). Some of this evidence involves patients in whom the absence of V1 has not been verified using both anatomical and functional MRI, and paradigms in which eye movements were not controlled for (e.g. Ffytche & Zeki, 2011). However, such claims have also been made in relation to the blindsight subject GY, in whom no doubt exists as to the complete lack of V1 functionality (e.g. Goebel et al., 2001). Specifically, it was claimed that GY "...was able to discriminate correctly and faultlessly and to have conscious awareness of having seen the particular visual stimulus" (Barbur et al., 1993). GY himself denies having phenomenal visual experience in his blind field. In an interview with Weiskrantz (1997), he described his blind field experience as follows: "you don't actually ever sense anything or see anything...its more an awareness but you don't *see* it....it is a sense that I haven't got....if you said something to try to describe sight to a blind man, we don't have the words to do it because he does not have the receptors or the reception, and that is the same with me. I mean I can't describe something I don't understand myself". From this it appears that GY can have a nonvisual sense of events occurring in it; however, this "awareness" does not appear to be phenomenal in nature.

There are, however, circumstances in which patients without V1 can clearly experience visual qualia in their blind field. In hemianopic completion, which can occur when visual stimuli are presented across the vertical meridian such that one half of the figure falls within the blind hemifield, some patients are able to perceive the complete figure (e.g., Bender & Kahn, 1949). For example, in a study by Marcel (1998), when both the normal and the blind hemifield of patients were stimulated in combination, such that the stimulus formed a good Gestalt, a visual after-image appeared in both hemifields (see Fig. 4). In contrast, when the blind field was stimulated in isolation, no afterimage was perceived. Hemianopic completion has been associated with activation anterior to retinotopic cortex in the lingual gyrus in the right occipital cortex, contralateral to the lesion, ipsilateral to the illusory edge of the stimulus (Weil, Plant, James-Galton, & Rees, 2009). Further but less systematically investigated examples of phenomenal conscious experience in hemianopia include hallucinations, which can occur especially in the early stages of recovery from stroke (Kölmel, 1985; see also Silvanto & Rees, 2011, for a review).
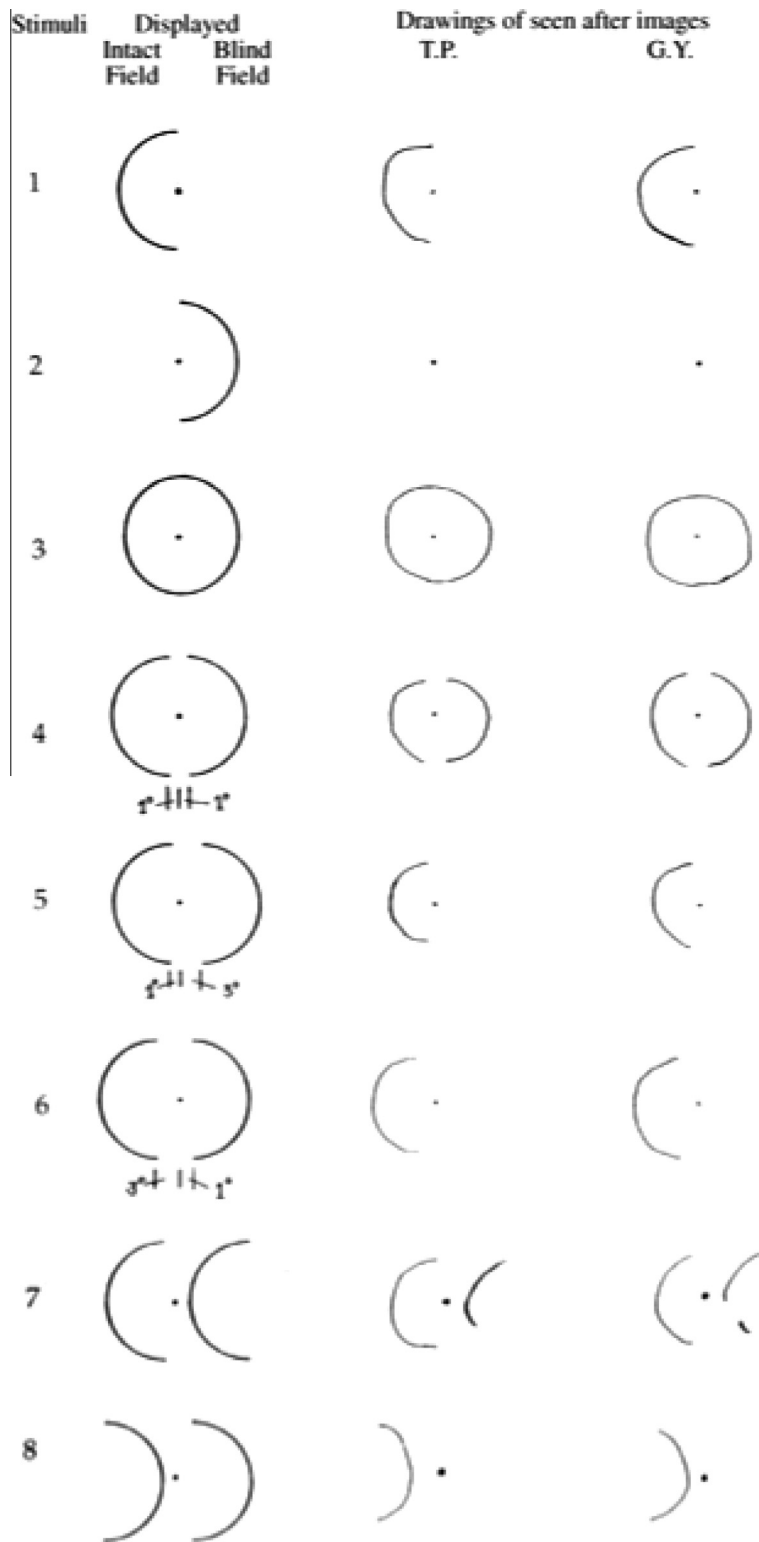
Fig. 4. Hemianopic completion in blindsight (from Marcel, 1998). On the left, stimuli used to induce after-image, and on the right, the most frequent drawing out of three series made by each subject to depict what they had seen. In certain conditions, the blindsight participants perceived afterimages in their blind field.
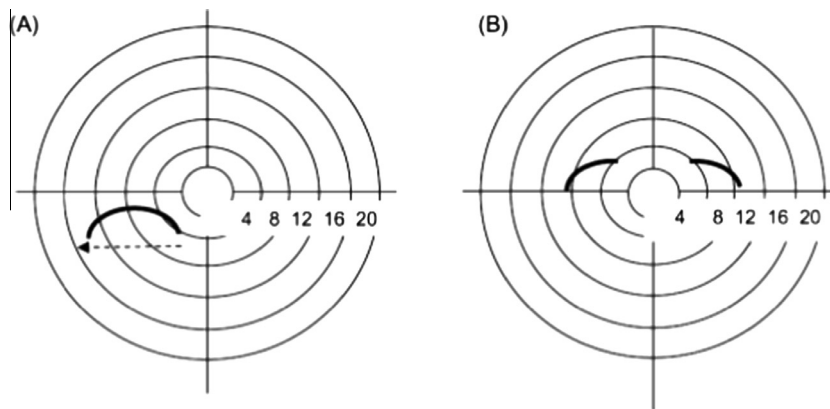
Fig. 5. Phosphene appearance in GY, based on his drawings (from Silvanto et al., 2008). (A) Unilateral stimulation of V5/MT in the normal hemisphere induced a contralateral moving phosphene in the intact visual field. (B) Bilateral stimulation of GY's V5/MT induced bilateral phosphenes.

3.5. TMS-induced phosphenes in blindsight

There is also evidence of TMS-induced percepts in blindsight patients. As discussed above, the application of TMS over the ipsilesional extrastriate cortex of the blindsight subject GY does not induce the experience of phosphenes in the blind field (Cowey & Walsh, 2000), consistent with the view that extrastriate activation cannot reach awareness in the absence of ipsilateral V1. However, subsequently it was shown that his ipsilesional activation can reach awareness under specific circumstances. GY could perceive a phosphene intruding into both the intact and the blind visual field when TMS was applied over V5/MT bilaterally (Silvanto, Cowey, Lavie, & Walsh, 2007; see Fig. 5). Two characteristics of GY's blind field phosphenes suggested that a percept in the intact visual field was necessary for their induction. Firstly, GY did not consistently perceive phosphenes that were restricted to his blind field, and secondly, when the intact hemisphere was stimulated below phosphene threshold, no phosphenes were induced, indicating that the activity level of the intact hemisphere determined phosphene presence.

This finding was not restricted to achromatic pecepts, as in a subsequent study, by combining bilateral application of TMS with a chromatic adaptation paradigm (Silvanto, Muggleton, Cowey, & Walsh, 2007), it was shown that GY can also perceive chromatic color in his blind field (Silvanto, Cowey, & Walsh, 2008). In this study, GY was adapted to color displays prior to phopshene induction. Without adaptation, unilateral stimulation of V5/MT in GY's intact hemisphere induced a moving phosphene similar to those reported by normal subjects, whereas unilateral stimulation of the ipsilesional V5/MT never induced a phosphene. After adaptation, phosphenes induced with unilateral TMS in the control subjects as well as from GY's intact hemisphere appeared in the color of the adapting stimulus (see Cattaneo, Devlin, Salvini, Vecchi, & Silvanto, 2010; Cattaneo, Rota, Walsh, Vecchi, & Silvanto, 2009; Silvanto & Muggleton, 2008; Guzmán-López, Silvanto, & Yousif, et al., 2011, Guzmán-López, Silvanto, & Seemungal, 2011 for examples of state-dependent TMS approach). In control subjects, when TMS was applied over both hemispheres to induce a biltateral phosphene, the component of the phosphene overlapping the adapted hemifield appeared colored, and the component overlapping the unadapted hemifield was lacking in chromatic color. In contrast, in GY the bilateral phosphene appeared uniformly colored after adaptation was restricted to the intact field. Furthermore, when adaptation was restricted to GY's blind field, the phosphene always appeared colorless. As adaptation of the blind field had no influence on phosphene color, it must have been adaptation of the wavelength/color-selective regions in GY's normal hemisphere that enabled color experience in the blind field. These TMS findings may be related to hemianopic completion, in which the contralesional extrastriate cortex has been implicated (Weil et al., 2009).

GY's blind field phosphenes may possibly result from the spread of ipsilesional activation to the contralesional V5/MT through callosal connections, thereby increasing and/or modulating the activation level of neurons in V5/MT of the normal hemisphere that have a representation of the ipsilateral visual field. The existence of callosal pathways in GY has been demonstrated using probabilistic diffusion-based tractography (Bridge et al., 2008). This study also revealed that GY possesses a prominent novel pathway between the LGN in the intact hemisphere and V5/MT in the damaged hemisphere. This could account for the finding that when TMS is applied over V5/MT of the damaged hemisphere, it influences the appearance of phosphenes evoked by TMS applied above V1 of the normal hemisphere, an effect not found in controls (Silvanto, Walsh, & Cowey, 2009). The existence of such abnormal connections warrants caution when drawing conclusion regarding the neural basis of visual awareness in the normal brain. While conscious perception is possible in GY, the implications to understanding visual awareness in the normal brain may be limited.

Intriguingly, a very recent study has demonstrated that even unilateral application of TMS over intact regions of the damaged hemisphere can induce phosphenes in the absence of V1. In a study by Mazzi et al. (submitted for publication), the authors attempted to induce phosphenes by applying TMS over the parietal cortex of the lesioned hemisphere in two hemianopic patients with a complete destruction of V1. Importantly, both patients could perceive phosphenes with this stimulation (see Fig. 6), and a psychophysical threshold function on these phosphenes could be created, which was not
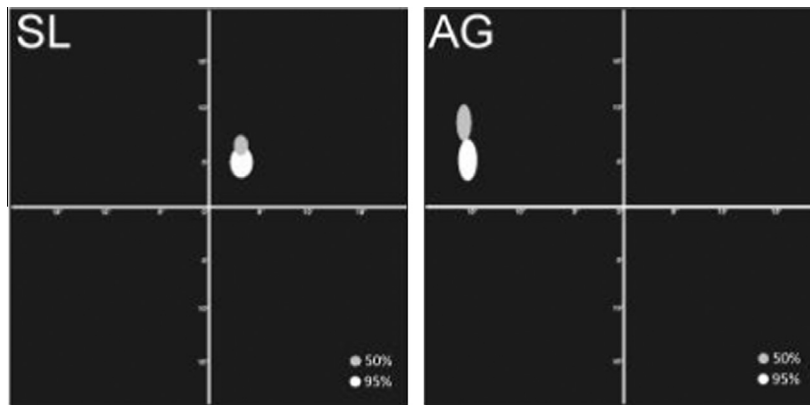
Fig. 6. Phosphenes induced in two patients with V1 lesions (from Mazzi et al., submitted for publication), at two different TMS intensities (55% and 95% of TMS output).

different from those obtained from neurologically normal controls. Thus, in the absence of V1 phosphenes can be induced from the ipsilesional hemisphere, with properties similar to those induced in neurologically normal observers. This is a novel result with important theoretical implications, and does not suffer from at least two limitations present in the findings of Silvanto et al. (2007, 2008). Firstly, the induction of a phosphene from patients' normal hemisphere was not required for the blind field phosphene to arise; thus, unlike the phosphenes experienced by GY, they cannot be exclusively explained in terms of blind field percepts being experienced through activation reaching awareness in the normal hemisphere. Secondly, unlike GY (who sustained his injury at the age of 8 and participated in the phosphene studies decades later), both of these patients sustained their injuries in adulthood, and the TMS studies were carried out relatively soon (6 and 30 months) after the neurological event. It is thus unlikely that novel and abnormal connectivity underlies their phosphene perception in these patients.

The key implication of these studies is that feedback to ipsilateral V1 does not seem to be the only pathway for extrastriate activation to reach awareness. One could argue that it is V1 in the intact hemisphere which enabled conscious experience to arise. However, as V1 contains a representation of only the contralateral visual field, activity of the intact V1 cannot underlie the blind field percepts. Furthermore, it is not a parsimonious explanation of the findings of Mazzi et al. (submitted for publication) where there is no evidence of the necessity of the intact hemisphere.

Overall, the evidence reviewed in Section 3 suggests the following. Firstly, while extrastriate-V1 feedback appears to play an important in visual perception, it does not seem to be uniquely associated with awareness. This is consistent with electrophysiological evidence indicating that the importance of extrastriate feedback on V1 activity is stimulus-dependent (Hupe et al., 1998), which would not be the case if they were required for *all* awareness. Secondly, demonstrations of conscious experience in the absence of V1 argue against the possibility that this region is the only gateway to awareness.

4. Theoretical implications of blindsight: evaluating the key arguments

As discussed in Section 2, theories linking V1 feedback with visual awareness rely on two main tenets deriving from blindsight. Firstly, the highly accurate detection performance in the absence of awareness has been interpreted as a selective effect of V1 damage on visual awareness. Secondly, it is argued that the loss of awareness cannot simply be due to a loss of extrastriate functioning, as extrastriate regions retain a relatively normal responsiveness in the absence of V1. These arguments are of great theoretical importance: if extrastriate neural responses were severely impoverished by the V1 lesion and only few visual functions survived, it would be more parsimonious to attribute the loss of awareness to reduced functioning of the extrastriate visual cortex, resulting from the loss of excitatory feedforward input from V1. The few residual visual functions could be explained in terms awareness being more vulnerable to disruption. These arguments will be evaluated next.

4.1. Is blindsight a true dissociation of awareness and visual detection?

It is often stated that V1 lesions abolish conscious perception selectively; a wide range of visual functions remain even though phenomenological experience is lost fully. However, this is an oversimplification, as the visual functions that survive V1 lesions are severely impoverished (see Cowey, 2004, for review). Human blindsight subjects are unable to discriminate gratings when luminance contrast is reduced to 0.1 Michelson contrast, even when tested at most effective spatial frequencies at which performance of control participants is at ceiling. Orientation discrimination is no more than about 10 degrees (Morland, Ogilvie, Ruddock, & Wright, 1996; Weiskrantz, 1986), and wavelength discrimination is increased by tenfold (Stoerig & Cowey, 1992). Many visual functions such as shape discrimination are absent in blindsight subjects. Even true motion perception is not preserved (Azzopardi & Cowey, 2001); although blindsight subjects can discriminate the direction

of a moving bar, this does not require motion processing per se as it can be accomplished by a sequence detector that has detected the presence of the bar in a given position, and shortly afterwards in another position. With global motion stimuli, in which the moving dots are randomly regenerated from moment to moment but have an overall vector motion, this is not possible. Blindsight subjects (including GY) fail to discriminate the direction of such motion stimuli (Azzopardi & Cowey, 2001); thus blindsight is motion-blind.

The residual visual functions are in fact so poor that a seeing person with such acuity would be legally classified as blind (Cowey, 2004). Strictly speaking, therefore, blindsight is not a dissociation of conscious perception and visual detection; rather it is a case of severely impoverished residual visual function in the absence of visual awareness. This is a neglected issue but an important one, because the lack of an absolute dissociation between conscious and unconscious visual functions could be explained in terms of conscious perception merely being more susceptible to disruption than low-level detection and discrimination.

Nevertheless, blindsight is unlike near-threshold vision in that its hallmark is a robust dissociation between performance in Yes–No tasks assessing conscious experience and forced-choice detection tasks which is absent in healthy controls (Azzopardi & Cowey 1997). As discussed in Section 3, when assessed through SDT analyses, the blindsight subject GY is approximately half as sensitive to targets during the Yes–No task as during the forced-choice task, a difference not found in the healthy controls (Azzopardi & Cowey 1997). While the range of stimuli with which this dissociation occurs is limited, the question of why it happens at all remains; why is blindsight blind? Section 5 will attempt to resolve this issue.

4.2. Neural responsiveness of the visual cortex in the absence of V1

It is often argued that the loss of awareness in blindsight cannot simply be due to lack of excitatory input from V1, as extrastriate regions can be activated in the absence of V1. What is the effect of a V1 lesion on neuronal responses in the ipsilesional extrastriate cortex? Initial electrophysiological evidence suggested that extrastriate regions V2, V3 and V4 are silenced while dorsal stream areas V5/MT and V3a remain responsive (e.g. Schiller & Malpeli, 1977; Rodman et al., 1989a; Girard & Bullier, 1989; Girard et al., 1991). However, subsequent fMRI and electrophysiological studies carried out in macaques have shown that also V2, V3, V4, superior temporal sulcus and lateral intraparietal area show residual responsiveness in the absence of V1 (Schmid et al., 2009, 2010); the discprecancy with earlier studies might be due to the former assessing V2 and V3 functionality using cooling (Girard & Bullier, 1989; Girard et al., 1991), rather than surgical lesions (see Schmid et al., 2009 for detailed discussion on the source of this discrepancy). The neural responses in V5/MT have been studied in most detail and thus offer the most detailed clues on how extrastriate neurons can function in the absence of V1.

Rodman et al. (1989a) presented bars moving through the receptive fields of V5/MT neurons in macaques after unilateral removal of V1. Evoked responses of neurons with receptive fields within the lesion zone were weak; while 66% of the neurons were still responsive, only 5% gave responses classified as strong. Whereas average response magnitude of V5/MT in an unlesioned monkey is approximately 19 spikes per second, in the lesion zone the responses of the majority of neurons were less than half of this (Rodman et al., 1989a). Single units within the lesion zone also tended to exhibit abnormally bursty spontaneous activity and high response variability. While the briskness of the responses was lost, direction selectivity was present, and tuning sharpness was within parameters reported to be within the normal range. This residual activation is likely to be driven by inputs from superior colliculus, as its lesion silenced V5/MT completely (Rodman et al., 1989b). Intriguingly, none of the cells with receptive fields *outside* of the lesion zone had response magnitudes as high as the highest values found in unlesioned monkeys. This might be due to the loss of local excitatory connections from cells in the lesion zone, pointing to a general loss of excitation after a V1 lesion.

Rosa et al. (2000) investigated the residual responses in the middle temporal region of marmoset monkeys. 17 of the 36 cells with receptive fields inside the scotoma showed no significant response to stimulation of the visual field inside the scotoma; 13 cells did so, and also showed evidence of direction selectivity. In contrast, another study on marmoset monkeys found no significant residual responses after a V1 lesion (Collins et al., 2003). The latter study investigated neuronal responses *immediately* after a V1 lesion, whereas post-lesion recovery period in the study by Rosa et al. (2000) was 4–6 weeks.

Azzopardi, Fallah, Gross, and Rodman (2003) examined the temporal dimension of the residual activity by measuring V5/MT responses in four macaques after unilateral V1 removal. Global motion stimuli were used. In the damaged hemisphere, V5/MT responses were consistently much weaker, irrespective of the speed of the stimulus or whether or not the stimulus was positioned mainly inside, or mainly outside the blind field. While every recorded neuron responded significantly to visual stimulation, on average neurons were only as responsive to moving stimuli as V5/MT neurons in unlesioned monkeys to the presentation of a non-moving bar. In the unlesioned hemisphere, median response latencies to stimuli moving at 4 and 20 degrees per second were 78 ms and 83 ms, respectively. In V5/MT ipsilateral to a striate cortex lesion, these latencies were longer by an average difference of between 30 and 320 ms, These abnormally long response latencies were not merely associated with slow moving stimuli to which V5/MT responses are weaker but were also found with fast motion. Therefore, the long latencies were not merely a consequence of low response magnitude (cf. Raiguel et al., 1999).

Thus the existing evidence does not support the view that extrastriate activity can be rather normal in the absence of V1. In V5/MT, neurons show abnormally bursty spontaneous activity and high response variability, and the normally brisk evoked responses are replaced by sluggish long-latency neural firing. Importantly, direction selectivity does remain, and can be within normal range. While the functionality of other extrastriate regions has been less studied, there is evidence that

neural tuning is somewhat preserved, and that the magnitude of the evoked responses (measured with either fMRI or multiunit activity) in V2, V3, V4 to stimuli in the blind field are approximately 20–30% of pre-lesion levels (Schmid et al., 2009, 2010, 2013).

In summary, blindsight is not a pure dissociation of conscious perception and visual detection; rather it is a case of severely impoverished residual visual function in the absence of visual awareness. Furthermore, while extrastriate neurons retain certain levels of neural tuning in the absence of V1, their responses are reduced. Thus the two main tenets of the view linking V1 to visual awareness do not hold; V1 lesions do not induce a selective effect on visual awareness, and extrastriate activity is not normal in the absence of V1.

5. Emerging view: Explaining blindsight in terms of a distinction between "local" vs "global" visual functions

Existing literature does not support the view that awareness arises in V1 in response to feedback from extrastriate cortex. Nevertheless, important questions remain: why is blindsight blind? Why is awareness lost after a V1 lesion, and why are the diminished V5/MT responses able to give rise to visual detection in the absence of awareness? An explanation is offered here between two types of visual processes: those that can be accomplished "locally" within a single cortical region, and those of "global" nature, defined by inter-areal communication. It is proposed that the former are possible in blindsight. The latter, of which consciousness is one, cannot occur due to the loss of recurrent activity involving V1.

5.1. "Local" vs. "global" neural computations and visual functions

A "local" visual function, as defined here, is one for which the key neural process takes place within *one cortical area*. Of course, all brain regions are dependent on regions lower down in the anatomical hierarchy for input to drive their neural activity; for this reason a visual function cannot, strictly speaking, be exclusively "local". However, the point is that the key neural computation underlying the visual function is limited to one brain region. In the case of a "local" visual function, it is the readout from neural firing rates in response to an external stimulus which guides behavior. While it is challenging to make a direct link between neural activity and behavioral output, a number of criteria have been put forward (e.g. Parker & Newsome, 1998). Fundamentally, it is important that the neural activity not only correlates with perceptual reports independently of the motor response, but that manipulation of that activity has a corresponding effect on those reports.

A good example of a "local" function is motion detection in V5/MT. The ratio of spiking activity between V5/MT neurons of different direction tunings has been shown to predict behavioral responses in monkeys in motion detection tasks (e.g. Adelson & Movshon, 1982). In neural readout models, each V5/MT neuron is thought of as having a vote for the vector of its preferred tuning, with the strength of its vote being proportional to its firing rate (e.g. Groh, Born, & Newsome, 1997). For example, in the vector average model, the preferred tunings are averaged together in proportion to the firing rate of each neuron, whereas in the "winner-take-all" model, the winner is the single vector receiving the most votes. It has been shown that in V5/MT, neurons with nearly opposite preferred directions compete in a manner consistent with a "winner-take-all" model, whereas a coding scheme resembling vector averaging is used for neurons with less extreme differences in preferred direction tunings (Nichols & Newsome, 2002). Furthermore, microstimulation of V5/MT neurons biases motion judgments towards the direction of the stimulated neuron, demonstrating the causal link between the neural activity and observer's judgments (Groh et al., 1997; Nichols & Newsome, 2002). The key point is that once information reaches V5/MT, it is the firing rate of neurons in this region that determines the behavioral consequence of the stimulus. It is a *local* computation which underlies the behavioral output.

While detection of simple visual features such as motion, orientation and brightness can be accomplished locally, more complex visual tasks requiring the binding of stimulus features, semantic processing and working memory are likely to additionally require coordination between numerous brain areas, as they cannot be fully encoded by any one visual region. For such "global" visual functions, the key neural signature is inter-areal communication which may be accomplished through oscillatory activity.[5] The foundation for such communication is that activated neuronal populations have the intrinsic property to oscillate, which gives rise to rhythmic modulations in neuronal excitability affecting both the sensitivity to synaptic input and likelihood of spike output (Fries, 2005). According to one influential model (Fries, 2005), only coherently oscillating neuronal groups can communicate effectively, because their communication windows for input and for output are open at the same time. Long-range inter-areal oscillatory synchrony is characterized by phase synchronization and thought to arise via feedforward and feedback connections (Lachaux et al., 2000). A good example of a "global" visual function is binding, which requires inter-areal communication because different features of the object (e.g. color, shape, motion) are analyzed by different extrastriate regions. The outcome of those separate analyses has been proposed to be combined via neuronal synchrony (Singer & Gray, 1995), such that all neurons in different visual areas that are involved in the processing of a given object synchronize their firing to signal they are operating on that object. There is evidence consistent with this view. For example, when observers are presented with an object crossing the vertical meridian, gamma oscillations in the two hemispheres become phase-locked (Rose

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[5] While synchrony also plays an important role at the "local" level within individual cortical regions, it is between-area synchrony which is the focus here. Such "inter-areal" mechanism is postulated due to the "global" nature of consciousness, i.e. the availability of information to large number of perceptual and cognitive systems (e.g. Baars, 1988).

et al., 2006). Furthermore, the latency of the oscillations correlate with the behavioral latencies of object recognition (Martinovic, Gruber, & Muller, 2007).

Consciousness is a "global" function as its hallmark is the availability of information to a large number of perceptual and cognitive systems. It has been associated with a global workspace (GW) where information is broadcasted to multiple non-conscious specialized networks, and globally available for cognitive function (Baars, 1988). In its neuronal implementation, consciousness is a consequence of global ignition of large-scale frontoparietal systems (Dehaene & Changeux, 2011; Dehaene & Naccache, 2001). Specifically, a subset of cortical pyramidal cells with long-range excitatory axons together with thalamo-cortical loops form a horizontal "neuronal workspace" and the hallmark of conscious access is the simultaneous increase in high-frequency power and synchrony involving prefrontal cortex and other high-level associative cortices (Dehaene & Changeux, 2011). Also in other theories of awareness, inter-areal synchrony underlies conscious experience. As discussed in Section 2, Pollen (1999) proposed that for conscious perception to arise, a consensus needs to be reached between the visual areas that process the given stimulus; this is achieved through feedforward and feedback pathways linking multiple cortical areas, with synchronous neuronal activity on a short timescale throughout the visual cortex serving as a marker that a "steady state" (or consensus) has been achieved.

There is an increasing amount of empirical evidence for this view (see e.g. Fries, 2005; Dehaene & Changeux, 2011 for reviews). For example, in a study by Melloni et al. (2007), both perceived and nonperceived words caused a similar increase of local gamma oscillations in the EEG, but only perceived words induced a transient long-distance synchronization of gamma oscillations across widely separated regions of the brain. Wyart and Tallon-Baudry (2008) showed that conscious perception is associated with increased mid-frequency gamma-band activity over the contralateral visual cortex. In their study, trial-to-trial fluctuations of activity in the gamma-band not only correlated with visual awareness in stimulus-present trials but also correlated well with false-alarm reports on stimulus-absent trials.

5.2. Explaining blindsight in terms of "local" vs. "global" visual functions

After a V1 lesion, extrastriate regions can still receive a limited amount of feedforward input. Although the neuronal responses are severely impoverished, the sharpness of neural tuning can be in normal range: for example, in V5/MT, direction selectivity can be preserved (Rodman et al., 1989a). This is important because it enables neural computations in which the firing rates of neurons of different tunings are averaged in order to obtain an estimate of stimulus properties, such as motion direction (cf. Groh et al., 1997; Nichols & Newsome, 2002). Thus neural read-out of low-level visual information, which can guide behavior, is possible in the absence of V1. That such meaningful neural computations take place in V5/MT after a V1 lesion is supported by the finding that the disruption of this region with TMS impairs blindsight (Cowey & Alexander, 2009).

The situation is fundamentally different for "global" processes relying on inter-areal synchrony. An important characteristic of V5/MT responses in adult-lesioned monkeys is the sluggish nature of the responses and abnormally long response latencies (Azzopardi et al., 2003), indicating that the brisk nature of V5/MT firing found in the normal brain is driven by excitatory input from V1. Furthermore, V5/MT neurons exhibit abnormally bursty spontaneous activity and high response variability (Rodman et al., 1989a). While the timing of extrastriate responses in other ipsilesional regions has not been explicitly investigated, it is likely that similar results are found, given that V1 is their main source of excitatory input and residual responses in these regions are no more than 20–30% of the response magnitude of pre-lesion levels (Schmid et al., 2009, 2010, 2013). The critical issue is that the sluggishness and the variability in neuronal responses are likely to prevent the formation of inter-areal neural networks via oscillatory activity, as these require fine temporal resolution. Furthermore, the abnormally burst spontaneous activity is likely to prevent the representation of information by varying the timing of spikes relative to ongoing neuronal oscillations.

This immediately leads to the question of why V1 damage has such a detrimental effect on neural responses, given that information can still reach extrastriate cortex through subcortical pathways. Fundamentally, this could be due to its role as a key hub in the visual cortical hierarchy: it engages in feedforward/feedback activity with a large number of visual regions. Firstly, V1 provides the vast majority of excitatory extrastriate input; normal functioning might not be possible with a mere fraction of the normal amount of feedforward activation. Especially the brisk, short-latency responses may be lost. A second answer relates to the importance of recurrent activity, which modulates neural activity not only in V1 but also in the extrastriate cortex. For example, as V5/MT-V1 feedback can enhance the activity of V1 neurons projecting to V5/MT, this recurrent loop allows V5/MT to shape the responses of neurons providing its input, acting as a form of gain control (see e.g. Sillito, Cudeiro, & Jones, 2006). Thirdly, recurrent activity may be important for maintaining the appropriate baseline oscillatory activity, in the context of which spiking occurs; there is indeed evidence that recurrency plays a role already in the early parts of the neural responses (Hupe et al., 2001), and its loss may explain the abnormal nature of spontaneous activity in the absence of V1. The precise mechanisms by which feedback connections enable neural synchrony require further study. The key point is that in the absence of recurrent activity involving V1, inter-areal synchrony and therefore conscious perception cannot arise.

In this view, the content of awareness reflects the activity of individual visual areas which form a network through synchronous activity (cf. Pollen, 1999). Extrastriate-V1 feedback is not the pathway to awareness; rather, it plays a role in various neural processes which are needed for inter-areal synchrony between visual areas to arise. V1 is central due to its position in the visual cortical hierarchy; it is the most important source of extrastriate input and engages in recurrent loops

with a large number of visual areas, thereby enabling normal neural responsiveness throughout the visual cortex and modulating activity in a large number of extrastriate regions simultaneously. However, it is not V1 per se which is critical. If sufficiently robust and temporally specific neural activity can arise via other routes, then V1 would not be required. This may be occur when patients without V1 can experience conscious percepts in their blind field, whether through hemianopic completion or by TMS. Interestingly, recent evidence suggests that the brisk, short-latency responses of V5/MT neurons can be preserved if V1 lesion occurs in early age (Yu, Egan, Reser, Worthy, & Rosa, 2013); this might be related to greater preservation of conscious vision after V1 lesion in childhood relative to later in life, and might be due to another cortical area replacing the role of V1 as a key node in recurrent activity (see Silvanto & Rees, 2011, for a review). When V1 is lesioned in early development, another region may be able to take over its role as a key node in the visual cortical hierarchy.

In summary, it is proposed that blindsight is blind because in the absence of V1, the extrastriate neurons are unable to establish inter-areal cortical networks via neural synchrony. However, as some extrastriate neuronal selectivity is preserved, neural computations in which the firing rates of neurons of different tunings are compared can still operate. This "local" neural read-out can guide behavior and give rise to blindsight. The range of visual stimuli that can be detected without the establishment of inter-areal communication is likely to be limited. In this context, it is important to bear in mind that rather than being a clear dissociation between visual awareness and forced-choice detection, blindsight is a case of severely impoverished residual visual function in the absence of visual awareness. As discussed in Section 4.1, only very basic visual features can be detected by blindsight patients.

At least two complicating issues are worth mentioning. Firstly, "local" and "global" processes are likely to be by no means mutually exclusive processes, and inter-areal synchrony is likely to modulate the outcome of the "local" neural readout, although the mechanisms of oscillatory activity and spiking can be to some extent independent (Ardid et al., 2010). Furthermore, if the tuning of V5/MT neurons arises gradually during the neural response through recurrent activity, then it is clear that the accuracy of the "local" readout is enhanced by it. However, the key point is that a basic form of readout is likely to be possible in their absence. A second important caveat relates to the distinction between *intra-areal* and *inter-areal* synchrony. Both play an important role in visual cognition and their separate measurement is not always straightforward. For example, it has been suggested that oscillatory activity in MEG may more likely to reflect local synchrony because of its similarity to patterns of local synchrony observed in intracranial recording (Tallon-Baudry, 2003).

6. Conclusions

Blindsight is an important neuropsychological phenomenon for the study of consciousness (Weiskrantz, 1997; Cowey, 2004, 2010). This review aimed to evaluate one specific theoretical framework derived from blindsight, according to which recurrent/feedback activity in V1 is the neural signature of phenomenal awareness. There are a number of problems with this view. Firstly, in neurologically normal observers, feedback activity is important not only for visual awareness but for visual perception in general, a view proposed by some feedback models (e.g. Bullier, 2001). Secondly, conscious experience in blindsight patients through hemianopic completion and by TMS indicates that the ipsilateral V1 cannot be the only pathway to visual awareness. Thirdly, the main tenets on which theories of V1 feedback are based can be questioned; the dissociation between conscious and unconscious visual function in blindsight is not as robust as often stated, there is doubt as to whether extrastriate neurons remain sufficiently responsive to support awareness after a V1 lesion.

In the view presented here, visual awareness is a "global" phenomenon requiring inter-areal synchronous oscillatory. For these oscillations to arise, a specific temporal profile of neuronal activity is required, established through recurrent feedback activity involving V1 and the extrastriate cortex. When V1 is lesioned, inter-areal networks on the basis of oscillatory activity cannot be established. Thus visual awareness cannot arise. However, as some extrastriate neuronal selectivity is preserved, computations in which the responses of neurons of different tunings are compared can still operate. This enables "local" neural read-out from specific brain regions which can guide behavior unconsciously, and give rise to blindsight. V1 is important for awareness not because it acts as a gatekeeper to conscious experience, but because its position in the visual cortical hierarchy: it is the most important source of excitatory extrastriate input and engages in recurrent loops with a large number of visual areas.

Acknowledgments

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