The Timing and Neuroanatomy of Conscious Vision as Revealed by TMS-induced Blindsight

Christopher P. G. Allen, Petroc Sumner, and Christopher D. Chambers

Abstract

■ Following damage to the primary visual cortex, some patients exhibit "blindsight," where they report a loss of awareness while retaining the ability to discriminate visual stimuli above chance. Transient disruption of occipital regions with TMS can produce a similar dissociation, known as TMS-induced blindsight. The neural basis of this residual vision is controversial, with some studies attributing it to the retinotectal pathway via the superior colliculus whereas others implicate spared projections that originate predominantly from the LGN. Here we contrasted these accounts by combining TMS with visual stimuli that either activate or bypass the retinotectal and magnocellular (R/M) pathways. We found that the residual capacity of TMS-induced blindsight occurs for stimuli that bypass the R/M pathways, indicating that such pathways, which include those to the superior colliculus, are not critical. We also found that the modulation of conscious vision was time and pathway dependent. TMS applied either early (0–40 msec) or late (280–320 msec) after stimulus onset modulated detection of stimuli that did not bypass R/M pathways, whereas during an intermediate period (90–130 msec) the effect was pathway independent. Our findings thus suggest a prominent role for the R/M pathways in supporting both the preparatory and later stages of conscious vision. This may help resolve apparent conflict in previous literature by demonstrating that the roles of the retinotectal and geniculate pathways are likely to be more nuanced than simply corresponding to the unconscious/conscious dichotomy. ■

INTRODUCTION

Blindsight (Weiskrantz, 1986) and TMS-induced blindsight (e.g., Boyer, Harrison, & Ro, 2005; Jolij & Lamme, 2005) are intriguing phenomena in which observers can successfully identify visual stimuli for which they deny awareness. This dissociation between ability and awareness typically follows damage or disruption to the early visual cortex and offers insights into the neural bases of consciousness and subliminal perception (Dretske, 2000). Despite decades of research, a consensus explanation of blindsight remains elusive, and two central problems remain unsolved. The first, long-standing question concerns which neural pathways are crucial for vision lacking in awareness (Cowey, 2010). The second, more recent, question concerns the temporal dynamics of vision—whether unconscious and conscious processing are supported, respectively, by feedforward and recurrent phases of processing (Lamme, 2001).

The classic anatomical debate in blindsight research is whether residual reportedly "unseen" vision depends on the ancient retinotectal pathway via the superior colliculus (SC) or whether it instead arises from other spared projections that are thought to predominantly involve the LGN. Empirical support has emerged for both the retinotectal (Leh, Ptito, Schonwiesner, Chakravarty, & Mullen, 2010; Tamietto et al., 2010; Leh, Johansen-Berg, & Ptito, 2006; Leh, Mullen, & Ptito, 2006; Ro, Shelton, Lee, & Chang, 2004; Rafal, Smith, Krantz, Cohen, & Brennan, 1990) and geniculate hypotheses (Railo, Salminen-Vaparanta, Henriksson, Revonsuo, & Koivisto, 2012; Schmid et al., 2010; Radoeva, Prasad, Brainard, & Aguirre, 2008; Wessinger, Fendrich, & Gazzaniga, 1997). Although some authors have offered a more nuanced view (e.g., Cowey, 2010; Danckert & Rossetti, 2005), many have argued for one standpoint over the other, with the majority favoring the retinotectal account.

Two previous TMS studies probed the retinotectal versus geniculate basis of blindsight but arrived at opposite conclusions (Boyer et al., 2005; Ro et al., 2004). The first of these studies (Ro et al., 2004) showed that reportedly "unseen" distractors slowed saccadic responses, but not manual button presses. Because the retinotectal pathway is thought to drive saccades, the authors concluded that the preserved capacity of TMS-induced blindsight necessarily originated from retinotectal information. However, the absence of a manual distractor effect was not definitive, and subsequent evidence demonstrates that saccadic distractor effects can also be generated via geniculate pathways (Bompas & Sumner, 2009; Sumner, Nachev, Castor-Perry, Isenman, & Kennard, 2006). In contrast to Ro et al. (2004), Boyer et al. (2005) used chromatic stimuli that did not activate the retinotectal pathway and demonstrated preserved capacity under TMS, suggesting

Cardiff University

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a geniculate role. However, they also showed that the confidence participants placed in discrimination judgments correlated with the capacity they claimed to represent TMS-induced blindsight, and during the collection of these data some of their participants acknowledged partial awareness of the stimuli. Partial conscious perception, as opposed to blindsight, may therefore be a more plausible interpretation of their results. Clear interpretations of both studies are further hampered by a lack of control for nonspecific effects of TMS (e.g., sham TMS condition or a control site) and the absence of robust evidence that TMS was effective in suppressing awareness during the conditions designed to demonstrate blindsight. Together with the divergence in conclusions, these limitations leave open the question as to the anatomical basis of blindsight.

Here we directly contrasted the retinotectal and geniculate accounts in TMS-induced blindsight by comparing responses to visual stimuli that activate or bypass the retinotectal pathway (Sumner, Adamjee, & Mollon, 2002). The retinotectal pathway has previously been demonstrated not to receive color opponent input from shortwave cones (s-cones) of the retina (Sumner et al., 2002; de Monasterio, 1978; Schiller & Malpeli, 1977). This means that s-cone-specific stimuli, when accompanied by luminance noise, will be invisible to the retinotectal route (Sumner et al., 2002). Therefore, if the residual capacity of blindsight requires retinotectal processing, it should not be demonstrable with "s-cone" stimuli. Importantly, our question addresses whether critical information is carried in the fast and direct pathway to the SC, rather than concerning all processing within the SC. Information can reach the SC via the visual cortex as well as via the direct retinotectal pathway. Accordingly, chromatic information can activate the SC when it is the target for an eye movement, and does so with a delay consistent with the cortical route (White, Boehnke, Marino, Itti, & Munoz, 2009). The geniculate account of blindsight does not, therefore, require that there is no SC processing, but simply that the retinotectal pathway is not the critical conduit for blindsight.

Additionally, it is noteworthy that magnocellular and possibly parvocellular projections of the LGN are also blind to s-cone stimuli (Dacey, 2000; Mollon, 1989). Thus, if TMS-induced blindsight were eliminated for s-cone stimuli, then this finding would be consistent with retinotectal mediation but also consistent with spared magnocellular fibers. On the other hand, if TMS-induced blindsight were the same for s-cone stimuli compared with luminance stimuli, then this would rule out a critical role for the retinotectal pathway. It would not rule out a retinotectal contribution for other types of stimulus that we do not test here, but it would be sufficient to show that retinotectal mediation is not always required.

The anatomical basis of blindsight may also depend critically on temporal dynamics. According to a popular account (Lamme, 2001; Lamme & Roelfsema, 2000), visual stimuli trigger an initial feedforward volley, likely including retinotectal signals, that is sufficient for some degree of unconscious processing; this is then followed (from ~100 msec poststimulus) by recurrent feedback that facilitates awareness. By manipulating both the stimulus color (s-cone vs. luminance) and timing of TMS, we sought to test how the functional anatomy of blindsight interacts with these putative phases of visual processing.

Both the retinotectal and geniculate hypotheses predict that later occipital TMS (≥100 msec) should disrupt recurrent processing, suppressing conscious awareness while leaving reportedly "unseen" abilities for luminance stimuli above chance (and thus demonstrating TMS-induced blindsight; see Figure 1B). According to the retinotectal hypothesis, "unseen" abilities under these conditions should be relatively suppressed for s-cone stimuli because the retinotectal route is blind to these stimuli. In contrast, if the geniculate hypothesis is correct, then blocking the retinotectal pathway should be inconsequential and blindsight should be observed for s-cone stimuli. Because an equivalence of TMS-induced blindsight for s-cone and luminance stimuli would be theoretically informative, we adopted Bayesian statistical methods that assess the likelihood of the null hypothesis (Dienes, 2008b, 2011) in addition to more common Neyman–Pearson analyses.

METHODS

Participants

Sixteen neurologically healthy participants with normal color vision were recruited (seven men, mean age = 24.0 years, SD = 3.04). All procedures were approved by the research ethics committee at the School of Psychology, Cardiff University. Initially, a further 10 participants were enlisted but did not participate in the main experiment because occipital TMS did not elicit phosphenes within the safety limits set by ethics approval (seven participants) or because TMS applied at the required intensity caused large contractions of peripheral facial nerves that could cause an experimental confound (two participants). One participant withdrew from the study following a mild adverse reaction to the TMS (Maizey et al., 2013).

Task

In our paradigm, active or sham (control) TMS was applied in 25-Hz pulse pairs over the occipital cortex. To modulate the earliest phases of visual processing, TMS was applied at 0–40 msec and 40–80 msec after stimulus onset (Corthout, Hallett, & Cowey, 2002). Recurrent processing was targeted by applying TMS at 90–130 msec (e.g., Amassian et al., 1989) and during a later period at 280–320 msec (e.g., Chambers, Allen, Maizey, & Williams, 2012). These four periods were tested to capture possible independent early and late phases (Koivisto, Mantyla, & Silvanto, 2010). TMS applied at ~100 msec poststimulus

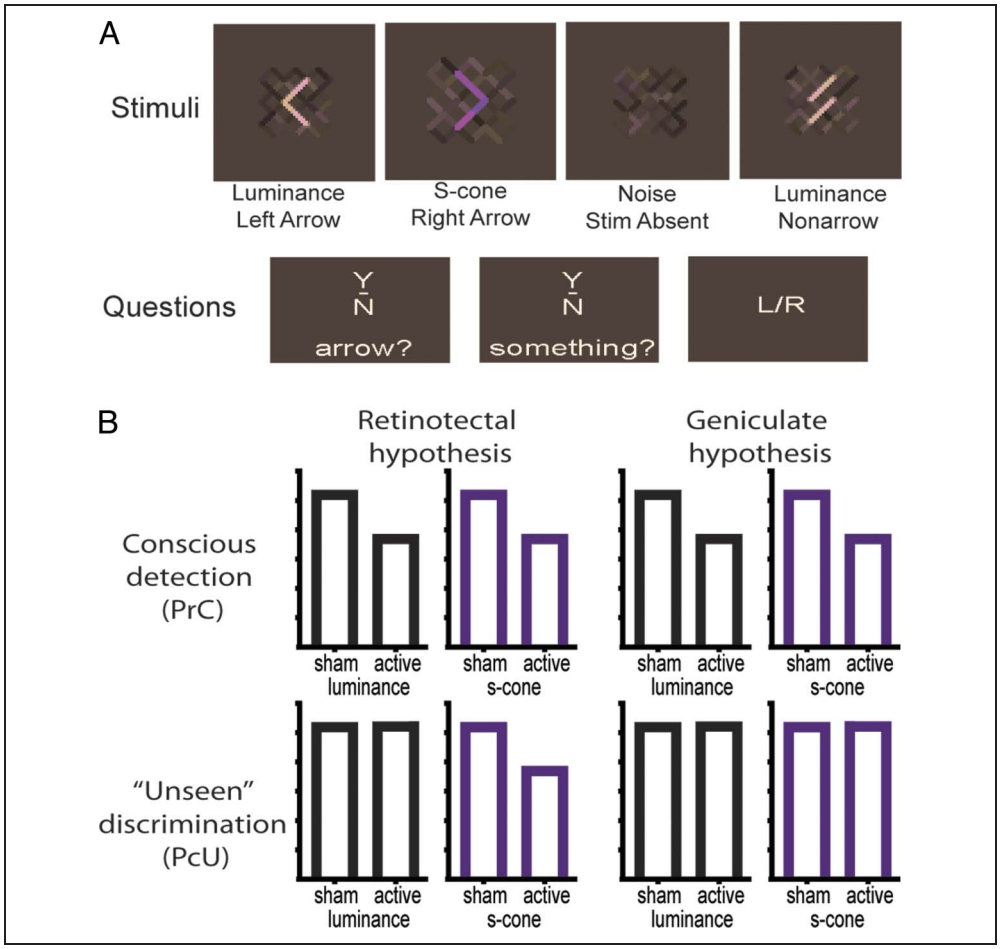

Figure 1. (A) Examples of stimuli and questions. The measure of conscious detection was derived from application of signal detection theory (Corwin, 1994) in response to the questions: "Did you consciously see the arrow?" (Arrow Y/N) and "Did you see something that might have been a stimulus?" (Something Y/N). The measure of "unseen" discrimination was the proportion of correct discriminations of arrow direction (L/R) on trials in which participants reported "No" to both the "arrow?" and "something?" questions. The nonarrow condition ensured that participants perceived an arrow when reporting one, as opposed to simply responding to a change in luminance or s-cone saturation. (B) Illustration of predictions. TMS-induced blindsight as defined by a statistically significant suppression in the measure of conscious detection (PrC) for both stimulus types, in conjunction with above-chance "unseen" discrimination (PcU) for luminance stimuli. Under the retinotectal hypothesis, "unseen" discrimination for s-cone stimuli may not be demonstrated as being greater than chance. Furthermore, according to the retinotectal account, concurrent "unseen" discrimination in the presence of s-cone stimuli should be suppressed relative to sham, and any change in capacity may differ from that observed in the presence of luminance stimuli. According to the geniculate hypothesis, concurrent "unseen" discrimination ability for s-cone stimuli should be relatively preserved and comparable to that observed in the presence of luminance stimuli.

has formed the basis for many of the previous demonstrations of TMS-induced blindsight (Christensen, Kristiansen, Rowe, & Nielsen, 2008; Boyer et al., 2005; Jolij & Lamme, 2005; Ro et al., 2004).

The target stimulus was an arrow embedded in luminance noise, presented on 50% of all trials (Figure 1A). Trials could include an arrow target, a nonarrow target, or no target. On each trial, participants were asked if they were aware of the arrow stimulus and in which direction it was pointing (left or right). In addition, we asked participants whether they were aware of "something" having been presented (including the nonarrow target). This question probed a lower level of awareness than the "arrow?" question (Overgaard, Rote, Mouridsen, & Ramsoy, 2006). Using negative responses to both questions to indicate lack of awareness is more stringent than merely using the "arrow?" question alone. Forced-choice discrimination judgments (left/right) on these "double negative" awareness trials constituted the "unseen" measure (PcU). This discrimination capacity therefore represents a relatively conservative form of unconscious processing (see Dienes, 2008a). The conscious awareness measure was participants' sensitivity in their reported awareness of stimuli (PrC). In this design, blindsight would thus manifest as a TMS-induced impairment of conscious detection (PrC) while leaving the ability to discriminate reportedly "unseen" arrows (PcU) above chance and relatively unaffected by TMS.

Two classes of target stimuli were employed (Figure 1A): luminance stimuli, which were defined by an increase in luminance, and s-cone stimuli, which were defined by an increase in color saturation that stimulated s-cones, but not (or minimally) long and medium wave cones (Sumner, 2006; Sumner, Nachev, Vora, Husain, & Kennard, 2004; Sumner et al., 2002). In addition to retinotectal pathways, magnocellular layers of the LGN—and possibly parvocellular layers (Dacey, 2000; Mollon, 1989)—are also blind to s-cone stimuli embedded in luminance noise (Derrington, Krauskopf, & Lennie, 1984); thus, these retinotectal and magnocellular pathways bypassed by s-cone stimuli are collectively denoted hereafter as "R/M." Because of the isolation of magnocellular pathways, any

s-cone-dependent effect cannot be attributed exclusively to the retinotectal pathway. However, if a capacity were to depend upon retinotectal input, then a deficit would be expected when s-cone stimuli are used. To obscure the s-cone stimuli from R/M channels (Bompas & Sumner, 2009; Sumner et al., 2006), all stimuli were presented against a background of luminance noise (mean luminance of noise and background: 25 cd/m^2; range of noise: 10 cd/m^2). To our knowledge, this range is larger and therefore has a greater capacity to obscure a luminance transition than any previous comparable study. The noise occupied a 1.91° × 1.91° area at fixation, whereas the arrows and nonarrows subtended 0.81° × 1.43°. To aid comparability of stimulus classes, the noise also contained low levels of color noise along the tritan axis (range: ±8% of mean s-cone saturation of stimuli). Arrow stimuli were present on 50% of all trials, whereas nonarrows and stimulus-absent trials comprised 25% of trials each.

Note that here we use "s-cone stimuli" to denote the transition between gray and purple in the context of luminance noise, rather than the presence of the purple arrow per se. This is because the retinotectal pathway is thought to be blind to such a transition: Even if a small s-cone signal were to enter the pathway, there are no chromatically opponent cells in this pathway to distinguish a chromatic change from a luminance change (Smithson, Sumner, & Mollon, 2003; Sumner et al., 2002; Marrocco & Li, 1977; Schiller & Malpeli, 1977). Any chromatic information that does reach the SC is likely to be indirect, initially entering, and therefore dependent upon cortical areas (White et al., 2009). Consistent with this, Sumner et al. (2006) showed that s-cone stimuli (calibrated and embedded in luminance noise using the same methods we employ here) affect eye movements in a pattern distinct from any strength of luminance stimulus (i.e., the effect of s-cone stimuli could not be accounted for by weak retinotectal signals). Additionally, White et al. (2009) showed that, although chromatic information can activate the SC when it is the target for an eye movement, it does so with a delay consistent with an indirect cortical route. Therefore, as noted in the Introduction, our question addresses whether critical information is carried in the retinotectal pathway to the SC, rather than assessing SC processing per se. The geniculate account of blindsight does not require an absence of SC processing, but rather that the retinotectal pathway is not critical for the residual capacity.

Trials commenced with a fixation cross (1.5 sec) followed by luminance noise, which was produced by alternating the luminance of the bars at 50 Hz. The noise continued for 800 msec before onset of the target stimulus (20 msec; or noise of equal duration on stimulus-absent trials). Following target offset, the noise continued for a further 380 msec. The three questions were then presented, and responses were collected on a standard computer keyboard.

The pattern of behavior that characterizes blindsight could potentially be attributed to changes in response bias (Campion, Latto, & Smith, 1983; Nisbett & Wilson, 1977). To address these concerns, we applied signal detection theory (SDT) to responses to the "something?" and "arrow?" questions. This provided a measure of conscious detection (PrC) that is largely independent of response criteria and corresponds to the rate at which participants report awareness of the arrow when it is present (hit) minus the rate at which they report awareness when no arrow was presented (false alarm; Corwin, 1994; MacMillan & Creelman, 1990). This measure was predominantly driven by responses to the arrow question, in line with task instructions and discursive reports made by the participants about their experience of the task (Gallagher, 2003; Varela, 1996). For example, responding "No" to "arrow?" and "Yes" to "something?" in the presence of a nonarrow was classified as a "correct rejection" of a potential arrow target as opposed to being a "hit" for a nonarrow target. This categorization of responses into SDT classes is summarized in Table 1 and was established before data analysis. Nonparametric versions of SDT were used because the imbalance in stimulus-absent conditions violated the assumptions of classic SDT (Pr; see Corwin, 1994). "Unseen" discrimination performance was defined as the proportion correct when participants responded negatively to both "arrow?" and "something?" questions (PcU).

Procedure

Following calibration, participants completed six experimental sessions of ~1 hr. Each session consisted of four blocks, one for each of the experimental conditions: s-cone stimuli or luminance stimuli combined with sham or active TMS, with the order randomized between sessions. Blocks consisted of 80 trials, including, in a randomized order, the four stimulus types (left arrow, right arrow, nonarrow,

Table 1. SDT Classes for Measures of Conscious Awareness (PrC)

	Response		
Stimulus	*Something?*	*Arrow?*	*SDT Class*
Arrow	Yes	Yes	HIT
	No	No	MISS
Nonarrow	Yes	Yes	FA
	Yes	No	CR
Nothing	Yes	Yes	FA
	Yes	No	FA
	No	No	CR

FA = false alarms; CR = correct rejections. Hit Rate = Hits/(Hits + Miss); False Alarm Rate = FA/(FA + CR); Pr = Hit Rate − False Alarm Rate (Corwin, 1994).

and stimulus-absent) and four TMS onset times (0–40, 40–80, 90–130, and 280–320 msec).

The order of the response questions (Arrow? Something? Left/Right?) was different for each session, so that across the experiment each participant completed a fully counterbalanced set of question orders. Participants were given 10 practice trials at the start of each session to become familiar with the order. The sequence in which these question orders were presented was randomized between participants.

Task Calibration

Calibration sessions lasted 2–4 hr and took place at least 24 hr before experimental sessions. During this session, stimulus levels were calibrated to ensure that all participants performed at comparable levels of detection for both luminance and s-cone stimuli and participants were screened for suitability for TMS. For each participant we also established the TMS phosphene threshold (PT; Franca, Koch, Mochizuki, Huang, & Rothwell, 2006) and tritan line in color space for calibration of s-cone stimuli (Smithson et al., 2003).

Detection thresholds for stimuli were calculated using a psychophysical method of constants. Following a period of familiarization with the task, participants completed approximately 10 blocks over a range of stimulus intensities, separately for s-cone and luminance stimuli. For luminance stimuli, the threshold was obtained by adjusting stimulus intensity, whereas for s-cone stimuli, the color saturation was varied along the s-cone axis/tritan line. Data were then regressed using a sigmoidal or linear function (depending on fit quality), solving for the intensity, which produced a PrC of 0.5; this corresponded to the stimulus level at which participants reported consciously seeing the stimulus on 50% of target-present trials if no false alarms were reported. Following the initial calibration, participants then completed several blocks at the derived intensity, and minor adjustments were made to the stimulus levels to compensate for any learning effects, thus maintaining PrC $\cong$ 0.5. At the start of each experimental session, participants completed at least one preblock with sham TMS (40 trials), and similar adjustments were made as required (criteria $>$ $\pm$0.1 PrC from 0.5). If participants completed two sessions in 1 day, then the mean value for the previous sham block was taken as the starting point for subsequent recalibration. The mean luminance for luminance stimuli was 36.5 cdm^2 (*SD* = 3.5). Participant-specific s-cone stimuli were produced following the methods described in Smithson et al. (2003) and used in several previous studies (Bompas & Sumner, 2008, 2009, 2011; Anderson, Husain, & Sumner, 2008; Bompas, Sterling, Rafal, & Sumner, 2008; Sumner, 2006; Sumner et al., 2002, 2004). This procedure yielded individual s-cone colors with the following coordinates in CIE 1931 space: mean ($\pm$*SD*): 0.249 (0.009), 0.186 (0.022), 25.067 (0.809).

The method used to determine PT closely resembled that of Franca et al. (2006). First, we assessed each participant's susceptibility to phosphenes within safety limits set by our ethics committee and international guidelines for 25-Hz stimulation (160% of motor threshold). The coil was oriented with the handle pointing upward and side "B" facing the participant, so that the induced current passed initially in a left-to-right direction. Coil positioning was initially based on anatomical proximity to the midhemispheric convergence of the calcarine sulci, localized in individual structural MRI scans. Immediately before each active TMS block, the intensity was set to 120% of PT (or ~130% of motor threshold if a PT had not yet been established) and the coil was moved so that it produced a phosphene that the participant reported as being "reasonably clear" and "at least in part, covering the center of their visual field," with their eyes closed. This procedure usually required ~10 pulses. The coil position was then recorded using a Brainsight system (Rogue Research, Inc., Montreal, Canada) and used for the subsequent block of trials. If the participant moved beyond a 5-mm tolerance of the original position, then the block was paused and the coil repositioned to the recorded site. Single pulse TMS was applied approximately every 5 sec. An approximate PT was obtained using an up–down staircase method, starting at 50% of maximum output and adjusting TMS intensity in reducing steps of 5%, 2%, and 1%, so that participants verbally reported seeing five phosphenes from 10 pulses. This level was then used as the basis of a more accurate threshold where the number of reported phosphenes arising from 10 pulses was recorded at $-$10%, $-$5%, 0%, +5%, +10%, and +15% of the approximate PT. The orders of these sets of 10 pulses were randomized, and the full range of intensities was repeated three times in separate blocks with breaks in between. The coil was repositioned at the start of each block. Averaging across blocks yielded a function representing the number of phosphenes out of 10 over a range of intensities, to which a curve was fitted (sigmoid or linear depending on goodness of fit). Solving this curve for 5/10 phosphenes thus provided the PT used in the subsequent experiments.

Equipment

Cortical stimulation was delivered with a Magstim Super Rapid biphasic stimulator in conjunction with a Magstim high-power 90 mm round coil. Round coils have previously been shown to effectively produce TMS-induced blindsight (e.g., Christensen et al., 2008; Boyer et al., 2005; Jolij & Lamme, 2005) and are more likely to affect both hemispheres than are figure-eight coils. TMS delivery was controlled with a Cambridge Research Systems Visage running Real-time Sequencer software on a Matlab platform, which also governed stimulus presentation on a gamma-corrected 21-in. Mitsubishi CRT monitor (100 Hz vertical refresh rate). Pairs of TMS pulses were applied at

95% of PT (group mean = 54.4% of maximum stimulator output, *SD* = 11.5%). This protocol was adopted because higher TMS intensities in pilot experiments caused blinks, twitches, and phosphenes that the participants reported as visible during the task. In pilot experiments, lower intensities and single pulses of TMS were found not to produce reliable effects on performance. The coil was positioned using the same procedure as described for the calibration procedure. An approximation of this position was used in the sham condition but with the coil perpendicular to the scalp so that the rim pointed toward the head, with a 9-mm plastic spacer inserted between the scalp and coil to replicate the contact artifact.

To exclude effects of TMS-induced blinks on performance, eye tracking was undertaken throughout the experiment using a Cambridge Research Systems chin-rest mounted infrared eye tracker (250 Hz). Trials were excluded on the basis of blinks identified by a shift in the trace of pupil position followed by a transitory loss of pupil signal, coincident with the stimulus presentation. Overall, 241 of a total 30,720 trials were excluded from the analysis (<0.8%).

Statistical Analyses

The temporal and anatomical dynamics of effects were assessed using a combination of Neyman–Pearson significance tests and complementary Bayesian analyses, which can directly estimate the likelihood of the null hypothesis (Gallistel, 2009; Rouder, Speckman, Sun, Morey, & Iverson, 2009; Dienes, 2008b). We adopted this combined approach because our hypotheses (Figure 1B) required testing for a specific pattern of results that included both variance and invariance between experimental conditions.

TMS-induced blindsight is defined here as a significant suppression (i.e., for active TMS vs. sham) in conscious detection, whereas concurrent reportedly "unseen" discrimination remains above chance and ideally unperturbed by the TMS. We expected to find this pattern for luminance stimuli at least for the mid timed intervention at 90–130 msec (Christensen et al., 2008; Boyer et al., 2005; Jolij & Lamme, 2005; Ro et al., 2004). Only under such conditions, where conscious detection is suppressed for both stimuli types, can the retinotectal and geniculate hypothesis be tested by assessing whether blindsight occurs in the presence of s-cone stimuli—that is, when direct input via the SC is withdrawn. If "unseen" perception is not preserved, then the retinotectal hypothesis is upheld, but if "unseen" capacity is demonstrated, then the geniculate hypothesis is supported. Note that the geniculate hypothesis does not require the extent to which "unseen" performance is above chance for s-cone stimuli to be the same as for luminance stimuli, because there may be differences in the way the different geniculate pathways contribute to perception (Merigan & Maunsell, 1993). However, the conclusions would be strengthened if the two stimulus types produce equivalent effects. Additionally, pilot experiments demonstrated that participants were able to discriminate "unseen" s-cone stimuli above chance levels when no TMS was applied. This is a prerequisite of the current paradigm because the lack of such capacity in the baseline condition would make it impossible to ask which pathways support residual capacity when early visual areas are disrupted with TMS.

Therefore, following the observation of TMS-induced blindsight with luminance stimuli and the observation that conscious detection of s-cone stimuli is suppressed by TMS, the critical question is whether or not "unseen" discrimination performance is maintained above chance in the presence of s-cone stimuli. Second, any potential disruption of "unseen" discrimination of s-cone arrows was assessed by comparing active to sham "unseen" performance and by comparing any change in "unseen" performance between the stimuli types.

Effects were assessed using repeated-measure ANOVAs with TMS site (two levels: active and sham), stimulus type (two levels: luminance and s-cone), and time of TMS relative to stimulus onset (four levels: 0–40, 40–80, 90–130, and 280–320 msec) as factors, conducted separately on measures of conscious detection (PrC; hit rate − false alarm rate) and "unseen" discrimination (PcU; proportion correct when negative responses to both "arrow?" and "something?" questions were given). Analyses of simple main effects exploited the relevant Greenhouse–Geisser corrected error terms from the ANOVAs (Winer, Brown, & Michels, 1991) and the sham condition as a baseline. "Unseen" discrimination performance was assessed relative to chance using single-sample *t* tests, which are reported in the text. All *t* tests and analyses of simple main effects applied the Holm–Bonferroni method of correction for multiple comparisons (Holm, 1979).

The positive comparisons described above only assess the likelihood of differences between conditions. By using a Bayesian approach, we can additionally assess the opposing hypothesis that two conditions are equivalent (Dienes, 2008b). This approach involves deriving a Bayes factor (*B*) which represents the strength of support for the alternative hypothesis (H1) relative to the null (Dienes, 2008b), with $B > 1$ indicating evidence in favor of H1 and $B < 1$ indicating evidence in favor of the null. In practice, values of *B* greater than 3 or smaller than 1/3 are regarded as providing substantial evidence in favor of H1 or the null hypothesis, respectively (Dienes, 2011; Jeffreys, 1961).

Bayesian analyses, complementary to the Neyman–Pearson statistics, were based upon prior models representing the critical hypotheses. Bayesian tests were conducted separately on both positive and negative changes from the sham baseline with the hypotheses represented by a uniform distribution (Dienes, 2008b). Because the critical measures were calculated relative to a baseline, zero was selected as the starting point for the distributions. The maximum reasonable shift on both measures was 0.5 and was therefore used as the upper limit for the theoretical distributions. For PcU, these

values ranged from 1 to 0.5 (chance performance) and for PrC from the calibrated level of 0.5 to chance at 0. Because it is conceivably possible that performance could fluctuate beyond this range (e.g., if false alarm rates were found to be higher than hit rates or if discrimination performance fell well below chance), the adoption of the 0.5 limit does not unfairly favor evidence for the null (see Dienes, 2008b). Sham baselined data (active − sham) is denoted as Δ sham.

To assess TMS-dependent effects, we analyzed the change in measures from sham at each level of TMS onset time and stimulus condition. The vector for comparison between stimuli types, as affected by the TMS, was calculated as ($Luminance_{active-\ sham}$) − ($s\text{-}cone_{active-sham}$). The hypothesis for this analysis was represented by a uniform distribution between 0 and 0.5, corresponding to a benefit bestowed by the additional input of luminance stimuli. In addition, for these comparisons, the complementary *t* tests were reported, as were the *B* statistics representing effects in the opposite direction. Because each comparison involved 16 independent data points (participants), appropriate standard error adjustments were applied, as recommended by Dienes (2008b).

Tests for outlier rejection were applied at a participant level using Chauvenet's criterion (Taylor, 1997). No participants were excluded.

RESULTS

The two principal measures were conscious detection and "unseen" discrimination ability. As an overview, conscious detection was suppressed by TMS during the middle and later periods, whereas "unseen" performance remained above chance and statistically unaffected. This pattern indicates characteristic TMS-induced blindsight. Our key result was that s-cone stimuli produced blindsight during the principal epoch (~100 msec, Figure 2C and D), indicating that it does not rely on the retinotectal pathway. Moreover, the pattern for s-cone stimuli in this epoch was indistinguishable from that for luminance stimuli. Beyond this, we also found that TMS facilitated rather than impaired conscious detection at the earliest time point (0–40 msec) for luminance stimuli only (Figure 2A and B). Finally, TMS impaired conscious detection at the latest time point (280–320 msec), again for luminance

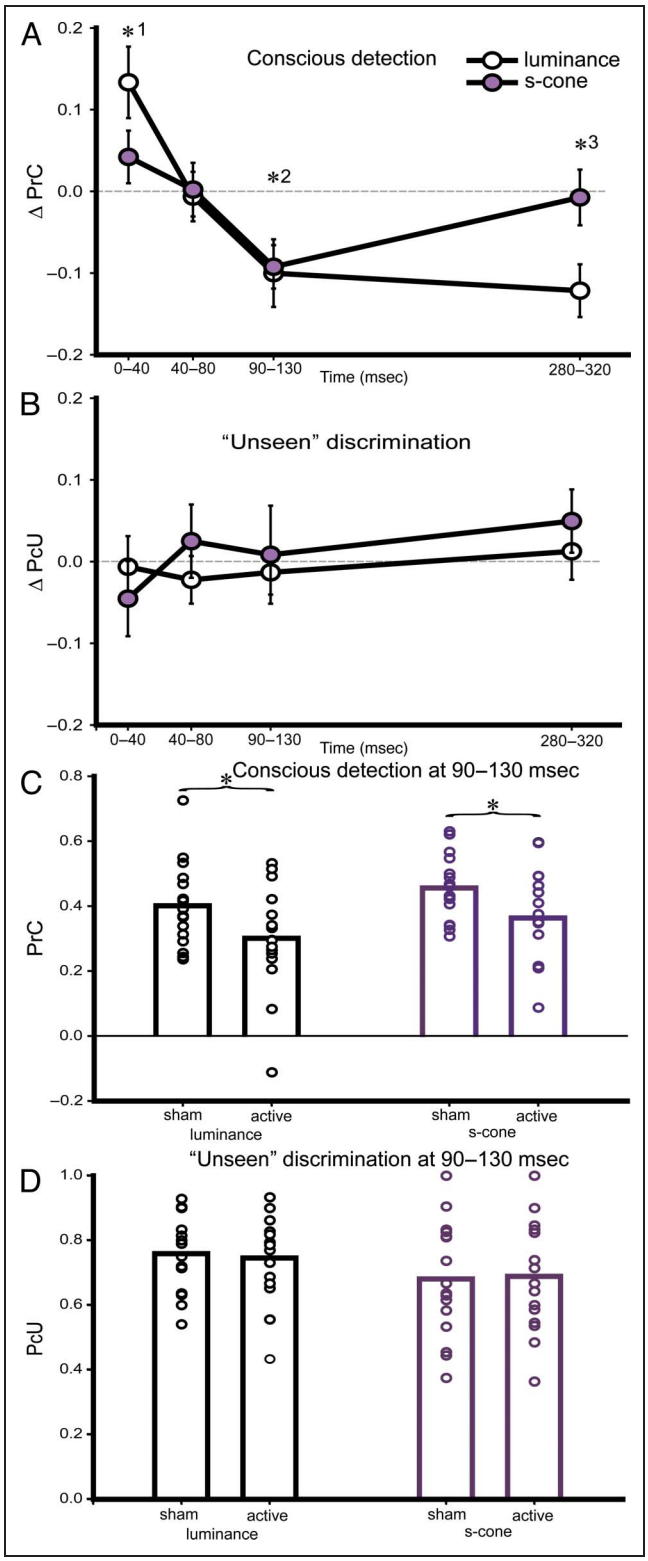

Figure 2. Sham-normalized effects of occipital TMS on (A) conscious detection (PrC) and (B) "unseen" discrimination (Proportion correct "Unseen," PcU), plotted according to stimulus type (s-cone or luminance) and TMS onset time. PrC is calculated as a nonparametric equivalent of d' (Corwin, 1994). Error bars are the within-subject *SEM* (Loftus & Masson, 1994). An impairment in conscious detection for both stimulus types, combined with preserved "unseen" abilities (TMS-induced blindsight) was observed only at the 90–130 msec epoch (*2). Occipital TMS selectively enhanced (0–40 msec, *1) or impaired PrC (280–320 msec, *3) for luminance stimuli relative to s-cone stimuli. No significant effect of TMS was observed for PrC at 40–80 msec or for PcU at any TMS onset times. C and D illustrate raw data during TMS-induced blindsight at the 90–130 msec period, where the measure of conscious detection is suppressed (C) in the active TMS condition relative to sham for both stimulus types. D depicts the preservation of "unseen" discrimination ability in this condition, again for both stimulus types. Symbols denote individual data points. *$p < .05$.

stimuli only. Data associated with this article can be downloaded from dx.doi.org/10.6084/m9.figshare.802837.

The modulation of awareness by TMS was thus both time- and stimulus-dependent: A significant three-way interaction, $F(3, 45) = 3.3, p = .031$ (see Figure 2A), was observed between TMS Condition (occipital, sham) × TMS Onset Time (0–40, 40–80, 90–130, 280–320) and Stimulus Type (luminance, s-cone). Meanwhile, discrimination performance on "unseen" trials was consistently above chance at all 16 combinations of TMS condition, TMS onset time, and stimulus type (mean proportion correct = 0.71, $SD = 0.15$, all $t(15) > 3.14, p < .007$ with Holm–Bonferroni correction). A three-way ANOVA of "unseen" discrimination revealed no significant interaction of TMS Condition × TMS Onset Time × Stimulus Type, $F(3, 45) = 0.36, p = .75$ (Figure 2B), and no significant main effects or lower-order interactions (all $F < 2.37$, all $p > .142$). Given the significant three-way interaction for awareness, we now consider these data in relation to the anatomical and temporal hypotheses by detailing the effects of occipital stimulation during each TMS epoch.

0–40 msec

The application of early TMS did not disrupt conscious detection or "unseen" discrimination. Rather, occipital stimulation significantly facilitated conscious detection relative to sham in the presence of luminance stimuli only (Figure 2A *1, PrC luminance, active vs. sham $t(15) = 5.59$, $p < .001$, $B_{(\text{active} > \text{sham})} = 8.97$, $B_{(\text{active} < \text{sham})} = 0.03$; PrC s-cone, active vs. sham $t(15) = 1.76, p = .10$, $B_{(\text{active} > \text{sham})} = 0.31$, $B_{(\text{active} < \text{sham})} = 0.04$; PrC Δ sham luminance vs. s-cone $t(15) = 2.70, p = .02$, $B_{(\Delta \text{ sham, luminance} > \text{s-cone})} = 1.20$, $B_{(\Delta \text{ sham, luminance} < \text{s-cone})} = 0.05$). A TMS-induced early suppression of "unseen" abilities would have supported a link between the feedforward sweep of activity and unconscious processing. Additionally any stimulus specificity of such an effect would implicate the role of the retinotectal pathway in such processing (Lamme, 2001). In contrast, "unseen" performance during this intervention was above chance (for luminance stimuli $t(15) = 6.25, p < .001$, for s-cone stimuli $t(15) = 3.55, p = .003$) and was unaffected by the TMS (see Figure 2B, PcU luminance, active vs. sham $t(15) = 0.16, p = .88$, $B_{(\text{luminance, active} > \text{sham})} = 0.10$, $B_{(\text{luminance, active} < \text{sham})} = 0.12$. PcU s-cone, active vs. sham $t(15) = 0.92, p = .37$, $B_{(\text{s-cone, active} > \text{sham})} = 0.08$, $B_{(\text{s-cone, active} < \text{sham})} = 0.31$). Moreover, no discernible difference in "unseen" discrimination was observed between stimuli types (PcU Δ sham luminance vs. s-cone $t(15) = 0.55, p = .59$, $B_{(\Delta \text{ sham, luminance} > \text{s-cone})} = 0.30$, $B_{(\Delta \text{ sham, luminance} < \text{s-cone})} = 0.13$). Therefore, these results do not directly support a correspondence between early occipital processing and unconscious vision. The dissociation between stimulus types for conscious detection, however, does provide confirmation that the s-cone stimuli were successfully excluded from luminance pathways. That this dissociation occurred at the earliest time point is consistent with s-cone stimuli being transmitted by a slower pathway, as we would expect (e.g., Bompas & Sumner, 2008, 2009; Anderson et al., 2008; Sumner et al., 2002, 2004, 2006).

40–80 msec

No significant effects were observed as a result of applying TMS at 40–80 msec. Neither conscious detection nor "unseen" discrimination showed any difference from sham, and both stimulus types were equally unaffected by the TMS (see Figure 2A and B, all $t < 0.99$, all $p > .34$, all $B < 0.32$). "Unseen" discrimination was above chance under active TMS (for luminance stimuli $t(15) = 5.62, p < .001$, for s-cone stimuli $t(15) = 4.84, p < .001$).

90–130 msec

TMS applied at 90–130 msec produced a blindsight effect for both stimulus types and hence adjudicates between the retinotectal versus geniculate accounts. A reliable suppression of conscious detection was observed relative to sham for both s-cone and luminance stimuli (see Figure 2A *2 and C, PrC luminance, active vs. sham $t(15) = 4.19$, $p < .001$, PrC s-cone; active vs. sham $t(15) = 3.87, p = .002$, $B_{(\text{luminance, active} < \text{sham})} = 1.58$, $B_{(\text{s-cone, active} < \text{sham})} = 59.98$). This effect did not significantly dissociate between stimulus types (PrC Δ sham luminance vs. s-cone $t(15) = 0.23, p = .82$, $B_{(\Delta \text{ sham, luminance} > \text{s-cone})} = 0.15$, $B_{(\Delta \text{ sham, luminance} < \text{s-cone})} = 0.18$).

The key question in this study is whether above-chance "unseen" discrimination is maintained in the context of such a TMS-induced impairment of conscious detection —that is, whether TMS-induced blindsight occurred— and whether these "unseen" abilities are stimulus independent (as predicted by the geniculate hypothesis) or differentially impaired in the presence of s-cone versus luminance stimuli (as predicted by the retinotectal hypothesis). "Unseen" discrimination at 90–130 msec was maintained above chance for both stimulus types (luminance: $t(15) = 7.51, p < .001$, s-cone: $t(15) = 4.47, p < .001$; Figure 2D). This indicates the characteristic signature of TMS-induced blindsight and is the primary indication that residual capacity is preserved even when direct input via the SC is withdrawn. "Unseen" ability was unaffected by occipital TMS in the presence of both s-cone stimuli and luminance stimuli (PcU luminance, active vs. sham $t(15) = 0.39, p = .70$, $B_{(\text{luminance, active} < \text{sham})} = 0.13$, $B_{(\text{luminance, active} > \text{sham})} = 0.07$. PcU s-cone, active vs. sham $t(15) = 0.14, p = .89$, $B_{(\text{s-cone, active} < \text{sham})} = 0.15$, $B_{(\text{s-cone, active} > \text{sham})} = 0.18$). Furthermore, comparisons between sham-normalized discrimination performance confirmed no reliable difference between stimulus types (see Figure 2B and D, PcU Δ sham luminance vs. s-cone $t(15) = 0.30, p = .77$, $B_{(\Delta \text{ sham luminance} > \text{s-cone})} = 0.16$, $B_{(\Delta \text{ sham luminance} < \text{s-cone})} = 0.24$). The strongest trend in this comparison was a tendency toward reduced

performance for luminance relative to s-cone stimuli ($B = 0.24$), contrary to the pattern predicted by the retinotectal hypothesis. Taken together, the findings demonstrate the persistence of "unseen" discrimination ability when informative retinotectal input is prevented.

280–320 msec

The latest TMS intervention suppressed conscious detection but only in the presence of luminance stimuli (see Figure 2A *3, PrC luminance, active vs. sham $t(15) = 5.09$, $p < .001$, $B_{(\text{luminance, active < sham})} = 168.5$, $B_{(\text{luminance, active > sham})} = 0.02$. PrC s-cone, active vs. sham $t(15) = 0.31$, $p = .76$, $B_{(\text{s-cone, active < sham})} = 0.11$ $B_{(\text{s-cone, active > sham})} = 0.08$, PrC Δ sham luminance vs. s-cone $t(15) = 3.38$, $p = .004$, $B_{(\Delta \text{ sham, luminance > s-cone})} = 0.03$, $B_{(\Delta \text{ sham, luminance < s-cone})} = 10.62$). This later disruption of conscious processing, together with the disruption caused by TMS at 90–130 msec, is consistent with the notion that conscious processing is supported by recurrent activity (Lamme, 2001). "Unseen" discrimination was above chance under active TMS (for luminance stimuli $t(15) = 6.98$, $p < .001$; for s-cone stimuli $t(15) = 6.90$, $p < .001$). Again "unseen" discrimination appeared to be unaffected by the TMS and did not differ between stimulus types (see Figure 2B, PcU luminance, active vs. sham $t(15) = 0.35$, $p = .73$, $B_{(\text{luminance active < sham})} = 0.08$, $B_{(\text{luminance active > sham})} = 0.13$, PcU s-cone, active vs. sham $t(15) = 1.13$, $p = .28$, $B_{(\text{s-cone active < sham})} = 0.06$, $B_{(\text{s-cone active > sham})} = 0.35$, PcU Δ sham luminance vs. s-cone $t(15) = 0.60$, $p = .55$, $B_{(\Delta \text{ sham luminance > s-cone})} = 0.11$, $B_{(\Delta \text{ sham luminance < s-cone})} = 0.28$).

Discrimination performance when participants acknowledged awareness of both the "arrow" and "something" was close to ceiling (proportion correct; 0.97 ± 0.05 *SD*). Performance when partial awareness of stimuli was acknowledged, through a positive responses to the "something" question but denial of having seen the "arrow," was also high (proportion correct: 0.88 ± 0.15 *SD*). This apparent correlation between level of awareness and discrimination capacity is to be expected but does not violate the zero correlation criteria for "unconscious" processing (see Dienes, 2008a). This is because the claim is that unconscious processing and blindsight are only observed here when participants report "no" to both the "arrow" and the "something" questions and therefore is consistent with the "guessing criterion" definition of unconscious capacity (Dienes, 2008a).

It is notable that PcU is calculated from "unseen" trials only, whereas more trials contribute to the measure of conscious detection (PrC and PcU were calculated from a total of 22,275 and 5525 trials, respectively). Thus, it might be argued that the analysis of PcU is hindered by a relative lack of statistical power at an individual level. To test this hypothesis, estimates of standard error based on these specific trial numbers (the number of data points convolved for the number of contributing trials) can be compared with test whether heterogeneity of variance could be responsible for the observation of significant effects on one measure and not another. Keppel (1982) recommends a 3:1 ratio of such variances (Fmax ratio) as the cut-off point, beyond which the variances should be considered heterogeneous and the conditions noncomparable. For our data, the Fmax ratio was calculated as 1.84:1 (0.032/0.017), thus satisfying this requirement.

DISCUSSION

Our results provide evidence that the residual abilities of TMS-induced blindsight shown here are predominantly geniculate (e.g., Schmid et al., 2010; Boyer et al., 2005) rather than retinotectal in origin (e.g., Leh et al., 2010; Ro et al., 2004). Blindsight was demonstrated most clearly by the suppression of reported awareness at 90–130 msec for both stimulus types, in the presence of above chance "unseen" discrimination. This corresponds to the expected epoch of visual suppression by occipital TMS (Amassian et al., 1989). The central question posed was whether the concurrent "unseen" abilities were dependent upon stimulus type, that is, whether the residual abilities were maintained for stimuli that are invisible to the retinotectal route. The preservation of such abilities for s-cone stimuli indicates that chromatic geniculate pathways rather than the retinotectal route supported the residual abilities of TMS-induced blindsight shown here. Moreover, it seems unlikely that the absence of stimulus-specific effects on the measure of "unseen" discrimination was due to the stimuli not isolating the R/M pathways or the ineffectiveness of TMS, because both TMS effects and stimulus specificity were reliably demonstrated upon the contrasting measure of conscious detection.

How can we reconcile previous evidence in favor of the retinotectal hypothesis with the current evidence in support of the geniculate hypothesis? Much of the evidence consistent with retinotectal mediation does not in fact logically exclude a geniculate role, but in patients where the geniculate pathways are surgically cut the evidence for retinotectal mediation is particularly strong (Leh et al., 2010; Tamietto et al., 2010; Leh, Mullen, et al., 2006). Our results are not inconsistent with a role for the retinotectal pathway in certain kinds of residual ability (i.e., for certain types of stimuli), particularly following permanent brain injury where plasticity may alter the functional contribution of different pathways (Silvanto & Rees, 2011; Huxlin, 2008; Mittmann & Eysel, 2001).

Rather than exclusively supporting the geniculate hypothesis and disconfirming the retinotectal hypothesis, we view our data as disconfirming the core idea implicit in the long debate between these theories—that because blindsight has been categorized as a single phenomenon we should expect a single pathway to support it. Different pathways are likely to support unconscious abilities that depend on different types of information (Cowey, 2010; Danckert & Rossetti, 2005). Some residual abilities for

some types of stimuli, within the wider scope of blindsight, may be supported by input through the SC. However, the positive demonstration of residual abilities when such input is withdrawn indicates that the SC cannot be necessary for the preserved capacity of TMS-induced blindsight, as has been previously claimed (Ro et al., 2004).

The proposed correspondence between early feedforward activity and the residual "unseen" abilities was neither supported nor refuted by the current data (Lamme & Roelfsema, 2000). Our results do, however, indicate that this period and these abilities are not wholly dependent upon initial input provided by the SC. In contrast to unconscious abilities, R/M pathways did make a time-specific contribution to conscious vision, thus validating the efficacy of the chromatic intervention. No specific predictions were cast in terms of s-cone dependency; hence, the conclusions that can be drawn in relation to these effects are qualified as speculative. However, such effects upon conscious awareness are clearly a possibility given the significant contribution of R/M pathways to cortical input (Nealey & Maunsell, 1994).

Interestingly, the earliest effect we observed (0–40 msec) was a facilitation of conscious detection. Early TMS has been reported to cause either perceptual suppression (Corthout, Uttl, Walsh, Hallett, & Cowey, 1999; Corthout, Uttl, Ziemann, Cowey, & Hallett, 1999) or enhancement (Abrahamyan, Clifford, Arabzadeh, & Harris, 2011; Schwarzkopf, Silvanto, & Rees, 2011). Such enhancement effects have been attributed to the addition of neuronal noise, producing stochastic resonance and driving cortical activity across a threshold for detection (see Schwarzkopf et al., 2011). Earlier potentiation and later suppression of functionality can be further understood if the role of the early period is seen as preparatory (Marr, 1982), potentially in the service of later conscious processing. Because the state of activation leading up to the arrival of information is crucial in determining whether or not that information is perceived (Romei, Gross, & Thut, 2010; Mathewson, Gratton, Fabiani, Beck, & Ro, 2009), the application of early TMS may possibly provide a pedestal of activity, priming the region for subsequent processing. This might not be the case for later applications because increasing activity without interfering with information is only possible when TMS is applied before the arrival of that information. Here this rapid facilitation was only realized in the presence of luminance stimuli, indicating that this early period was dependent upon input provided by R/M pathways. This is consistent with the relatively high speeds at which these pathways transmit information (Bompas & Sumner, 2008; McKeefry, Parry, & Murray, 2003; Maunsell et al., 1999; Cottaris & De Valois, 1998), suggesting that the R/M pathways may play a particularly prominent role in the early preparatory period, potentially in the service of conscious processing.

The observed disruption of conscious detection at later TMS onset times is consistent with existing proposals (Lamme, 2001) that delayed re-entrant processing supports conscious vision. The latest of these effects (280–320 msec) occurred only in the presence of luminance stimuli, indicating that information conveyed via R/M pathways supports this later period. What then provides this late R/M input? Our findings provide no definitive answer to this question. However, it seems unlikely to be dependent upon feedforward or recurrent activity that has first passed through the affected early visual cortex because s-cone inputs are believed to be integrated with other signals within these regions (Sincich & Horton, 2005; Moutoussis & Zeki, 2000, 2002; Cottaris & De Valois, 1998). It is more plausible to speculate that R/M information is first passed through unaffected areas—possibly via the aforementioned extrastriate pathways—and is only then fed back to interact with feedforward processing. In this way, the later stage of conscious processing may have been susceptible to later TMS because it is supported by R/M inputs, present for luminance stimuli, that initially enter unaffected frontoparietal networks (Corbetta, Patel, & Shulman, 2008) before later feeding back into the disrupted occipital cortex.

In conclusion, the residual visual abilities of TMS-induced blindsight shown here originate primarily from spared geniculate projections. Our findings thus stand in contrast to the explanations of blindsight that rely on the retinotectal pathway (Ro et al., 2004) and instead add weight to evidence that spared regions in the geniculate pathway can support unconscious vision following occipital disruption (Schmid et al., 2010; Fendrich, Wessinger, & Gazzaniga, 1992). Our results also indicate that conscious vision depends on activity within the early occipital cortex from ~100 msec after stimulus onset and that R/M pathways make a contribution to awareness during early preparatory (0–40 msec) and later feedback (280–320 msec) stages of visual processing.

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Reprint requests should be sent to Christopher P. G. Allen, Cardiff University Brain Research Imaging Center, School of Psychology, Cardiff University, Park Place, Cardiff CF10 3AT, UK, or via e-mail: allencp@cardiff.ac.uk.

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