Research Focus

Zap! Magnetic tricks on conscious and unconscious vision

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Blindsight, the remarkable capability to react to unseen visual stimuli, has thus far only been demonstrated in patients and monkeys with a lesion to primary visual cortex. A recent study by Boyer, Harrison and Ro demonstrates blindsight in normal human observers, using TMS to block visual processing. Combined with other work, this opens new vistas towards understanding mechanisms of conscious and unconscious vision.

Unconscious vision

Everyone will have had the experience of grasping a bottle that falls from the table 'before you knew it was falling' or ducking for an imminent blow from something that 'you didn't even see'. These are your unconscious visual reflexes at work. Typically, however, your actions are (or at least seem) guided by what you consciously see. What distinguishes conscious from unconscious vision? This important question is central in the quest for understanding consciousness in general. A recent study by Boyer, Harrison and Ro [1], sheds new light on the problem.

Boyer and colleagues studied blindsight in normal observers. Classically, blindsight is seen in patients following a lesion to V1 (primary visual cortex, or striate cortex) (see Figure 1). These patients have no conscious visual experiences in the part of the visual field corresponding to the lesion. What sometimes remains however, is the remarkable ability to react to invisible stimuli. If prompted to do so (this is a prerequisite) patients can make eye movements towards unseen objects or even point to and grasp them. In forced-choice experiments, patients can discriminate orientations, shapes, colours, directions of motion and even emotional expression of faces, high above chance levels [2–5].

Boyer and colleagues tried to induce this phenomenon in normal subjects, using transcranial magnetic stimulation (TMS). With TMS, it is possible to disturb ongoing neural processing for very brief periods of time, at selected regions of the cortex, in normal human subjects. TMS can thus be used as a 'virtual lesion' technique, with the advantage of inducing reversible 'lesions' that last only milliseconds [6]. In the past, TMS has been used to induce visual field defects in normal subjects by applying TMS pulses to the occipital pole [6]. Until recently, however, these field defects were never shown to be accompanied by blindsight phenomena like those seen in patients with clinical lesions.

TMS induced blindsight

Boyer and colleagues applied TMS pulses to the occipital pole, targeting V1. The pulses were delivered at about 100 ms after the presentation of visual stimuli. Two types of stimuli were used: small oriented bars or faint patches of colour. On each trial, the subjects first had to indicate whether they had seen the orientation of the bar, or the colour of the patch. If not, they were then probed to guess the orientation or colour. Finally, subjects were asked to rate their confidence in guessing the unseen stimulus attribute.

As expected, subjects were unaware of the orientation or colour on the majority of trials in which TMS pulses were delivered. This showed that the brief disruption of activity in V1 by the TMS pulse at the right time after stimulus onset was sufficient to block conscious visual percepts. Crucially though, the authors also showed that on trials where subjects reported no awareness of the stimulus attributes, their guesses about orientation or colour were nevertheless highly above chance. This result therefore replicated the classical blindsight phenomenon but in normal subjects.

There was also a difference however. In lesion-induced, so called type-I blindsight, there is usually no awareness of a stimulus whatsoever, and subjects have no confidence at all in their guesses, even though they might guess correctly on 80% of trials. Therefore, there is no relation between confidence ratings and percentage correct in guessing [3,7]. The TMS study, on the other hand, showed a positive correlation between percentage correct and confidence rating. Subjects also indicated that on many trials they saw that 'something' was presented, but were just not aware of the stimulus attribute being asked for (orientation or colour). This type-II blindsight can occur in patients as well, for example with high-luminance flashes or fast-moving stimuli [2,3].

A previous study from the same group showed that objects made invisible by TMS could also influence the latency of saccades made to seen objects, replicating another blindsight observation made in patients [8]. And Jolij and Lamme recently showed TMS-induced blindsight for affective content: the emotional expression (happy or sad) of faces that could not be detected or localized could still be guessed at far above chance levels [9]. Thus, it now

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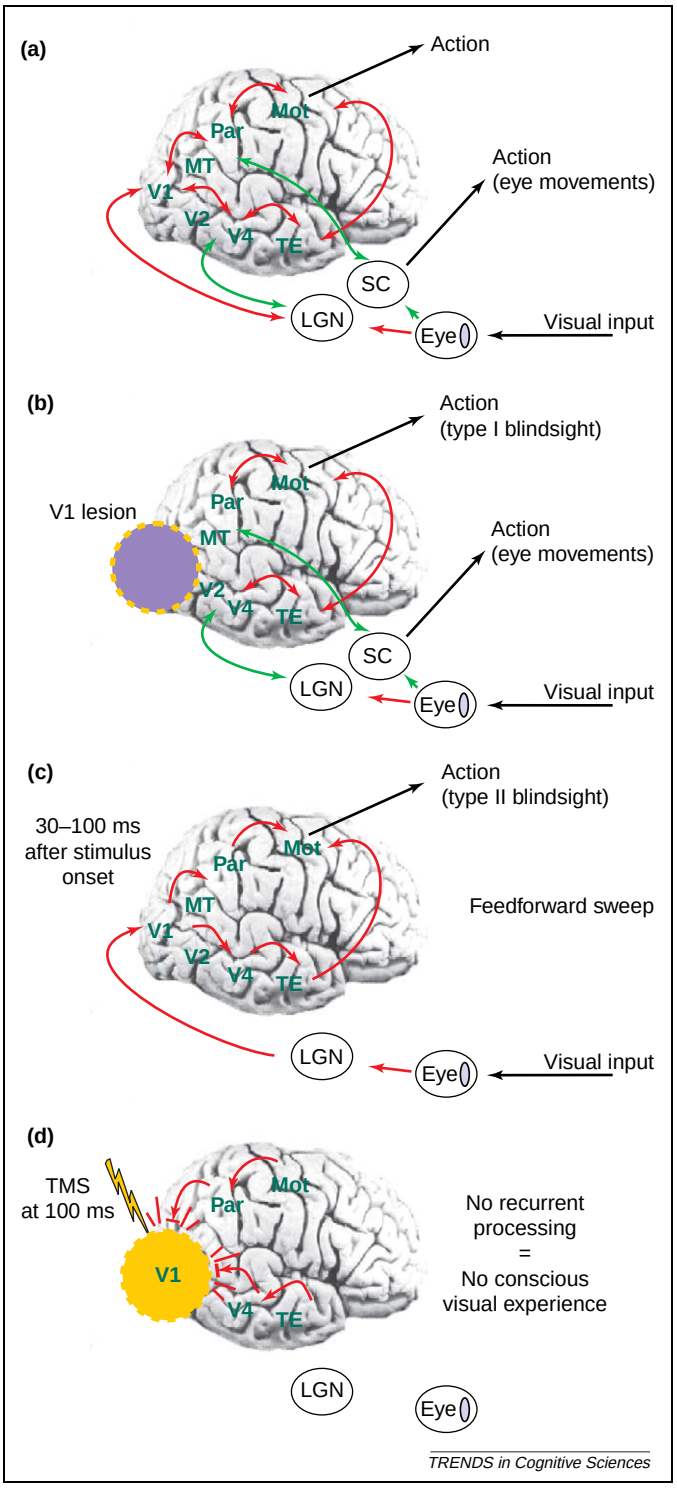

Figure 1. Neural pathways enabling conscious and unconscious vision. **(a)** The major neural pathways transferring visual information from the eye to the visual cortex. LGN: lateral geniculate nucleus, SC: superior colliculus, V1, V2, V4, MT, TE: cortical visual areas, Par: parietal cortex; Mot: motor cortex. Red arrows indicate the most important, geniculo-striate and cortical pathways. Green arrows indicate the alternative routes to the cortex. **(b)** The remaining routes after a lesion to V1 (purple), which potentially mediate blindsight behaviour. **(c)** and **(d)** An alternative explanation for blindsight induced by TMS. **(c)** Feedforward activation is still possible until the delivery of the TMS pulse (~100ms after stimulus onset), thus enabling blindsight behaviour. **(d)** When the TMS pulse is delivered to V1 at 100 ms latency (yellow), visual signals have already passed the feedforward sweep. However, recurrent interactions between higher visual areas and V1 are disrupted, which results in a loss of the conscious percept.

seems that many of the classical blindsight findings can be replicated in normal subjects using TMS.

Unconscious pathways

What do these TMS results add to what we already know from blindsight patients? Primarily, it tells us something about the visual pathways that have been suggested to mediate blindsight behaviour (Figure 1b). The first is a route that bypasses the lateral geniculate nucleus (LGN), flowing directly from the retina to the superior colliculus, a midbrain structure important for eye movements and orienting behaviour. The second are projections from the LGN to areas beyond striate cortex, such as V2, V4 and MT. Different types of blindsight behaviour might arise through one or other of these pathways [1–4]. The TMS studies tell us that these routes are also functional in normal subjects, and not just in patients, in whom such routes might have developed as a result of brain plasticity after the lesion.

Revealing the mechanisms of consciousness?

What remains an open question, is why activation of these pathways, all of which project to cortical areas, does not lead to conscious experience. Studies in patients have so far not revealed the answer to this intriguing question. The TMS study lends support to a potential answer, however. It can be argued that the TMS pulses delivered at latencies of around 100 ms after stimulus onset do not completely block the transfer of information via primary visual cortex. Visual signals have been shown to arrive in V1 as soon as 30 or 40 ms after stimulus presentation [10]. At 100 ms latency, the visual signal is well under way towards extrastriate areas, and areas in parietal and temporal cortex [10]. So why is there no conscious experience when TMS is applied that late after the stimulus?

It has been suggested that the key to understanding the conscious/unconscious dichotomy lies in the distinction between feedforward and recurrent processing [10,11]. This hypothesis states that the 'feedforward sweep', that is, the initial volley of visual information transfer through the successive visual areas, can generate motor responses but is not generating conscious experience. Only after higher visual areas send signals back to lower areas, using recurrent processing, is an organized visual percept thought to arise [12]. This would fit exactly with the TMS results of Boyer *et al.*: activation via the feedforward sweep is unimpaired by the TMS pulses at ~100ms latency, and could therefore allow the blindsight behaviour (Figure 1c). But the feedback signals arrive in V1 at exactly this latency, and will be disrupted by the electromagnetic pulses of the TMS (Figure 1d). Similarly, in patients, signals bypassing V1 will generate feedforward activation of extrastriate areas. Owing to the lesion, feedback signals cannot reach V1, hence there is no recurrent processing and no conscious experience [13].

Following the demonstration by Boyer *et al.* of TMS-induced blindsight, this hypothesis can now be tested: if indeed TMS does not impair visual signals reaching the extrastriate areas via the feedforward sweep, this should

be measurable by recording EEG or fMRI signals. It could also be tested whether disrupting the feedforward activation, using TMS pulses delivered at 30–50 ms latency, would abolish the blindsight capabilities. TMS-induced blindsight therefore offers us new opportunities to study the role of the dynamics of visual processing in conscious and unconscious vision, at the same time yielding a better understanding of the blindsight phenomenon in patients.

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Letter

A module for syntactic processing in music?

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Music and language have rules governing the structural organization of events. By analogy to language, these rules are referred to as the 'syntactic rules' of music. Does this analogy imply that the brain actually performs syntactic computations on musical structures, similar to those for language and based on a specialized module [1–3]? In contrast to linguistic syntax, which involves abstract computation between words, rules governing musical syntax are rooted in psychoacoustic properties of sound: syntactically related events are related on a sensory level and involve only weak acoustical deviance. For example, the dominant and tonic chords (referred to as V and I in Figure I, Box 1 of [1]), whose succession forms the most fundamental syntactic unit of Western tonal music, have pitch commonality values [4] two times higher than less related dominant and supertonic chords (V and II).

Musical syntax and sound properties are so strongly entwined that psychoacoustic and cognitive approaches provide highly correlated accounts of Western musical structures [4–6], the former providing a parsimonious account. Long-standing evidence for syntax-like processing in music has been challenged by psychoacoustic approaches: a short-term memory model, operating on echoic images of periodicity pitch, can account for the musical functions of tones in tonal contexts [5]. Does a more abstract computation occur beyond this sensory processing? Some studies provide evidence that cognitive components linked to musical syntax processing can overrule sensory components in music processing [7,8]. However, even these studies acknowledge the predominance of sensory factors during early processing stages.

A key issue is therefore to evaluate whether the contribution of syntactic processing in music is minor (if present at all), as suggested by psychoacoustic approaches, or whether it is as essential as linguistic syntactic processing is for understanding language. Moreover, to conclude in favour of a syntactic module, neuroscientific studies should manipulate orthogonally syntactic and acoustical deviances in musical stimuli, and should confirm that both manipulations result in distinct neural signatures. The findings available in the literature demonstrate neural correlates responding to a combination of musical and acoustic irregularities, but do not provide sufficient evidence for us to conclude that the effects elicited by these irregularities reflect purely syntactic processing. The article by Koelsch and Siebel [1] points to very promising issues but asserting the existence of a module of musical syntax processing is premature.

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