# Modulation of cortical excitability can speed up blindsight but not improve it

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**Abstract**

Blindsight has been widely investigated and its properties documented. One property still debated and contested is the puzzling absence of phenomenal visual percepts of visual stimuli that can be detected with perfect accuracy. We investigated the possibility that phenomenal visual percepts of exogenous visual stimuli in patient GY might be induced by using transcranial direct current stimulation. High contrast and low contrast stimuli were presented as a moving grating in his blind hemifield. When left area MT/V5 was anodally stimulated during the presentation of high contrast gratings, he never reported a phenomenal percept of a moving grating but showed perfect blindsight performance. When applied along with low contrast gratings, for which accuracy was titrated to 60-70%, performance did not improve but responses were significantly faster. Cathodal stimulation had no effect. Results are explained in the framework of GY's reorganised cortical connexions and oscillatory patterns known to be involved in awareness in GY. The apparent presence of phenomenal visual percepts in earlier studies is shown to be a semantic confusion about what he means when he says that he sees in his blind field.

Key words: blindsight; tDCS; visual qualia; motion

**Introduction**

Blindsight is the term used to describe the paradoxical ability of many patients with unilateral destruction of all or part of the striate cortex - resulting in a field defect of apparent total blindness - to detect and localise and even discriminate between visual stimuli confined to the field defect. Since they claim to see nothing, their residual ability can only be revealed by forced-choice responding (Weiskrantz et al. 1974). After nearly 40 years of investigation, much of it on a small number of intensively studied patients, some aspects of blindsight remain controversial, especially whether the patient can experience phenomenal visual percepts (visual qualia) or whether the successful performance is based on cerebral events that are not accessible to visual consciousness but can provide the basis for a verbal or motor response (Cowey, 2010). A further possibility is that moving stimuli, which are more readily detected in blindsight, lead to real phenomenal visual percepts (Riddoch, 1917; Barbur et al., 1993; Zeki and ffytche, 1998). In the case of the latter there is strong evidence that the cortical motion area complex MT/V5 is functionally activated by moving stimuli in the blind field and that it is this activity that underlies excellent performance and, perhaps, leads to real phenomenal visual percepts.

Much of the investigation of blindsight over three decades has involved the hemianope GY, who is the subject of the present investigation. In an attempt to produce visual phosphenes by stimulating different regions of his extra-striate cortex, notably MT/V5, Cowey and Walsh (2000) applied bursts of repetitive transcranial magnetic stimulation, rTMS, When applied above MT/V5 of the intact (right) hemisphere GY always experienced moving visual phosphenes in the contralateral hemifield, like intact subjects. But the same procedure over the 'blind' hemisphere never produced a phosphene. However, when both hemispheres were simultaneously stimulated, with slight stimulation onset asynchronies, he reported phosphenes in both seeing and blind hemifields at roughly mirror image positions (Silvanto et al. 2007). However, the bilateral phosphenes were always close to the vertical midline and it is possible that the blindfield phosphenes arose from activation in the normal hemisphere of neurons that are part of the bilateral representation of the vertical midline retina (Lavidor & Walsh, 2004). Therefore, TMS in GY can induce a phenomenological awareness of internally generated phosphenes within the central visual field, the mechanism for which is unclear. One aim of the present experiment was to see if we could induce phenomenological visual awareness of external visual stimuli (i.e. visual stimuli that he can respond to but has no visual awareness of in his blind field). Hence, we carried out an experiment to examine the effect of modulation of GY's cortical excitability to answer three questions. First, would anodal transcranial direct current

stimulation (tDCS) raise the excitability of neurons in MT/V5 of the blind (left) hemisphere to a point where a high-contrast moving visual grating began to induce a phenomenal visual percept? Second, and irrespective of the answer to the first question, would tDCS improve blindsight performance on a motion discrimination task where the stimuli were sufficiently faint to make the discrimination difficult? Third, even if performance was unaltered would the stimulation influence reaction times, which might indicate faster processing in MT/V5 of the blind hemisphere? GY's performance on a visual task in which he had to make discriminations about targets in his blind field was assessed after anodal stimulation of MT/V5 and cathodal stimulation of V5 on separate days. This performance was compared with results of sham stimulation done on each testing day. tDCS allows us to investigate the neural networks involved in GY's awareness of visual stimuli as the excitability of areas connected to left MT/V5 is also modulated (Peña-Gómez et al., 2011). Therefore, tDCS of MT/V5 will also presumably increase or decrease excitability in his altered neural connectivity.

## Method

### *Subject*

Subject GY displays blindsight, following almost complete destruction of his left striate cortex (V1) as a consequence of a traffic accident suffered at the age of 8. There was also slight damage to extrastriate areas V2 and V3. The damaged regions show no responsiveness to visual stimulation (Azzopardi and Cowey, 2001; Barbur at al., 1993). GY was 60 years old at the time of the present testing and was naïve as to the precise aims of the study. Informed consent was given before his participation in the study which was approved by the Durham University Ethics Committee in agreement with the Declaration of Helsinki, 1964.

### *Transcranial Direct Current Stimulation, tDCS*

The two rubber electrodes were placed in two sponge pouches (7 cm x 5 cm) which had been soaked in a physiologically active saline solution. A rubber strap around the skull was used to hold the two electrodes in place. tDCS was applied using a Magstim Eldith DC stimulator for 15 minutes at a current intensity of 1.5 mA. The stimulation protocol complied with the current safety guidelines for tDCS (Nitsche et al., 2003). Stimulation was carried out over three testing days. On the first testing day, GY's perception of a physically prominent and moving visual grating was tested. tDCS was applied in two montages. Sham tDCS was first applied followed by the anode being situated over left MT/V5 and the cathode over right MT/V5. Several hours later, tDCS was applied with the anode placed over left MT/V5 and the cathode (reference) over

the right frontal pole. Each of the second and third testing days comprised of a sham session and an experimental session in which the cathode was placed over left MT/V5 or in which the anode was placed over left MT/V5. In each case, the reference electrode was placed over right frontal pole. Sham stimulation had the same electrode placement but in this case the stimulator was switched off following 30 seconds of stimulation which is not sufficient to generate any excitability changes. However, this allowed GY to experience the initial itching sensation associated with real stimulation, making him unaware of which stimulation condition he was experiencing. The sham condition preceded the experimental condition on each testing day.

On the first day, prominent and longer-lasting visual stimuli were used in order to see whether anodal tDCS over left MT/V5 of his damaged hemisphere might allow him to experience real visual percepts in his hemianopic field. On the second and third days the visual stimulus was brief, 200 ms, and its mean luminance was the same as that of the surround for reasons explained below.

The left MT/V5 location was measured as being 3 cm dorsal and 5 cm lateral to the left of the mastoid-inion in agreement with previous functional location of MT/V5 with GY (Silvanto et al. 2009) with the right MT/V5 at the homologous co-ordinates. However, as the area of stimulation was defined by the size of the electrodes (Peterchev et al., 2011), precise functional localisation of the sites of interest was not necessary and centring the electrode over the known regions was sufficient.

*Visual task*

The task and procedure have been described in detail before (Cowey and Alexander, 2012). A cartoon of the visual display is shown in Figure 1. GY sat in front of the display with his head on a chin-rest and his eyes 47 cm from the centre of the screen. The room was dimly-lit and the display was in silhouette against the white wall behind it.

The stimuli were presented on a 17" colour monitor (Phillips UP2799), subtending $50^{\circ}$ x $34^{\circ}$ at the viewing distance of 47 cm. The stimuli were generated and controlled by a Pentium 4 computer. Luminance was measured with a Minolta LS-110 digital photometer. The gamma function of the screen was determined with an Optical OP200-E (Cambridge Research Systems), providing a table of values, accessible in software, that allowed us to select appropriate input voltages to the three guns in order to present achromatic stimuli of known luminance and

contrast. The face of the VDU was a touch sensitive screen that recorded where and when the display was touched with the subject's forefinger. The effective response area on the screen coincided with the area of the target stimuli and with the central 2° start-light. The display is shown schematically at the top of Figure 1.

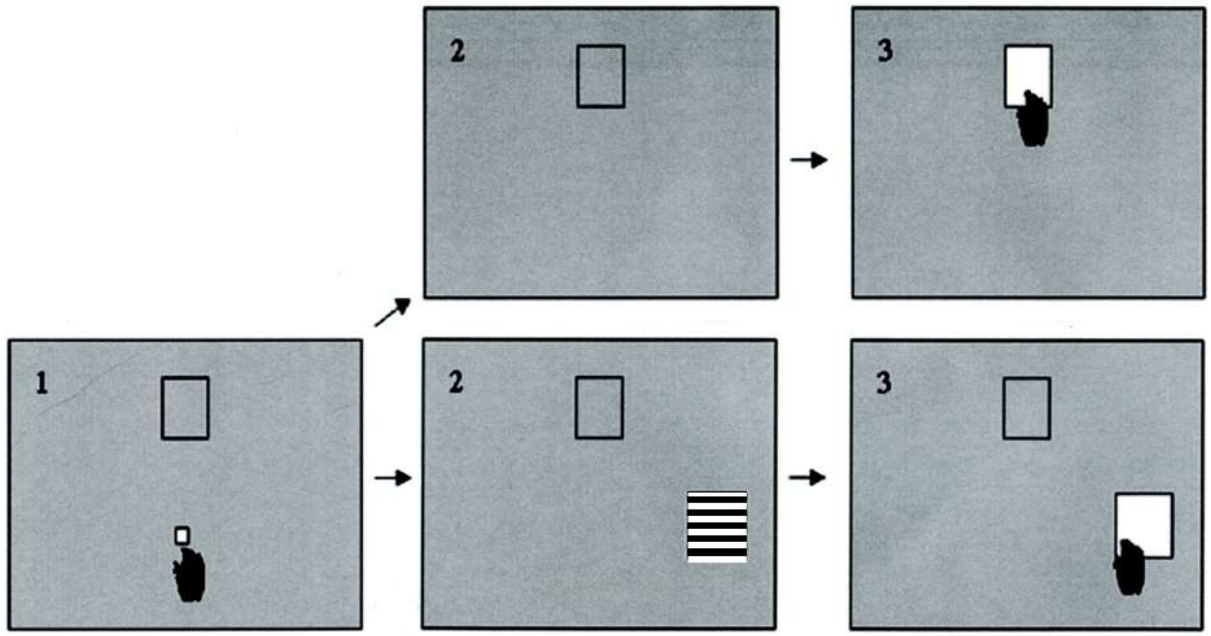


Figure 1: Schematic of each trial type.

Immediately beneath the screen was an SMI RED-11 infra-red eye tracker (Sensorimotoric Instruments) which monitored one eye of the subject so that any deviations from central fixation of more than about 2-3 degrees during a trial could be detected and the trial discarded. Therefore, visual stimuli could reliably be presented at, and confined to, particular retinal positions. As the nearest edge of the grating in the blind hemifield was approximately 10 degrees from the start light any eye movement large enough to reach it was easily detected.

When testing with prominent stimuli on day 1 the moving grating lasted 1.0 sec and its mean luminance was 31 Cd/m2 against a surround of 7 Cd/m2. On days 2 and 3 the contrast between the mean luminance of the grating target and the surround was always zero. The contrast between the surround and the individual bright bars of the target was maximally 0.3, meaning that luminance artefacts from intra-ocular scatter should be undetectable. Nor were there any detectable extra-ocular artefact such as specular or diffuse reflections from any part of the display and its surroundings. The left edge of the screen was covered with a vertical strip of black cardboard to cover any raster artefacts that are prone to occur there.

*Procedure*

When GY touched the start-light near the centre of the display the light was extinguished and the target grating (except on blank trials) was presented for 1 sec (day 1) or 200 ms, during which period it moved smoothly downwards at 18 deg/sec. GY's task was to respond as quickly as possible by touching the position of the right target on target trials and the square at the top of the display if it was a blank trial. Correct responses were signaled by briefly filling the response area with bright white light. Incorrect responses, made by pressing blank on a target trial or vice versa, were indicated by briefly turning the entire screen black. Trials were self-paced and were given in blocks of 100. Target and blank trials were equiprobable but randomly presented. Four blocks of the visual task were administered in each experimental block (see Figure 2).

On days 2 and 3 the appropriate target contrast for visual detection of 60-70% correct was first determined using a contrast titration procedure. This required several hundred trials and its purpose was to make the task sufficiently difficult for any improvement in performance attributable to tDCS to be revealed. The first block was administered at 5 minutes post tDCS onset with subsequent blocks at 5 minute intervals. Therefore, blocks 1 and 2 fell within the stimulation period whereas blocks 3 and 4 were carried out after the stimulation had ended. Each block comprised of 100 trials and took 5 minutes to complete.

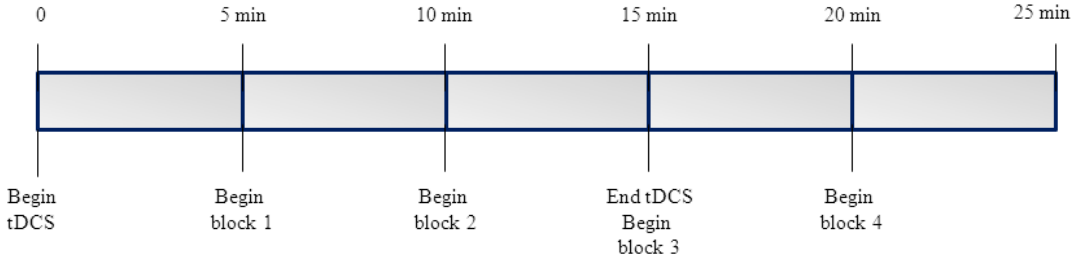


Figure 2: Protocol of tDCS application and visual stimulus blocks.

**Results**

*Subjective experience*

One purpose of the experiment was to see whether GY might experience a physically prominent moving visual grating as some form of visual quale in his blind hemifeld if tDCS was administered over area V5 of his left 'blind' hemisphere The results in all three sessions (sham tDCS, anodal tDCS over left MT/V5 with the cathode situated over right MT/V5 and anodal tDCS over left MT/V5 with the cathode over right frontal pole) were the same: almost perfect performance in 1200 trials (GY once categorized a blank trial as a target in the blind field). But the outstanding result was that not once did he report a visual quale in his blind field despite attending to the blind right hemifield and correctly detecting the blind-field target on every one of 600 target trials and expressing awareness, i.e.Type 2 blindsight.

Equally, when less prominent stimuli were displayed, neither anodal nor cathodal stimulation administered over GY's left MT/V5 resulted in an increase of phenomenological awareness of visual stimuli.

*Reaction time and accuracy scores*

For data related to less prominent stimuli (days 2 and 3), a two factor (stimulation $_{[\text{sham v tDCS}]}$ x block $_{[1\text{-}4]}$) repeated measures ANOVA was carried out for both anodal and cathodal stimulation for accuracy and reaction time measures to assess its effect on GY's performance.

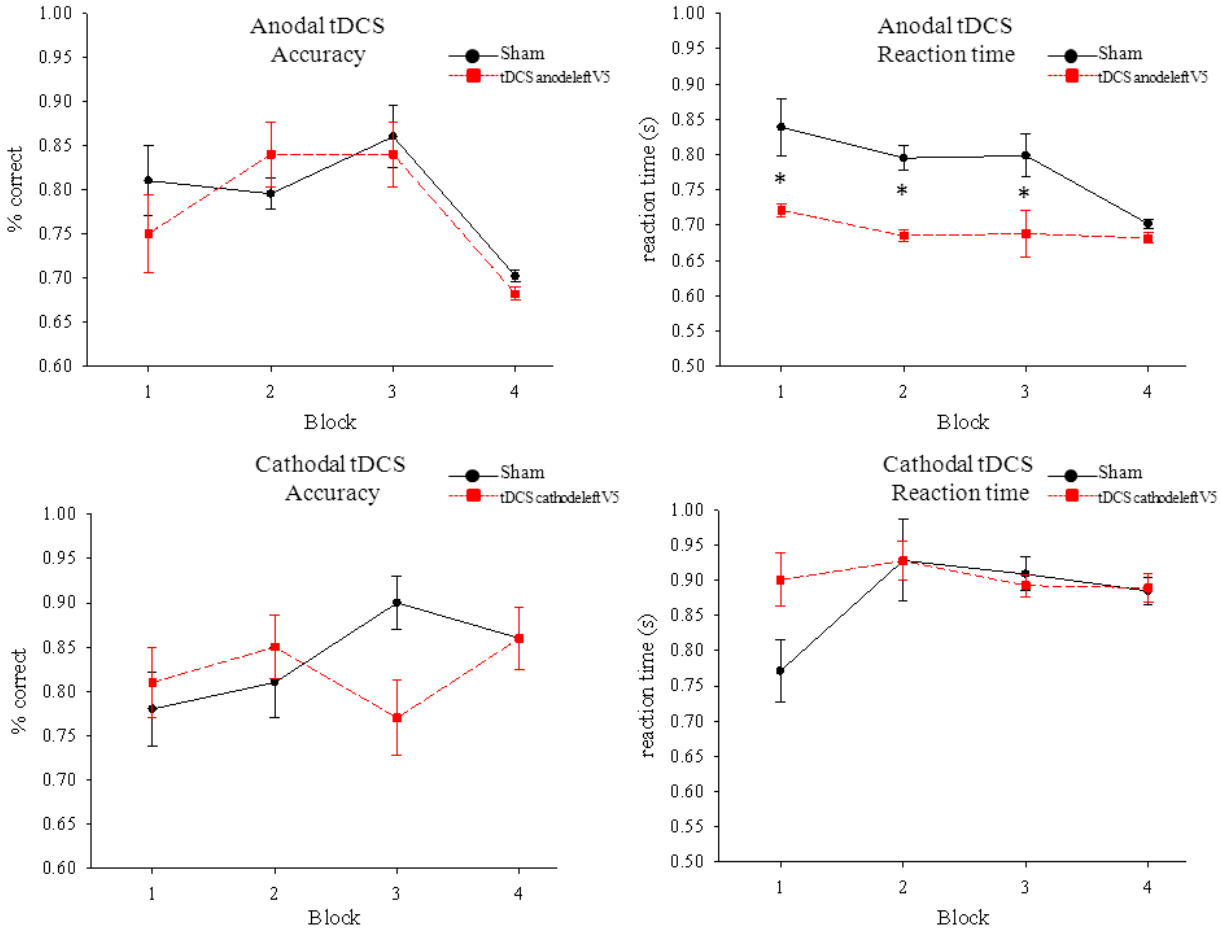


Figure 3: Accuracy and reaction time scores in the sham and tDCS conditions. Error bars represent the standard error of the mean. * denotes $p < 0.05$

As shown in Figure 3a & c, there was no significant main effect of tDCS on accuracy scores when either anodal or cathodal tDCS was administered despite accuracy being lower with cathodal tDCS in block 3. With respect to reaction times, there was a main effect of anodal stimulation ($F_{(1,16)} = 9.407$, $p = 0.007$) but no main effect for block ($F_{(3,48)} = 0.068$, $p = 0.436$) or interaction between stimulation or block ($F_{(3,48)} = 0.090$, $p = 0.290$). Post-hoc pairwise Bonferroni tests revealed that anodal stimulation significantly decreased reaction times in blocks 1 to 3 but not 4 (Block 1: $t = 2.425$, $df = 61$, $p = 0.018$; Block 2: $t = 5.840$, $df = 70$, $p = 0.000$; Block 3: $t = 2.323$, $df = 73$, $p = 0.023$; Block 4: $t = 1.701$, $df = 63$, $p = 0.094$), see Figure 3b.

In the cathodal condition, there was no main effect of stimulation ($F_{(1,15)} = 0.497$, $p = 0.492$) or block ($F_{(3,45)} = 0.657$, $p = 0.583$) and no interaction between stimulation and block ($F_{(3,45)} = 0.014$, $p = 0.934$) on reaction times, see Figure 3d.

## Discussion

Despite abundant evidence that anodal tDCS increases the excitability of cortical neurons, it had no effect on the quality of what GY experienced when a high contrast moving grating was presented in his blind hemifield. When low contrast stimuli were presented, GY was pre-titrated to 60-70% accuracy respectively. After every block of 100 trials he insisted that he never experienced a phenomenal visual percept when he "saw" a visual moving stimulus. Yet his ability to discriminate between the target and blank trials was almost perfect when high contrast visual moving stimuli were presented across 1200 trials and still excellent even when the contrast was adjusted downwards to make the task more difficult with performance stable at 70-80% (see Cowey 2010). In attempting to explain this paradox, which lies at the heart of blindsight and with which GY is familiar, he said that with the high contrast moving grating he knew that it had been presented because he experienced a feeling that something happened and was confident that his judgement was correct but that he did not see anything. In other words he was displaying Type-2 blindsight (as opposed to Type-1 blindsight in which there is no awareness whatsoever).

How then can one account for previous reports that he experiences something like degraded normal vision (e.g. Zeki and ffytche, 1998; Barbur et al, 1993; Stoerig and Barth, 2001)? The likeliest explanation, which GY said was correct and agreed to have his confirmation published, is that since he knows the detectable targets are visual he uses the word 'see' when describing his experiences, just as people without eyes nonetheless use the word see in this metaphorical sense. For example, he said "In my head I do see it move, but what do I see? If there is nothing to see, no shape, no texture, no colour etc., how can I see 'nothing' move? Strange concept but is it possible to see movement itself? In aware mode I KNOW there is movement but there is no awareness of anything there to move, bit like trying to explain colour to a blind person." It is even possible that since GY knows what the target looks like in his seeing field, top-down processing induces its mental image when he detects it in his blind field. Doubtless this debate will continue but this simple explanation has long existed, for example "Looking on darkness

which the blind do see/ Save that my soul's imaginary sight/ Presents their shadow to my sightless view," Shakespeare, Sonnet 27). GY has imaginary sight.

There was also no effect of tDCS on the accuracy of his performance in trials in which he was not initially at ceiling. However, there is evidence that phenomenological awareness of moving stimuli can be induced in GY. GY has reported TMS-induced visual phosphenes in his blind hemifield within a few degrees of the vertical meridian but beyond his macular sparing (Silvanto et al, 2007; 2009). But these phosphenes occurred only when TMS was applied over MT/V5 of both hemispheres or over MT/V5 on the left and V1 on the right. Their existence almost certainly reflects the abnormal hypertrophic connexions between MT/V5 of the blind hemisphere and the normal hemispheres and/or the enhanced connexions between the pulvinar, superior colliculus and amygdala, with their subsequent connexions with cortex, in GY's blind hemisphere as revealed by diffusion tensor imaging (Bridge et al, 2008; Tamietto et al, 2012). Such changes in connectivity may underlie GY's totally non-conscious performance (blindsight) and also contribute to his somewhat emotional "feeling" that something appeared in his blind field. However, increasing the excitability of one node on this network (left MT/V5) is not sufficient to induce a phenomenological awareness with respect to external visual stimuli or indeed make him better at their detection in a non-ceiling task.

Anodal tDCS did, however, speed GY's detection in trials in which he correctly responded to moving stimuli in his blind field with respect to a sham stimulation condition. Sham stimulation is accepted in the literature to be an appropriate control in tDCS of the strength used in this experiment due to the naivety of the subject to the condition type and has recently been used as a control in clinical studies (e.g. Blumberger et al., 2012; Wu et al., 2012). Reaction times to the anodal condition were also significantly faster than those in the cathodal condition, further evidence that the results seen were not due to non-specific effects of stimulation. Reaction times were significantly faster in blocks one through three. The lack of effect in block four may be related to two equally possible factors, namely a) as block four occurred 20 minutes post tDCS onset, tDCS was no longer affecting neuronal processing and b) reaction times in the sham condition had improved by block four such that tDCS could not improve the measure any further. The faster reaction times seen in the earlier blocks may be related to the increased gamma activity on trials when he reports awareness in a difficult visual discrimination task (Schurger et al. 2006, 2008) even though accuracy, i.e. percentage correct, was no different in the aware and unaware conditions. In the 2006 study, where reaction times were measured GY

responded faster on trials where he was aware than unaware, even though accuracy was no different. If anodal tDCS potentiates gamma band activity in MT/V5, which is unknown, it may speed response time under this condition. However tDCS as applied here is not sufficient to engage these neurons, for if it did we would expect anodal tDCS to affect GY's awareness as evidenced by his accuracy. A further puzzle is why cathodal tDCS did not have the opposite effect, i.e. to lengthen reaction times. One way of investigating this would be to make the task even more difficult, either by reducing baseline performance to, say, 65% correct by reducing contrast even further or by shortening the presentation time. But the simplest explanation is that cathodal stimulation scarcely changes the resting discharge rate of visual cortical neurons, especially if that rate is already low in area MT/V5 deprived of its major input from V1 thus leading to a lack of functional effect.

Therefore, whatever tDCS does to cortical neurons it did not, in the present experiment, facilitate accuracy in blindsight. Due to the known action of tDCS (increasing the likelihood neurons under the anodal electrode will fire) a decrease of threshold and quicker activity of neurons in left MT/V5 should increase activity in the network GY uses for non-visual awareness. Such changes in network activity have been reported elsewhere between anatomically connected parietal and motor cortices and motor regions (for example Feurra et al., 2011; Boros et al., 2008). However, as already discussed, such activity changes are not sufficient to bring about a functional awareness, but can potentiate faster reaction times. The functional contribution of GY's demonstrated unusual connectivity could be tested by applying anodal tDCS to either right MT/V5 or right V1 to examine the possibility that increasing neuronal excitability here can affect performance.

It is possible that the reason why activity changes at left V5 are not sufficient to provide phenomenal visual awareness is because consciousness may be a two-step feedforward/feedback process. Feedback connections from V5 to V1 are required for conscious perception in the undamaged brain as was demonstrated using transcranial magnetic stimulation (Pascual-Leone & Walsh, 2001). However, a recent MEG study has shown that despite the absence of his left V1, GY does display feedback activity, albeit abnormally, but does not have any initial feedforward activity from V1 to V5 (Ioannides et al., 2012). It may therefore be that the feedforward activity, absent in GY, is necessary for conscious awareness. This seems intuitive as if V5 is receiving abnormal information (i.e. not from V1) then feedback activity (required for consciousness in the normal brain) cannot engender awareness. Our intervention with tDCS

provides one important fact to the argument. Activity at V5 may determine the speed of correct responses, but not the number of correct responses.

In conclusion the effect of tDCS on GY's blindsight performance is limited to reaction time, which may be mediated by his reorganised cortical connectivity and/or oscillatory condition.

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