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DOI: 10.1523/JNEUROSCI.2282-16.2016

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Document Version
Publisher's PDF, also known as Version of record

Citation for published version (Harvard):

Link to publication on Research at Birmingham portal

Publisher Rights Statement:
Article published as above and available online at: http://dx.doi.org/10.1523/JNEUROSCI.2282-16.2016

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Contributions of the Ventral Striatum to Conscious Perception: An Intracranial EEG Study of the Attentional Blink

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The brain is limited in its capacity to consciously process information, necessitating gating of information. While conscious perception is robustly associated with sustained, recurrent interactions between widespread cortical regions, subcortical regions, including the striatum, influence cortical activity. Here, we examined whether the ventral striatum, given its ability to modulate cortical information flow, contributes to conscious perception. Using intracranial EEG, we recorded ventral striatum activity while 7 patients performed an attentional blink task in which they had to detect two targets (T1 and T2) in a stream of distractors. Typically, when T2 follows T1 within 100–500 ms, it is often not perceived (i.e., the attentional blink). We found that conscious T2 perception was influenced and signaled by ventral striatal activity. Specifically, the failure to perceive T2 was foreshadowed by a T1-induced increase in α and low β oscillatory activity as early as 80 ms after T1, indicating that the attentional blink to T2 may be due to very early T1-driven attentional capture. Moreover, only consciously perceived targets were associated with an increase in θ activity between 200 and 400 ms. These unique findings shed new light on the mechanisms that give rise to the attentional blink by revealing that conscious target perception may be determined by T1 processing at a much earlier processing stage than traditionally believed. More generally, they indicate that ventral striatum activity may contribute to conscious perception, presumably by gating cortical information flow.

Key words: attentional blink; consciousness; intracranial EEG; oscillations; perception; striatum

Significance Statement

What determines whether we become aware of a piece of information or not? Conscious access has been robustly associated with activity within a distributed network of cortical regions. Using intracranial electrophysiological recordings during an attentional blink task, we tested the idea that the ventral striatum, because of its ability to modulate cortical information flow, may contribute to conscious perception. We find that conscious perception is influenced and signaled by ventral striatal activity. Short-latency (80–140 ms) striatal responses to a first target determined conscious perception of a second target. Moreover, conscious perception of the second target was signaled by longer-latency (200–400 ms) striatal activity. These results suggest that the ventral striatum may be part of a subcortical network that influences conscious experience.

Introduction

A fundamental question in the study of conscious perception relates to the neural basis of conscious access: what brain mechanisms determine whether a stimulus becomes available for explicit report? Research shows that only consciously perceived stimuli are associated with sustained, synchronized activity

The authors have the following interests: P.R.S. is an independent consultant for Medtronic Inc. on educational matters and received travel grants from the company. D.D. has received occasional fees from Medtronic for educational purposes and has served as a consultant for Lundbeck. The DBS intervention was supported by an unrestricted investigator-initiated research grant by Medtronic Inc. There are no patents, products in development or marketed products to declare.

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DOI:10.1523/JNEUROSCI.2282-16.2016

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within a distributed network of cortical regions (Rees et al., 2002; Haynes, 2009; Dehaene and Changeux, 2011; Lau and Rosenthal, 2011; Aru et al., 2012; van Gaal and Lamme, 2012). Based on this work, influential theories propose that conscious access stems from a cognitive architecture with an evolved function: the flexible sharing of information throughout the cortex (Baars, 1993; Dehaene and Naccache, 2001). Yet, the cortex operates in close interaction with subcortical regions, specifically the thalamus and the basal ganglia. The thalamus has rich and widespread reciprocal connections to the cortex and thus can synchronize activity across distant cortical regions. Several theories propose that this vast thalamocortical architecture may shape the boundary conditions for both the level of wakefulness (Vijayan and Kopell, 2012; MacDonald et al., 2015) as well as conscious perception to occur (Newman and Baars, 1993; Crick, 1995; Tononi and Edelman, 1998; Baars, 2005; Dehaene and Changeux, 2005). Notably, thalamocortical interactions are modulated by the basal ganglia, which tonically inhibit the thalamus and thereby cortical activation. This raises the possibility that the basal ganglia may also contribute to conscious perception, through their influence on activity within the network of frontoparietal regions that gives rise to conscious experience.

The basal ganglia are connected to many frontal regions in parallel loops through the thalamus, and hence capable of modulating a wide range of associated processes. Loops connecting the basal ganglia to motor cortex have long been implicated in action selection. Notably, converging evidence indicates that these parallel loops connecting the basal ganglia and frontal cortex may serve a more generic “selection” function and may resolve conflicts not only in the motor domain, but also among cognitive resources (Redgrave et al., 1999a). Evidence from animal studies suggests that the striatum can act as an early gating system that enables the prioritization of salient stimuli by triggering frontal systems to orient attention (Redgrave and Gurney, 2006; Overton et al., 2014). Moreover, the striatum receives longer-latency input from the hippocampus, which may provide longer-duration, context-dependent gating of prefrontal activity (Newman and Grace, 1999). By gating cortical information flow, the striatum could hence be part of a subcortical network that provides a “gateway” to conscious experience, for example, by biasing attention (van Schouwenburg et al., 2015), and guide which information is selected for global broadcasting and conscious access.

Although the striatum is perfectly situated to influence conscious information processing, little is currently known about its exact contribution to conscious perception. This gap in knowledge is related to our inability to record subcortical activity with high temporal precision in healthy humans and difficulties in studying consciousness in animals. Human neuroimaging studies have provided some support for a role for the striatum and its irradiation by dopamine in conscious perception (Slagter et al., 2010, 2012; Van Opstal et al., 2014; Bisenuis et al., 2015). For example, a recent PET study (Slagter et al., 2012) revealed an association between striatal dopamine D2 receptor binding and the attentional blink (AB): a deficit in consciously perceiving the second target of two targets (T1 and T2) whenever it follows T1 within 100–500 ms in a rapid stream of distractors. Yet, these neuroimaging methods lack temporal precision and provide indirect measures of neural activity, leaving the specific contribution of the striatum to conscious perception unclear.

To shed light on the potential role of the striatum in conscious perception, we used the unique opportunity to directly record electrophysiological activity from the ventral striatum in patients while they performed an AB task (Raymond et al., 1992). This task has two unique features. First, it has proven useful for studying the neural correlates of conscious perception by comparing the neural processing of T2 between T2-seen versus T2-unseen trials. Second, it allows investigation of the conditions that are necessary for a stimulus to reach awareness by revealing how neural processing of T1 affects the ability to consciously perceive T2 (Dux and Marois, 2009; Martens and Wyble, 2010). By linking conscious perception to real-time basal ganglia measurements, we aimed to elucidate the role of the ventral striatum in conscious perception.

Materials and Methods

Participants. Eight therapy-resistant patients eligible for deep brain stimulation (DBS) participated in the study. One patient had difficulty seeing any target, and the experiment had to be aborted prematurely. Of the 7 remaining patients (mean age: 39.5 years; age range: 22–63 years), 6 were female. Five patients had a diagnosis of obsessive-compulsive disorder, one patient had a diagnosis of major depressive disorder, and another patient had a drug addiction. They participated in the experiment 1 d (1 patient) or 3 d (6 patients) after undergoing DBS lead placement surgery and before DBS stimulator implantation. All the patients underwent careful screening before being included for DBS as described by Denys et al. (2010). Patients were implanted following standard procedures with a four contact electrode (model 3387 with contact points 1.5 mm long and separated from adjacent contacts by 1.5 mm; Medtronic) in each hemisphere. The surgical technique has been described previously (Denys et al., 2010; van den Munckhof et al., 2013). The electrodes were implanted following the anterior limb of the internal capsule (ALIC), with the deepest contact targeted at the core of the nucleus accumbens and the three upper contacts in the ventral ALIC (Fig. 1). DBS leads were externalized at the back of the head based on individual anatomy to facilitate the
recordings. Correct stereotactic position of the DBS leads was verified with postoperative stereotactic CT. During the subsequent recordings, patients received normal medical treatment in addition to antibiotics and analgesics when requested or deemed medically necessary. However, to reduce the risk of brain hemorrhage, any administration of selective serotonin response inhibitors was suspended. This study was approved by the Institutional Review Board of the Academic Medical Center of the University of Amsterdam.

**Task and stimuli.** Stimulus presentation was performed using Presentation (version 14.3; Neurobehavioural Systems) and a laptop (Hewlett Packard 6730b) with a 15.4 inch display at a resolution of 1024 × 768 pixels (refresh rate of 60 Hz). The distance from the screen to the participants was kept at ~60 cm. Participants performed a standard AB task in which they had to identify two digits (T1 and T2) presented in a rapid stream. Stimulus presentation was performed using Presentation (version 14.3; Neurobehavioural Systems) and a cluster size of 25 time-frequency pixels. In case of a significant t-value corresponding to a z-value of 0.05 as the threshold for significance and a cluster size of 25 time-frequency pixels. In case of a significant effect, post hoc analyses were conducted to determine whether effects were driven by changes in T2-seen or T2-unseen trials by comparing mean activity in the significant time-frequency cluster in T2-seen (or T2-unseen) trials to mean baseline activity in a corresponding frequency range in T2-seen (or T2-unseen) trials. **Results.** Patients displayed a robust AB: they more often failed to perceive T2 when it followed T1 after 200 ms than after 800 ms (Fig. 2; t(6) = 4.0, p = 0.007). Comparable with previous studies with healthy adults, T1 accuracy was high (on average, 85%) regardless of the interval between the two targets (t(6) = 0.04, p = 0.972).

To examine the role of the ventral striatum in the AB, our primary analysis focused on differences in target-induced oscillatory activity (or power) between T2-seen (no-blink) and T2-unseen (blink) trials. Time-frequency representations of power and phase were calculated for each trial using sliding Hanning tapers with an adaptive time window of two cycles for each frequency (1–30 Hz in logarithmically spaced steps). This approach has been used often in previous studies (e.g., Mazaheri et al., 2009). Time-frequency representations were computed separately for each condition (blink, no-blink), interval, (short, long), contact point (left, right), and participant, and baseline corrected using the time window 0–500 ms before T1. **Statistics.** Given the relatively small number of patients, we used a nonparametric permutation procedure used in previous studies to assess statistical significance of the time-frequency data (Cohen et al., 2009). For the time-frequency data, we first randomly relabeled T2-unseen and T2-seen trials and created 1000 permuted difference maps of power. Next, at each time-frequency point, the true observed difference between T2-unseen and T2-seen trials was subtracted from the mean permuted difference and divided by the SD of the permuted differences to obtain a z-value. This z value reflects the standard deviation away from the distribution of time-frequency power expected by chance. We chose a z value corresponding to a p value of 0.05 as the threshold for significance and a cluster size of 25 time-frequency pixels. In case of a significant effect, post hoc analyses were conducted to determine whether effects were driven by changes in T2-seen or T2-unseen trials by comparing mean activity in the significant time-frequency cluster in T2-seen (or T2-unseen) trials to mean baseline activity in a corresponding frequency range in T2-seen (or T2-unseen) trials. **Results.** Patients displayed a robust AB: they more often failed to perceive T2 when it followed T1 after 200 ms than after 800 ms (Fig. 2; t(6) = 4.0, p = 0.007). Comparable with previous studies with healthy adults, T1 accuracy was high (on average, 85%) regardless of the interval between the two targets (t(6) = 0.04, p = 0.972).
missed, did T1 induce this short-latency increase in \( \alpha \)-/low \( \beta \) activity (Figs. 3B, 4). Post hoc analyses confirmed that this early response was only induced by T1 in T2-unseen trials \( (t_{(6)} = 2.75, p = 0.055) \), not in T2-seen trials \( (t_{(6)} = 0.55, p = 0.59) \). This effect was not observed for the right ventral striatum.

Second, conscious T2 perception was associated with an increase in \( \theta \) (4–8 Hz) oscillatory activity between \( \sim 150 \) and 400 ms in both the left and right ventral striatum (Fig. 3). For the left ventral striatum, it can clearly be seen that when both targets were consciously perceived (T2-seen trials), this increase in \( \theta \) activity was observed between 200 and 400 ms after each target onset (Fig. 4B). However, when only T1 was seen (T2-unseen trials), only the first increase in \( \theta \) activity was present (Fig. 4A). Of further note, when T2 followed T1 after a long interval, the second increase in \( \theta \) activity was correspondingly shifted in time (Fig. 4C), confirming that the second increase in \( \theta \) power observed in short-interval T2-seen trials is related to (conscious) T2 processing. The difference in T2-induced \( \theta \) power between T2-seen and T2-unseen trials was significant between 215 and 400 ms post-T2 in the left ventral striatum and between 150 and 280 ms in the right ventral striatum (Fig. 3). Post hoc analyses confirmed that the \( \theta \) response to T2 was only significantly increased from baseline in the left ventral striatum in trials in which T2 was seen \( (t_{(6)} = 2.64, p = 0.05) \), and not in trials in which T2 was not seen \( (t_{(6)} = 1.05, p = 0.33) \) (Fig. 3B). However, in the right ventral striatum, the increase in \( \theta \) activity to T2 was not significantly different from baseline in T2-seen trials \( (t_{(6)} = 1.87, p = 0.1) \), indicating that the effect was less robust in the right hemisphere. Thus, only consciously perceived T2’s were signaled by \( \theta \) oscillatory activity in the ventral striatum. This effect was also visible at the two most dorsal contact points (3 and 4) in the left ventral ALIC (data not shown).

Last, conscious T2 perception was associated with a greater increase in \( \beta \)-band (15–30 Hz) activity in T2-seen versus T2-unseen trials between 210 and 260 ms after T2 in the right ventral striatum (Fig. 3). This \( \beta \) increase was not significantly increased from baseline in either condition (T2-seen trials: \( t_{(6)} = 1.84, p = 0.2 \); T2-unseen trials: \( t_{(6)} = 0.18, p = 0.86 \); Figs. 3B, 4).

Thus, the AB was associated with differences in both T1 and T2 processing; T1 processing influenced the ability to consciously perceive T2, as indicated both by stronger \( \alpha / \beta \) activity between 80 and 140 ms after T1 in the left ventral striatum. Moreover, only perceived T2’s elicited a \( \theta \) response between 150–280 ms in the right ventral striatum and 200–400 ms in the left ventral striatum, and a transient increase in \( \beta \) activity between 210 and 260 ms in the right ventral striatum.

As T1 could have one of two colors (red or green), we ran a control analysis to ensure that observed differences in T1 processing between T2-seen and T2-unseen trials could not be accounted for by differences in the relative contribution of red T1 versus green T1 trials between trial types. Specifically, we ran an additional repeated-measures ANOVA with T1 color and interval (short vs long) as within-subject variables. This analysis importantly showed that T2 detection was not affected by T1 color (no main effect of T1 color: \( F_{(1,6)} = 0.012; p = 0.915 \)), nor was AB size (no interaction between T1 color and Interval: \( F_{(1,6)} = 0.336; p = 0.583 \)), excluding this possibility.

**Discussion**

This study aimed to shed more light on the role of the ventral striatum in conscious perception. We found that conscious T2 perception was not only reflected in, but also influenced by, ventral striatal activity in that responses to T1 foreshadowed the AB to T2. Specifically, only in T2-unseen trials, T1 elicited a short-latency (80–140 ms) increase in \( \alpha \) and lower \( \beta \) activity (8–16 Hz) in the left ventral striatum. This novel finding suggests that the AB to T2 is determined by T1 processing at a much earlier processing stage than commonly assumed (Dux and Marois, 2009; Martens and Wyble, 2010). Our second main finding was that only consciously perceived T2s were associated with an increase in \( \theta \) activity between 215–400 ms and 150–280 ms in the left and right ventral striatum, respectively, as well as with transient \( \beta \)-band activity between 210 and 260 ms in the right ventral striatum. Thus, we also observed signals related to conscious experience in the ventral striatum before, or in the time range in which the global network of frontoparietal regions implicated in consciousness is activated (Sergent et al., 2005; Dehaene and Changeux, 2011). Together, these findings suggest that the ventral striatum may contribute to conscious perception and provide first insight into the time course of its contributions. They may also shed novel light on the mechanisms that give rise to one of the most studied phenomena in the consciousness literature: the attentional blink.

Strikingly, the AB was associated with differences in T1 processing as early as 80 ms after T1 (i.e., much earlier than previously shown with scalp-EEG) (Sergent et al., 2005; Slagter et al., 2007). Specifically, this work has shown that in trials in which T2 goes undetected, the T1-elicited P3b, a brain potential with a latency of \( \sim 300–400 \) ms, is delayed or larger in amplitude (Sergent et al., 2005; Slagter et al., 2007). As the P3b has been linked to several, albeit related, cognitive functions, including working memory updating (Donchin, 1981), event categorization (Kok, 2001), and decision making (Twomey et al., 2015), these findings have been taken as support for major AB theories that propose that T1 encoding renders some mechanism unavailable for T2 processing until T1 encoding is completed and, thus, that the AB is related to some later-stage information processing bottleneck (Duncan et al., 1994; Chun and Potter, 1995; Bowman and Wyble, 2007; for reviews, see Dux and Marois, 2009; Martens and Wyble, 2010; Marti et al., 2015). Yet, our findings relate the AB to differences in T1 processing starting at 80 ms. This could suggest that T1 consolidation starts much earlier, as soon as sufficient evidence has been collected. Alternatively, the AB may be affected by T1 processing at a much earlier processing stage. Notably, animal work has shown that the ventral striatum responds to salient stimuli within 100 ms. This response is thought to reflect a
signal to frontal regions to orient attention to potentially relevant information for further processing (Redgrave et al., 1999b; Horvitz, 2000; Overton et al., 2014). Although this warrants further research, the short-latency increase in \(\alpha/\beta\) activity may thus reflect an early alerting signal triggered by a salient stimulus, the strength of which determines the extent of higher-order stimulus processing, and ability of the brain to pick up on a subsequently presented target stimulus (Slagter et al., 2009). This possibility receives initial support from a study in which early evoked activity (80–180 ms) in the nucleus accumbens related to deviancy detection predicted the amplitude of the scalp P3b (Dürschmid et al., 2016). Yet, future studies that manipulate, for example, T1 saliency are necessary to draw firm conclusions regarding the functional significance of our early \(\alpha/\beta\) effect.

Intriguingly, the short-latency difference in T1 processing was observed in the ventral striatum. This raises the question how the ventral striatum, which does not directly receive visual input, can in some trials “know” so quickly after T1, which differed from the distractors in color and shape/identity, that a salient stimulus has been presented. Animal research shows that short-latency visual in-
formation is provided by the superior colliculus to dopaminergic neurons, which in turn project to the striatum (Overton et al., 2014). Yet, the superior colliculus is relatively primitive and allegedly not capable of distinguishing between colors (McPeek and Keller, 2002) or complex visual stimuli (Schiller and Koerner, 1971; Goldberg and Wurtz, 1972), making it unlikely that the here observed short-latency response reflects input from this region. At a slightly longer latency (after $\sim 150$ ms), the ventral striatum can receive the results.
of complex stimulus processing via a recently discovered route by which cortical areas project to dopaminergic neurons via the superior colliculus (Overton et al., 2014). Yet, the α/β effect occurred earlier. Although the ventral striatum also receives visual information via the thalamus, this is primarily from higher-order thalamic nuclei, which receive little direct sensory input. Therefore, our findings, which indicate very short-latency information processing in the ventral striatum dependent on nonspatial visual features, cannot easily be explained by existing ideas about subcortical visual processing and warrant further research. Moreover, the fact that the early α/β effect was only triggered by T1 in T2-unseen trials further suggests that it does not simply reflect sensory T1 processing. This observation also raises the question as to why a physically identical T1 only elicited this response in T2-unseen, but not T2-seen, trials. Several studies have shown that differences in attentional state can influence T1 processing and AB magnitude (Olivers and Nieuwenhuis, 2005; Slagter et al., 2007; van Vugt and Slagter, 2014), raising the possibility that trial-to-trial fluctuations in attentional state may have contributed to observed effects. Notably, α and β oscillations have been specifically related to top-down, feedback-related processing (Bastos et al., 2015). Thus, although speculative, the extent to which T1 captured attention or placed demands on consolidation processes may have depended on pretarget attentional state. Alternatively, differences in bottom-up T1 strength across trials, caused by variability across trials in the specific target-distracter sequence and hence distractor masking, may have also influenced T1 processing (Bowman and Wyble, 2007). Future studies that manipulate attentional state and T1 saliency are necessary to determine the functional significance of our early α/β effect. Nevertheless, our findings indicate that the AB is related to T1 processing at a much earlier processing stage than is commonly assumed, at the subcortical level. They may also extend recent findings that indicate that, although visual consciousness and endogenous (top-down) attention may be neurally dissociated (Koch and Tsitschewa, 2007; Wyart and Tallon-Baudry, 2008), exogenous spatial attention (Chica and Bartolomeo, 2012) and phasic alertness (Kusnir et al., 2011) are important antecedents of conscious experience and facilitate conscious access by calling upon frontoparietal networks to orient attention.

Importantly, striatal responses to T1 not only prevented conscious access to T2, conscious T2 perception was also signaled by striatal activity in the θ band. This is notable as most theories of consciousness focus exclusively on the cortex and/or thalamocortical interactions (Rees et al., 2002; Haynes, 2009; Dehaene and Changeux, 2011; Lau and Rosenthal, 2011; Ari et al., 2012; van Gaal and Lamme, 2012), and are agnostic on striatal contributions. θ oscillations are associated with the active intake of sensory stimuli (Bastos et al., 2015), but are also observed in structures further down the processing stream, including the hippocampus (Colgin, 2013). Hippocampal-generated θ oscillations are key in setting the dynamics for memory encoding and retrieval within cortical circuits (Hasselmo and Stern, 2014). As the ventral striatum allegedly does not generate θ oscillations itself (van der Meer and Redish, 2011) and intracranial EEG measures local field potentials (i.e., neuronal input), the observed T2 detection-related θ response may thus reflect hippocampal-dependent processes. The nucleus accumbens, which also receives inputs from the amygdala, prefrontal cortex, and the ventral tegmental area, projects to both the mediodorsal nucleus of the thalamus and the thalamic nucleus reticularis, which in turn project to the prefrontal cortex. The nucleus accumbens is thus well positioned to integrate information from different sources and select which information is broadcasted through the brain (via the thalamic nucleus reticularis) (Scheibel, 1980) or selected for sustained cortical representation (via the mediodorsal nucleus of the thalamus) (Goto and Grace, 2008). This is significant as several influential theories propose that consciousness is related to the “broadcasting” of sensory information to the whole brain and that thalamocortical circuits serve as an important mediator of such broadcasting (Newman and Baars, 1993; Crick, 1995; Tononi and Edelman, 1998; Baars, 2005; Dehaene and Changeux, 2005, 2011). Future studies are necessary to determine how the striatum may precisely influence thalamocortical interactions and conscious perception.

As for the T2 perception-related increase in β activity, a recent iEEG study reported reduced β-band activity in the ventral striatum at a similar latency to rare versus frequent scene images (Zaehle et al., 2013), suggesting that β-band activity may signal contextual deviancy. Yet, we observed an increase in β activity to perceived T2s. This discrepancy in findings might relate to differences in the specific task or reference procedure used.

Our results extend findings from neuroimaging studies (Christensen et al., 2006; Slagter et al., 2010, 2012; Van Opstal et al., 2014; Bisenius et al., 2015; Chica et al., 2016) by revealing the time course of conscious perception-related striatal activity. Importantly, some of these studies used simple backward masking tasks with only one stimulus (Christensen et al., 2006; Van Opstal et al., 2014; Bisenius et al., 2015), in which conscious access is not dependent on attentional selection (Dehaene and Changeux, 2011) as in the AB task. Yet, many fMRI studies did not report conscious-related activity in the striatum. Possibly, fMRI does not provide a sensitive measure of consciousness-related activity in the basal ganglia due to the nature of the BOLD response; BOLD activity mostly reflects local field potential activity between 20 and 60 Hz (Goense and Logothetis, 2008) and correlates negatively with α power (Laufs et al., 2003; Scheeringa et al., 2011). Low-frequency oscillatory activity, as observed here, may hence not be reflected in increased BOLD activity or even result in decreased BOLD activity. Combined with the fact that basal ganglia activity cannot be measured with scalp-EEG (Cohen et al., 2011), this might explain the exclusion of a striatal contribution in many theories of consciousness. Yet, the striatum is well positioned to gate cortical information flow and integrate the massively parallel and distributed information capacity of the cerebral cortex into the limited-capacity, sequential mode of operation required to form a coherent percept of our environment. Albeit speculative, our observations may provide initial support for this idea. A role for striatum-dependent gating mechanisms in the AB fits with influential theories that attribute the AB to dysfunctional gating of information (Di Lollo et al., 2005; Bowman and Wyble, 2007; Olivers and Meeter, 2008). For example, in the episodic simultaneous type serial token model (Bowman and Wyble, 2007), target perception critically relies on the capacity to rapidly detect that a stimulus is salient when a fleeting representation of it arises among (temporally) competing stimuli, and the ability to sustain this representation through transient attentional enhancement for memory encoding. In this model, ongoing consolidation of T1 has an inhibitory effect on T2 attentional selection, resulting in impaired T2 detection. According to another influential theory (Olivers and Meeter, 2008), the AB reflects the workings of a rapidly responding gating system. Specifically, T1 elicits transient excitatory feedback activity within 100 ms meant to provide access to working memory. However, accidentally, the subsequent post-T1 distracter is “boosted,” resulting in a strong inhibitory feedback response, which closes the gate to working memory for T2. This theory actually postulated a possible role for the basal ganglia in the AB.

Although behaviorally our mixed-patient data are very similar to typical findings in healthy adults, the question remains how
our findings translate to the healthy human brain. Answering this question requires the development of methods that can noninvasively measure subcortical activity with high temporal precision in healthy humans.

In conclusion, our results shed new light on the mechanisms that give rise to the AB, by revealing that the AB may be due to very early T1-driven attentional capture. More generally, by showing that conscious perception is modulated by ventral striatal activity, they suggest that the neural mechanisms underlying conscious access may not be confined to the thalamicocortical complex alone.

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