

Ventral striatum

The ventral striatum is a portion of the [striatum](#).

It consists of the [nucleus accumbens](#) and the [olfactory tubercle](#).

Some sources also include the ventromedial parts of the [caudate nucleus](#) and [putamen](#).

It is considered a [reward](#) center.

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.

Slagter et al., examined if the ventral striatum, given its ability to modulate cortical information flow, contributes to conscious perception. Using intracranial EEG, they 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 100-500ms, it is often not perceived (i.e., the attentional blink).

They 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 alpha and low beta oscillatory activity as early as 80ms post-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 theta activity between 200-400ms. 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 ¹⁾.

[Deep brain stimulation](#) (DBS) of the ventral capsule/ventral striatum (VC/VS) region has shown promise as a neurosurgical intervention for adults with severe treatment-refractory [obsessive compulsive disorder](#) (OCD). Pilot studies have revealed improvement in obsessive-compulsive symptoms and secondary outcomes following DBS. Fayad et al sought to establish the long-term safety and effectiveness of DBS of the VC/VS for adults with OCD.

A long term follow-up study (73-112 months) was conducted on the six patients who were enrolled in the original National Institute of Mental Health pilot study of DBS for OCD. Qualitative and quantitative data were collected.

Reduction in OCD symptoms mirrored the one-year follow-up data. The same four participants who were treatment responders after one year of treatment showed a consistent OCD response (greater than 35% reduction in Yale Brown Obsessive Compulsive Scale (YBOCS)). Another subject, classified as a non-responder, achieved a 26% reduction in YBOCS score at long term follow-up. The only patient who did not achieve a 25% or greater reduction in YBOCS was no longer receiving active DBS

treatment. Secondary outcomes generally matched the one-year follow-up with the exception of depressive symptoms, which significantly increased over the follow-up period. Qualitative feedback indicated that DBS was well tolerated by the subjects.

These data indicate that DBS was safe and conferred a long-term benefit in reduction of obsessive-compulsive symptoms. DBS of the VC/VS region did not reveal a sustained response for comorbid depressive symptoms in patients with a primary diagnosis of OCD ²⁾.

¹⁾

Slagter HA, Mazaheri A, Reteig LC, Smolders R, Figee M, Mantione M, Schuurman PR, Denys D. Contributions of the ventral striatum to conscious perception: An intracranial EEG study of the attentional blink. *J Neurosci*. 2016 Dec 16. pii: 2282-16. [Epub ahead of print] PubMed PMID: 27986925.

²⁾

Fayad SM, Guzick AG, Reid AM, Mason DM, Bertone A, Foote KD, Okun MS, Goodman WK, Ward HE. Six-Nine Year Follow-Up of Deep Brain Stimulation for Obsessive-Compulsive Disorder. *PLoS One*. 2016 Dec 8;11(12):e0167875. doi: 10.1371/journal.pone.0167875. PubMed PMID: 27930748.

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