Subiculum stimulation

Evidence has been provided that the subiculum may play an important role in the generation of seizures.Electrostimulation at this target has been reported to have anticonvulsant effects in kindling and pilocarpine rat models, while in a clinical study of hippocampal deep brain stimulation (DBS), contacts closest to the subiculum were associated with a better anticonvulsive effect.

Vázquez-Barrón et al. from the Unit of Functional Neurosurgery, Stereotaxy and Radiosurgery, General Hospital of Mexico City, evaluated the effect of Electrostimulation of the subiculum in patients with refractory mesial temporal lobe epilepsy (MTLE) who have hippocampal sclerosis (HS).

Six patients with refractory MTLE and HS, who had focal impaired-awareness seizures (FIAS) and focal to bilateral tonic-clonic seizures (FBTCS), had DBS electrodes implanted in the subiculum. During the first month after implantation, all patients were OFF stimulation, then they all completed an open-label follow-up of 24 months ON stimulation. DBS parameters were set at 3 V, 450 µs, 130 Hz, cycling stimulation 1 min ON, 4 min OFF.

There was a mean reduction of 49.16% (±SD 41.65) in total seizure number (FIAS + FBTCS) and a mean reduction of 67.93% (±SD 33.33) in FBTCS at 24 months. FBTCS decreased significantly with respect to baseline, starting from month 2 ON stimulation.

Subiculum stimulation is effective for focal to bilateral tonic-clonic seizures (FBTCS) reduction in patients with mesial temporal lobe epilepsy (MTLE) and hippocampal sclerosis (HS), suggesting that the subiculum mediates the generalization rather than the genesis of mesial temporal lobe seizures. Better results are observed at longer follow-up times [1].

Glangetas et al. reported that high-frequency stimulation of the vSUB (HFSvSUB) over-activates ventral tegmental area (VTA) dopamine neurons in vivo and triggers long-lasting modifications of synaptic transmission measured ex vivo. This potentiation is caused by NMDA-dependent plastic changes occurring in the bed nucleus of the stria terminalis (BNST). Finally, we report that the modification of the BNST-VTA neural circuits induced by HFSvSUB potentiates locomotor activity induced by a sub-threshold dose of cocaine. This findings unravel a neuronal circuit encoding behavioral effects of cocaine in rats and highlight the importance of adaptive modifications in the BNST, a structure that influences motivated behavior as well as maladaptive behaviors associated with addiction [2].

References

1)
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