

Pallidal Deep Brain Stimulation Side effects

Some possible side effects of pallidal DBS may include:

Infection: Any surgical procedure carries a risk of infection, which can be a serious complication.

Bleeding: There is a small risk of bleeding during or after the surgery.

Device-related complications: The device used for DBS may malfunction or move out of place, which may require additional surgery.

Speech difficulties: Some people may experience difficulty with speech or changes in their voice after the surgery.

Balance problems: DBS may affect a person's balance and coordination, which can lead to falls.

Mood changes: Some people may experience mood changes or develop depression or anxiety after the surgery.

Cognitive changes: In rare cases, pallidal DBS may cause changes in a person's cognitive function, such as memory or attention.

It is important to note that not everyone who undergoes pallidal DBS will experience side effects, and the procedure's benefits may outweigh the risks. It is important to discuss any concerns or questions with a qualified healthcare provider.

In Parkinson's disease, hypokinetic symptoms have been associated with increased beta oscillations (13-30 Hz). We hypothesize that this pattern is symptom-specific, thus accompanying DBS-induced slowness in dystonia.

Methods: In 6 dystonia patients, pallidal rest recordings with a sensing-enabled DBS device were performed and tapping speed was assessed using marker-less pose estimation over 5-time points following cessation of DBS.

Results: After cessation of pallidal stimulation, movement speed increased over time ($P < 0.01$). A linear mixed-effects model revealed that pallidal beta activity explained 77% of the variance in movement speed across patients ($P = 0.01$).

Conclusions: The association between beta oscillations and slowness across disease entities provides further evidence for symptom-specific oscillatory patterns in the motor circuit. Our findings might help DBS therapy improvements, as DBS devices able to adapt to beta oscillations are already commercially available ¹⁾.

[Globus pallidus internus deep brain stimulation](#) induces [tremor](#) in [Parkinson's disease](#): A [paradoxical phenomenon](#) ²⁾.

Findings suggest that STN could be the preferred target for DBS in patients with advanced Parkinson's

disease ³⁾.

The success of deep brain stimulation (DBS) of the internal segment of the globus pallidus (GPi) depends on accurately placing the electrode into the GPi motor territory. Direct targeting can be difficult as GPi laminar borders are not always clearly identifiable on MRI.

Stimulation-induced hypokinetic [gait disorders](#) with [freezing of gait](#) (FOG) have been reported as adverse effects of deep brain stimulation (DBS) of the globus pallidus internus (GPi) in patients with [dystonia](#).

Wolf et al., prospectively performed computerized gait analysis in ten consecutive patients (mean age 57.8+/-14.3 years) with segmental [dystonia](#) but without involvement of lower trunk or legs who were treated with bilateral GPi DBS. Using pressure sensitive insoles, several parameters were measured preoperatively (pre-OP) and at a median of 7 months postoperatively.

The mean step length significantly decreased from 60.0+/-6.9cm pre-OP to 54.3+/-6.4cm with GPi DBS ($p<0.01$). Due to only small changes of walking distance and gait velocity, the cadence correspondingly increased from 105.6+/-9.2 steps/min to 111.3+/-11.4 steps/min ($p<0.05$). More importantly, the variance of several gait parameters significantly decreased postoperatively.

In patients with segmental dystonia, chronic DBS of the posteroventral lateral GPi is associated with only mild hypokinesia of gait, but with a relevant decrease in gait variability. Given other recently reported hypokinetic effects of GPi DBS for dystonia and recent results of electrophysiological coherence studies, these findings support the hypothesis of a general alteration of neuronal activity in striato-pallido-thalamo-cortical motor pathways following chronic stimulation of the posteroventral lateral GPi ⁴⁾.

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