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Bradykinesia induced by pallidal neurostimulation in dystonia: clinical risk factors and anatomical mapping.

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Pallidal deep brain stimulation (GPi-DBS) is a highly effective therapy for dystonia, yet many patients develop stimulation-induced motor slowing that can compromise overall benefit. Across a retrospective cohort of 55 patients, our study shows that bradykinesia is common, present in 60% of cases. Despite the generally mild magnitude of this effect, several consistent clinical predictors emerged. Female sex, older age at dystonia onset, and shorter disease duration were associated with a greater susceptibility to stimulation-induced bradykinesia at the univariate level. Crucially, pulse width was identified as the only independent stimulation parameter predicting bradykinesia severity in multivariate modeling: wider pulse widths significantly increased the risk of motor slowing, whereas amplitude and frequency did not independently contribute to risk. These findings establish pulse width as a modifiable programming factor with direct clinical relevance.

A prospective cohort of 11 dystonia patients allowed us to examine the phenomenon with millisecond-level kinematic precision. Reducing stimulation amplitude by 50% led to consistent improvements in movement frequency during finger tapping and hand opening tasks, while movement amplitude remained unchanged. This dissociation (slowing without hypokinesia) suggests selective disruption of neural circuits governing motor speed rather than amplitude. The pattern is reinforced by correlations between clinical UPDRS bradykinesia ratings and objective measures of speed and frequency, but not amplitude decay. Importantly, connectivity analyses revealed no relationship between bradykinesia and activation of major white-matter tracts, including the pyramidal tract, even when assessed using patient-specific DTI. Instead, the data point to local GPi gray-matter modulation as the primary substrate of stimulation-induced slowing. Together, these results provide convergent evidence for a targeted physiological mechanism rather than a nonspecific spread of stimulation.

Using voxel-wise probabilistic mapping, we identified a discrete “sour spot” in the posterolateral GPi where stimulation significantly increased bradykinesia risk. This region is strikingly segregated from the established dystonia “sweet spot,” indicating near-complete anatomical dissociation. Leave-one-out cross-validation confirmed patient-level predictive power for forecasting bradykinesia severity. These findings enable risk stratification and demonstrate that alternative programming strategies, adjusting contact selection and reducing pulse width, may reduce bradykinesia without compromising dystonia control. Computational simulations further suggest that steering stimulation away from the sour spot could substantially mitigate motor slowing while preserving therapeutic benefit. Clinically, this work reframes stimulation-induced bradykinesia as a predictable and preventable phenomenon and provides an anatomical and programming framework for improving DBS outcomes in dystonia.

Dr. Florian Lange

Florian Lange is an advanced clinician scientist in the visualDBSlab at Universitätsklinikum Würzburg (UKW). He has contributed to the development of quantitative, computer-vision–based assessments of motor function and to data-driven approaches that refine DBS targeting and programming.

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