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Tending the source of parkinsonism through deep brain microstimulation

Simon M Vogt*, Felix Njap and Ulrich G Hofmann

Address: Institute for Signal Processing, University of Lübeck, Lübeck, D-23562, Germany Email: Simon M Vogt* - vogt@isip.uni-luebeck.de

* Corresponding author

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Introduction

Descriptive models of basal ganglia operation have seen a recent increase in interest from the classical idea of direct and indirect pathways towards a more feedback-oriented view of statistic optimality [1]. These new views may prove to be valuable in explaining and finding new treatments for common basal ganglia disorders such as Parkinson's disease beyond current techniques of pure symptom fighting through widespread deep brain stimulation or chemical regulators for increasing tonic levels of dopamine.

It is known that phasic changes in dopamine signals from the substantia nigra pars compacta (SNc) and ventral tegmental area (VTA) seem to present a reward prediction error to the striatum [2] and likely play an important part in procedural learning. Some (rate-based) models of basal ganglia learning procedures have been suggested and await further integration with biological evidence [3].

Our current interest here is to examine the firing variability between SNc neurons depending on their afferent inputs and general projections to within the rat striatum. We have therefore created a new design of self-fabricated tetrode-like nine-wire electrodes [4] to assist in current spike clustering techniques [5]. Our results with this new design have shown a triple increase in detectable basal ganglia neurons per probe tip in a confined area.

One long-term goal is to use reward information accumulating in the SNc to guide fine-grained electrical stimulation of dopaminergic cells placed in the striatum to accommodate for reduced dynamic rage of a procedural learning signal.

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