indicate that the AT and PM subdivisions of the human HCN respond differently to theta-burst stimulation, possibly related to the endogenous theta oscillations identified in the anterior versus posterior hippocampus. Our findings provide insight into how brain stimulation may be used to target network oscillations and motivate future interventions utilizing brain stimulation to treat disorders and impairments with associated disruptions in network oscillatory activity.

Keywords: theta, hippocampal network, iEEG, stimulation

P1.092

NEUROVERSION: A POSSIBLE MECHANISM OF ACTION OF ECT IN ACUTE MANIA

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Abstract

The first-line treatments for acute mania are lithium, antiepileptic mood-stabilizers, and antipsychotic drugs. Electroconvulsive therapy (ECT) is reserved for drug-resistant patients, although it is the first-line treatment for severe or delirious mania with life-threatening physical exhaustion. Contrary to depression, there is a paucity of evidence for the use of ECT in acute mania. However, available literature indicates that ECT seems effective in treating acute mania, with response rates between 80% and 90%, even in drug-resistant patients. Yet, its electrophysiological and molecular pathways remain unknown.

We present the curious case of a 63-year-old woman, followed at our psychiatric outpatient clinic for bipolar disorder type I, admitted to our inpatient unit in a manic state with psychotic features. Due to bradycardia episodes secondary to the psychiatric medication, she was submitted to ECT. After a single session, her symptoms improved, despite no evoked seizures. We compare this curious phenomenon to the well-known procedure of cardioversion and name it *neuroversion* – in other words, a possible normalization of brain activity and behavior triggered by a unique session of electrical stimulation. However, we acknowledge that further research, including randomized clinical trials, are needed to study this reported event.

Keywords: Neuroversion, Electroconvulsive therapy, Mania, Bipolar Disorder

P1.093

A COMPUTATIONAL BASAL GANGLIA-THALAMOCORTICAL CIRCUITRY MODEL FOR PARKINSON'S DISEASE

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Abstract

This work presents a computational *motor pathway* model of the *basal ganglia* to explain alterations in neuronal firing rates, firing patterns, and synchrony in Parkinson's disease (PD). Although the literature relates low dopamine levels to PD symptoms, the underlying mechanisms that explain these alterations are not fully understood. Based on changes in dopamine levels, our model describes the progression of PD via the active-cell-ratio (ACR) in the *substantia nigra par compacta (SNc)* throughout the patient's life.

The model implements the classical *motor circuit* topology with the *SNc*, *D1* and *D2* receptors in the *striatum* (*STRD1* and *STRD2*), *internal* and *external globus pallidus* (*GPi* and *GPe*), the *subthalamic nucleus* (*STN*), the *thalamic ventral lateral nucleus pars oralis (VLo)* and the *cortex* (*CTX*).

Using firing rates and patterns, synaptic weights, and synchrony values from the literature for healthy subjects and PD patients, the MATLAB model describes the motor circuit as a multiple-feedback oscillatory network with varying frequency per nucleus. This defines healthy and parkinsonian boundary conditions for a non-linear multiple-input multiple-output (MIMO) system representing the network, with time-varying outputs in state-space representation.

The preliminary results satisfactorily predict -for the first time to our knowledge- frequencies, patterns, and synchrony of the neurons, throughout a patient's life. A decreased activity in the GPe, increased in the STN, GPi, and VLo, as well as elevated synchrony in these subpopulations and the CTX, are observed in advanced parkinsonian stages.

The model mathematically suggests the existence of self-regulatory mechanisms in the STN, GPi, and VLo, forcing the network convergence with low ACR at the SNc. This theorizes how the lack of dopamine alters neuronal patterns, leading to the PD symptoms through the thalamocortical pathway. The validation of this model would allow defining early-diagnosis indicators for PD and stimulation parameters for neuromodulation therapies, including DBS and TMS.

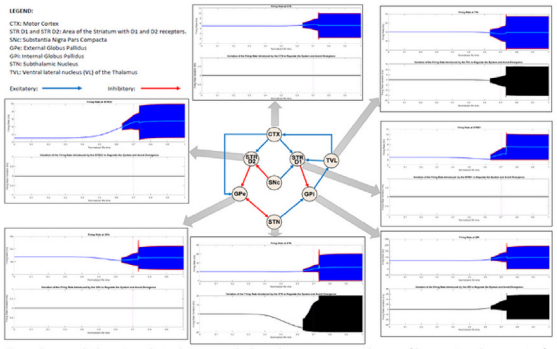

Fig. 1 – Topology of the simulated network for the motor pathway (figure in the center). In the surroundings, the plots of the firing rates are shown in blue with the upper and lower envelopes in red on the oscillatory (parkinsonian) side. Notice the trend line (in cyan) in all the cases is the average between the envelopes, showing how the firing rate either increases or decreases from the healthy to the parkinsonian condition. The plots in black indicate the effort that neurons of each population do to regulate the firing rates in their outputs, being observed only in the STN, GPi, and TVL regions.

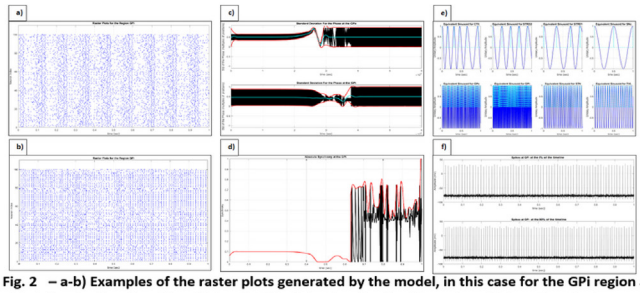

Fig. 2 – a-b) Examples of the raster plots generated by the model, in this case for the GPi region a 0% of the timeline (healthy side) and 80% of the timeline (parkinsonian side). Notice the increase of the synchrony in the parkinsonian side. c) Example of the change of standard deviation used to model the probability that a neuron in the GPi region fires at a certain random instant, or as a consequence of the last common input that produces synchrony. d) Resulting synchrony at the GPi region along the timeline. Notice how the model predicts a significant increase of the synchrony from equal or lower to a 10% to in the healthy side to a value around 65% on the parkinsonian side. f) Examples of the spike plots generated by the model for the GPi region at 0% and 80% of the patient's timeline. It is relatively easy to observe the increase in the firing rate counting the number of spikes in an interval of 0.1 s and multiplying the result by 10. This shows an increase in this region

Keywords: Parkinson's disease, movement disorders, computational model, motor pathway

P1.094

DISPARITIES SURROUNDING DBS SURGERY FOR PARKINSON'S DISEASE AND ESSENTIAL TREMOR IN HAWAII

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