**Introduction**

Anesthetics such as propofol likely exert their influence by altering functional connectivity within different brain circuits. However, network level analyses of anesthesia, particularly in humans have been sparse due to limited access to concurrent intracranial and subcortical recordings. Here, we aim to gain insight into the effects of propofol on pallidocortical circuits in Parkinson disease (PD) patients, with specific attention to alpha (8-12 Hz) and beta (13-35 Hz) oscillations, implicated in the “akinetic” state of the basal-ganglia thalamocortical (BGTC) motor network.

**Methods**

We recorded local field potentials from sensorimotor cortices and pallidum, in twelve PD patients undergoing DBS implantation surgery targeting globus pallidus internus (GPI), while subjects were resting with their eyes open and after propofol-induced loss of consciousness (LOC). We used the modified observer's assessment of alertness/sedation scale (MOAA/S) to evaluate the level of alertness. We measured spectral power and phase based connectivity (de-biased weighted phase lag index) between GPI and sensorimotor cortices during 1 minute of rest and similarly after complete LOC.

**Results**

At GPI and sensorimotor cortices propofol shifts the dominant spectral peak toward alpha frequencies (8-12 Hz) while increasing low frequency (<20 Hz) power. Specific to the GPI, power in frequencies >20 Hz decreases. At the cortical level however, there is broadband power increase (20-100 Hz) similarly at both sensory and motor/premotor cortices with power suppression in frequencies > 200 Hz specific to the motor/premotor cortex.

Propofol reduces sensorimotor high beta connectivity despite local high beta power increase at both cortices.

**Conclusions**

Alpha oscillations are suggested to functionally inhibit unneeded neuronal networks during movement. Our findings suggest that propofol-induced increase in alpha synchronization may be the underpinning for propofol-induced immobility, in addition to suppression of beta synchrony (both local and inter-regional). Moreover, recent discoveries in PD pathophysiology suggest that it is most likely excessive coupling between nodes of the BGTC motor network that are related to disease symptoms. Reduction of pallidocortical high beta coupling with propofol further supports this hypothesis.

**Learning Objectives**

1) Understanding the effects of propofol on sensorimotor cortical electrophysiology
2) Understanding the effects of propofol on subcortical (pallidal) electrophysiology
3) Understanding changes in pallidocortical synchrony as they relate to propofol and Parkinson disease

**References**
