role of each area in this fronto-parietal network is unclear. Previously, data collected from these areas were limited to single neuron electrophysiological recordings. Single neuron recordings are not sufficient to elucidate the interaction of neurons at a population level during the formation of a motor plan. To capture network dynamics, we recorded single and multi-unit activity in parallel from chronically implanted electrode arrays in AIP and F5 while two macaque monkeys (female) performed a delayed grasping task (using one of two grip types) that also involved a grip selection component. Firstly, implementing the 'initial condition hypothesis' of movement preparation developed by Afshar et al. (2011), we predicted behavior of the animal on a single trial basis. This hypothesis posits that neural population activity prior to movement is predictive of subsequent reaction times (RTs) of the animal on single trials. In support of Afshar et al., who recorded on the border of dorsal premotor cortex and primary motor cortex in a reaching task, we found this method was able to explain significantly more variance in RT compared to classical methods. We introduce here a new prediction method based on a data-driven selection of relevant neurons and neural population averaging designed to extract a consistent relationship between neural activity and RT. Our method was able to explain the most variance in RTs overall and was the most parsimonious in that it does not rely on complex high-dimensional analyses. No decision related difference was found between conditions where the grip type was instructed and when the monkey chose freely. Furthermore, we were able to compare the information content of areas AIP and F5. We found that F5 was able to predict RT significantly better than AIP. Interestingly, multivariate regression of F5 and AIP together was able to significantly improve prediction using our proposed method. Taken together, these results lend support to the hypothesis that trial-to-trial movement preparation is strongly encoded in F5 and that AIP represents a step in the visuo-motor transformation not well encoding the temporal characteristics of upcoming movements such as RT. Reference: Afshar, A., Santhanam, G., Yu, B. M., Ryu, S. I., Sahani, M., & Shenoy, K. V. (2011). Single-trial neural correlates of arm movement preparation. Neuron, 71(3), 555-564.

Information flow in optogenetically stimulated macaque motor cortex: simulation and experiment

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Optogenetics is a powerful tool for performing spatiotemporally precise perturbations to ongoing cortical dynamics in behaving primates. However, current methods allow for only small numbers of neurons to be recorded from simultaneously. In this work, we present a biomimetic spiking network model of macaque motor cortex, including an opsin channel model, in order to extend experimental optogenetics results to the large number of cells required for determining interlaminar information flow. Experimental data were recorded from the primary motor cortex of a male macaque. Optogenetic stimulation targeted excitatory neurons, likely preferentially affecting deeper layers, via the excitatory opsin C1V1TT, with either continuous (200 ms duration) or periodic (20, 40, or 80 Hz) pulses. The network model consisted of 3100 spiking Izhikevich neurons, consisting of regular-firing and bursting pyramidal neurons and fast-spiking and low-threshold-spiking interneurons, with connectivities and proportions of each cell type across each cortical layer drawn from experimental mammalian literature. Opsin channel properties were also based on empirical estimates. The network model was calibrated to reproduce the dynamics of the experimental data, including firing rates of approximately 10 Hz in the quiescent state, 60 Hz following a continuous light pulse, and 20 Hz during periodic stimulation. Applying spectral Granger causality to the LFPs produced in the different cortical layers of the model showed that, in the absence of optogenetic stimulation, the strongest projection was from layer 2/3 to layer 5A. This finding is consistent with the hypothesis that descending excitation is the primary driver of dynamics in the motor cortex. This pathway had roughly double the total Granger causality as the strongest ascending pathway, from layer 5A to layer 2/3. Strong Granger causality from layer 5A to layer 5B was also observed. Across all layer pairs, Granger causality showed a pronounced peak in the mu rhythm band (~9 Hz), with a small, broad gamma peak (~40 Hz) also observed in pathways from layer 2/3 to other layers. Optogenetic stimulation in the model increased Granger causality from layer 5 to other layers in a narrow band near the stimulation frequency. Optogenetic stimulation also increased the amplitude of Granger causality from layer 2/3 to other layers in the mu rhythm band, while decreasing it in the gamma band. In summary, this work demonstrates that (1) biomimetic modeling allows data from optogenetic stimulation experiments to be explored on a network level, and (2) optogenetic stimulation may be used to enhance and suppress information flow in particular frequency bands and between particular cortical layers.

Common low-frequency dynamics in movement and sleep

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It has been known for over a century that upper-limb movements are often composed of discrete submovements, but the origin of this intermittency remains unclear. While neural correlates of submovement frequencies around 2-3 Hz can be found in the primary motor cortex (M1), the temporal profile of movement kinematics is usually assumed to be determined by extrinsic factors such as limb biomechanics and sensory feedback delays. However, another possibility is that movement intermittency arises from an intrinsic rhythmicity in motor networks that causes low frequencies in behavior. Delta activity recorded in the electroencephalogram during slow-wave sleep and from isolated cortical slices points to the existence of neural oscillators at frequencies similar to those found in behavior. However, to our knowledge the low-frequency dynamics of brain activity during movement and sleep have not been directly compared. We therefore used chronic multi-electrode arrays to record neural activity and local field potentials (LFPs) from M1 and ventral premotor cortex (PMv) in monkeys during an isometric wrist movement task, natural sleep and ketamine sedation. We used principal component analysis to project the low-frequency LFP onto a plane, and observed cyclic trajectories in M1 that were phase-locked to each submovement. The areal velocity of trajectories increased for faster submovements, but the angular frequency remained constant at around 3 Hz (the frequency of submovements). During sleep, LFP activity traversed cycles with the same frequency and direction of rotation (albeit with larger amplitude) and under ketamine sedation these were phase-locked to K-complexes occurring at the transition from down- to up-states of the cortex. Neural activity was locked to LFP cycles within the same cortical area under all behavioral conditions, and became synchronized across areas during sleep and sedation. Since the same cortical dynamics are observed during movement and in the absence of behavior during sleep and sedation, we suggest that the motor networks controlling the upper-limb possess intrinsic, low-frequency rhythmicity. In the awake state, periodic descending drive from M1 constrains the temporal structure of tracking movements, while widespread synchronization across cortical areas generates the well-known delta rhythms associated with slow-wave sleep. This work was funded by the Welcome Trust.

Posterior parietal cortex in predictive sensorimotor control

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Interaction with the evolving world largely relies on accurate prediction of body and environment based on dynamic interplay between sensory inflow and motor outflow. Numerous studies have shed light on sensorimotor integration in the posterior parietal cortex (PPC), but most have emphasized purely reactive movements toward static targets, in which sensory and motor