The constraints of neural plasticity during acquisition of Brain-Machine-Interface Skill

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Abstract

Interaction with a Brain-Machine Interface (BMI) results in different neuronal tunings as compared to naturalistic movements. We hypothesize that we are able to improve our prediction of these changes based on the neuronal changes during the process of BMI learning. We analysed the monkeys’ neural tuning up to 20Hz and under ketamine-induced sedation as they learned BMIs with arbitrary mappings between neural firing rates and cursor position over several weeks. As performance improves, we used Principle Component Analysis (PCA) of brain electrical activity as an estimate of possible constraints on neural plasticity. We also applied a novel algorithm[1] which extracts the dynamical structure of activity modulation to further predict constraints. Due to the short span of time and problems writing the custom Matlab code, we were unable to complete the analyses and have no significant findings.

Methods

Brain control

\[ y = \Sigma_{\text{up}} - \Sigma_{\text{down}} \]

Wrist control

2D torque

We recorded the spiking activity from neurons in the primary motor cortex (M1) and ventral premotor cortices (pMV) of two rhesus macaques, monkey D and monkey R, performing 2D wrist- and 1D brain-controlled cursor tasks over multiple sessions. During brain control, neurons were assigned to up, down and off ensembles according to an arbitrary mapping (Map 1 or Map 2). Instantaneous cursor position was determined from neuronal firing rates.

Results

Figure 1. Data Histogram for experiment 1 reveals no predictive tuning changes of 34 neurons over the course of 11 sessions.

Figure 2. Data Histogram for experiment 2 reveals no predictive tuning changes of 34 neurons over the course of 11 sessions.

Figure 3. Data Histogram for experiment 3 also reveals no predictive tuning changes of 34 neurons over the course of 14 sessions.

Aims

To further predict constraints on neural plasticity, we will:

1. Examine the Correlation structure during natural movements in different frequency bands, including sensorimotor rhythms at 10-20 Hz up to millisecond spike synchrony.

2. Examine whether a novel algorithm [1] which extracts the dynamical structure of activity modulations can further predict constraints on neural plasticity.

3. Examine whether similar correlation structure as observed during awake behaviour is present under ketamine-induced sedation, which includes periods of intense ‘up-state’ activity.

Conclusion

The project could not be finished within the stipulated duration. The data, while inconclusive at this point of time, show promising statistical findings.

References