Volitional Control of Beta Band Power in Parkinsonian patients

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Introduction: Previous work has demonstrated that subjects can volitionally control the power of their endogenous local field potential signals given real-time feedback [1], [2]. Further work has demonstrated that volitional modulation of cortical motor beta band local field potentials prior to performance of a reaching task effects performance of the reach [3]. Specifically, reduction in beta power contributes to faster reaching, supporting the hypothesis that motor system beta power serves as an inhibitor of upcoming movements [4]. Since beta LFP signals are hypothesized to also be involved in motor symptoms related to Parkinson's disease [5], [6] we sought to replicate the sequential neurofeedback and movement task in PD patients implanted with a Medtronic Activa PC + S Neural Stimulation device. Here we present results from initial neurofeedback-only sessions from three patients.

Material, Methods and Results: We studied 3 subjects with diagnosed PD. Subjects all had one Activa PC + S devices, each that supports two four-contact leads. Subjects with bilateral therapy also had a separate Activa SC unit (for clinical therapy only). Subjects all had one lead laying over cortical areas approximately spanning premotor cortex to somatosensory cortex [7]. Subjects either came into the UCSF clinic (2 subjects) or completed the beta band power controlled neurofeedback task at home (1 subject) for a single 1-2 hour session. Subjects performed an instructed movement task as a baseline to assess the range of beta power. Table 1 summarizes the signal inputs used for neurofeedback control for each patient. All patients gave their written informed consent to participate in this study under a protocol approved by the Institutional Review Board.

Patient	Home or UCSF	Stim On / Off	Time Domain vs. Power Channel	Beta Power Calculation Method	Cursor Prediction
1	UCSF	Off	Time Domain	Multi-Taper	Linear Reg.
2	Home	On	Power Channel	n/a	Linear Reg.
3	UCSF	Off	Time Domain	Welch	Kalman Filter

Table 1: Signal Properties Driving Beta Cursor For Each Patient

For the neurofeedback task, a fixed mapping between subject neural activity and cursor position was calculated using the movement task as training data. The task required subjects to modulate the cursor to hit one of four instructed targets per trial.

We find that for all three subjects there were slight asymmetries in the mapping between neural activity and cursor position that made 1-2 targets challenging yet possible, and the other 2-3 targets either too easy or unattainable. For the targets that were achievable yet challenging, during late training all three subjects exhibited above chance performance, and one subject exhibit significant improvement in time it took them to modulate to that target (Chance calc. above bootstrapped distribution: Patient 1: p<0.01, Patient 2: p < 0.01, Patient 3 p < 0.001, Improved time to target two-tailed Student's t-test: Patient 3: p < 0.05).

Discussion: We found that subjects were able to improve performance for at least one neurofeedback target, demonstrating a proof of concept of volitional control using signals acquired by the Activa PC + S device in various modes of operation. These modes include subcortical stimulation on or off, power channel signals or time domain signals, and use of a linear regression method or a Kalman Filter to estimate cursor position (see Table 1). Moving forward, we will be completing longer training sessions with patients and eventually coupling the neurofeedback trials with bradykinesia-inducing motor tasks to investigate effects of modulation on disease-related motor symptoms.

Significance: This is the first demonstration to our knowledge of using the Activa PC + S device for volitional neurofeedback control using cortical leads.

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References (*8pt*) [1] B. Engelhard et. al. *Neuron*, vol. 77, no. 2, pp. 361–375, Jan. 2013.[2] K. So, S. Dangi, et. al *J. Neural Eng.*, vol. 11, no. 2, p. 026002, Apr. 2014. [3] P. Khanna and J. M. Carmena, in *2015 7th International IEEE/EMBS Conference on Neural Engineering* (*NER*), 2015, pp. 340–343. [4] A. K. Engel and P. Fries, *Curr. Opin. Neurobiol.*, vol. 20, no. 2, pp. 156–165, Apr. 2010. [5] C. de Hemptinne, et. al *Nat. Neurosci.*, vol. advance online publication, Apr. 2015. [6] A. L. Crowell, et. al. *Brain J. Neurol.*, vol. 135, no. Pt 2, pp. 615–630, Feb. 2012. [7] E. Ryapolova-Webb, et. al. *J. Neural Eng.*, vol. 11, no. 1, p. 016009, Feb. 2014.