The neural representation of fine hand movements in an individual approaching locked-in syndrome

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Introduction: For individuals who are paralyzed due to amyotrophic lateral sclerosis (ALS), brain-computer interfaces (BCIs) based on electrocorticography (ECoG) may restore communication [1, 2]. BCI control is driven by attempted movements of for example the hand, and next to the primary motor cortex (M1), the somatosensory cortex (S1) may contribute to decoding movements [1, 2]. Yet, in these BCI studies most participants were incompletely paralyzed, meaning they could still move parts of their body. To our knowledge, BCI users with locked-in syndrome (i.e., complete paralysis; LIS) due to ALS controlled the BCI using a simple attempted hand movement [2]. It remains unclear how the degeneration of neurons in M1 due to late-stage ALS and the absence of movement-related sensory feedback affect the neural representation of attempted movements in individuals who are locked-in as a result of ALS. In the current study, we explored the representation of attempted fine hand movements of an individual who was approaching LIS due to ALS.

Material, Methods and Results: We implanted four 32-channel subdural high-density ECoG grids with 3 or 4 mm pitch in an individual (41 years old, female) who was quadriplegic and anarthric due to ALS. Grid placement was based on pre-surgical functional MRI activation in response to attempted hand and mouth movements. For ECoG data collection, the participant performed a task where active trials were interleaved with rest trials. Prior to the task, the participant was instructed to attempt one out of six predefined movement types (classes) at the onset of each active trial, being index, thumb or little finger flexion/extension, or grasp, pinch or American sign language 'Y' movement (18 active trials during each of 3 sessions). After standard preprocessing, we calculated high-frequency band power (HFB, 60-120 Hz) per trial, for each electrode. HFB power was compared to a regressor, with the correlation coefficient calculated per class, averaged across the three sessions. Each class resulted in a different pattern of activation, with the strongest response generally over the central sulcus or postcentral electrodes (Fig. 1).



Figure 1. Electrode locations on the brain of the BCI participant. For the upper two grids, the correlation coefficients are shown for electrodes where the HFB showed a positive correlation to attempted movement in at least one of the three sessions. The electrode radius increases with the magnitude of the correlation coefficient.

Conclusion: The pronounced post-central response, even in the absence of *coefficient.* movement-related sensory feedback, warrants further investigation to better understand the neural representation of attempted movements in LIS, and its implications for BCI control.

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