

Electrocorticographic Decoding of Ipsilateral Reach in the Setting of Contralateral Arm Weakness from a Cortical Lesion

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Abstract— Brain machine interfaces have the potential for restoring motor function not only in patients with amputations or lesions of efferent pathways in the spinal cord and peripheral nerves, but also patients with acquired brain lesions such as strokes and tumors. In these patients the most efficient components of cortical motor systems are not available for BMI control. Here we had the opportunity to investigate the possibility of utilizing subdural electrocorticographic (ECoG) signals to control natural reaching movements under these circumstances. In a subject with a left arm monoparesis following resection of a recurrent glioma, we found that ECoG signals recorded in remaining cortex were sufficient for decoding kinematics of natural reach movements of the non-paretic arm, ipsilateral to the ECoG recordings. The relationship between the subject's ECoG signals and reach trajectory in three dimensions, two of which were highly correlated, was captured with a computationally simple linear model (mean Pearson's r in depth dimension = 0.68, in height = 0.73, in lateral = 0.24). These results were attained with only a small subset of 7 temporal/spectral neural signal features. The small subset of neural features necessary to attain high decoding results show promise for a restorative BMI controlled solely by ipsilateral ECoG signals.

I. INTRODUCTION

The fast growing field of brain machine interfaces (BMI) provides the opportunity to both expand our working knowledge of the human nervous system and rehabilitate disabled persons. Electrocorticography (ECoG) is a promising source of feature-rich neural signals that provides a compromise between the highly invasive and non-stationary spike recordings from microelectrode arrays and the low bandwidth recordings from electroencephalography (EEG) [1]. ECoG electrode grids and strips are clinically used for localizing the seizure focus in patients with intractable epilepsy. While patients wait to have a seizure recorded to plan resective surgery, researchers are afforded the opportunity to recruit volunteers for experimental tasks in which the subject's behavior and neural signals are monitored simultaneously. Previous work has shown ECoG

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signals from humans can be used to successfully decode two-dimensional cursor control [2] or arm trajectory [3, 4]. In this way patients undergoing ECoG recordings can help test BMI systems being developed for patients with motor impairments from a variety of causes.

Prior BMI studies have focused primarily on decoding limb movements contralateral to the motor areas from which ECoG electrodes are recorded [5-10]. This approach is aimed at restoring upper limb function in amputees and patients with lesions of the spinal cord and peripheral nerves. In this case, the best approach is assumed to consist of an interface with upper limb representations in M1 and nearby premotor cortices. However, when designing a BMI for patients with motor impairments due to brain lesions, as in stroke, trauma, or brain tumors, these cortical motor systems may not be available. Such a BMI might instead rely on motor signals from ipsilateral motor cortex. It has been shown that hemiparetic subjects with unilateral lesions have significant deficits in the movement and muscle activity of the ipsilateral wrist [11]. It has been conjectured that this is due to the interruption in the pathway of the ipsilateral corticospinal motor projections. This raises the possibility of cortical representations for ipsilateral limb movements that could be activated and recorded by ECoG.

Another rationale for testing the viability of ECoG for ipsilateral limb control is that amputees cannot train a BMI using contralateral arm movements. One possible solution for these patients is to train the decoding algorithms using ECoG signals generated during movements of the intact ipsilateral arm, and then transition to control of the contralateral prosthetic arm by mirroring the movements of the ipsilateral arm. While this may be counterintuitive for the subject at first, it might help minimize the amount of necessary operant conditioning.

It has been shown previously that two-dimensional hand position can be decoded ipsilaterally using ECoG [12, 13]. However, it remains to be investigated whether or not natural, unconstrained three-dimensional ipsilateral hand trajectory can be decoded from ECoG signals in humans. Here we investigated this question in a subject with contralateral arm weakness performing blocks of a discrete reaching task with their ipsilateral arm.

II. METHODS

A. Recordings

The volunteer for this study was a 36 year old right handed man with medically refractory epilepsy associated with a low-grade oligodendroglioma in the right frontal lobe. He had already undergone 4 previous resections of the

tumor, but he was still having frequent secondarily generalized seizures beginning in the left hand, followed by Todd's paresis of the left arm and leg. Simultaneous with surgical implantation of subdural electrodes to guide epilepsy surgery, a nodule of recurring tumor was resected from the posterior lip of the previous resection cavity, at the anterior border of the precentral gyrus near the anatomically defined hand knob. During the subsequent week of intracranial monitoring, the patient's left arm was paretic, especially in distal limb muscles. The subject was recruited for this study because his intracranial electrodes had been placed over hand and arm areas of motor cortex. His electrode placement had been determined solely by clinical needs. The ECoG studies performed with this subject had been approved of by the Johns Hopkins International Review Board.

ECoG recordings from a total of 47 platinum subdural electrodes (Adtech Medical Instrument Corp., Racine, WI), referenced to a single subdural electrode, were used in our offline analysis. Electrode placement is seen in Figure 1. Electrodes were 4 mm in diameter with 10 mm inter-electrode spacing. Signal acquisition was performed by a 64-channel Neuroscan Synamps² (Compumedics, Charlotte, NC). The electrodes were implanted in the right hemisphere, ipsilateral to the patient's right (dominant) hand, which was used for the reaching task. The ECoG signals were bandpass filtered with cutoff frequencies at 0.15 Hz and 200 Hz, then sampled at 1000 Hz.

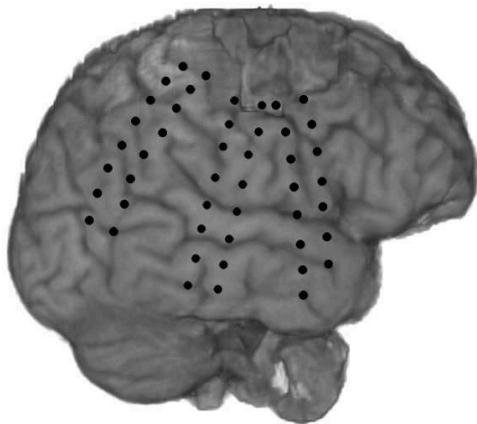


Figure 1. Reconstruction depicting the locations of the 47 electrodes used for analysis

The subject's hand and shoulder position were optically tracked using the Optotrak system (Northern Digital, Inc.; Ontario, Canada) with a sampling frequency of 100 Hz. Tracking was done in height, depth, and lateral axes, which corresponded to the arm moving up or down, forward or backward, and left or right respectively. All recording was done within the system outlined in [14]. The arm position was synchronized with the neural data using parallel port triggers sent through a split cable to Neuroscan's trigger inputs and the Optotrak Data Acquisition Unit.

B. Experimental Paradigm

The subject made discrete natural reaches in three dimensional space to touch the tip of a wooden dowel being moved by the experimenter. The target location was determined by the experimenter in order to fully probe the

three-dimensional workspace. Reaches with the contralateral arm were not performed because it was too weak to perform the task. The subject performed each reach by pinching or touching the dowel tip with his ipsilateral index finger, then returning his hand to a comfortable resting position in his lap. The subject's hand remained in the resting position for 1.7-6.6 seconds, as the target was moved to another point in three dimensional space signaling the next reach to begin. The only cue for onset of the next reach was cessation of movement of the target to each new location. This was done to avoid confounding variables such as audio cues and to keep the reach dynamics as natural as possible. The dowel was always placed in front of and above the hand's resting position. The cue was presented on both the left and right side of the subject. Data analysis included 2 blocks of 20 and 22 trials, lasting for 164 and 163 seconds, respectively. These blocks were performed as part of an array of tasks lasting a few hours.

C. Feature Extraction

The MATLAB (MathWorks, Natick, MA) Signal Analysis Toolbox was used for offline analysis of the ECoG recordings. After removing bad electrodes via visual inspection, we common average referenced (CAR) the data. The local motor potential (LMP) was computed by applying a 2-second moving average filter both forward and backward to remove any group delay. Previous studies have indicated that this smoothed time domain feature contains movement-related information [2, 15, 16].

To obtain movement-related ECoG signal features in the spectral domain, we used forward and backward filtering with 400-order bandpass FIR filters with Hamming windows to segregate the different frequency bands from the CAR filtered data without phase distortions. The cutoff frequencies corresponded to delta (0-4 Hz), mu (8-12 Hz), beta (12-30 Hz), low gamma (30-50 Hz), high gamma 1 (70-110 Hz) and high gamma 2 (130-200 Hz) bands. The resulting signals were squared to yield band-specific power estimates. It has been previously shown that changes in the power of ECoG signals in both low and high frequency bands are associated with cortical processing during visual-motor tasks [17, 18]. We smoothed this data using a 2-second moving average forward and backward to remove noise. The beginning and end of each block was truncated to remove filter transients and the data was log-transformed to normalize the distribution.

D. Decoding Framework

Five-fold cross validation was used to validate our decoding model. Approximately 131 seconds of data was used in the training sets. Both ECoG signal features and kinematic data were normalized by subtracting their means and dividing by three standard deviations based on the training data. The means and standard deviations were recomputed during every fold of cross-validation. This normalization was done to ensure the magnitude of the features did not impact how heavily they were weighted in the decoding models.

ECoG signals from each of the 47 recording channels were decomposed into 7 different spectral/temporal features, leading to 329 features. This high dimensional data set

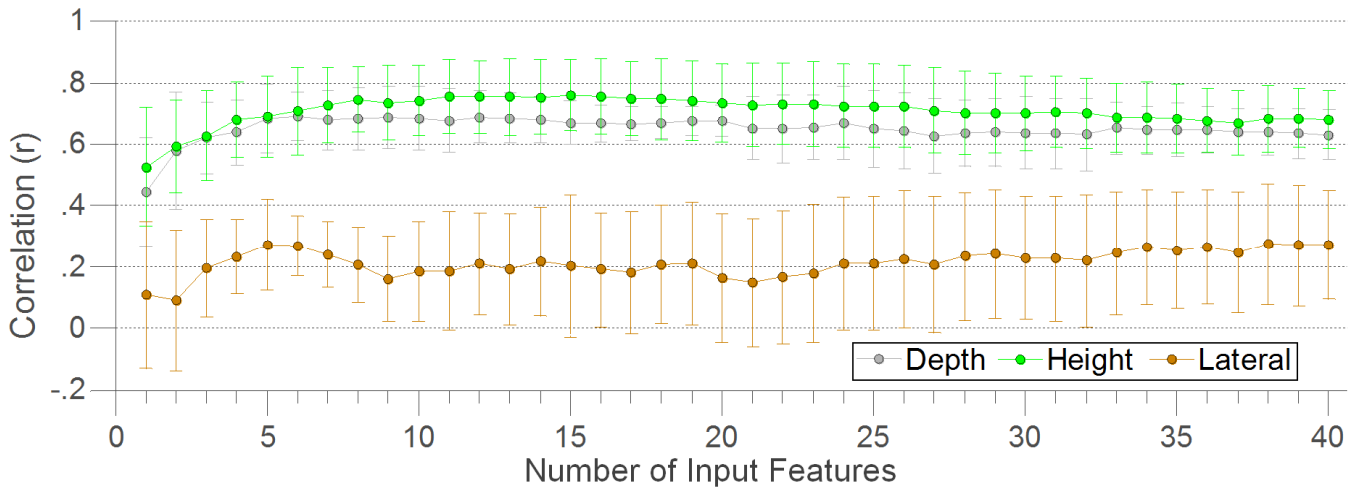


Figure 2. Correlation (Pearson’s r) of predicted and actual arm trajectory as a function of input features used. Each point corresponds to the mean of 5-folds across 2 sessions of data, and each bar represents the standard error of the mean.

caused overfitting with our time-limited data set, so it was necessary to select a subset of the features. This selection was done every fold by finding the features with the highest raw correlations to the subject’s hand trajectory across multiple time lags in the training set. We looked at the correlations for the features at time lags varying between -100 and 100 ms with respect to the kinematics at 5 ms intervals. We did not consider the same feature at multiple time lags as inputs to the model, though the model could include multiple feature types from the same electrode.

Each of the outputs was modeled as a linear combination of the input features with a constant offset and a Gaussian noise term:

$$y_k(t) = \beta_{0k} + \beta_k X(t) + \varepsilon_k$$

where $y_k(t)$ denotes the predicted output of the k^{th} dimension of the kinematic output at time t , $X(t)$ is the vector of feature values at time t , β_k is the linear weight associated with each neural feature, β_{0k} is the offset, and ε is zero-mean Gaussian noise. These parameters were calculated using the `glmfit` function in MATLAB which follows the formula:

$$\beta_k = (X^T X)^{-1} X^T y_k$$

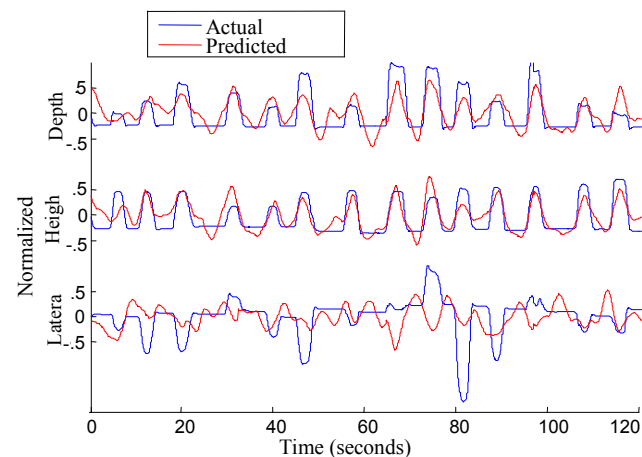


Figure 3. Sample of actual (blue) vs. predicted (red) kinematics. Position was normalized by subtracting the mean and dividing by three standard deviations based on the training dataset.

Model weights were recalculated for the features selected from the training set during each fold of the cross-validation.

III. RESULTS

Sample performance of the model using one to forty input features is shown in Figure 2. Performance is qualitatively high with one feature input ($r > 0.4$ for depth and height). Increasing the number of features appears to provide a small increase in performance until around 7 features, when saturation occurs.

The addition of features does not result in a monotonic increase in performance, possibly due to extraneous features being fitted by the model to aspects of the training dataset that do not generalize well. We report results using 7 temporal/spectral neural signal features as model inputs, since this was the minimal set that confidently provided saturation for each dimension.

Figure 3 shows a sample of the actual and decoded kinematics. The depth and height axes are tracked fairly well, but the lateral axis had much poorer performance. The depth and height axis had very similar performance, probably resulting from the high degree of correlation

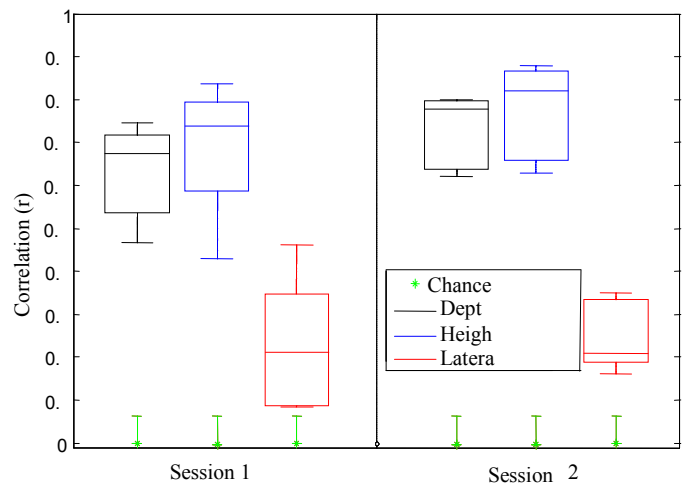


Figure 4. Correlation (Pearson’s r) for each dimension. Chance decoding is the mean accuracy of 1024 models based on shuffled neural data with a bar corresponding to 3 standard deviations.

between the two kinematic traces.

The overall decoding performance of the linear model is shown in Figure 4. This shows that while the lateral decoding is substantially lower than depth or height, it is still significantly above chance levels. The chance decoding results were found by randomly permuting the neural data and doing feature selection, training, and testing using this randomly shuffled data. This was repeated 1024 times for both sessions.

IV. DISCUSSION

We have shown that it is possible to predict natural reaching movements in three dimensional space with accuracy significantly higher than chance. This was done using less than three minutes of ECoG data from the ipsilateral cortex of a subject with contralateral arm weakness from a unilateral cortical lesion. Further, we have shown that a simple linear model is sufficient for capturing a significant portion of the relationship between the neural signals and unidirectional hand position. This was accomplished with a model using only 7 temporal/spectral neural signal features as inputs.

Our model was able to attain high accuracies in the depth and height axes. The markedly lower performance in the lateral axis can most likely be explained by the fact that there were bidirectional deflections relative to the rest position of the hand. A possible resolution for this would be to create a classifier for predicting movement direction while the linear regressor predicts movement magnitude. It may be possible to accomplish this by looking at aspects of the neural data overlooked by our model, such as nonlinearities present in the signals. Alternatively, higher resolution neural recordings may be necessary for decoding bidirectional movement deflections from ipsilateral cortex. This might involve using ECoG grids with smaller inter-electrode distances or using microelectrode arrays for spike train and/or LFP analysis.

The low number of neural features required is in agreement with previous work [16], which showed that saturation of performance occurs with a very small feature set. This indicates that a very small footprint would likely be necessary when implanting an ECoG grid for a brain machine interface to reach peak decoding performance.

All our decoding was based on a limited area of precentral gyrus in a subject where most of the premotor cortex anterior to precentral gyrus had been resected. This successful decoding in the context of damage to important components of cortical motor systems, suggests a possible new avenue for rehabilitating victims of brain lesions. Sufficient decoding accuracy could potentially be obtained from electrodes placed on and around damaged cortex. This obviates the need to expose undamaged cortical areas to the risks of electrode implantation. In addition, electrode implantation could be done concurrently with experimental therapies (e.g. stem cells) for restoring neural populations and enhancing functional reorganization. A potential obstacle for this strategy is that it won't be possible to train any decoding algorithms if the patient can't move their arm. However, one could bootstrap the process by first controlling ipsilateral arm movements, and then robotically

animating or assisting the contralateral arm to mirror the movements of the ipsilateral limb under BMI controlled. Eventually the BMI could wean off ipsilateral arm control as contralateral arm control improves through functional reorganization and neural plasticity.

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