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Electrocorticogram encoding of upper extremity movement trajectories

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***Abstract*— Electrocorticogram (ECoG)-based brain computer interfaces (BCI) can potentially control upper extremity prostheses to restore independent function to paralyzed individuals. However, current research is mostly restricted to the offline decoding of finger or 2D arm movement trajectories, and these results are modest. This study seeks to improve the fundamental understanding of the ECoG signal features underlying upper extremity movements to guide better BCI design. Subjects undergoing ECoG electrode implantation performed a series of elementary upper extremity movements in an intermittent flexion and extension manner. It was found that movement velocity, $\dot{\theta}$, had a high positive (negative) correlation with the instantaneous power of the ECoG high-γ band (80-160 Hz) during flexion (extension). Also, the correlation was low during idling epochs. Visual inspection of the ECoG high-γ band revealed power bursts during flexion/extension events that had a waveform that strongly resembled the corresponding flexion/extension event as seen on $\dot{\theta}$. These high-γ bursts were present in all elementary movements, and were spatially distributed in a somatotopic fashion. Thus, it can be concluded that the high-γ power of ECoG strongly encodes for movement trajectories, and can be used as an input feature in future BCIs.**

I. Introduction

Brain-computer interface (BCI)-controlled upper extremity prostheses are a much sought-after application to restore upper extremity function and independence after paralyzing conditions such as cervical spinal cord injury, subcortical stroke, or brainstem lesions. Recently, there has been a growing interest in using electrocorticogram (ECoG) as a long-term signal acquisition platform for BCI-control of upper extremity prostheses. Several studies have shown that ECoG signals can be used to decode movement trajectories of the arm and fingers, thereby indicating that the ECoG-based BCI platform for upper extremity prosthesis control is promising. Prior studies used local motor potentials (LMPs) [1], [2], [3], [4], [5] and the high-γ band [2], [3], [6], [7], [5] of ECoG to decode trajectories of repetitive finger or arm movements. The maximum correlation coefficients between the actual and decoded finger trajectories averaged across all subjects within each study ranged from 0.32 to 0.64. Similarly, the correlation between the actual and decoded 2D arm trajectory was 0.3 in [8], and varied from 0.50 to 0.62 in [9].

The development of an ECoG-based BCI-controlled upper extremity prosthesis to restore motor function and independence to paralyzed individuals must still overcome many limitations. First, with the exception of [2] and [7], the existing decoders were unable to accurately predict idling periods, or these idling periods were completely omitted. Hence, it remains unclear how well idling periods can be decoded from ECoG signals. Second, the ability to decode movement trajectories has mostly been studied in the context of repetitive movements. In everyday life, however, intermittent movements of upper extremities are much more common, so it remains unclear if existing decoders can be generalized to these types of movements. Third, the majority of ECoG decoding studies have focused on finger or 2D arm movement trajectories [8], [9]. However, since activities of daily living require many unique configurations of upper extremities, a BCI-controlled upper extremity prosthesis will require at least 6 degrees-of-freedom (DOF) to restore independence to a user [10]. Therefore, the moderate decoding accuracies reported in the current literature may not be viable for online BCI control of an upper extremity prosthesis.

To address the above limitations and unknowns, a better fundamental understanding of how ECoG encodes upper extremity movements is required. This may reveal more salient features underlying upper extremity movements, and may ultimately lead to the design of superior decoding algorithms. In this exploratory study, the authors examine the time-frequency characteristics of ECoG signals during 6 elementary upper extremity movements to increase the fundamental understanding of ECoG motor encoding.

II. Methods

A. Signal Acquisition

The study was approved by the Institutional Review Boards of the University of California, Irvine and the Rancho Los Amigos National Rehabilitation Center. Subjects were recruited from a patient population undergoing temporary subdural electrode implantation for epilepsy surgery evaluation. Subject selection was limited to those with electrodes involving the primary motor cortex (M1) upper extremity representation area. Up to 64 channels of ECoG data were recorded using a pair of linked NeXus-32 bioamplifiers (Mind Media, Roermond-Herten, The Netherlands), and signals were acquired at 2048 Hz with common average referencing.

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The subjects performed six elementary arm movements on the side contralateral to their ECoG electrode implant [11]: **1.** pincer grasp and release (PG); **2.** wrist flexion and extension (W); **3.** forearm pronation and supination (PS), **4.** elbow flexion and extension (E); **5.** shoulder forward flexion and extension (SFE); **6.** shoulder internal and external rotation (SR). The trajectories of PG and W were measured by a custom-made electrogoniometer [12], while the movement trajectories of PS, E, SFE, SR were measured by a gyroscope (Wii Motion Plus, Nintendo, Kyoto, Japan). The trajectory signals, including position, $\theta(t)$, and velocity, $\dot{\theta}(t)$, were acquired using an integrated microcontroller unit (Arduino, Smart Projects, Turin, Italy). ECoG data were synchronized with the trajectory signals using a common pulse train sent to both acquisition systems.

B. Task

The above elementary movements were performed sequentially from **1** to **6**. Prior to each movement, the appropriate physical sensor was mounted and calibrated using conventional goniometry at 10° intervals throughout the joint's range of motion. Subjects then performed intermittent alternating flexion and extension movements. A flexion movement was performed until the end of the range of motion. This was followed by an idling period (while in the fully flexed position) for 3-5 seconds. Subjects then extended to the end of the range of motion, and idled in this fully extended position for 3-5 seconds. This was repeated 25 times for each elementary movement.

C. Time-Frequency Analysis

The temporal relationship between the γ-band power and trajectory was explored by first calculating the ECoG instantaneous power:

$$P_n(t) = f(x_n^2(t)) \tag{1}$$

where $x_n(t)$ is the bandpass filtered ECoG signal (80-160 Hz) at channel n and $P_n(t)$ is its power, enveloped by a 1.5-Hz low-pass filter, $f(\cdot)$. Subsequently, $P_n(t)$ was segmented into flexion, extension, and idle epochs based on $\dot{\theta}(t)$. The cross-correlations between $P_n(t)$ and $\dot{\theta}(t)$ were then calculated during flexion, extension, and idling epochs. The cross-correlations during flexion and extension epochs were lag-optimized, while idling cross-correlations were calculated at zero lag. The procedure was repeated for all channels and for all 6 elementary movements in all subjects.

III. Results

Two subjects undergoing subdural electrode implantation for epilepsy surgery evaluation were recruited for this study. Subject 1, a 27-year-old female, was implanted with a 6×8 ECoG electrode grid on the right frontal-parietal area. Subject 2 was a 49-year-old female with a left frontal-temporal 8×8 ECoG grid and a posterior frontal-anterior parietal 1×6 strip. Their electrode placements are shown Fig. 1 (Note that electrode numbers with the "G" prefix are from the main ECoG grid, while "S" denotes the ECoG strip). Each subject completed the motor tasks described in Section II-B, and the ECoG signals were analyzed by the above procedure.

Visual inspection of the $P(t)$ signals revealed a burst of power that was time-locked to every intermittent flexion or extension event, while the $P(t)$ signal during idling appeared noisy and chaotic. Additionally, the waveform of $P(t)$ during these bursts closely matched the visual appearance of the extension and flexion waveforms seen in $\dot{\theta}(t)$. A representative set of tracings can be seen in Fig. 2. To quantify this similarity, the cross-correlation between $P(t)$ and $\dot{\theta}(t)$ during flexion, extension, and idling epochs were calculated (see Section II-C). Based on the visual appearance of $P(t)$ and $\dot{\theta}(t)$, the results were as expected: high positive cross-correlation for flexion epochs, low correlation for idle epochs, and high negative cross-correlation for extension epochs. Representative correlation-lag diagrams for M1 electrodes are shown in Fig. 2. The electrodes located over M1 were ranked based on the above correlation pattern, and the top 1 to 3 electrodes were reported in Tables I and II.

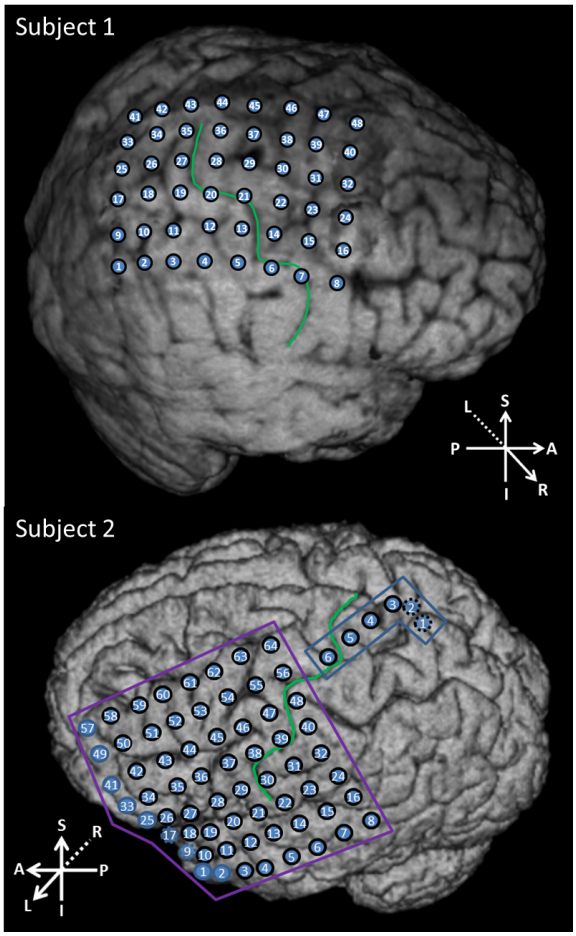

Fig. 1. (Top) Magnetic resonance imaging (MRI) with electrodes localized using the technique described in [13] (Subject 1). The black outlined circles are electrodes that were recorded from (limited by amplifier channel capacity). The green line delineates the central sulcus. (Bottom) A similar image for Subject 2.

IV. Discussion

Based on the visual similarity between $P(t)$ and $\dot{\theta}(t)$, as well as the high positive (negative) cross-correlation values

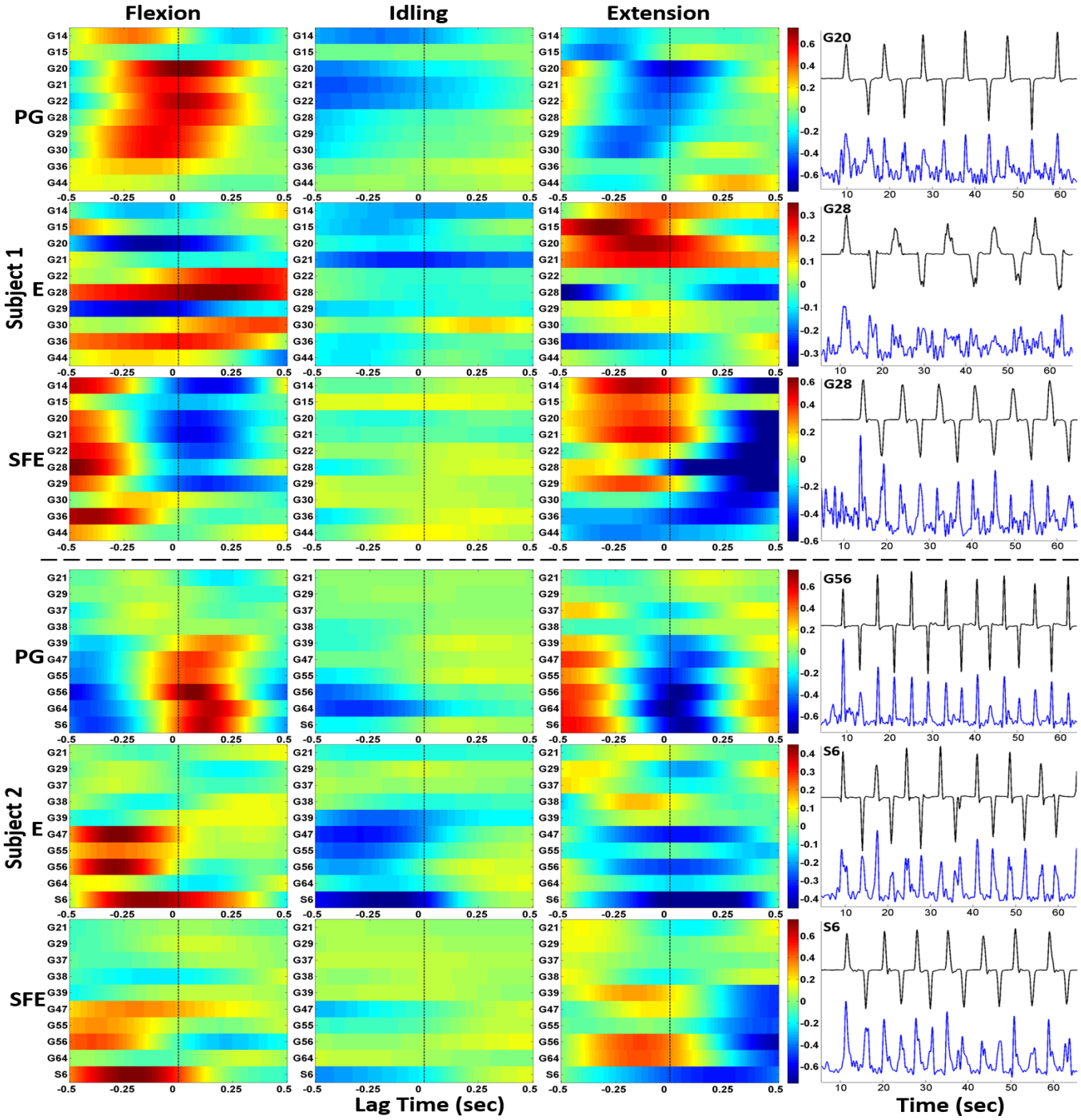

Fig. 2. Representative lag-correlation diagrams of Subject 1 (top) for movement types PG, E, and SFE, and of Subject 2 (bottom) for movements types PG, E, and SFE. For each M1 electrode, the colors represent the cross-correlation at various lag times. The dotted line at time 0 sec, indicates the initiation of movement. Flexion, idling, and extension have their own lag-correlation diagram. On the right is a representative segment of $P(t)$ (blue trace) and corresponding $\dot{\theta}(t)$ (black) at the best M1 electrode.

during flexion (extension) epochs, it can be hypothesized that ECoG high-γ power strongly encodes for elementary upper extremity velocities. Conversely, idling periods are characterized by a lack of correlation, and the $P(t)$ signal appears desynchronized (lower amplitude, noisy, and chaotic). Tables I and II indicate that the electrode(s) on M1 exhibits this correlation-lag pattern for all elementary movements. The top-ranked electrode(s) tends to overlap across movement types; they are more lateral for movements at distal joints (PG, W, and PS), and are progressively more medial for more proximal joints (E, SFE, and SR). These findings point to the existence of separate, but somatotopically arranged neuronal generators that drive each movement type. When active, these generators appear to behave in a similar manner by producing high-γ bursts. Finally, although maximum cross-correlation for some flexion/extension epochs were found at positive lags, Fig. 2 indicates that these values begin increasing before the onset of movement, indicating that the high-γ bursts

TABLE I

CROSS-CORRELATION RESULTS OF THE TOP CHANNELS FOR SUBJECT 1 DURING INTERMITTENT FLEXION (F) AND EXTENSION (E) MOVEMENTS, AND IDLE (I). LAG TIME IN SECONDS ARE PROVIDED IN PARENTHESES.

	Electrode	$\rho(P_F, \dot{\theta}_F)$	$\rho(P_I, \dot{\theta}_I)$	$\rho(P_E, \dot{\theta}_E)$
PG	G20	0.75 (0.02)	-0.21	-0.64 (0.01)
W	G20	0.73 (0.44)	-0.17	-0.55 (-0.11)
	G28	0.56 (-0.11)	0.00	-0.58 (-0.03)
PS	G28	0.47 (0.00)	0.03	-0.62 (-0.18)
	G36	0.58 (0.43)	-0.12	-0.45 (-0.42)
E	G28	0.36 (0.18)	-0.07	-0.38 (-0.56)
SR	G28	0.45 (0.45)	0.10	-0.47 (-0.30)
	G36	0.32 (0.50)	0.06	-0.39 (-0.24)
SFE	G28	0.60 (-0.52)	0.05	-0.83 (0.27)
	G36	0.56 (-0.41)	0.08	-0.47 (0.20)

TABLE II

CROSS-CORRELATION RESULTS OF THE TOP CHANNELS FOR SUBJECT 2.

	Electrode	$\rho(P_F, \dot{\theta}_F)$	$\rho(P_I, \dot{\theta}_I)$	$\rho(P_E, \dot{\theta}_E)$
PG	G56	0.76 (0.07)	-0.10	-0.75 (0.04)
	G47	0.49 (0.07)	0.03	-0.50 (0.10)
W	S6	0.71 (0.09)	0.18	-0.65 (0.07)
	G47	0.42 (0.02)	0.07	-0.41 (-0.02)
	G56	0.61 (0.08)	0.02	-0.30 (-0.08)
PS	S6	0.61 (0.08)	0.05	-0.60 (0.07)
	G47	0.40 (-0.05)	0.05	-0.58 (0.00)
E	S6	0.44 (-0.16)	-0.37	-0.59 (0.13)
	G47	0.45 (-0.28)	-0.21	-0.34 (0.05)
SR	S6	0.51 (0.18)	0.08	-0.32 (-0.34)
SFE	S6	0.75 (-0.21)	-0.20	-0.55 (0.23)

likely precede movement.

Although not explicitly shown in Fig. 2, a high correlation between $P(t)$ and $\dot{\theta}(t)$ can be seen in electrodes in brain areas outside M1. When the $P(t)$ signal from these channels is visually inspected, a pattern of high-γ bursts can be seen with each flexion and extension event. For example, the power bursts exist in the supplementary motor area and posterior parietal cortex in all elementary movements types in Subject 1 (i.e. G39 and G19, respectively). Also, in the auditory cortex (i.e. G12-G16) in Subject 2, these power bursts may be due to the auditory cues given to the subject. These findings suggest that other brain areas behave similarly to M1 when activated despite subserving different functions.

Despite minimal processing, the correlation between $P(t)$ and $\dot{\theta}(t)$ at a single channel is already as high as (and occasionally higher than) those reported in the prior literature [1], [2], [3], [6], [7], [4], [5], [8], [9]. Hence, the authors hypothesize that using $P(t)$ as an input feature for future BCI decoding algorithms may significantly boost the decoding accuracies. However, an additional fundamental understanding of ECoG neurophysiology may be necessary before a useful and generalizable model of upper extremity movements can be designed. Specifically, it is unclear if further spatial or spectral separation of individual movement types, or flexion and extension generators, is possible. Currently, it seems that the same 2-3 M1 channels are involved across all movements in both subjects, indicating that the separate neuronal generators of upper extremity movements are densely packed in a small area of M1, which may make it difficult to resolve them [11]. This warrants further investigation to determine how these generators can be better distinguished, and subsequently exploited for BCI control. This will require the application of more sophisticated signal processing techniques, or possibly higher resolution signals, such as those from mini- or micro-ECoG grids.

V. CONCLUSION

The time-frequency characteristics of ECoG signals may be a good input feature for BCI decoders to control upper extremity prostheses. Future work will focus on developing methods that exploit the characteristics of $P(t)$ to accurately identify when movement is occurring, which movement(s) is (are) occurring, and the direction of movement.

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