NeuroImage 101 (2014) 695–703

Contents lists available at ScienceDirect

NeuroImage

journal homepage: www.elsevier.com/locate/ynimg

Extracting kinetic information from human motor cortical signals

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ARTICLE INFO

Article history:
Accepted 22 July 2014
Available online 2 August 2014

Keywords:
Electrocorticography
Force
Decoding
EMG
Brain–machine interface
Motor cortex

ABSTRACT

Brain machine interfaces (BMIs) have the potential to provide intuitive control of neuroprostheses to restore grasp to patients with paralyzed or amputated upper limbs. For these neuroprostheses to function, the ability to accurately control grasp force is critical. Grasp force can be decoded from neuronal spikes in monkeys, and hand kinematics can be decoded using electrocorticogram (ECoG) signals recorded from the surface of the human motor cortex. We hypothesized that kinetic information about grasping could also be extracted from ECoG, and sought to decode continuously-graded grasp force. In this study, we decoded isometric pinch force with high accuracy from ECoG in 10 human subjects. The predicted signals explained from 22% to 88% (60 ± 6%, mean ± SE) of the variance in the actual force generated. We also decoded muscle activity in the finger flexors, with similar accuracy to force decoding. We found that high gamma band and time domain features of the ECoG signal were most informative about kinetics, similar to our previous findings with intracortical LFPs. In addition, we found that peak cortical representations of force applied by the index and little fingers were separated by only about 4 mm. Thus, ECoG can be used to decode not only kinematics, but also kinetics of movement. This is an important step toward restoring intuitively-controlled grasp to impaired patients.

Introduction

The ability to grasp is critical in daily life, but the neural control of grasping is still not fully understood (Castiello and Begliomini, 2008; Davare et al., 2011). Better knowledge of grasp encoding in the brain could lead to restoration of grasp to people who have lost it because of amputation or paralysis from spinal cord injury, stroke, or amyotrophic lateral sclerosis. Brain machine interfaces (BMIs) use directly-decoded brain signals to control an external device such as a computer cursor (Hochberg et al., 2006; W. Wang et al., 2013), prosthetic arm or hand (Collinger et al., 2013; Fifer et al., 2014; Hochberg et al., 2012; Yanagisawa et al., 2011), or functional electrical stimulation (FES) of paralyzed muscles (Ethier et al., 2012; Moritz et al., 2008). In particular, restoring movement to a paralyzed hand via FES has the potential to grant intuitive control over grasp, which could greatly improve the quality of life for patients with spinal cord injury or stroke (Andersen et al., 2004).

Most existing BMI studies involving grasp have concentrated on decoding kinematics from cortical signals. However, grasping involves a combination of kinematic and kinetic factors (Danion et al., 2013; Krakauer et al., 1999). While a hand neuroprosthesis could be controlled by a BMI that classifies discrete hand grasps or continuous finger movements, fine control of grasp force is essential for accurate manipulation of objects. Thus, for patients to safely and successfully interact with their environment, BMIs will need to enable continuous and accurate control of grasp force by the neuroprosthesis.

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http://dx.doi.org/10.1016/j.neuroimage.2014.07.049
1053-8119/

In monkeys, hand shape (Spinks et al., 2008) and individual finger movement (Aggarwal et al., 2008) can be decoded discretely from neuronal action potentials (spikes), local field potentials (LFPs), or cortical surface potentials (electrocorticography, ECoG; see Chestek et al., 2013; Kubanek et al., 2009) in the primary motor cortex (M1). In addition, spikes in the premotor cortex modulate with grasp type (Townsend et al., 2011). Continuous finger joint positions can also be decoded using spikes (Aggarwal et al., 2013; Ben Hamed et al., 2007; Vargas-Irwin et al., 2010), LFPs (Zhuang et al., 2010), and ECoG (Acharya et al., 2010; Miller et al., 2009). Spike-based BMIs have been used to control continuous grasp aperture (Collinger et al., 2013; Hochberg et al., 2012). An ECoG-based BMI has been used by a few subjects to move a prosthetic hand to one of two discretely-decoded hand postures, but not manipulate objects (Yanagisawa et al., 2011).

To manipulate objects, control of grasp kinetics (force and muscle activation) is critical. However, less is known about the cortical control of grasp kinetics than about the control of kinematics. Spikes have been shown to correlate with grasp force in M1 (Boudreau et al., 2001; Carmena et al., 2003; Evarts et al., 1983; Hendrix et al., 2009), and a spike-based BMI has been used to control grip force in monkeys (Carmena et al., 2003). The representation of force in the dorsal premotor cortex is not as clear, with some finding modulation of spikes with grasp force (Hepp-Reymond et al., 1999), while others did not (Boudreau et al., 2001). Using ECoG from motor and premotor cortices, a study requiring subjects to lift objects with two different weights showed little effect of weight on grasp type decoding, and modest ability to decode the discrete weights (Pistohl et al., 2012). It is unclear if the inconsistencies among studies reflect a complex relationship between force and neural activity, or if the spatial distribution of the brain's force representation has not yet been accurately specified.

Ideally, a neuroprosthetic hand would continuously modulate the force it applies by decoding the user's intended grasp force continuously. Alternatively, FES could be used to activate paralyzed muscles. Studies in monkeys have shown that BMIs could restore movement to a paralyzed hand by using decoded muscle activity from M1 spikes to drive FES (Ethier et al., 2012; Moritz et al., 2008; Pohlmeyer et al., 2009). These arm and finger muscle activations can also be decoded using intracortical M1 LFPs in monkeys (Flint et al., 2012a). Here, we use ECoG to decode the continuous grasp force and finger muscle activity produced by 10 human subjects while they perform an isometric grasp task. In 3 subjects, we decoded force and muscle activity using microwire-ECoG data.

Methods

Subjects and surgical implantation

This study included 10 human participants (4 females, 6 males, ages 20–49, referred to in chronological order as S1, S2,…S10) who were undergoing intracranial monitoring prior to surgery for treatment of medication-refractory epilepsy. All experiments for S1–S4 were performed under protocols approved by the institutional review board of Northwestern University (S1 through S4, protocol #00013311). Experiments for S5–S10 were performed at the University of California at Irvine (IRB protocol #2009-7114) or Rancho Los Amigos National Rehabilitation Center (study #BCI-11-02). All subjects gave written informed consent to participate in the study. Electrode placement was determined by clinical need. Subjects were recruited for the study if their monitoring arrays were expected to cover the hand area of the primary motor cortex. During surgery, arrays were placed in reference to anatomical landmarks, using intraoperative stealth MRI co-registration. In 7 patients (S1, S3–S7, S9), we used standard clinical arrays with 2.3 mm exposed area, 10 mm interelectrode spacing (PMT, Inc. for S1, S3 and S4, and Integra, Inc., for S5–S7 and S9). We implanted subjects S8 and S10 with 8 × 8 "medium-density" ECoG arrays, with 1.5 mm disks spaced 4 mm apart (Integra). In 3 subjects (S2–S4), we implanted surface microwire arrays, with 75 μm diameter and 1 mm interelectrode spacing (16 channel arrays in a diamond configuration, PMT, S2–S4). Post-operative array locations were confirmed using co-registration of pre- or post-operative 1.5 T MRI and post-operative CT images. We performed cortical surface reconstruction and electrode colocalization using either a modification of the techniques presented in Hermes et al. (2010), or according to the method of P.T. Wang et al. (2013). Subjects S8 and S10 could not have MRIs due to metal in the body, so we localized the electrodes from X-rays using the technique of Miller et al. (2007).

Experimental protocol

During each experimental session, subjects were instructed to squeeze a force sensor between their thumb and index finger in a precision grasp. Subjects S8, S9, and S10 also squeezed with their thumb and fifth (or "little") finger in separate experiments. We recorded the isometric force produced simultaneously with ECoG (see Signal acquisition section). The isometric force behavior was performed with the hand contralateral to the recording array. We used a custom-built force sensor based on a 1 DOF load cell (Futek LRF350). Force signals were amplified with a gain of 10,000 (Honeywell model UV in-line amplifier) before being digitized. Beginning with S2, we gave subjects at Northwestern continuous visual feedback of applied force via a computer cursor, and instructed them to perform a 1D random force target-pursuit task. During this task, the subjects attempted to acquire and hold the cursor in each force target for 0.1 s. Feedback was provided using a customized module in BCI2000 (Schalk et al., 2004). Data from subjects S5–S10 was recorded at the University of California at Irvine (UCI), or at Rancho Los Amigos National Rehabilitation Center. Our acquisition hardware at those locations did not allow us to utilize the visual feedback software, so S5–S10 performed self-paced squeezes of self-determined, varying force levels.

Signal acquisition

For subject S1, ECoG signals were digitized at 500 Hz (Nihon Kohden EEG-1100) and force signals were sampled at 500 Hz using a TDT RZ2 Bioamp (Tucker Davis Technologies). The Nihon Kohden and TDT data were synchronized using a TTL pulse prior to analysis. For subjects S2, S3, and S4, both ECoG and force were analog high-pass filtered at 1 Hz and sampled at 1 kHz using the TDT Bioamp and BCI2000 software. For S5–S10, ECoG was sampled at 2048 Hz using 2 linked NeXus-32B amplifiers (20× pre-amp gain, linear phase digital low-pass filtered at 553 Hz; Mind Media). ECoG was common average referenced by the NeXus amplifier before digitization. Force signals for S5–S10 were digitized at 4 kHz with a Biopac MP150 (1000× pre-amp gain), interfaced with custom MATLAB software. The ECoG and force data acquisition devices were synchronized via TTL pulse. Both ECoG and force data were downsampled to 1 kHz in S5–S10 and digitally high-pass filtered at 0.1 Hz (2nd order Butterworth FIR, forward and backward) prior to further analysis.

In S2–S5, S7, S9, and S10 we recorded electromyograms (EMGs) from extrinsic finger flexors (flexor digitorum superficialis). EMGs for S2, S3, and S4 were acquired with surface electrodes (Delsys Bagnoli-8), pre-amplified with gain of 1000 and then digitized using the TDT system. For S5, S7, S9, and S10 EMGs were digitized with a Biopac MP150 EMG amplifier.

Decoding continuous kinetics

We decoded force from ECoG using techniques similar to those we have previously used to decode movement kinematics (Flint et al., 2012b) and muscle activation (Flint et al., 2012a) from intracranial spikes and LFPs. Briefly, we divided each channel of ECoG into its smoothed time-domain representation, the local motor potential