Brain & Language 169 (2017) 1–7

Contents lists available at ScienceDirect

Brain & Language

journal homepage: www.elsevier.com/locate/b&l

Short communication

Activity associated with speech articulation measured through direct cortical recordings

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ARTICLE INFO

Article history:
Received 5 August 2016
Revised 1 December 2016
Accepted 26 January 2017

Keywords:
Articulation
Electrocorticography
Insula
Speech production

ABSTRACT

The insula has been credited with a role in a number of functions, including speech production. Here, we recorded electrocorticography (ECoG) signals from the left insula during pseudoword articulation in two patients undergoing pre-surgical monitoring for the management of medically-intractable epilepsy. Event-related band power (ERBP) activity from electrodes implanted in the superior precentral gyrus of the insula (SPGI) was compared to that of other left hemisphere regions implicated in speech production. Results showed that SPGI contacts demonstrated significantly greater ERBP within the high-gamma frequency range (75–150 Hz) during articulation compared to a listening condition. However, frontal and post-central regions demonstrated significantly greater responses to the articulation task compared to the SPGI. Results suggest the SPGI is active during articulation, but frontal and post-central regions demonstrate significantly more robust responses. Given the small sample size, and number of electrodes implanted in the SPGI, further study is warranted to confirm these findings.

1. Introduction

The insular cortex has been credited with a number of roles, ranging from communication (e.g., Ackermann & Riecker, 2004, 2010; Ardila, 1999; Ardila, Benson, & Flynn, 1997; Baldo, Wilkins, Ogar, Willock, & Dronkers, 2011; Dronkers, 1996; Dronkers, Ogar, Willock, & Wilkins, 2004; Nagao, Takeda, Komori, Isozaki, & Hirai, 1999; Ogar et al., 2006), visceral functions (e.g., Augustine, 1985; Craig, 2002, 2009; Mayer, Naliboff, & Craig, 2006; Moisset et al., 2010), conscious awareness (Craig, 2009), addiction (Naqvi & Bechara, 2009, 2010), and psychiatric disorders (Klein, Ullsperger, & Danielmeier, 2013). The participation of the insula in visceral, emotional, and conscious processes is supported by theoretical models (e.g., Craig, 2002, 2009). However, the relationship between insular function and communication is less rooted in models of language production (e.g., Hickok, 2014; Tourville & Guenther, 2011), even though several studies have reported that the insula is implicated in communication disorders, such as apraxia of speech (AOS; a disorder of motor speech planning and programming that results in off-target articulation, speech sound distortions, and prosodic abnormalities) and aphasia.

To date, lesion-deficit studies of individuals with AOS have informed the study of the neuroanatomical correlates of speech production processes (e.g., Baldo et al., 2011; Basilakos, Rorden, Bonilha, Moser, & Fridriksson, 2015; Dronkers, 1996; Dronkers & Ogar, 2004; Dronkers et al., 2004; Graff-Radford et al., 2014; Hickok et al., 2014; Hillis et al., 2004; Itabashi et al., 2016; Richardson, Fillmore, Rorden, Lapointe, & Fridriksson, 2012). The earliest systematic, quantitative study that revealed a role of the insula in speech was conducted by Dronkers (1996). That study showed 100% lesion overlap in the superior precentral gyrus of the insula (SPGI) in patients with AOS, but 0% lesion overlap in the SPGI among patients without AOS. The relationship between the insula and speech was subsequently supported by functional imaging (Moser et al., 2009; Wise, Greene, Büchel, & Scott, 1999) and lesion (Dronkers et al., 2004; Nagao et al., 1999; Ogar et al., 2006) studies.

The insula as the primary region implicated in AOS has not been a unanimous finding. In a sample of acutely post-stroke patients, Hillis et al. (2004) found that insula damage was not a prerequisite for AOS. Instead, in their sample of patients with AOS (n = 31) over half (n = 19) did not demonstrate hypoperfusion to the insula;

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http://dx.doi.org/10.1016/j.bandl.2017.01.013
0093-934X/

rather, hypoperfusion to the left inferior frontal gyrus *pars opercularis* (IFGpo) was more likely (n = 26/31 patients). These findings were confirmed by an independent group of individuals at the chronic stage of stroke (≥6 months post-onset; Richardson et al., 2012). However, more recent studies suggest a role of the frontal motor and post-central areas in AOS. Collectively, these studies have suggested that pre- and post-central regions may instead be the areas crucially involved in planning, monitoring and executing the motor aspects of speech (Basilakos et al., 2015; Graff-Radford et al., 2014; Hickok et al., 2014; Josephs & Duffy, 2008; Josephs et al., 2012; Whitwell et al., 2013).

With its high temporal and anatomical resolution, electrocorticography (ECoG) has the advantage of providing rare data from brain activity within multiple target regions. Although several prior studies have used ECoG to investigate the role of auditory and motor cortices during speech production (e.g., Behroozmand et al., 2016; Chang, Niziolek, Knight, Nagarajan, & Houde, 2013; Greenlee et al., 2013; Kingyon et al., 2015), relatively fewer studies have investigated regions that are involved during overt production (e.g., see Bouchard, Mesgarani, Johnson, & Chang, 2013; Flinker et al., 2015).

Until relatively recently, direct cortical recordings from the insula were less feasible due to the insula's anatomical intricacies, being concealed from the lateral surface of the brain by the frontal and temporal opercula and covered by multiple branches of the middle cerebral artery. Advanced improvements in stereotaxic surgical techniques have resulted in successful ECoG electrode implantation in the insula (Isnard, Guénot, Sindou, & Mauguière, 2004) through stereoEEG (sEEG). To our knowledge, no published studies thus far have provided accounts of direct cortical recordings from the insula, or the SPGI specifically, during speech production.

Here, we report ECoG recordings from the SPGI in two patients without visible structural brain lesions who were undergoing pre-surgical monitoring of medically intractable epilepsy. The purpose of this study was to measure the SPGI's response to speech production, compared to other grey matter regions of interest previously implicated in speech production based on the results of lesion studies (e.g., Baldo et al., 2011; Basilakos et al., 2015; Dronkers, 1996; Graff-Radford et al., 2014) and fMRI studies in non-brain damaged individuals (e.g., Wise et al., 1999) (see regions listed in Table 1). To this end, we aimed to test: (1) whether the SPGI would demonstrate greater response during articulation when compared to a non-articulation task, and (2) the relative magnitude and timing of cortical responses from the SPGI compared to other left hemisphere frontal and post-central regions during articulation (see Fig. 1).

Table 1
Anatomical coordinates for each channel of interest.

Lobar region	Anatomical region	Patient	MNI coordinate
Frontal 1	IFGpt	1	−36, 27, 25
Frontal 2	SFG	1	−21, 44, 44
Frontal 1	SFG	2	−17, 20, 65
Frontal 2	MFG	2	−40, 54, 6
Insular	SPGI	1	−29, 2, 4
Insular 1	SPGI	2	−30, 12, 10
Insular 2	Posterior insula	2	−31, −16, 15
Post-Central	PoCG	1	−20, −30, 60

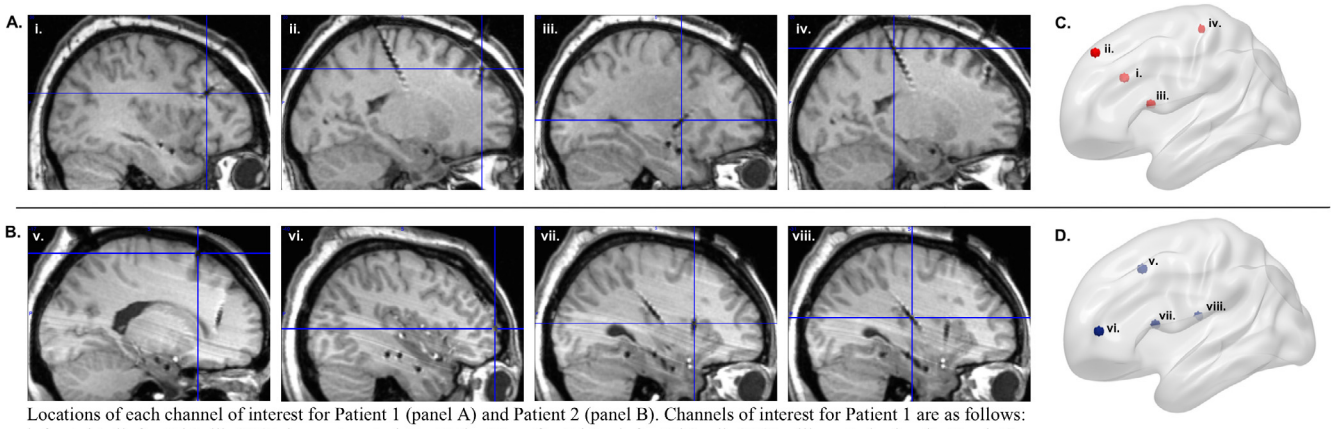
Abbreviations: IFGpt: inferior frontal gyrus *pars triangularis*; SFG: superior frontal gyrus; MFG: middle frontal gyrus; SPGI: superior precentral gyrus of the insula; PoCG: post-central gyrus.

2. Method

2.1. Participants

Two patients with surgically implanted electrodes undergoing monitoring for intractable epilepsy were recruited for study. Both patients were female, right-handed, ages 33 (Patient 1) and 31 (Patient 2). Epilepsy onset was six years prior to testing for Patient 1, and 14 months for Patient 2. Neither patient reported premorbid speech and/or language difficulties or concomitant neurological impairment in addition to epilepsy. Clinical pre-surgical neuroimaging was unremarkable for any structural brain abnormalities. During pre-surgical evaluation of their epilepsies, both patients had poorly localized and poorly lateralized seizure onsets during ictal scalp EEG monitoring, leading to broad bilateral sEEG coverage to further elucidate seizure onset. Results from sEEG monitoring revealed that both patients had seizures localized to medial temporal lobes (MTL): Patient 1 had independent seizure onset on the right and left hippocampi, whereas Patient 2 had right hippocampal seizure onset. The insula was neither the location of ictal onset nor was it involved in seizure propagation in either one of the patients. None of the recorded seizures was poorly localized.

A WADA test was not indicated for Patient 1 since resective surgery was not possible and the patient subsequently underwent



Locations of each channel of interest for Patient 1 (panel A) and Patient 2 (panel B). Channels of interest for Patient 1 are as follows: i: frontal 1; ii: frontal 2; iii: SPGI; iv: post-central. For Patient 2: v: frontal 1; vi: frontal 2; vii: SPGI; viii: posterior insula. Panels C and D show all eletrodes overlaid on a normal brain template for patients 1 and 2, respectively.

Fig. 1. Locations of each channel of interest for Patient 1 (panel A) and Patient 2 (panel B). Channels of interest for Patient 1 are as follows: i: frontal 1; ii: frontal 2; iii: SPGI; iv: post-central. For Patient 2: v: frontal 1; vi: frontal 2; vii: SPGI; viii: posterior insula. Panels C and D show all electrodes overlaid on a normal brain template for Patients 1 and 2, respectively.