# Inhibition of Manual Movements at Speech Arrest Sites in the Posterior Inferior Frontal Lobe

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**BACKGROUND:** Intraoperative stimulation of the posterior inferior frontal lobe (IFL) induces speech arrest, which is often interpreted as demonstration of essential language function. However, prior reports have described "negative motor areas" in the IFL, sites where stimulation halts ongoing limb motor activity.

**OBJECTIVE:** To investigate the spatial and functional relationship between IFL speech arrest areas and negative motor areas (NMAs).

**METHODS:** In this retrospective cohort study, intraoperative stimulation mapping was performed to localize speech and motor function, as well as arrest of hand movement, hand posture, and guitar playing in a set of patients undergoing awake craniotomy for dominant hemisphere pathologies. The incidence and localization of speech arrest and motor inhibition was analyzed.

**RESULTS:** Eleven patients underwent intraoperative localization of speech arrest sites and inhibitory motor areas. A total of 17 speech arrest sites were identified in the dominant frontal lobe, and, of these, 5 sites (29.4%) were also identified as NMAs. Speech arrest and arrest of guitar playing was also evoked by a single IFL site in 1 subject.

**CONCLUSION:** Inferior frontal gyrus speech arrest sites do not function solely in speech production. These findings provide further evidence for the complexity of language organization, and suggest the need for refined mapping strategies that discern between language-specific sites and inhibitory motor areas.

**KEY WORDS:** Speech arrest, Brain mapping, Negative motor areas, Language organization

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Accumulating research indicates that the anatomical organization of language function is more complex than previously thought.[1-3] Several studies have, for example, challenged concepts surrounding the role and location of "Broca's area."[4,5] The identification of sites that elicit arrest of ongoing speech or counting has served to minimize iatrogenic language deficits.[6-10] However, stimulation of the dominant IFL has also been shown to disrupt motor functions other than speech production. For example, in awake patients, dominant IFL stimulation disturbs ongoing fine motor performance as well as repetitive movements (eg, hand or foot tapping), without eliciting responses while the patient is inactive.[11,12]

**ABBREVIATIONS: IFL,** inferior frontal lobe; **NMA,** negative motor areas

Numerous reports have described "negative motor areas" (NMAs), ie, cortical sites where stimulation causes arrest of ongoing motor activity.[11,13-20] NMAs were first reported in 1949 by Penfield and Rasmussen,[11] and then characterized in more detail by Lüders et al.[12,21] Stimulation of these sites elicits no response when the subject is at rest, however, during voluntary movement of the fingers, hand, foot or tongue, stimulation slows or halts the ongoing movement.[12] These sites can be effector specific (ie, only arrest hand movement) or nonspecific (stimulation results in arrest of multiple effectors). NMAs are suspected to be the ictal onset focus for negative motor seizures.[22] By incorporating evidence from functional imaging studies showing an important role for IFG in response inhibition,[23-25] Filevich and collegues[26] argued that stimulation of NMAs may lead to activation of "physiologically inhibitory pathways" involved in normal action

control and praxis for fine movements. Given that speech production requires highly coordinated action of multiple articulators, it is possible that clinical stimulation of these physiologically inhibitory sites may also induce speech arrest.

Whether stimulation of the dominant IFL elicits speech arrest via focal disruption of a speech-specific center, or instead activates tissue involved in an inhibitory motor network (ie, NMA) is thus a question of great clinical and scientific interest. In this study, we sought to determine whether dominant IFL speech arrest sites were specific to language production, or whether they instead functioned as NMAs that also involved musculature outside the speech apparatus. Secondly, we examined the relative frequencies of speech arrest sites and negative motor sites, and characterized their spatial relationships to one another.

## METHODS

### Patient Population and Surgical Procedures

This retrospective study was approved by the Human Research Committee at the University of California, San Francisco. Patient consent was not required for this minimal risk study, per institutional protocol. All 11 patients underwent craniotomy with awake language and motor mapping for treatment of dominant hemisphere pathologies. The senior author (E.F.C.) performed all surgeries between May 2015 and July 2016.

### Intraoperative Mapping Procedures

After exposure of the peri-Sylvian cortex and emergence from intravenous sedation (either dexmedetomidine or propofol), intravenous fentanyl was titrated for optimal balance of patient pain control and arousal during the mapping procedure. Exposed cortex was completely mapped for motor and speech function following our previously published protocol.[27,28] An Ojemann stimulator (bipolar electrodes spaced 5 mm apart) was used with the following stimulation parameters: current range 1 to 3.5 mA, pulse frequency 60 Hz, pulse width 1 ms, stimulus duration 500 to 1500 ms. Behavioral effects of stimulation on each trial were considered valid only in the absence of after-discharges or seizure activity on electrocorticography, which was monitored and reported in real time by an epileptologist. The entire mapping procedure was recorded on 2 HD video cameras–one that provided an unobstructed video of the operative field/exposed cortex, and a second that provided a detailed video of the patient's face or hand, as appropriate for different portions of the mapping procedure. Each stimulation site was registered on the patient's MRI using a neuronavigation system (BrainLab®, Westchester, Illinois), as well as with a photograph. The behavioral response corresponding to each stimulation site was recorded and later confirmed by review of the intraoperative videos.

Following completion of routine sensorimotor and speech mapping, patients were systematically stimulated to identify NMAs. Mapping for NMAs was performed anterior to the central sulcus, where they have generally been localized by previous studies.[12] Inhibitory motor area mapping was initiated at those sites where speech arrest had been elicited, if identified, and then proceeded over the surface of the exposed frontal lobe. During NMA mapping, patients were instructed to perform 2 tasks: the first task involved continuous voluntary "wiggling" of their contralateral thumb at a steady rate, while stimulation was intermittently applied. The second task involved the maintenance of contralateral finger extension (first and second digits), during which stimulation was intermittently delivered. If stimulation arrested or slowed either thumb wiggling or finger extension during more than two-thirds of the trails at a given site (ie, ceased digit movement without causing loss of tone), and induced no sensorimotor response when the patient was inactive, then that site was considered an NMA. Electrocorticography was used to ensure current spread did not explain any motor/speech arrest. In 1 patient (Patient 1), stimulation was intermittently applied while the patient played guitar with his contralateral hand, and sites that caused arrest were noted.[29]

### Analysis of Mapping Results

Speech arrest sites and NMAs were plotted onto a surface reconstruction of the patients' brain which was derived from the MRI using FreeSurfer (http://surfer.nmr.mgh.harvard.edu/). The 3-dimensional coordinates were obtained using stored points from the neuronavigation, and photographs detailing their relationship to surface anatomy were used to ensure accuracy. The algorithm for warping the 3-dimensional coordinates of all the stimulation sites from the individual patients' brains into a common coordinate space for composite visualization on the MNI template brain is detailed in Hamiton et al.[30]

## RESULTS

### Patient Population and Surgical Procedures

Patient demographics are described in Table. The mean patient age was 43.0 ± 13.0 yr. Slightly over half of the patient population was male (6 of 11; 54.5%), and the remainder was female (5 of 11; 45.5%). Nearly all patients (10 of 11; 90.9%) presented with seizures; while 1 patient (9.1%) presented with speech disturbance. Nearly half of the patients (5 of 11; 45.5%) presented with a brain tumor (3 with grade II oligodendrogliomas, 2 with glioblastoma multiforme), 4 patients (33.3%) presented with mesial temporal sclerosis, and 2 patients (16.7%) presented with vascular malformations (1 arteriovenous malformation and 1 cavernous malformation)

Nine of 11 patients (81.8%) were right handed, and 2 patients (18.2%) were left handed. All patients underwent surgery involving the left cerebral hemisphere. Both left-handed patients underwent preoperative Wada testing or magnetoencephalography, which indicated left hemispheric language dominance.

### Intraoperative Mapping Results

Positive language sites (sites where stimulation disrupted or prevented either counting, repetition, naming, or comprehension in the absence of positive motor phenomenon) were identified in 90.9% of patients (10 of 11). In each of these 10 patients, stimulation of the positive sites induced speech arrest. Additionally, stimulation elicited other speech disturbances, including anomia, perseveration, phonological errors, hypophonia, and comprehension errors. Positive motor sites (sites where stimulation elicited involuntary motor movements) were identified in 54.5% of patients (6 of 11).

**TABLE. Features of the patient population**

| Patient | Age (yr) | Sex | Pathology | Frontal lobe speech arrest sites | Frontal lobe motor arrest sites | Speech arrest sites that overlapped with motor arrest sites (ie, NMA) |
|---|---|---|---|---|---|---|
| 1 | 27 | M | Oligodendroglioma (insula) | 1 | 1 | 1 |
| 2 | 38 | F | Mesial temporal sclerosis | 2 | 1 | 1 |
| 3 | 45 | F | Mesial temporal sclerosis | 2 | 2 | 1 |
| 4 | 27 | M | Cavernous malformation (fusiform gyrus) | 2 | 1 | 0 |
| 5 | 46 | M | Mesial temporal sclerosis | 2 | 1 | 0 |
| 6 | 50 | F | Arteriovenous malformation (frontal operculum) | 2 | 1 | 0 |
| 7 | 61 | M | Glioblastoma multiforme (temporal) | 1 | 0 | 0 |
| 8 | 27 | F | Mesial temporal sclerosis | 1 | 1 | 1 |
| 9 | 55 | M | Oligodendroglioma (temporal) | 2 | 1 | 1 |
| 10 | 35 | F | Oligodendroglioma (frontal operculum) | 0 | 0 | 0 |
| 11 | 58 | M | Glioblastoma multiforme (insula) | 2 | 0 | 0 |

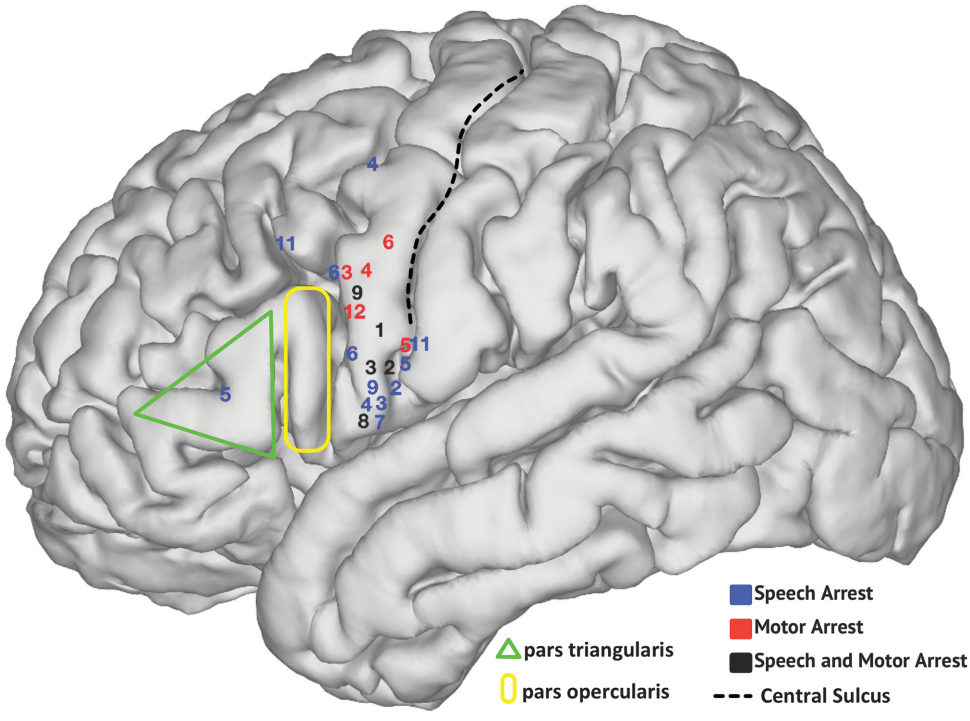
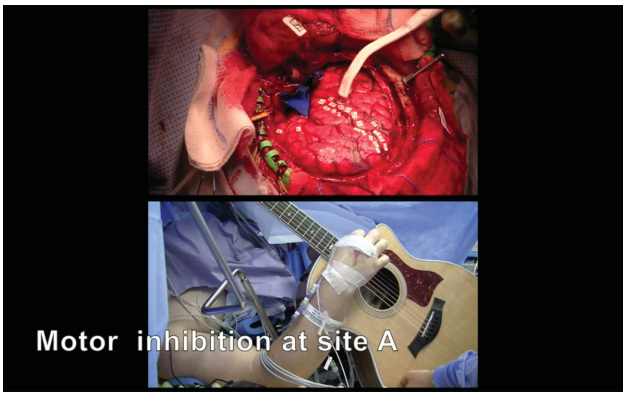



**FIGURE.** *Positive stimulation sites for speech arrest (blue), thumb/finger motor arrest (red), or both (black) from 11 subjects are plotted in a common left hemispheric coordinate space. Altogether, positive stimulation sites were predominantly localized to the inferior precentral gyrus. Sites are labeled by subject number.*

NMAs (sites where stimulation halted ongoing, voluntary hand movements or postures) were identified in 72.7% of patients (8 of 11). Stimulation of sites outside the IFL had no effect on the frequency or amplitude of these ongoing thumb movements. However, stimulation of specific sites in the IFL (depicted in Figure) caused the thumb movements to slow significantly, diminish in amplitude, or halt altogether. When stimulation was stopped, patients resumed the movements/postures




**VIDEO.** *Simultaneous video recording of the cortical surface and patient during intraoperative mapping as Patient 1 plays the guitar illustrating arrest of a complex motor task occurring when a IFL speech arrest site is stimulated.*

that had been halted. Patients reported that during stimulation they still had intent to perform the movements but were unable to do so.

Inferior frontal lobe (IFL) stimulation also produced motor arrest in one patient who was asked to perform a complex motor task. Patient 1 was a 27-yr-old guitarist who underwent awake craniotomy for resection of a low-grade glioma from the dominant frontal operculum and insula. Preoperatively, he expressed concern that resection might impair his ability to play guitar, so stimulation intraoperative mapping was performed while the patient played guitar. First, he underwent the standard mapping procedure as described above. Then, he was given his guitar and instructed to play while cortical stimulation was carried out (Video). In the absence of brain stimulation, he played guitar without hesitation or error. However, upon stimulation of an isolated IFL site (which had caused speech arrest earlier in the mapping procedure; Figure), the patient's right-handed strumming movements were halted, and his hand remained stationary. There were no tonic contractions or abnormal postures elicited by the stimulation. When stimulation was stopped, the patient stated, "I can't play or talk." He then immediately resumed playing as before the stimulation. Repeated stimulation trials were performed, and stimulation of this IFL site elicited the same response in 4 subsequent trials.

### Speech Arrest Sites are Commonly Negative Motor Areas

Across all patients, a total of 25 sites of speech arrest were identified. The majority of these sites (17 of 25; 68%) were located in the frontal lobe. A total of 9 NMAs were identified (all in the frontal lobe). The spatial distributions of speech arrest sites, NMAs, and overlapping speech/motor inhibition sites are depicted in Figure. Positive stimulation sites, regardless of type, were predominantly located in the precentral portion of the inferior frontal gyrus. Notably, 29.4% (5 of 17) frontal lobe speech arrest sites were identified as inhibitory motor sites as well. The site that caused arrest of guitar playing in Patient 1 also caused speech arrest.

In 4 of 11 patients (36.4%), distinct (ie, nonoverlapping) speech arrest and inhibitory motor sites were identified. In 3 of these 4 patients (75%), speech arrest sites were located ventral to the inhibitory motor sites (Figure).

## DISCUSSION

The archetype of language organization suggests that a region of the IFL is essential for speech preparation and production. However, this concept has been challenged on 2 fronts: first, there is increasing evidence that speech function (production and perception) is widely distributed and highly networked across numerous brain regions.[1-3,31] For example, direct cortical stimulation studies have shown that the distribution of speech arrest sites is bilateral.[11,32] Second, numerous functional imaging studies suggest that portions of the IFL participate in a physiologically inhibitory network.[23-25,33-36] In this study, we focused on the relationship between stimulation sites that disrupted ongoing hand movement (ie, NMAs) and sites that caused speech arrest in the IFL. It was initially postulated that stimulation of these sites disrupted a normal neural process producing motor action.[21] However, in light of fMRI evidence that this region is activated during action inhibition, an alternate interpretation is that stimulation activates a physiologically inhibitory network important for "praxic control of fine details of action execution," albeit in a nonphysiologic way, thus arresting speech or ongoing movement.[26]

We found that speech arrest sites were not specific to speech. In 29% of cases, stimulation of speech arrest sites also caused arrest of ongoing thumb movements, as well as more complex hand movements (eg, patient 1, playing guitar). Previous studies have also demonstrated that stimulation of IFL sites can cause pure speech arrest or the arrest of additional motor effectors.[12] One possible unifying explanation of these direct cortical stimulation and neuropsychology functional imaging findings is that the historically conceptualized "Broca's area" (an IFL area that, when stimulated, causes pure speech arrest) may actually be an example of an effector-specific inhibitory motor area essential for speech praxis. Consistent with fMRI results showing activations in the IFL during stop-signal tasks where participants must inhibit an action just before it begins,[23] pure speech arrest sites show arrest of speech in both preparatory and ongoing stages.[27,28] It should be noted that, while direct cortical stimulation with bipolar (eg, Ojemann) electrodes provides a functional map of relatively high (eg, subcentimeter) resolution, it may not be able to discern between language and motor sites that are separated by small distances. We thus cannot completely exclude the possibility that NMAs identified here in fact represent 2 distinct functional sites (ie, 1 with language function and another with inhibitory motor function).

From a clinical perspective, our results suggest that speech arrest sites may be erroneously misidentified as language-specific (ie, as Broca's area) if testing of other motor effectors is not also performed. Intraoperative testing for tongue, hand, arm, or leg action inhibition can be used to differentiate speech-specific NMAs, (which may be essential for fine praxic control of speech), from nonspecific NMAs. Our results suggest that this misclassification could occur in about one-third of cases. A strong body of empiric data shows that postoperative aphasia can be avoided if speech arrest areas are preserved, making the distinction seem trivial in current clinical practice.[8,37-39] However, the observation that there are 2 types of IFL speech arrest sites–one that is speech-specific, and another that is nonspecific–leads to a clinically important question: are both of these sites essential to language function? In previous studies, resection of nonspeech NMAs has caused only transient clumsiness and no permanent motor deficits, although postoperative testing of action inhibition was not reported.[13,16,18] It remains unknown whether resection of nonspecific NMAs associated with speech arrest produces deficits similar to those that occur with resection of or injury to speech-specific NMAs. Resectability of the former, if well tolerated in terms of resultant language effects, could allow for greater extent of resection during neurosurgical procedures. Importantly, no speech arrest site, whether speech-specific or not, was resected in this study population.

Future studies should systematically test various effectors (such as arm, leg, and foot, in addition to hand) during the identification of NMAs, and, in particular, nonverbal tongue movements. In a previous study of NMAs, a mixture of both general and effector-specific inhibitory sites was described, but their distributions were not well characterized.[12] In the current study, only thumb movements were tested; whether these sites were involved in inhibition of other movements (besides those involved in speech production) is unknown. Additionally, to clarify if NMA mechanisms involve actions under preparation, future studies may vary the timing of stimulation relative to movement.[26] Given the results described here, we speculate that NMAs can inhibit effector actions at both preparatory and execution stages. Finally, the small sample size in this study limits the generalizability of our conclusions.

## CONCLUSION

Evidence suggests language organization is more complex and distributed than once believed. Our results indicate IFL stimulation-induced speech arrest is not actually specific to speech: in approximately 30% of cases, stimulation of these sites also inhibited hand motor function. These findings indicate that the IFL may serve a more general role in action inhibition. Further detailed neurosurgical mapping studies, informed by contemporary speech neuroscience and the functional imaging literature, are needed in order to elucidate the complexities of speech organization and ultimately improve the sophistication of our treatment of neurosurgical patients.

## Disclosure

The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

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## COMMENT

This is an interesting, albeit small, series of patients that contributes to our understanding of dominant hemisphere inferior frontal lobe (IFL) language and motor function organization. The authors studied 11 patients with a variety of pathologies who were accumulated over just over 1 year, focusing on the distribution of inferior frontal speech arrest versus inhibitory motor sites. They found that ~30% (5/17) of frontal lobe speech arrest sites were also inhibitory motor sites, while 4/11 patients had distinct, non-overlapping speech arrest and inhibitory motor sites. While this paper is not particularly novel, it does increase overall knowledge of a complex area of the brain that is involved in speech production/inhibition, as well as motor inhibition. As the authors note, the mixed pathologies and relatively small number of patients limits the ability to generalize the findings. However, the study builds on past work and raises several interesting and testable questions for future investigation.

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