Resection of dominant opercular gliosis in refractory partial epilepsy

Report of two cases

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Frontal opercular gliosis in the dominant hemisphere caused medically refractory partial epilepsy in two patients. Both patients were aphasic during their seizures, but otherwise had normal speech. Magnetic resonance images showed well-demarcated lesions resembling tumors in each patient; on heavily T_{1}-weighted images, the lesions were hyperintense compared with normal brain. Cortical mapping with subdural grids localized speech to the area of the lesions; therefore, the resections were performed under local anesthesia and speech was tested throughout the procedure. Postoperatively, both patients were seizure-free and had no new neurological deficits. Well-demarcated lesions, even in the dominant operculum, can be safely removed in patients with medically refractory partial epilepsy.

KEY WORDS • gliosis • epilepsy • language • operculum • magnetic resonance imaging

The frontal, parietal, and temporal opercula in the dominant hemisphere have important speech, motor, and sensory functions. Lesions in these areas often cause debilitating neurological deficits. Penfield and Faulk emphasized that during insular resection for focal epileptic seizures, the frontoparietal operculum could not ordinarily be elevated for fear of producing severe neurological damage and that manipulation of this tissue is dangerous. Rasmussen and Milner stated that frontal excision for epilepsy could be safely performed without loss of speech if the two frontal opercular gyri immediately in front of the precentral gyrus remained untraumatized and without vascular compromise. However, Ojemann, et al., found that the site of language function in the dominant lateral cortex varies greatly and cannot be localized accurately without cortical mapping. Similarly, Lesser, et al., observed speech arrest during electrical stimulation at sites far outside classic speech areas.

Resection of opercular gliosis causing intractable partial seizures in the dominant hemisphere has not been described. We report two cases of well-demarcated gliosis in the left frontal operculum simulating tumors on magnetic resonance (MR) images in patients with medically refractory partial epilepsy. In both patients, removal of the lesion guided by electrocorticography (ECoG) under local anesthesia eliminated clinical seizures and did not cause new postoperative neurological deficits.

Case Reports

Case 1

This 18-year-old right-handed Korean man had been born at term without complication in Korea and reached normal developmental milestones. At 3 years of age, he began having episodes of right-hand weakness followed by a sensation that arose in the epigastric region and moved to his throat. These symptoms were associated with drooling, choking, and the inability to speak, but there was no loss of consciousness, no difficulty with comprehension, and no urinary incontinence. The episodes lasted for 5 to 10 seconds and occurred two to three times per week. Approximately twice a year he had serial episodes at 30-minute intervals that disabled him for several days. He was treated with vitamins in Korea and was told he was deliberately "doing these things to get his way" with his parents.
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FIG. 1. Case 1. Preoperative T-weighted magnetic resonance images (TR 2800 msec, TE 30 msec), axial (left) and coronal (right) views, showing increased signal intensity in the left frontal operculum (arrows).

After moving to the United States at 10 years of age, he was treated with carbamazepine and phenobarbital, which reduced the severity but not the frequency of the seizures. There was no history of trauma or infection, no clear etiology for the seizures, and no family history of seizures or neurological disorders.

Examination. The patient was referred to our institution at the age of 14 years. Physical and detailed neurological examinations showed no abnormalities. Magnetic resonance images revealed a focal area of thickened gray matter in the left frontal operculum that displayed increased signal intensity on the first echo of long-TR/TE sequences (Fig. 1). On the second echo, the intensity of the lesion decreased relative to the surrounding gray matter. Evidence of remodeling of the calvaria directly over the lesion suggested a diagnosis of low-grade glioma. Interictal electroencephalographic (EEG) studies on two occasions showed no abnormalities.

The patient underwent prolonged telemetric EEG recording. After his anticonvulsant medication was discontinued, he had two stereotypical seizures that were clinically compatible with partial seizures arising in the left central opercular region, but lacked evidence of any EEG abnormality. He was treated with a variety of medications, including carbamazepine, phenytoin, phenobarbital, and valproic acid, for 3 years without improvement. Serial MR images showed no significant change in the left frontal opercular lesion. Neuropsychological testing and a Wada test revealed left hemispheric speech and intact memory on the left. His full-scale intelligence quotient was 90.

Operations. When he was 18 years old, the patient underwent left frontotemporoparietal craniotomy while under general anesthesia to place a subdural EEG grid. A protruding mass was seen in the left frontal operculum (Fig. 2). Electrocorticography demonstrated occasional spikes from the left opercular central region just anterior to the lesion. The 64-contact subdural grid was centered over the rolundic sulcus with at least two rows below the sylvian fissure.

Telemetric recordings from the subdural grid were obtained for 10 days. During this time, the patient had four stereotypical seizures, which were characterized by suddenly sitting up, gagging, increased salivation, and drooling; during these episodes he did not respond, verbalize, or follow commands, and his right arm and hand were flexed. About 1 minute after the onset of the seizures, he began to verbalize and follow commands and had no notable weakness in his right arm. The EEG recordings showed that the seizure activity arose from the left midsagittal postcentral gyrus and spread to contiguous contacts (Fig. 3). At no time during the ictus did contacts overlying the cortical lesion show evidence of seizure activity. Cortical mapping with the subdural electrode grid was also performed (Fig. 4).

A left frontotemporoparietal craniotomy was performed with the patient under local anesthesia to remove the subdural grid and resect the focus of the seizures. Electrocorticography did not reveal consistently well-defined epileptiform activity. A portion of the superior postcentral gyrus was resected that corresponded to the area of apparent seizure onset recorded during subdural grid telemetry. Cortical mapping showed that language was localized in the area of the frontal opercular lesion. Two biopsy specimens of the mass were obtained outside the areas over which electrical stimulation caused speech arrest; speech and motor function were monitored throughout the procedure. A protruding lesion in the left frontal operculum (solid arrow) and adjacent to the sylvian fissure (open arrow).

FIG. 2. Case 1. Intraoperative photograph showing a protruding lesion in the left frontal operculum (solid arrow) and adjacent to the sylvian fissure (open arrow).
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FIG. 3. Electroencephalographic recordings from the subdural grid in Case 1. Numbers refer to grid electrodes as shown in Fig. 4. Left: Recording at seizure onset. The first arrow above the time line indicates the onset of well-defined sharp waves from Electrode 47, followed soon after by similar activity in Electrode 48. The second arrow indicates the behavioral onset of the seizure. Right: Recording 20 seconds later, well into the clinical seizure. The electrodes over and near the mass lesion are still not showing evidence of seizure activity. Resection of tissue under Electrodes 47 and 48 did not result in seizure control.

FIG. 4. Case 1. Schematic representation of the cortical map derived from recordings obtained with the subdural grid. The seizure focus appeared to be located in the area of the postcentral gyrus, at a site distant from the opercular lesion. The circle with broken lines shows the extent of cortical resection at the first operation; the circle with a solid line shows the area of the protruding lesion resected at the second operation. The numbers refer to the grid electrodes from which the recordings in Fig. 3 were obtained.

seizure-free for the remainder of his hospitalization; he was discharged on a course of phenytoin. The final pathological diagnosis of the frontal opercular lesion was astrocytic gliosis (Fig. 5).

After discharge, the seizures returned. Five months later, a repeat left frontotemporoparietal craniotomy was performed with the patient under local anesthesia to remove the left opercular lesion. Electrical stimulation of the area over the frontal opercular lesion caused tongue and pharynx movement and speech arrest. The lesion was resected piecemeal; speech function was tested throughout the procedure. The mass was very firm and was totally resected except for an area at the posteroinferior margin. The patient had no change in his ability to speak during the resection. The pathological diagnosis was gliotic cerebral cortex.

Postoperative Course. Postoperatively, he had no new neurological deficits and no seizures while taking phenytoin. Postexcision ECoG showed no evidence of epileptiform activity. At 18 months after surgery, he is seizure-free while taking phenytoin.

Case 2

This 10-year-old left-handed Chinese girl was referred at 7 years of age for treatment of medically refractory partial seizures. She was born without complication at 37 weeks' gestation and reached all developmental milestones appropriately. The first seizure, characterized by tonic extension of her right arm, occurred at 18 months of age. There was no history of head trauma, infection,
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![Figure 5](image1.png)  
**Fig. 5.** Photomicrographs of the lesion in Case 1. PTA-hematoxylin, × 20. *Left:* Section showing dense perivascular gliosis in the cortex. *Right:* Section showing dense gliosis in the cortex.

![Figure 6](image2.png)  
**Fig. 6.** Case 2. Preoperative **T**$_1$-weighted magnetic resonance images (TR 2000 msec, TE 70 msec), axial (left) and coronal (right) views, showing increased signal intensity in the left frontal operculum (arrows).

or clear etiology for her seizures, nor was there a family history of seizures or neurological disorders. The seizures were of two types. One type was characterized by tonic posturing of the right arm and a "dreamy" state in which she was able to follow simple commands; she occasionally had an aura described as a tingling sensation in her entire body. These episodes lasted 5 to 30 minutes, occurred one to 10 times per day, and were not associated with postictal confusion. The second type involved staring and occasional loss of consciousness; these episodes lasted 5 to 30 seconds, were less frequent than the other type, and did not cause postictal deficits. Treatment with phenobarbital, carbamazepine, and valproic acid alone and in various combinations did not control the seizures. Magnetic resonance images showed an abnormality in the left frontal opercular region, but hexamethyl propylamine oxime single-photon emission computerized tomography (CT) and numerous interictal EEG recordings revealed no abnormalities.

**Examination.** Prolonged inpatient telemetry showed evidence of seizure activity arising from the left hemisphere, but a clear epileptic focus could not be identified. The patient was treated with anticonvulsant drugs for 1 year but did not improve. Neuropsychological testing and a Wada test revealed left hemispheric language and intact memory on the left. Her full-scale intelligence quotient was 74. Her physical examination was normal except for hyperreflexia and bilateral ankle clonus. A repeat MR image showed an area of increased signal intensity on the first echo of long-TR **T**$_1$-weighted sequences in the left opercular region (Fig. 6) that was unchanged from previous studies.

**Operations.** A left frontotemporoparietal craniotomy was performed with the patient under general anesthesia. Intraoperative cortical recordings showed rare brief spikes from the left opercular and mid-central region, just superior to an area of firmness noted by palpation. Ultrasound examination showed no discrete lesion in this area. Cortical mapping demonstrated a few motor responses in the tongue and lower part of the face, but was limited by after-discharges. Intravenous infusion of etomidate produced bursts of rhythmic, large-amplitude spikes from the central region. A 64-contact subdural grid was centered over the central sulcus with two rows over the superior temporal gyrus.

Telemetric recordings with the subdural grid were obtained for 7 days. During this time, the patient had several seizures. The recordings demonstrated frequent spikes from the left opercular central region. Brief after-discharges centered in the same region were occasionally recorded during electrical stimulation. Her pre- and postcentral gyri and language areas were identified by cortical mapping (Fig. 7). Electrical stimulation directly over the firm lesion caused tongue and pharyngeal movement and arrested her speech.

A left frontotemporoparietal craniotomy was performed with the patient under local anesthesia to remove the subdural grid and resect the opercular mass. Intraoperative ECoG showed epileptiform activity in the left frontal opercular region. A firm, well-demarcated lesion was completely removed in piecemeal fashion; speech was monitored repeatedly. Postexcision
Anatomical Considerations

"Operculum," meaning lid or cover, describes a small portion of telencephalic mantle covering the insula of Reil. The operculum is divided into the frontal, parietal, and temporal opercula. Lesions in the frontal operculum often cause speech dyspraxia or faulty intonation, stress, and phrasing of words and sentences. If the adjacent sensorimotor cortex is involved, dysarthria and dysphonia are more prominent. Although many believe that functions related to motor speech are located only in the inferior portion of the third frontal convolution, electrical stimulation of various areas of the dominant lateral cortex can arrest speech.

Both of our patients had well-demarcated gliotic lesions in their dominant frontal opercula without associated neurological deficits. Their seizures were typical of those associated with frontal opercular lesions. Both had speech arrest or aphasia (although in Case 2 comprehension was spared), and both had right-arm weakness and dysfunction. Case 1 also had typical visceral symptoms of insular stimulation adjacent to the frontal operculum, including epigastric sensations, choking, excessive salivation, and drooling. Gustatory hallucinations have been induced by electrical stimulation of the opercular opercula in patients with gustatory seizures.

Ojemann, et al., found great individual variation in the location of language in the dominant hemisphere. In 10 patients with medically refractory seizures, the only area in which naming errors were consistently evoked by electrical stimulation was the gyrus anterior to the inferior portion of the sensorimotor cortex; stimulation of other areas traditionally referred to as speech cortex had quite variable effects. In a review of 117 patients undergoing electrical stimulation mapping of the left, dominant hemisphere, Ojemann, et al., found that individual language centers were usually highly localized, forming multiple mosaics of 1 to 2 sq cm in 67% of the patients studied. However, these mosaics were identified in numerous sites throughout the lateral cortex, often far removed from the traditional Broca's or Wernicke's areas. Naming errors were consistently evoked only by stimulation of the inferior posterior frontal cortex, but 21% of patients mapped in this area did not have significant naming difficulty. The authors concluded that language cannot be localized accurately by anatomical considerations alone and that techniques such as stimulation mapping must be used. This variability also suggests that cortex in some classic language areas may be safely resected without causing aphasia in selected patients.

Imaging Studies

Magnetic resonance imaging is the preferred method for evaluating patients with intractable epilepsy.

ECOg showed no evidence of well-organized epileptiform activity.

Postoperative Course. The postoperative course was uneventful and without seizure activity. The patient was discharged 5 days after surgery and placed on a regimen of valproic acid and carbamazepine. She was neurologically normal except for bilateral ankle clonus. The final pathological diagnosis was reactive astrocytic gliosis.

Magnetic resonance images obtained 2 months after the operation showed encephalomalacia at the site of resection. The patient had an occasional aura but had no seizures for 4 months postoperatively (Fig. 8). However, she was readmitted for nonstereotypical seizures 5 months after her corticectomy. Clinical evaluation and inpatient EEG monitoring were most consistent with pseudoseizures. With counseling for numerous psychosocial problems within her family, these episodes have been controlled. She has remained seizure-free for 3 years.

Discussion

Anatomical and Functional Considerations

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Surgical Management

The surgical management of frontal lobe epilepsy in the dominant hemisphere has been described by Rasmussen and Milner. They stated that the cortex near the speech area can be safely removed by gentle suction while the patient performs repeated verbal tasks, but at least one opercular convolution in front of the anteriormost gyrus that produces aphasia when stimulated should be preserved. If aphasia cannot be elicited, the first three dominant opercular gyri anterior to the lower end of the precentral gyrus should be preserved. The authors emphasized that lack of speech disruption by electrical stimulation does not guarantee that the stimulated cortex is not involved in speech function. Similarly, Olivier recommended that 2 cm of the posterior extent of the third frontal gyrus be left undisturbed to avoid speech deficits. He pointed out that anatomical safety margins are necessary because electrical stimulation of the third frontal gyrus often does not yield speech disturbances.

Non-neoplastic frontal lobe lesions that cause medically refractory seizures are rare. To our knowledge, resection of dominant opercular gliosis causing intractable epilepsy has not been described. Gregorie and Goldring reported the subtotal resection of a left posterior frontal opercular glioblastoma multiforme in a 37-year-old woman with refractory partial seizures. Somatosensory evoked responses and cortical stimulation were used to localize the sensorimotor region while the patient was under general anesthesia. She had a postoperative hemiparesis and dysphasia, which improved until she died 11 months later. Berger, et al., used language-mapping techniques to guide the resection of a ganglioglioma in the sensorimotor region of a 4-year-old boy.

Resection of opercular lesions in the dominant hemisphere has been described in patients without seizures. Cosnett, et al., excised a 3- to 4-cm nocardial brain abscess in the right frontal operculum of a 67-year-old left-handed man who had bilateral opercular syndrome (and had apparently suffered an infarction in the contralateral operculum). General anesthesia was used, and EEG was not performed. Postoperatively, he had improving left-arm, lower-face, and tongue movement, and anarthric speech. Zangwill excised an astrocytoma occupying the entire left frontal operculum in a 70-year-old right-handed man who had speech difficulty, a change in personality, and a right-sided hemiparesis. Although the patient was mute for 2 days, evaluation 4 weeks later showed normal speech and writing, except for mildly decreased fluency. Zangwill also excised a large, deep, left frontal cystic tumor in a 37-year-old right-handed man in whom Broca's area was "totally undercut." This patient was also mute postoperatively, but within 3 months he had regained normal speech and writing, except for some mild loss of fluency. Cortical stimulation was not used in either case.

Hecaen and Consoli reported 12 right-handed patients in whom acquired lesions in Broca's area were

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**Fig. 8. Case 2. Postoperative T₁-weighted magnetic resonance images (TR 600 msec, TE 20 msec), axial (left) and coronal (right) views, obtained after administration of gadolinium, showing encephalomalacia at the site of surgical resection.**

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10,31 It is far more sensitive and specific than CT in determining the presence or absence of intracerebral lesions, but often it cannot demonstrate abnormalities due to gliosis found at surgery. Bergen, et al., found that MR imaging allowed clear discrimination between tumors and non-neoplastic lesions in 23 patients undergoing surgery for intractable epilepsy. The T₁-weighted studies were normal in all 12 patients with non-neoplastic lesions, nine of which were gliosis. Other authors have also failed to find any abnormalities on T₁-weighted images in epileptic patients with gliosis. In both of our patients, MR images showed a focal area of increased signal intensity on heavily T₁-weighted images that was most prominent on the first echo, possibly because both lesions were nodular and well demarcated. We did not use gadolinium-diethylentriamine penta-acetic acid (DTPA) in either case. It is unclear whether administration of gadolinium-DTPA increases the diagnostic yield. At least one study has shown no added benefit of gadolinium-DTPA in preoperative evaluation of patients with intractable partial epilepsy. Reactive gliosis may resemble glioma on both CT scans and MR images and must be considered in the differential diagnosis of nonenhancing lesions with increased signal intensity on T₁-weighted MR images. Our cases provide further evidence that MR imaging is probably sensitive, but not specific, for detecting lesions in patients with intractable epilepsy.

**Pathology**

It is possible that one or both of these lesions was a slow-growing neoplasm. This is of special concern in Case 1, in which MR images showed evidence of remodeling of the calvaria. However, the histopathological features of both lesions were entirely consistent with gliosis. Neither patient has shown clinical or radiographic evidence of recurrence 18 months (Case 1) or 3 years (Case 2) after resection of the lesion.

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Hecaen and Consoli reported 12 right-handed patients in whom acquired lesions in Broca's area were
resected. Postoperatively, five patients had mild articulatory disorders and/or mild dysprosody with agraphia, and seven patients had moderate disorders in expressive speech, writing, and calculation and impaired comprehension. The poorer outcome in the latter group was attributed to the deeper location of the lesions. In addition, it has been suggested that slow-growing tumors may displace convolutions and cause dysphasia, and thus excision of these masses may not affect Broca’s area.

Although gliosis is a common pathological finding in refractory epilepsy, the results of surgery for extratemporal lesions have usually been poor. Goldring reported that four of six children who underwent surgery for extratemporal focal neuronal dropout and astrocytosis of unknown etiology continued to have poorly controlled seizures; only two had a significant decrease in seizure frequency. He noted that better results could have been achieved if the resections had not been limited by motor or language cortex.

Both of our patients had superficial lesions that were visible or palpable on the brain surface and that did not significantly displace the convolutions. Both resections were performed with the patient under local anesthesia, because cortical mapping with subdural grids localized language function to the area of the lesions. In both patients, the opercular gliosis was resected without causing new neurological deficits. Perhaps the lesions were located in areas involved in but not essential for speech. In addition, there has been no recurrence of seizure activity during 18 months (Case 1) and 3 years (Case 2) of follow-up study. Although we realize that the length of the follow-up period is limited, these results strongly suggest that surgical resection of such lesions reduces seizure frequency.

The fact that both of our patients had well-demarcated lesions probably facilitated more complete resections. In Case 2, the seizures appeared to arise from the left frontal opercular lesion, which was therefore resected. However, in Case 1, subdural grid recordings during four clinical seizures showed no epileptiform activity adjacent to the lesion, but did show an epileptic focus at a distant site in the superior portion of the postcentral gyrus; therefore, a biopsy rather than a resection was performed on the left frontal opercular gliotic mass. The patient continued to have seizures. At reoperation, stimulation of the left frontal opercular nodule resulted in speech arrest. Piecemeal removal of the lesion with frequent speech testing allowed complete resection without causing a deficit and this eliminated the seizures. It appeared that the speech arrest elicited by electrical stimulation resulted from interference with motor function of the mouth and pharynx, not from interference with an area necessary for language. In addition, resecting a portion of somatosensory cortex did not cause a demonstrable sensory deficit. Rasmussen and Milner have reported that portions of the postcentral gyrus can be resected with little sensory deficit. Unfortunately, two-point discrimination, which might have shown a minor deficit, was not tested in this patient. This case demonstrates the potential inaccuracies of using subdural grids to localize an epileptic focus; moreover, the use of a subdural grid may not always eliminate the need for craniotomy using local anesthesia for surgery in areas critical for speech. Removal of well-demarcated lesions, even those in the dominant operculum, is necessary in the surgical management of refractory partial epilepsy.

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