Surgery for Frontal Lobe Epilepsy

The treatment of frontal lobe epilepsy is especially challenging due to the complexity of the seizures, the difficulty of using surface EEG for ‘hidden’ areas such as the medial frontal lobe, and the risk of motor or language deficit following resection of eloquent cortex. Surgery for frontal lobe epilepsy is nevertheless common, accounting for 10 to 20% of all surgical epilepsy cases1. Multimodal diagnostic data must be consistent to recommend surgical resection.

Semiology: Frontal lobe seizures are typically short, often nocturnal, and frequently occur without post-ictal confusion. An aura of arm or leg paresthesias often precedes focal clonic activity originating from motor cortex, or versive head or eye movements from frontal eye fields2. Early ‘hypermotor’ activity, i.e., complex vigorous coordinated bilateral limb movements, distinguish frontal lobe from temporal lobe seizures. At seizure onset, bilateral asymmetric posturing indicates medial frontal (supplementary motor area) involvement.

Drug therapy: About 40% of patients with epilepsy continue to have seizures despite medical management1, and can be considered for surgical treatment.

Evaluation for Surgery: Assessment includes review of:

- **EEG/Video Monitoring (Phase 1):** Recording from deep sites such as the medial frontal and orbitofrontal cortex is difficult, with ictal EEG localization or lateralization occurring only in 25% of mesial frontal epilepsy2, and interictal discharges occurring in only 60% of patients1. Ultimately, ictal surface EEG provides localization in only 30 to 40% of frontal lobe epilepsy patients1, and intracranial EEG (Phase II) recordings are often required.

- **MEG:** Magnetoencephalography can identify the location of epileptiform discharges and is best for dorsolateral frontal lobe seizure foci. MEG also provides non-invasive mapping of language and somatosensory cortex.

- **MRI:** MRI is often normal (‘non-lesional’) as in our case, but can reveal lesions such as cortical dysplasia, tumors and vascular malformations.

- **SISCOM:** SISCOM (Subtraction Ictal SPECT CO-registered to MRI) correctly identifies the seizure focus in about 63% of cases4.

- **PET:** Abnormal isotope uptake is found in 35 to 45% of non-lesional frontal lobe epilepsy cases1.

- **Neuropsychological testing:** Impairments in executive function can indicate frontal lobe dysfunction.

- **Phase II:** Grids (arrays of electric contacts) are implanted into the subdural space, and depth electrodes (wires containing electric contacts) into the brain parenchyma in order to more precisely monitor EEG when localization cannot be achieved non-invasively. These procedures allow more precise definition of the ‘epileptogenic’ and ‘irritative’ seizure zones, and provide access to those regions not well sampled by surface EEG. Extensive electrode coverage of the frontal areas yields localization in more than 90% of patients2.

Surgery: Epileptogenic areas are resected through a craniotomy by removal of cortical gray matter of the implicated gyri. Intraoperative navigation and electrocorticography allow the surgeon to precisely localize the area to be resected. For eloquent areas such as language and motor cortex, multiple subpial transection - placement of small, parallel cuts at 4 mm intervals transversely through the gyri - can interrupt seizure spread while minimizing the risk of long term impairment.

Outcome: Among frontal lobe epilepsy cases, the chance of becoming seizure-free after surgery is 55.7% at one year. Seizure-free rates have been reported as 62% for low-grade tumors, 52% for MRI-visible malformations of cortical development2, and are likely to be lower for non-lesional frontal lobe epilepsy.

Case

A 16 year old male presents with a five year history of seizures. He has an aura of right arm tingling, followed by right arm rhythmic jerking, versive head turning to the right, and secondary tonic-clonic activity. A right-sided two hour Todd's paresis usually follows afterwards. He has nightly seizures despite antiepileptic polytherapy.

Phase I

Surgical evaluation produced the following:

- **Video/EEG monitoring:** EEG recorded interictal bifrontal epileptiform discharges and three seizures. The seizures appeared as diffuse bursts followed by desynchronization and rhythmic fast activity from the left frontal-central area.
- **MRI:** Normal without cortical dysplasia.
- **PET:** Decreased bilateral posterior frontal lobe radioisotope uptake.
- **SISCOM:** Bilateral regions of increased radioisotope uptake.
- **MEG:** Clusters of dipoles were detected over the left pre-motor dorso-lateral frontal lobe (Fig 1).
- **Neuropsychological testing:** Subtle frontal lobe executive dysfunction.

Phase II

A craniotomy was performed to place subdural grids over the lateral and medial left frontal lobe (Fig 2).

Surgery

Resection of focus in the left medial frontal lobe, including part of the supplementary motor area. Multiple subpial transections over the medial premotor region (Fig 3). Pathological diagnosis was micro-dysgenesis with gliosis.

Outcome

He has had a significant reduction of seizures and no neurological deficits (follow-up 18 months).

For more information or to refer a patient, call 706-721-4626 or email epilepsy@georgiahealth.edu