Intracranial electroencephalographic monitoring is an integral component in localization of the epileptogenic zone in 25%–40% of patients who undergo surgical treatment of epilepsy, particularly in cases with abnormal intracranial anatomy and in pediatric patients. A stable anatomical relationship between the intracranial electrodes and the cortical surface is critical in guiding resection strategies. Migration of electrodes has been recognized as an impediment to proper identification of the epileptogenic zone. Some authors advocate fixation of an electrode grid to the dura mater while others depend on friction to hold a grid in place. Neither of these techniques is feasible when attempting to anchor the grid on the interior surface of a fluid-filled space, such as an arachnoid cyst.

Arachnoid cysts are frequently encountered in patients with epilepsy and significantly alter the surrounding anatomy. While there is no clear causal relationship between arachnoid cysts and seizures, in many cases patients may have both intractable localization-related epilepsy and arachnoid cysts in the suspected epileptogenic zone. Placing subdural electrodes within a decompressed arachnoid cyst poses a technical challenge due to the concave architecture of the cortical surface and the tendency of the electrodes to migrate away into the cavity. While not FDA-approved for use outside of cardiothoracic and abdominal surgery, fibrin sealant is used in neuro-surgery as a sealant and adhesive. In this report we describe the fixation of a grid of subdural electrodes to the cortical surface within a decompressed arachnoid cyst using fibrin sealant.

Case Report

History and Examination. A 4-year-old girl presented with a history of medically intractable, symptomatic, localization-related epilepsy consisting of complex partial seizures with and without secondary generalization. At the age of 3, she had developed headaches and underwent imaging, revealing a large left-sided arachnoid cyst and subacute subdural hematomas that were larger on the right side than on the left side. The right-sided subdural collection was drained via a bur hole. Several days later, she developed the acute onset of expressive aphasia and right lower-extremity weakness. She also began having staring episodes. An MR image revealed ischemic injury in the left frontal lobe, and scalp electroencephalography (EEG) demonstrated seizure activity in the left frontal region. The left-sided subdural collection was drained and the seizures were eventually controlled with multiple medications. She subsequently had undergone fenestration of the left arachnoid cyst without significant change in seizure activity (Fig. 1A). Clinically, surgery as a sealant and adhesive. In this report we describe the fixation of a grid of subdural electrodes to the cortical surface within a decompressed arachnoid cyst using fibrin sealant.

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her seizures consisted of staring with unresponsiveness, accompanied by lip-smacking and drooling. There was occasional rightward eye deviation and right-sided facial twitching. Episodes typically lasted 2 to 5 minutes. She underwent long-term video scalp EEG monitoring, which suggested a left temporoparietal seizure onset. Given the severity of the seizures and the focal scalp EEG findings, the patient’s family was offered surgery for the patient for placement of subdural electrode grids to further define the epileptogenic zone. They elected to proceed with the understanding that, depending on the findings, a resection of epileptogenic tissue may be offered.

**Operative Technique.** A large left frontotemporoparietal osteoplastic craniotomy was performed. The dura mater was opened and the underlying arachnoid cyst was clearly visualized. The lateral cyst wall, which was notably thickened (Fig. 2 left), was resected. The lateral portions of the frontal, temporal, and parietal lobes could be clearly visualized beneath the medial wall of the cyst. An 8 × 8–cm, 64-contact platinum electrode grid (Integra) was divided to create two 4 × 8–cm, 32-contact grids. Four contacts were trimmed from the superior frontal corner of 1 grid to accommodate the cerebral exposure.

The grids were positioned over the frontal, parietal, and temporal lobes to confirm a proper fit (Fig. 1C).

Strips of compressed gelatin sponge (Gelfoam, Pharmacia & Upjohn Co.) were applied to the margins of the grids to prevent fibrin sealant from seeping into the space between the electrodes themselves and the cortical surface. Fibrin sealant (Tisseel, Baxter Healthcare Corp.) was applied over the gelatin sponge, forming an adhesive bridge between the adjacent cortical surface and the grid. Five sentinel electrode strips were placed in the subdural space at the periphery of the exposed cerebrum. The electrical leads from the electrodes were secured to the margins of the dura with 4-0 braided nylon suture (Fig. 2 right) and tunneled through the skin to an adjacent exit site. The leads were secured at the skin. The electrodes were individually tested prior to wound closure. The dura mater was closed with dural substitute (Suturable DuraGen, Integra LifeSciences) and the bone was replaced without rigid fixation. The scalp was closed in anatomical layers with resorbable suture, the wound was dressed, and anesthesia was reversed.

Postoperatively, a CT scan was performed to evaluate for any immediate complications. The patient was ob-

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**Fig. 1.** Imaging findings before and after grid placement. Preoperative (A) and postoperative (B) T2-weighted axial (upper row) and coronal (lower row) MR images. Open arrows indicate the position of the grid against the cortical surface, whereas solid arrows indicate the position of the overlying DuraGen. A lateral skull radiograph (C) and 3D reconstruction of postoperative MRI and CT (D) reveal the grid location.
served in the intensive care unit overnight and MRI was performed the next morning to evaluate grid placement (Fig. 1B). Using a previously described technique, a 3D model of the electrodes in relation to the brain was built from these images (Fig. 1D). Continuous monitoring of the electrical activity was undertaken immediately post-operatively and antiepileptic medications were gradually stopped. The EEG recordings demonstrated adequate signal to noise without loss of signal from any of the grid electrodes.

Four electroencephalographic seizures were recorded during 6 days of monitoring. These seizures consisted of a focal, subfrontal, high amplitude spike-wave pattern that evolved into rhythmic focal spikes in adjacent grid contacts. Generalized rhythmic lower-amplitude fast-frequency activity occurred in association with the focal discharges. Spike activity persisted and became more discontinuous and widespread with polyspikes, then ended abruptly. The highest amplitude and earliest signal correlated with anterior frontal and orbitofrontal electrodes. An attempt at extraoperative stimulation language mapping during monitoring was not successful. Based on the intracranial EEG recordings, resection was planned for the anterior and inferior frontal lobe, sparing the lateral frontal region typically housing Broca’s area.

The patient was returned to the operating room 5 days after placement of the intracranial electrodes. Positioning and approach were identical to the initial procedure. The dural patch was removed and the electrodes had not appeared to have migrated (Fig. 3). The electrode grids were easily removed from the cortical surface without evidence of underlying injury. Resection of the frontal pole sparing the inferior frontal gyrus was performed and the dura mater was closed with dural substitute (Durepair Regeneration Matix, Medtronic). The cranium was replaced and fixated, and the remainder of the wound was closed without difficulty. The patient emerged from anesthesia in stable condition.

**Postoperative Course.** Postoperatively, the girl exhibited a mild right-sided facial droop that improved over the course of several days. Antiepileptic drugs were continued. The patient was discharged home on the 5th postoperative day without suffering any clinical seizures. Her last clinical evaluation was approximately 7 months following surgery. According to her parents, she had remained free of clear clinical seizures. At the discretion of the patient’s neurologist, she had continued on antiepileptic medications. Electroencephalography at that time revealed a background free of epileptiform discharges and neither photic stimulation nor hyperventilation induced a seizure.

**Discussion**

Arachnoid cysts are common and occur at a prevalence of 0.5% of the general population in autopsy studies. As in this case, cysts tend to have a predilection for the middle cranial fossa, particularly in patients with epilepsy. Despite multiple attempts, no consistent causal relationship has been established between arachnoid cysts and seizures. Fenestration of the cyst alone yields mixed results in control of seizures, emphasizing that correlation between EEG findings and anatomy is particularly important in the surgical treatment of these patients. A consistent relationship between the cortical surface and electrode array is paramount in accurately defining the epileptogenic zone. This relationship is often tenuous when attempting to fit a flat matrix of electrodes to a concave surface, as within a cyst cavity. In an effort to ensure greater anatomical fidelity between the electrodes and the cortical surface, Tisseel fibrin sealant was used.

While Tisseel only carries FDA approval for hemostasis and secondary sealing of anastomosis during bowel surgery, off-label use in neurosurgery is widely reported. No known adverse effects on the CNS have been reported with Tisseel use, but its use on brain parenchyma has not been evaluated by the FDA. Numerous investigators have evaluated its use for adjunctive closure of dura in cranial, skull base, and spine surgery. A few reports have also illustrated its use in closing corticotomy defects. The properties that allow fibrin glue to be effective in dural closure and sealing of corticotomy defects.
also make fibrin glue attractive for temporary anchorage of intracranial electrodes, including adherence to surrounding tissue, complete resorption with minimal reactive changes, and lack of neurotoxicity.\textsuperscript{4,5,11}

Per the manufacturer, Tisseel fibrin sealant consists of two components. The first component is thrombin obtained from pooled human plasma. The second is a mixture of fibrinogen and other proteins from pooled human plasma and purely synthetic aprotinin, a fibrinolysis inhibitor. When combined, the two components form a rubber-like mass of fibrin that adheres to surrounding tissue. The fibrin mass is degraded by normal fibrinolysis and is expected to be completely resorbed after 14 days.

Careful consideration was given to the risks of using fibrin "glue" in this application, particularly since the involved cortex must remain electrophysiologically unaffected during intracranial EEG identification of the epileptogenic zone. Rodent parenchyma exposed to thrombin exhibited cerebral edema, neuronal injury, and seizures.\textsuperscript{11} However, de Vries’s histological studies of fibrin sealant on rodent brain and Kassam’s study of Tisseel on nonhuman primate brain showed a lack of abnormal inflammation.\textsuperscript{4,11} Schlag and colleagues reported seizures following subdural application of a fibrin sealant in rats, but subsequently showed that tranexamic acid, a synthetic fibrinolysis inhibitor not contained in Tisseel, produced seizures.\textsuperscript{18} Development of antibodies against bovine thrombin and factor V resulting in prolongation of thrombin time during various surgical procedures has been reported, although none in neurosurgical use.\textsuperscript{3} Reports of clinical experience applying fibrin sealant to brain parenchyma argued for clinical safety.\textsuperscript{10,12} Although a compound containing bovine thrombin was sometimes used.

In practice, no complications were encountered related to the use of fibrin sealant. Of particular concern was removal of the electrodes without damage to the underlying tissue. In this case, the grid was easily removed without the need for excessive traction and there was no evidence of damage to the underlying pia (Fig. 3 right). Some petechial hemorrhage could be noted within the anterior temporal lobe, but was distant from the site of fibrin sealant use. Use of a gelatin sponge to separate the sealant from the electrode itself was helpful, as the sponge could be easily divided without attempting to remove all fibrin sealant from the parenchyma. The authors also noted slightly more acute blood product surrounding the grids than is typically encountered (Fig. 3 left). This appeared to be due to a lack of tamponade in the large potential space of the cyst cavity, rather than coagulopathy.

This was the first report of which these authors are aware of anchoring cortical electrode arrays with fibrin sealant during epilepsy surgery. The patient had an excellent outcome without complication related to the use of the sealant. While not advocating for the routine use of fibrin sealants to anchor intracranial electrodes, this technique may prove valuable when addressing electrode coverage of complex intracranial surfaces. Further use of this technique may provide a clearer understanding of the relationship between the structure of arachnoid cysts and the location of epileptogenic tissue in patients with both arachnoid cysts and seizures.

**Disclosure**

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Hoyt, Lew. Acquisition of data: Lew. Analysis and interpretation of data: LaViolette. Drafting the article: Hoyt. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Hoyt. Administrative/technical/material support: Hoyt.

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Fibrin sealant to prevent subdural electrode migration


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