A rachnoid cysts account for 1% of intracranial space-occupying lesions. They are thought to arise primarily from congenital splitting of arachnoid membranes during development or secondarily due to trauma or inflammation. However, the precise etiology of these cysts remains to be elucidated. Intraparenchymal hemorrhage has been reported as a very rare complication of open fenestration of an arachnoid cyst in the literature. Rapid evacuation of arachnoid cysts and chronic subdural hematomas, however, has been associated with intracerebral hemorrhage. It is thought that this rapid decompression causes an abnormality of cerebral vasculature, autoregulatory dysfunction, and subsequent reperfusion injury. While the pathogenesis of this complication remains unknown, there is some evidence that gradual slow decompression of large sylvian fissure arachnoid cysts may decrease the incidence of these complications.

We report the case of an 18-year-old female patient who presented with intractable progressive headaches with evidence of increased intracranial pressure (ICP). She was found to have a Galassi III arachnoid cyst (Table 1). The patient underwent craniotomy and open fenestration of the cyst. This case was complicated by postoperative intraparenchymal hemorrhage, which we speculate to be a sequela of reperfusion injury. This case highlights a complex, rarely reported postoperative complication of open fenestration.

**Hemorrhagic infarction following open fenestration of a large intracranial arachnoid cyst in a pediatric patient**

Tyler Auschwitz, MD, Michael DeCuypere, MD, PhD, Nickalus Khan, MD, and Stephanie Einhaus, MD

1Department of Neurosurgery, University of Tennessee Health Science Center; 2Semmes-Murphey Neurologic & Spine Institute; and 3Le Bonheur Children’s Hospital, Memphis, Tennessee

Intracranial arachnoid cysts are a rare condition thought to be congenital in nature. Treatment of intracranial arachnoid cysts remains controversial based on their variable presentation. Treatment options include CSF shunting, endoscopic fenestration, or craniotomy and open fenestration for larger cysts. The complications of these procedures can include hydrocephalus, subdural hematomas, hygromas, and—more rarely—intraparenchymal hemorrhage. The authors found very few reports of hemorrhagic infarction as a complication of arachnoid cyst fenestration in the literature. The authors report a case of an 18-year-old female patient who suffered an ipsilateral hemorrhagic infarction after craniotomy for open fenestration of an arachnoid cyst.

http://thejns.org/doi/abs/10.3171/2014.9.PEDS14126

**KEY WORDS** arachnoid cyst; infarction; craniotomy; reperfusion injury; congenital
headaches had been progressively worsening over recent years. She also reported some numbness and tingling periodically in her hands and feet. Bilateral papilledema was noted on fundoscopic examination. The remainder of the physical examination was unremarkable. Standard preoperative metabolic and coagulation studies were performed, and the values were within normal limits for age.

Operation

The patient was taken to the operating room and placed under general anesthesia. Normotensive blood pressure was maintained throughout the case. The patient was placed in a Mayfield head holding device, and the head was turned 30° to the right. A frontotemporal incision was made, and a small pterional craniotomy was performed. The dura was opened carefully, leaving the arachnoid cyst membranes intact. The arachnoid was visibly bulging and appeared to be under increased pressure. The arachnoid cyst self-decompressed, and CSF began draining from the dural incision. The lateral/outer membranes were resected, allowing direct visualization of the internal carotid and middle cerebral arteries (Fig. 2). The opticocarotid cistern was then fenestrated, and flow was established across this fenestration. Of note, the carotid artery was not manipulated, and there was no direct visual evidence of vascular spasm. The bone flap was replaced with titanium plates, dura and skin were closed in standard fashion, and the patient was extubated in the operating room. She was then taken to the recovery room in stable condition.

Postoperative Course

The patient was awake and had normal speech and stable vital signs immediately after surgery. She was noted to be moving all of her extremities with full strength. Ninety

<table>
<thead>
<tr>
<th>Type</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Small, spindle-shaped, limited to anterior middle cranial fossa, usually w/ free communication of CTC.</td>
</tr>
<tr>
<td>II</td>
<td>Superior extent along sylvian fissure w/ displacement of the temporal lobe.</td>
</tr>
<tr>
<td>III</td>
<td>Large, fills the whole middle cranial fossa, w/ displacement of not only the temporal lobe but also the frontal &amp; parietal lobes. Little communication w/ subarachnoid space on CTC.</td>
</tr>
</tbody>
</table>

CTC = CT cisternography.

FIG. 1. Preoperative axial T2-weighted (A and B) and coronal (C) and sagittal (D) T1-weighted MRI sequences demonstrating a large left frontotemporal arachnoid cyst.

FIG. 2. Intraoperative images of the arachnoid cyst before (upper) and after (lower) fenestration, allowing visualization into the opticocarotid cistern. Figure is available in color online only.
minutes after the conclusion of the surgical procedure, the patient suffered a partial seizure with secondary generalization in the recovery room. Emergency CT imaging of the head was performed and revealed 3 foci of intraparenchymal hemorrhage in the left middle cerebral artery territory (Fig. 3). It was thought that this most likely represented reperfusion injury. The hematoma was deemed inoperable due to its location within eloquent cortex and lack of mass effect. While in the radiology department, the patient had an apneic episode. She was urgently intubated and transferred to the ICU for further management.

In the ICU an ICP monitor was placed, which showed low-normal ICP. The patient was placed on a regimen of antiepileptic medications and was monitored closely. It was soon noted that the patient was hemiparetic on the right side. The patient remained under close observation in the ICU for an extended period.

The patient received extensive physical, occupational, and speech therapy while she was an inpatient. At discharge from the hospital, she had slight movement of her right upper and lower extremities and had some degree of expressive aphasia. She was transferred to a rehabilitation center for further care.

Follow-Up

The patient spent approximately 4 weeks at an intense inpatient rehabilitation hospital. The patient made substantial gains in function over this time period. At the 9-month follow-up, the patient was doing well with very few residual deficits (Fig. 4). Of note, the patient had symmetric strength in all 4 extremities and recovered excellent fine motor control in her right hand. She still has some mild word-finding difficulties.

Discussion

Treatment strategies for large intracranial arachnoid cysts remain controversial. Surgical options include open or endoscopic cyst fenestration, placement of a cystoperitoneal shunt, or marsupialization via craniotomy. Here, we demonstrate a rare complication after craniotomy for open fenestration of a large arachnoid cyst.1,6

The majority of arachnoid cysts are found in the supratentorial compartment. Of these, most have been noted to arise in the middle cranial fossa.2,8,17 This propensity of arachnoid cyst formation at this site has been attributed to failure of fusion of frontal and temporal arachnoid membranes within the sylvian fissure during fetal development.14 Of arachnoid cysts found in the middle fossa, up to 30% are considered large, occupying nearly the entire temporal fossa.4 There have been several theories proposed for possible mechanisms that lead to cyst enlargement. However, a precise understanding of this condition remains unknown.

The symptoms of arachnoid cysts are variable based on their size and anatomical location. The size of arachnoid cysts can be classified using the Galassi system (Table 1). Arachnoid cysts that enlarge may become symptomatic if they compress adjacent neural structures or interfere with CSF circulation.7 The most common symptom of a middle cranial fossa arachnoid cyst is headache,5,11 followed by proptosis, contralateral motor weakness, and seizures.9,10 Suprasellar arachnoid cysts may cause obstructive hydrocephalus, visual impairment, and endocrine dysfunction. Posterior fossa arachnoid cysts often present with cerebel-
lar symptoms, such as nystagmus and gait abnormalities. The correlation between the size of the arachnoid cyst and signs and symptoms remains poor.

Subdural hematomas and hygromas have rarely been reported as complications of arachnoid cysts. It is thought that the leptomeningeal vessels and bridging veins are susceptible to rupture due to a congenital weakness in the wall of the arachnoid cyst. Cases of minor trauma associated with an arachnoid cyst and subdural hematomas are widely reported throughout the literature.

Intraparenchymal hemorrhage following evacuation of a large arachnoid cyst may likely represent a reperfusion injury, although no definitive cause has yet been determined for this condition. This hypothesis assumes that these patients experience chronic high cerebral perfusion pressure and poor local autoregulatory response as a direct result of mass effect caused by the arachnoid cyst. Sgouros and Chapman demonstrated that arachnoid cysts may indeed interfere with cerebral blood flow and perfusion. Impedance of cerebral perfusion may indicate that patients presenting with headaches and papilledema (i.e., signs of increased ICP) are at increased risk for this phenomenon in the postoperative period. In this case, as rapid shift in local cerebral perfusion may lead to reperfusion injury, slow decompression of the arachnoid cyst may be the most appropriate treatment option.

If arachnoid cysts are found incidentally, without signs of increased ICP, rapid decompression may be safe. Based on our experience, patients who demonstrate signs of increased ICP are at risk for this phenomenon in the postoperative period. In this case, as rapid shift in local cerebral perfusion may lead to reperfusion injury, slow decompression of the arachnoid cyst may be the most appropriate treatment option.

References

Acknowledgment
We thank Andrew J. Gienapp for technical and copy editing, preparation of the manuscript and figures for publishing, and publication assistance with this manuscript.

Author Contributions
Conception and design: all authors. Acquisition of data: Auschwitz, DeCuyper, Khan. Analysis and interpretation of data: Auschwitz, DeCuyper, Khan. Drafting the article: Auschwitz, DeCuyper, Khan. Critically revising the article: DeCuyper, Khan. Reviewed submitted version of manuscript: DeCuyper, Khan. Approved the final version of the manuscript on behalf of all authors: Einhaus. Administrative/technical/material support: Einhaus. Study supervision: Einhaus.

Correspondence
Stephanie Einhaus, Semmes-Murphey Neurologic & Spine Institute, 6325 Humphreys Blvd., Memphis, TN 38120. email: seinhaus@semmes-murphey.com.