ELSEVIER
SAUNDERS

Neurosurg Clin N Am 15 (2004) 9–17

NEUROSURGERY
CLINICS
OF NORTH AMERICA

The endoscopic management of arachnoidal cysts

Rick Abbott, MD[a,b,*]

[a]*Department of Neurosurgery, INN, Beth Israel Medical Center, 170 East End Avenue, New York, NY 10128, USA*
[b]*Department of Neurosurgery, Albert Einstein School of Medicine, New York, NY, USA*

Arachnoidal cysts are a not uncommon lesion for a neurosurgeon, particularly a pediatric neurosurgeon, to be called on to treat. In one autopsy series, the incidence of these cysts was 0.1% [1]. Of lesions arising intracranially, arachnoidal cysts comprise approximately 1% [2]. There has been active debate as to how to manage these cysts best, with the controversy driven by their benign behavior, subtle clinical sequelae, and the potential for treatment failure or complications. Not surprisingly, the introduction of the neuroendoscope into a neurosurgeon's armamentarium typically leads to the consideration of its use to treat these entities in the hope of accomplishing what a craniotomy can while avoiding the associated morbidity. In the following article, the wisdom of such an approach is explored.

Pathology and pathogenesis

In 1831, Bright [3] first described an arachnoid cyst as "...a serous cyst forming in connection with the arachnoid, and apparently lying between its layers..." In his book, he discussed two cases, stating that these cysts seemed to be chronic, to have a low potential for growth, and to be of sizes varying from that of a pea to as large as or larger than an orange. During the past century, these observations were confirmed with the introduction of microscopic neuropathology, and in 1978, Rengachary et al [4] published a photomicrograph illustrating Bright's observation. This photomicrograph demonstrated splitting of arachnoidal membrane at the margin of the cyst and a lack of trabecula within the cyst, showing the cyst to arise within the arachnoidal membrane and not within the subarachnoid space. The cyst membranes contained hyperplasic arachnoidal cells and a thick layer of collagen.

The pathogenesis, however, has been more controversial. Until the 1970s, authors argued over arachnoid cysts being either secondary phenomena occurring in regions of agenesis of the brain or primary events of dysgenesis of the arachnoid investing the brain. In 1955, Robinson [5] published a series of 15 patients with middle fossa arachnoidal cysts, hypothesizing that they were cerebrospinal fluid (CSF) collections passively filling a space left by an agenesis of a portion of the temporal lobe. Starkman et al [6] put forth a countertheory in 1958 in a report of three autopsies done on individuals with middle fossa cysts, finding these cysts to be surrounded by arachnoidal membrane, which led him to conclude that the cysts had arisen as a result of splitting or duplication of the arachnoid during development. With the introduction of CT scanning and the ability to image the brain before and after treatment of these cysts, it became apparent that there was a capacity for expansion of the temporal lobe into space provided by decompression of the cyst. In 1971, Robinson [2] withdrew his hypothesis of agenesis, stating that the cysts were caused by maldevelopment of the arachnoid. Robinson [2], the primary proponent of these cysts being secondary phenomena, abandoned his position after observing that neurologic sequelae to these cysts were not in proportion to the cyst's size and that brain re-expansion could be seen on CT after the cysts were treated.

* Department of Neurosurgery, INN, Beth Israel Medical Center, 170 East End Avenue, New York, NY 10128, USA.

E-mail address: Rabbot@bethisraelny.org

doi:10.1016/S1042-3680(03)00071-8

Arachnoidal cysts typically arise within normal arachnoidal cisterns. They are believed to arise around week 15 of gestation after the rupture of the roof of the closed fourth ventricle and creation of the foramina of Luschka and Magendie. This allows for the escape of CSF into the subarachnoid space, where it replaces intracellular ground substance filling the subarachnoid space. Duplication or splitting of the arachnoid at the time of cistern formation provides the anlage for the arachnoidal cyst. Similarly, splitting or duplication of the ependymal lining of the lateral ventricles results in the formation of intraventricular or ependymal cysts. Arachnoidal cysts most commonly arise in the middle fossa (30%–50%), but 10% arise on the hemisphere convexity, 10% in the suprasellar cistern, 10% in the quadrigeminal cistern, 10% in the cerebellopontine (CP) angle, and 10% in the midline of the posterior fossa [7].

Intraventricular cysts typically arise in or near the atria of the lateral ventricles. Their walls can be formed by arachnoidal cells (intraventricular arachnoidal cysts), ependymal cells (ependymal cysts more correctly viewed as being periventricular), neuroepithelial cells, or choroidal cells. Most of these cysts, excluding the choroidal cysts, probably represent a dysgenic process and are therefore not uncommonly associated with dysfunction of adjacent brain parenchyma as manifested by focal seizures and cognitive disabilities [8].

Presentation

Intracranial CSF cysts typically become symptomatic in patients before the age of 20 years, with most doing so within the first decade [9]. There is some variation in age at presentation according to the location of the cyst, however. Arachnoidal cysts of the middle fossa present in patients before the age of 16 years [10]. Cysts of the quadrigeminal cistern present earlier, usually by the age of 12 months because of their compression of the aqueduct of Sylvius [8], whereas arachnoidal cysts of the suprasellar cistern present later in childhood, with more than 60% presenting in patients between 1 and 20 years of age [8]. Only 14% of suprasellar cysts present after the age of 20 years. The signs and symptoms at presentation are referable to the location and size of the cyst. With the exception of cysts of the CP angle, the most common symptoms present at presentation are those of a slowly growing mass or of hydrocephalus (ie, headache). The headache can be generalized, or it may localize to the site of the cyst.

Other symptoms at presentation point to the location of the cyst. Cysts of the middle fossa can be associated with partial or secondarily generalized seizures. Arai et al [11] found a 39% incidence in 77 patients having cysts of the middle fossa, whereas Ciricillo et al [12] found the same incidence in 39 patients. Boop and Teo [13] have reported that more than 80% of their patients with such cysts manifest behavioral abnormalities, attention deficit disorder, or other problems in school. Cysts of the quadrigeminal cistern can be associated with nystagmus, Parinaud's syndrome, hearing disturbances, and, rarely, motor deficits. Such symptoms are rare because of the relatively early onset of hydrocephalus associated with such lesions. Suprasellar cysts can be associated with visual disturbances (papilledema, optic atrophy, or bitemporal field cuts), endocrinopathies, and the "bobble head doll" sign that is pathognomic for lesions in this location. Cysts of the CP angle typically have a long history of slowly evolving symptoms referable to stretching of cranial nerves and distortion of the cerebellum. Vertigo, hearing disorders (eg, tinnitus, hearing loss), hemifacial spasm, facial paresis, trigeminal neuralgia, decreased corneal sensation, nystagmus, intention tremor, ataxia, and dysmetria can be features of these cysts. If ignored too long, obstruction of the outlets of the fourth ventricle can result in an obstructive hydrocephalus. Clival arachnoidal cysts distort the brain stem, causing compression of the corticospinal tracts (paresis in the extremities, hyperreflexia, and Babinski sign) and stretching of cranial nerves (diplopia). When these extend into the supratentorial space, endocrinopathies can evolve because of compression of the pituitary apparatus. Other than signs of increased intracranial pressure, intraventricular cysts can be associated with focal seizures, ataxia and other gait disturbances, blurred vision, or frank diplopia.

Diagnosis

The imaging tool of choice with these cystic CSF lesions is MRI (Fig. 1). Cysts of the middle fossa, clivus, and CP angle must be differentiated from epidermoid cysts. Diffusion-weighted MRI is useful in such cases, because the two types of cysts have different signal characteristics in the imaging sequence. Suprasellar arachnoid cysts need to be differentiated from craniopharyngiomas and Rathke cleft cysts. The suprasellar arachnoid cysts are typically larger; grow symmetrically and upward, resulting in the "Mickey

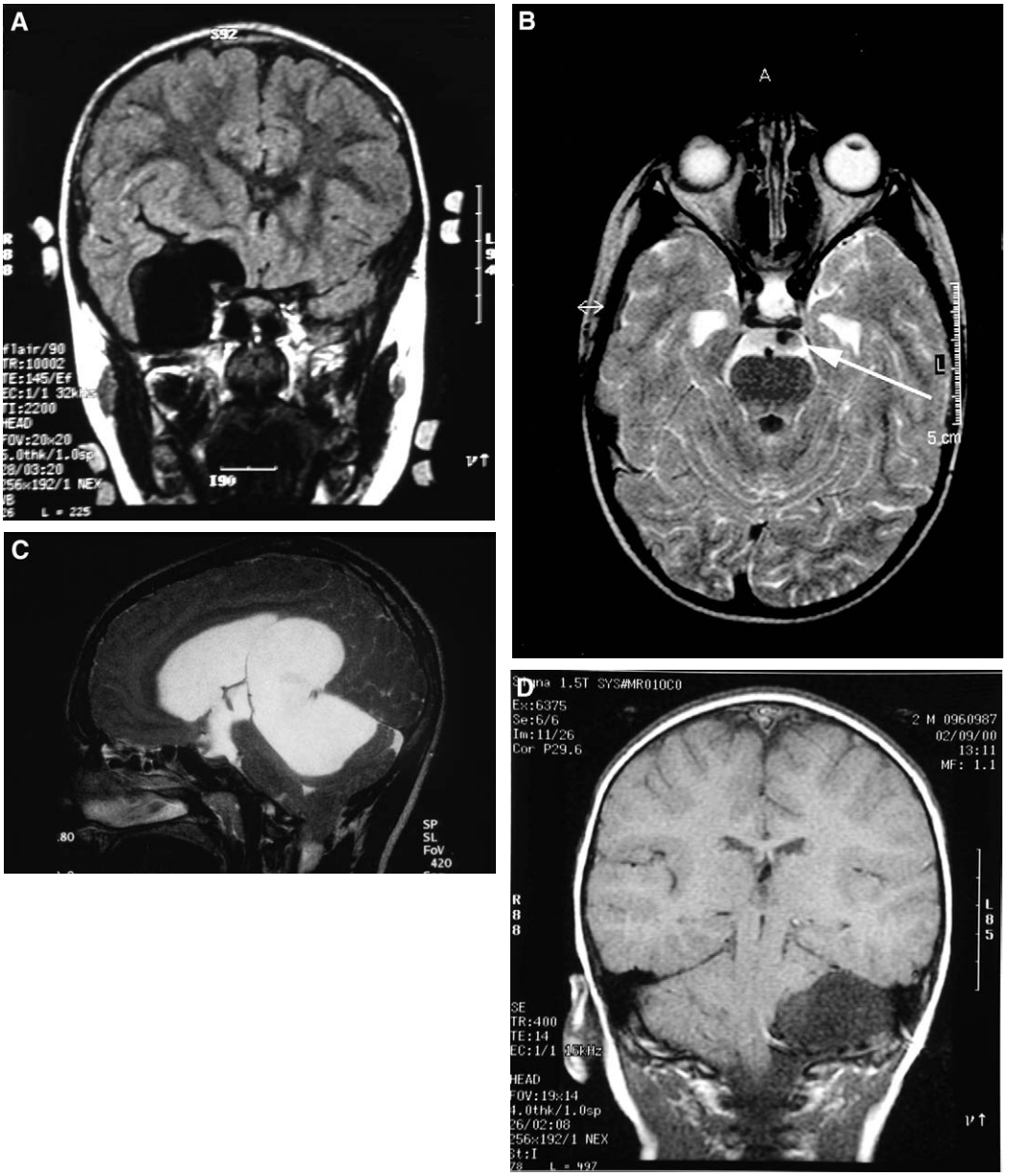

Fig. 1. (*A*) Coronal T1-weighted MRI scan of middle fossa arachnoid cyst. (*B*) Axial T2-weighted scan of suprasellar arachnoid cyst's base with flow void seen at point of fenestration (*arrow*). (*C*) T2-weighted sagittal MRI scan of quadrigeminal arachnoidal cyst. (*D*) T1-weighted coronal MRI scan of cerebellopontine angle arachnoidal cyst.

Mouse" sign; and do not contain calcification commonly seen in craniopharyngiomas. In this situation, CT may be warranted to exclude a craniopharyngioma given this modality's sensitivity for calcium.

Intraventricular CSF cysts can be differentiated from epidermoids, dermoids, and parasitic cysts using MRI. The differential diagnosis should be discussed with your neuroradiologist when ordering imaging of the lesion.

Treatment

The first question to be answered is that of whether any treatment should be offered. Implied in this question is whether further growth of the cyst is expected and whether symptomatic hydrocephalus is present. The latter question is easily addressed by careful history taking and examination of the patient. The former can be more difficult. There are few reports available that offer rules for treatment based on the observed natural history of these cysts. One based on a retrospective study of adults with arachnoidal cysts showed that cysts tended to grow over time if there was distortion of neural structures adjacent to the lobe containing the cyst or to bony structures [14]. This does not offer assistance in decision making for children, however, given its retrospective nature and the fact that because the study was conducted in a group of adults, it represented a more benign subgroup of patients with this condition. Most reports dealing with the pediatric population recommend treatment at the time of discovery of the cyst unless it is of a small size with minimal distortion of surrounding tissues and has been discovered incidentally [13,15,16]. Cysts that distort surrounding neural tissues have been shown to alter cerebral blood flow [10,17]. Presumably, this explains the atrophy that can occur over time, as shown when there is a failure of parenchymal re-expansion when cysts are treated in older individuals.

Next to be answered when treatment has been elected is how the cyst should be treated. In the 1980s, most articles discussing treatment of these cysts favored the use of shunts. Presumably, this was the result of a relatively high incidence of postoperative complications after craniotomies in the 1960s and 1970s. As operative techniques and the use of microneurosurgery have evolved, a reduction in the incidence of postoperative complications has occurred. With this and a greater appreciation of the life history of a shunted patient, there has been a natural shift in preference from treating these cysts with shunts to surgical fenestration [18]. Theoretically, to fenestrate a cyst is to cure it; thus, a lifelong dependence on a shunt is avoided. This shift in preference toward fenestration is only being accelerated with the introduction of the endoscope.

Endoscopic treatment of intracranial CSF cysts is technically challenging and should only be considered by experienced neuroendoscopists. The anatomy can be obscure, and the number and complexity of the instruments used are greater than those used in the more standard neuroendoscopic cases, such as third ventriculostomy. To start, careful planning is required. Thought should be given to the trajectory taken to the target(s) for fenestration. This is especially critical when multiple fenestrations are being considered, as is the case with a suprasellar cyst, or when a third ventriculostomy is needed in addition to cyst fenestration, as is case with a quadrigeminal cyst. An incorrectly placed burr hole because of a poorly planned trajectory results in an injury to the cortical tissues surrounding the scope as it is rocked back and forth to reach fenestration targets. When grossly off, reinsertion of the guide cannula to reach a second target may not even be possible and a new burr hole might be required. Ideally, when the target abuts or is in a ventricle, it is best to approach it via the ventricle so that normal anatomic structures can be used for guidance. It can be difficult to visualize structures on the other side of a cyst's wall, and it is a common occurrence to punch holes into brain tissue when attempting to fenestrate a cyst to an adjacent CSF space. Thought should also be given to structures that are to be avoided during the introduction and advancement of the scope. Ideally, the trajectory should be established to avoid these structures. If this is impossible, thought should then be given as to how to avoid their injury, such as establishing early visualization with the scope before encountering the structure so as to avoid injury (eg, visualization of the fornix at the foramen of Monro so as to avoid it as one traverses the foramen).

Next to be considered is whether a self-retaining holder for the scope is to be used. The use of a self-retaining system adds time and complexity to the case. In many cases, an assistant can navigate the scope while the surgeon works with instrumentation through the scope. In this setting, the assistant should be experienced in the use of the scope and familiar with the working habits of the surgeon so that the two work as a fluid team. The surgeon cannot do an adequate job if he or she is worrying about or fighting with the assistant. In such a setting, the surgeon might correctly elect to use a self-retaining system. The other setting where the use of such a system should seriously be considered is when one anticipates being at the target site for more than a few minutes because of the need for a delicate or extensive surgical dissection. Also, if one is considering the use of a second instrument channel, it is best to have the scope held by a self-retaining system.

With regard to the use of a second instrument channel, this refers to the introduction of a second catheter that approaches the target via a different trajectory than that of the endoscope [19]. It can be useful when one anticipates the need for instruments that are larger than the working channel of the scope or when there is a need to view the working of the instruments from an angle. This can be useful when one anticipates pulling material out of the surgical field, because such a maneuver not uncommonly blinds the endoscopist as the material reaches the head of the scope. The surgeon also gains a greater appreciation of the amount of distortion that occurs during the maneuvering of the instrumentation. Use of the second channel does add another level of complexity to the case, however. First, there is the issue of capturing and maintaining visualization of the channel. This becomes increasingly difficult as the distance between the burr holes of the scope and instrument channel increases and as the depth of the target increases. Second, there can be difficulty in coordinating the manipulation of the two channels. This takes some practice, and my advice is to make small movements that do not result in the complete loss of visualization of the instrument channel. The instrument channel is first moved partially out of the field of view in a desired direction, and the scope is then moved to recenter the instrument channel in its field of view. Computer-assisted guidance systems can be of great use in overcoming these problems, with trackers being placed on the scope as well as on the instrument channel.

At every step in the surgery, absolute attention must be paid toward homeostasis. It has been established that less than a teaspoon of blood within the intraventricular fluid space blinds the endoscopist. Consequently, after the burr hole has been drilled and before the dura is opened, homeostasis must be complete. This is also the case after the dura is opened before penetrating the arachnoid. Midline cysts, such as suprasellar and quadrigeminal cysts, are typically approached via the ventricles. A guide sleeve is typically introduced into the lateral ventricle, its stylet is removed, and the endoscope is then advanced into the lateral ventricle. During this approach, it is wise to remain oriented to the trajectory of the scope so as to appreciate the general location of the scope's tip. When the anatomy is disorienting, the first step should be to check that one's conceptualization of the scope's trajectory is correct. More than once, I have mistakenly thought that I was in the region of the foramen of Monro only to discover that the scope was pointing posterior with the tip in the ventricle's atrium. This is especially common with a burr hole placed too anteriorly. Normal anatomic structures are searched for to further establish orientation. Once the choroid plexus is found, it can be followed anteriorly to find the foramen of Monro or posteriorly to point to where a quadrigeminal cyst is herniating through the floor of the lateral ventricle. If the scope feels awkward in its movement, the most common reason is that the camera is not oriented to the scope (ie, the camera's 12-o'clock position is not the scope's 12-o'clock position). The scope should be withdrawn from the head and its camera's orientation checked.

Laterally projecting cysts, such as those of the middle fossa and CP angle, are approached through the overlying parenchyma laterally. This parenchyma can be present to a varying degree. Draped over the surface of the cysts are cortical veins, especially in the case of cysts of the middle fossa. Schroeder et al [20] have suggested not disturbing the lateral wall of such cysts so as to avoid traction of cortical veins stretched over their surface. Once within these cysts, arterial vessels of the Sylvian fissure can be seen coursing the medial wall. They can be followed down to the basilar cisterns, where the fenestrations can be made. The membrane of these cysts has been shown to be rich in collagen; consequently, it can be difficult to penetrate. Scissors or a knife can be used to cut an opening. It can then be enlarged with sharp dissection or by repeated inflation of a balloon. It has not been established what constitutes an adequate opening. Until such time as this has been established, one should attempt to duplicate what one would accomplish using microneurosurgery via a small craniotomy. If difficulty is encountered in doing this, judgment is used, but there should not be any hesitancy to convert to a craniotomy to accomplish one's surgical goals. On more than one occasion, we have done this on our service; the families of our patients are always told that this is a possibility, and consent is obtained for just such a possibility.

Earlier mention was made of the use of computer-assisted surgical guidance systems or frameless stereotaxis. These systems can be of great use when working within cysts containing no anatomic landmarks for guidance. By using such systems, one moves from navigating by dead reckoning based on surface landmarks to image-based navigation, where the tip of scope and or its channel or a second instrument channel can be seen

on MRI or CT. Before performing the surgery, such systems can be used for surgical planning. Structures to be avoided can be incorporated in planning the trajectory, and this information is, in turn, used to establish the optimum location for the burr hole. Depending on the guidance system being used, there are several options that can be employed for image guidance. Some systems require the use of dedicated pointing devices. In such settings, a special pointer can be manufactured to replace either the endoscope's guidance sleeve or its stylet. Another alternative is to have the company modify a rigid endoscope so that its system can track it directly. I have preferred tracking the stylet of a peel-away catheter used to establish a channel for the endoscope because it affords the greatest flexibility for scope selection and is the simplest and least expensive to manufacture. At least one system has removable tracking devices that can be attached to an instrument or catheter and then registered to the system to allow for tracking by the system. Some accuracy is sacrificed when using such a device, and it can take several minutes to establish registration. Also, more than once, I have been unable to accomplish registration when attempting to use these attachable trackers.

After registering the patient and pointing devices to the system, the burr hole is located using the system and the adequacy of the trajectory is confirmed before making a skin incision. It is wise to take several moments to confirm the appropriateness of instrument setup at this point so as to prevent discovering that movement of the scope is limited by something in the surgical field or tracking is lost when the scope is in a certain position within the head. Once this is done, the burr hole is made and the dura opened using the same care as mentioned previously. The appropriateness of the planned trajectory to the target is confirmed one final time using dead reckoning; the scope's channel is then advanced toward the target. Advancement toward the target is done slowly while retaining the appropriate trajectory by maintaining it on at least two image planes (Fig. 2). Some systems give a bird's eye view or so-called "target view" showing the tip being tracked as well

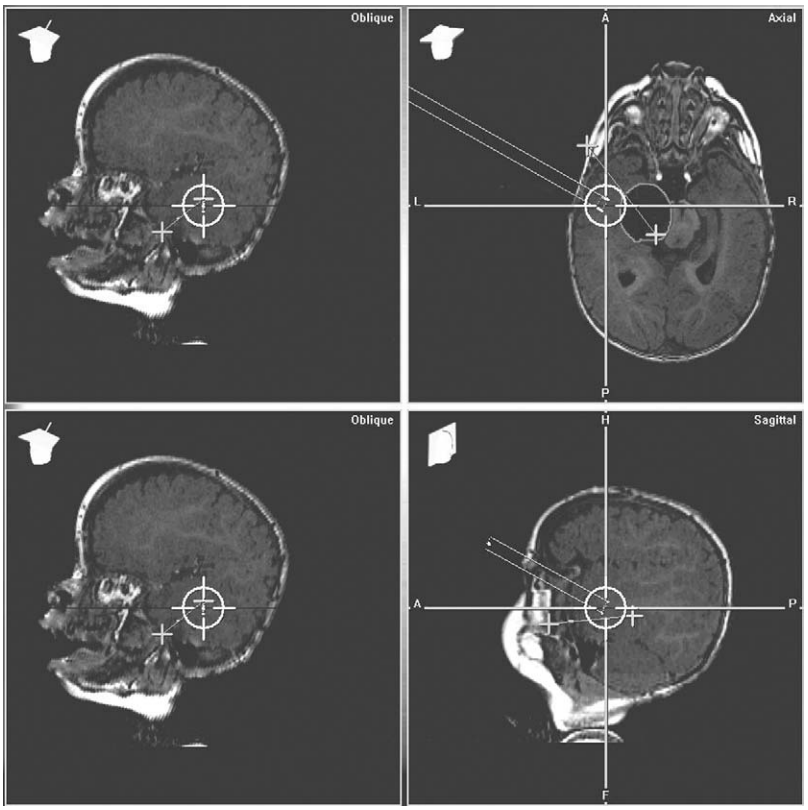

Fig. 2. Image showing approach to target (marked with +) by outer sleeve of endoscopy (its tip is the point of the cross hairs). For this cyst, we were targeting the point where the cyst abutted the ambient cistern.

as the target; this is quite helpful, because the goal is simply to overlay the two cross hairs. Side to side and upward or downward corrections in route to the target cause distortion in the brain's parenchyma, because the catheter cannot slice through brain tissue. This is critical to appreciate when a flexible catheter for the scope is being used, because once the stylet is removed to allow for the introduction of the scope, the catheter deflects as the parenchyma restores itself to its resting configuration. The result is that the catheter tip will no longer be on target. Consequently, the surgeon must judge when there has been too great a need for correction of trajectory. When there is concern about this, the catheter should be removed and a second attempt made to advance it to the target. Much has been made of the potential for brain shift imparting inaccuracy to the guidance data set as the surgery proceeds. Obviously, if one is to be working at only one target, this should not be of great concern if things move along at a pace such that little CSF escapes before the catheter is on target. At the point that the scope is looking at the target, the accuracy of the guidance data set is no longer of concern. Alternatively, if there is more than one target, thought should be given to the problem. In my experience, structures that are close to the center of the head and vertically inferior to my pointer seem to maintain accurate registration to the data set, whereas those near the surface or lateral to a deflating structure or lesion tend to lose registration as the case proceeds. This should be kept in mind when determining the order of targeting. It is also important to remember that there is always a tomorrow and to discuss with a family member the possibility of needing multiple sessions to accomplish the surgical goal.

There will be cases in which shunting of a cyst is required. We had one case of a quadrigeminal cyst that underwent three endoscopic fenestrations into the ventricles (twice into the lateral ventricle and once into the third) and two open fenestrations, only to fail all procedures and become symptomatic because of an increasing mass effect from the cyst (Fig. 3). It was then decided to insert a cystoperitoneal shunt. The placement of a catheter into an arachnoid cyst can be extremely difficult, because the cyst wall is extremely prone to deflecting the catheter to the side when insertion is attempted. Use of the endoscope to confirm entry into the cyst can avoid postoperative frustration. One can use a larger scope to cut a fenestration in the cyst wall. The scope and its guide sleeve are then advanced into the cyst, the

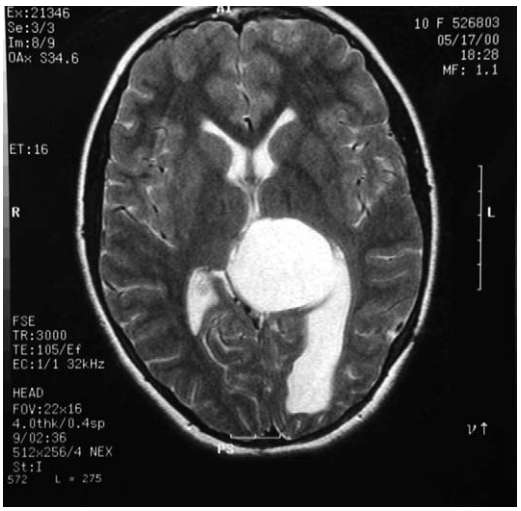

Fig. 3. Second recurrence of quadrigeminal cyst after two prior fenestrations into lateral and third ventricles. At the third operation, the cyst was fenestrated and a catheter placed for a cystoperitoneal shunt.

scope is removed, and a proximal catheter of a cystoperitoneal system is then fed down the guide sleeve into the cyst. It is wise to determine the depth of scope insertion to establish the length of catheter needed before removal of the scope in such a setting and to use a peel-away guide sleeve to simplify catheter placement. Alternatively, a small intraluminal scope can be used as the catheter's stint during attempted placement of the proximal catheter. Once within the cyst, the scope can be used to confirm cyst penetration. If the wall has not been penetrated, unipolar cautery can be applied to the scope to complete penetration unless there is concern about adjacent vessels, as might be the case for a quadrigeminal cyst. Before doing this, it is wise to back the scope out partially to visualize the wall better and confirm that it has indeed not been penetrated. On occasion, I have been fooled into thinking that I had not penetrated the cyst when, in fact, I had, and I was simply up against the far wall of the cyst. In such a case, one can immediately see the interior of the cyst as opposed to a trailing cyst wall.

Results

There are now a few papers describing outcomes in small series of patients who have undergone endoscopic management of their intracranial CSF cysts. Paladino et al [21] described 6 of their patients treated endoscopically. One had

a parasagittal cyst, and the other 5 had middle fossa cysts. Four of 6 cysts were successfully managed endoscopically, with 2 others requiring insertion of a cystoperitoneal shunt. Kim [22] reported on 7 patients with arachnoid cysts. Three had cysts in the posterior fossa, 2 had cysts in the suprasellar cistern, 1 had a cyst in the middle fossa, and 1 had a cyst on the convexity, with all experiencing symptomatic resolution. Radiographically, 4 of 7 cysts diminished in size, whereas 3 of 7 disappeared. Ruge et al [23] reported on successfully managing 2 children with quadrigeminal plate cysts endoscopically, and Furuta et al [24] reported a similar case managed successfully using an endoscope to place a cystoperitoneal shunt after a failed attempt to place the catheter stereotactically. Brunori et al [25] and Hayashi et al [26] have reported experiences similar to those of Ruge et al [23] in successfully communicating quadrigeminal cysts to the ventricles. Hopf and Perneckzy [27] reported on 36 patients with various forms of arachnoid cysts treated endoscopically, with 26 improving symptomatically. Wagner et al [28] reported on 2 children with arachnoid cysts managed successfully endoscopically, and Decq et al [29] reported on 2 children with suprasellar arachnoid cysts successfully managed. Walker et al [30] reported a 64% success rate in managing 14 children with arachnoidal cysts endoscopically. In summary, as series numbers increased, so did failures, with larger series reporting around a 33% failure rate in endoscopically managing arachnoid cysts. This compares with a 30% failure rate for shunted cysts and a 19% failure rate in cyst fenestration in the European Cooperative Study [9]. The more aggressive cyst resection surgery had a 7% failure rate in the same study. Fewel et al [18] reported a 27% failure rate in 102 arachnoidal cysts managed with either surgical fenestration or resection. Undoubtedly, there is a learning curve associated with this technique, and the failure rate currently being seen in the endoscopically managed cases will lessen over time. Until such time as the failure rate duplicates that seen with open surgery, thoughtful consideration should be given to the adequacy of the fenestration accomplished endoscopically and to whether or not the procedure should be converted to an open operation if there is concern about its adequacy. Additionally, a frank discussion should be undertaken with the family as to the pros and cons of the various approaches so that they can make an informed decision.

Complications

As with any surgery, there are potential complications when endoscopically managing arachnoid cysts. "Minimally invasive" should not be construed to mean minimal risk by the surgeon, and the family or patient should in no way be sold a bill of goods that this technique has fewer risks than open surgery. Kim [22] reported that 1 of his 7 patients treated endoscopically experienced significant bleeding during her surgery, requiring abandonment of the endoscopic procedure with conversion to an open procedure with successful control of the hemorrhage and completion of the surgical goal of fenestration. Hopf and Perneczky [27] reported a 14% complication rate in imaging 36 patients with arachnoidal cysts. Four patients experienced subdural hematomas or hygromas after their surgery, with 2 of them also developing meningitis. This may be an important observation, because we have noted infections in some of our patients who have experienced intraoperative hemorrhaging, which required extraventricular drainage after surgery. This is a discussion we have with all our families before surgery. Another of the patients reported on by Hopf and Perneczky [27] experienced hemorrhaging after treatment of a posterior fossa cyst, resulting in hydrocephalus that required a subsequent third ventriculostomy. Robinson and Cohen warn of the risks of injuring blood vessels and other structures by the guide sleeve when advancing the scope because of the sleeve not being visualized by the scope [8]. Finally, structures can be injured during the actual fenestration process when visualization is poor or excessive force is used to penetrate the tough membrane.

Summary

I have little doubt that most arachnoidal cysts will be managed endoscopically in the future given the advances we have seen over the last decade in our instrumentation. Our excitement to employ this new technology should be governed by the reality that we are still learning and that our current success rate is not quite as good as what can be expected when using microneurosurgery.

References

[1] Shaw C, Alvord EJ. Congenital arachnoid cysts and their differential diagnosis. In: Vinken P, Bruyn G, editors. Handbook of clinical neurology, vol. 31. Congenital malformations of the brain. Amsterdam: North Holland; 1977. p. 75–136.

[2] Robinson R. Congenital cysts of the brain: arachnoidal malformations. Prog Neurol Surg 1971;4: 133–74.
[3] Bright R. Reports of medical cases, selected with a view of illustrating the symptoms and cure of diseases by a reference to morbid anatomy, vol. 2. Diseases of the brain and nervous system. London: Lonham, Rees, Orme, Brown, Green, and Highley; 1831.
[4] Rengachary S, Watanabe I, Brackett C. Pathogenesis of intracranial arachnoid cysts. Surg Neurol 1978;9:139–44.
[5] Robinson R. The temporal lobe agenesis syndrome. Brain 1964;87:87–106.
[6] Starkman S, Brown T, Linell E. Cerebral arachnoid cysts. J Neuropathol Exp Neurol 1958;17:484–500.
[7] Hogg J, Peterson A, El-Kadi H. Imaging of cranial and spinal cerebrospinal fluid collections. In: Kaufmann H, editor. Cerebrospinal fluid collections. Park Ridge, IL: American Association of Neurological Surgeons; 1998. p. 19–57.
[8] Kaufmann H, editor. Cerebrospinal fluid collections. Park Ridge, IL: American Association of Neurological Surgeons; 1998.
[9] Oberbauer R, Haase J, Pucher R. Arachnoid cysts in children: a European cooperative study. Childs Nerv Syst 1992;8:281–6.
[10] Boop F, Young R, Scott R. Arachnoid cysts of the middle cranial fossa and convexity. In: Kaufmann H, editor. Cerebrospinal fluid collections. Park Ridge, IL: American Association of Neurological Surgeons; 1998. p. 67–96.
[11] Arai H, Sato K, Wachi A, Okuda O, Takeda N. Arachnoid cysts of the middle cranial fossa: experience with 77 patients who were treated with cystoperitoneal shunting. Neurosurgery 1996;39(6): 1108–12.
[12] Ciricillo S, Cogen PH, Harsh GR, Edwards MS. Intracranial arachnoid cysts in children. A comparison of the effects of fenestration and shunting. J Neurosurg 1991;74:230–5.
[13] Boop F, Teo C. Congenital cysts. In: McLone D, editor. Pediatric neurosurgery. Philadelphia: WB Saunders; 2001. p. 489–98.
[14] Becker T, Wagner M, Hofmann E, Warmuth-Metz M, Nadjmi M. Do arachnoid cysts grow? A retrospective CT volumetric study. Neuroradiology 1991;33:341–5.
[15] Artico M, Cervoni L, Salvati M, Fiorenza F, Caruso R. Supratentorial arachnoid cysts: clinical and therapeutic remarks on 46 cases. Acta Neurochir (Wien) 1995;132:75–8.
[16] Galassi E, Gaist G, Giuliani G, Pozzati E. Arachnoid cysts of the middle cranial fossa: experience with 77 cases treated surgically. Acta Neurochir Suppl (Wien) 1988;42:201–4.
[17] De Volder A, Michel C, Thauvoy C, Willems G, Ferriere G. Brain glucose utilisation in acquired childhood aphasia associated with a Sylvian arachnoid cyst: recovery after shunting as demonstrated by PET. J Neurol Neurosurg Psychiatry 1994;57: 296–300.
[18] Fewel M, Levy M, McComb J. Surgical treatment of 95 children with 102 intracranial arachnoid cysts. Pediatr Neurosurg 1996;25:165–73.
[19] Jallo G, Morota N, Abbott R. Introduction of a second working portal for neuroendoscopy. A technical note. Pediatr Neurosurg 1996;24:56–60.
[20] Schroeder H, Gaab M, Niendorf W. Neuroendoscopic approach to arachnoid cysts. J Neurosurg 1996;85:293–8.
[21] Paladino J, Rotim K, Heinrich Z. Neuroendoscopic fenestration of arachnoid cysts. Minim Invasive Neurosurg 1998;41:137–40.
[22] Kim M. The role of endoscopic fenestration procedures for cerebral arachnoid cysts. J Korean Med Sci 1999;14:443–7.
[23] Ruge J, Johnson R, Bauer J. Burr hole neuroendoscopic fenestration of quadrigeminal cistern arachnoid cyst: technical case report. Neurosurgery 1996;38:830–7.
[24] Furuta S, Hatakeyama T, Nishizaki O, Fukumoto S. Usefulness of neuroendoscopy in treating supracollicular arachnoid cysts—case report. Neurol Med Chir (Tokyo) 1998;38:107–9.
[25] Brunori AA, Chiappetta F. Endoscopy for cysts. J Neurosurg 1999;91:1067–8.
[26] Hayashi N, Endo S, Tsukamoto E, Hohnoki S, Masuoka T, Takaku A. Endoscopic ventriculocystocisternostomy of a quadrigeminal cistern arachnoid cyst. Case report. J Neurosurg 1999;90:1125–8.
[27] Hopf N, Perneczky A. Endoscopic neurosurgery and endoscope-assisted microneurosurgery for the treatment of intracranial cysts. Neurosurgery 1998; 43:1330–7.
[28] Wagner HJ, Seidel A, Reusche E, Sepehrnia A, Kruse K, Sperner J. A craniospinal enterogenous cyst: case report. Neuropediatrics 1998;29:212–4.
[29] Decq P, Brugieres P, Le Guerinel C, Djindjian M, Keravel Y, Nguyen JP. Percutaneous endoscopic treatment of suprasellar arachnoid cysts: ventriculocystostomy or ventriculocystocisternostomy? Technical note. J Neurosurg 1996;84:696–701.
[30] Walker M, Petronio J, Carey C. Ventriculostomy. In: Cheek W, Marlin A, McLone D, editors. Pediatric neurosurgery: surgery of the developing nervous system. Philadelphia: WB Saunders; 1994. p. 572–81.