Arachnoid cysts of the Sylvian fissure

Evidence of fluid secretion

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Morphological and enzyme ultracytochemical evidence is presented to support the contention that the walls of arachnoid cysts secrete fluid. Clinical evidence has already suggested this phenomenon, including intracranial pressure elevation and expansion in some cases, and the observation that arachnoid cysts constitute closed compartments with a fluid content that cannot be derived from other cerebrospinal fluid-containing spaces. Ultrastructurally, the cyst lining showed a similarity to subdural neurothelium and the neurothelial lining of arachnoid granulations in such morphological features as intercellular clefts with sinusoid dilatations, desmosomal intercellular junctions (upon which tonofilaments may be abutting), pinocytotic vesicles, multivesicular bodies, lysosomal structures, and the presence of a basal lamina. Some of these features, together with the presence of microvilli on the luminal surface, are consistent with fluid secretion. Moreover, enzyme cytochemistry demonstrated (Na\(^+\) + K\(^+\))\text{-ATPase in the plasma membranes lining the cavity, either directly (the apical membranes), or via the intercellular clefts (the basolateral membranes), and, with alkaline phosphatase occupying the opposite plasma membranes, this structural organization indicates fluid transport toward the lumen. It may be surmised that arachnoid cysts derive from subdural neurothelium differentiating towards arachnoid villus mesothelium.

**Key Words** • arachnoid cyst • leptomeningeal cyst • sodium pump • enzymes • (Na\(^+\) + K\(^+\))\text{-ATPase ultracytochemistry • subdural neurothelium • arachnoid granulation • Sylvian fissure

Some confusion still exists about the nature of arachnoid cysts. In the recent literature, however, these lesions have correctly been recognized as a separate entity, quite distinct from pockets of the true subarachnoid space which are sequestered by adhesions following infection, hemorrhage, or trauma. These pockets have also been designated as secondary or false arachnoid cysts, but should be called “leptomeningeal cysts,” a term employed by Taveras and Ransohoff for pockets of traumatic origin.

Two hypotheses still prevail as to the pathogenesis of arachnoid cysts. One suggests a primary origin, implying a developmental derangement of the arachnoid. The other proposes that agenesis of brain structures results in dilatation of the cerebrospinal fluid (CSF)-containing spaces to make up for loss of cerebral volume. This latter mechanism has been suggested by Robinson, and still has its proponents.

This paper presents our observations in a scanning electron microscopy (SEM) and transmission electron microscopy (TEM) study of the cyst lining in four cases, and reviews our findings in four other cases we have reported previously. Enzyme ultracytochemistry was performed in one case for the demonstration of (Na\(^+\) + K\(^+\))\text{-ATPase and alkaline phosphatase.

**Clinical Material and Methods**

**Specimen Preparation**

Immediately following biopsy in four patients, the specimens, consisting of flat sheets of cyst wall, were attached to pieces of cork by small entomology pins at the corners to prevent shrinking. They were then immersed in 2% glutaraldehyde solution, care being taken not to damage the surface of the specimen. After fixation, the specimens were cut into pieces for TEM and SEM study.

For SEM studies, the specimens were dehydrated in
an acetone series at room temperature, and then subjected to critical-point drying. Conducting cement was used to mount the dry specimens on edge on object holders. This placement allowed viewing of both surfaces. The specimens were coated with carbon and gold vapor, and studied with a JEOL JSM-U3 scanning electron microscope.

For TEM studies, the specimens were postfixed in 1% phosphate-buffered osmium tetroxide for 2 hours, washed and dehydrated in an alcohol series, and embedded in Epon. Ultra-thin sections were cut in a plane perpendicular to the membrane surface, counterstained with uranyl phosphate and lead citrate, and studied with a Philips EM 300 electron microscope.

**Enzyme Ultracytochemistry**

Enzyme ultracytochemistry was performed in one case. The sheets of cyst wall attached to cork were immersed for 1 hour at 4°C in a fixation fluid of 2% paraformaldehyde and 0.1% glutaraldehyde in 0.1 M sodium-cacodylate buffer with a pH of 7.38. After fixation, they were washed for 2 hours at 4°C in several changes of the buffer with 10% (v/v) dimethyl sulfoxide (DMSO) and 8% (w/v) sucrose with a pH of 7.38. Since the specimens were only several cell layers thick, whole specimens could be incubated without further section. For demonstration of transport (Na⁺ + K⁺)-ATPase (K⁺-nitrophenylphosphatase: K-NPPase) and alkaline phosphatase, we used the enzyme cytochemical method of Ernst et al. as modified by Mayahara, et al. A further modification consisted of the replacement of lead by cerium as a capturing agent in electron microscopy, as cerium has been proven to yield superior results in the cytochemical localization of many phosphohydrodases.

For the demonstration of K-NPPase in TEM using cerium as a capturing agent, the incubation medium (in final concentrations) consisted of a 60-mM Tris-maleate buffer with a pH of 9.0, 45 mM potassium hydroxide (KOH), 25% (v/v) DMSO, 10 mM p-nitrophenylphosphatase Mg²⁺-salt (Mg²⁺-pNPP), 2.5 mM levamisole, and 1 mM cerium chloride. With lead as a capturing agent for the demonstration of K-NPPase in TEM, both unstained and counterstained with 1% uranyl acetate in ethanol, were viewed with a Philips EM 300 electron microscope.

**Case Reports**

**Case 1**

This 26-year old woman had been complaining of episodes of dizziness for 1 year. At admission, neurological examination revealed no abnormalities, but cerebral angiography indicated a left temporal avascular lesion that caused upward displacement of the left middle cerebral artery (MCA). On computerized tomography (CT) scans, a large lesion with the low attenuation values of CSF occupied part of the anterior fossa and the entire middle fossa on the left side. After assessment of the extent of the cyst by transillumination, a left temporal craniotomy was performed. Upon opening the dura, a large cyst was visualized, occupying the Sylvian fissure and containing clear colorless fluid. The cyst was lined by thin translucent membranes resembling normal arachnoid, except at the floor, where an opaque, gliotic, scar-like membrane largely covered the exposed insula and the rudiments of the frontal and temporal lobes. Communication of the cyst cavity with the subarachnoid spaces was established along the MCA, which lay under the lower lining of the cyst. Postoperative recovery was uneventful.

Following incubation at room temperature for 15 minutes, the samples were washed three times for 10 minutes at 4°C in 0.1 M sodium-cacodylate buffer at a pH of 7.2 with 8% sucrose but without DMSO. After washing in distilled water, some samples were mounted for light microscopy and the others were postfixed in 1% osmium tetroxide, dehydrated, and embedded in epoxy resin. Sections 1-μm thick and ultra-thin sections were cut on an LKB ultramicrotome. Ultra-thin sections, both unstained and counterstained with 1% uranyl acetate in ethanol, were viewed with a Philips EM 300 electron microscope.
of neurothelial cells, one or more cells thick, on the surface of which occasional microvilli could be seen. The cells had a cytoplasm varying from light to very dark and a nucleus with marginated chromatin. Where the layer comprised more cells, the intercellular clefts showed interdigitations and sinusoid dilatations, with desmosomal junctions at some places. The layer of lining cells was separated from the dura by a more or less distinct basal lamina (Fig. 2 left).

Scanning electron microscopy of the cyst floor ad-
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FIG. 3. Case 2. Computerized tomography scan prior to craniotomy showing a hypodense arachnoid cyst in the temporal lobe. The cyst has displaced the lateral ventricles and extends into the posterior fossa.

joining the brain parenchyma showed a surface with slightly bulging cellular elements but lacking microvilli. On TEM of the floor lining, a layer of dark glial cells was apparent, separated from the lumen by a superficial basal lamina (Fig. 2 right). This layer resembled a pial glia limitans and presumably represented the gliotic floor of the cyst.

Case 2

This 2-week old baby boy was examined because of head enlargement, a bulging fontanel, and separation of the cranial sutures on skull films. A CT scan showed dilated ventricles and a sharply delineated large hypodense area in the posterior fossa, considered to be a Dandy-Walker cyst. The patient underwent placement of a Holter-type ventriculoatrial shunt. He did well until the age of 10 months, when he was readmitted because of drowsiness and vomiting. Upon revision of the shunt, both the ventricular and cardiac catheters appeared to be blocked. Removal of the ventricular catheter, which was obstructed by choroid plexus, caused a ventricular hemorrhage, whereupon he was treated with external ventricular drainage. Two weeks later, this was replaced by a ventriculoperitoneal shunt, which was thought to drain both ventricles, since CT scans showed an intracerebral hematoma that apparently occluded a dilated inferior horn.

At the age of 20 months, the child was admitted again because of lethargy and irritability. On CT scanning a hypodense lesion was again seen in the left temporal area (Fig. 3). This lesion had been thought on the scans taken after shunt placement to be a dilated temporal horn occluded by a hematoma. This area of hypodensity seemed to communicate with the hypodense lesion in the posterior fossa (considered to be a Dandy-Walker cyst on the admission scans), and was now suspected to be an arachnoid cyst. A left temporal craniotomy was performed. When the tense dura was opened, the exposed cortex of the temporal lobe showed widened convolutions and a bluish translucent spot (Fig. 4). Upon puncturing this area where the brain parenchyma was only a few millimeters thick, a cyst was entered, the floor of which consisted of the compressed insula, with the branches of the MCA running on its surface. The dome of the cyst was thus formed by the white matter of the temporal lobe, which was hollowed out on its medial side. The cyst reached the tentorium, and, according to the CT scans, apparently crossed the tentorial edge into the posterior fossa. However, this could not be visualized upon inspection. A blocked shunt catheter, with its tip embedded in the wall of the cyst (not in the ventricle as believed), was replaced by a new one. Communication was achieved by fenestration of the membrane separating the cyst cavity from the adjacent frontoparietal subarachnoid spaces. Postoperatively, the patient has done well.

On TEM of the thin layer of brain parenchyma that constituted the roof of the cyst, the luminal lining was shown to consist of a layer of cells with moderately dark cytoplasm, nuclei with margined chromatin, and tortuous intercellular clefts having desmosomal junctions. The layer was separated from underlying glial cells by a discontinuous basal lamina (Fig. 5 left). Scanning electron micrographs of the luminal surface of the sample revealed scattered stubby microvilli, 0.2 to 0.8 μm in height, and at some places fenestrations of 1-μm diameter through which the underlying structure was visible (Fig. 5 right).

Case 3

This 20-year-old man had been complaining of left temporal headaches since suffering a cerebral contusion in a car accident a year before. A CT scan showed a hypodense area in the left basal temporal region suggestive of an arachnoid cyst. Carotid angiography indicated an avascular temporal lobe lesion causing upward displacement of the left MCA. A left temporal craniotomy was performed. When the tense dura was opened, the exposed cortex of the temporal lobe showed widened convolutions and a bluish translucent spot (Fig. 4). Upon puncturing this area where the brain parenchyma was only a few millimeters thick, a cyst was entered, the floor of which consisted of the compressed insula, with the branches of the MCA running on its surface. The dome of the cyst was thus formed by the white matter of the temporal lobe, which was hollowed out on its medial side. The cyst reached the tentorium, and, according to the CT scans, apparently crossed the tentorial edge into the posterior fossa. However, this could not be visualized upon inspection. A blocked shunt catheter, with its tip embedded in the wall of the cyst (not in the ventricle as believed), was replaced by a new one. Communication was achieved by fenestration of the membrane separating the cyst cavity from the adjacent frontoparietal subarachnoid spaces. Postoperatively, the patient has done well.

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Arachnoid cysts of Sylvian fissure

opened, the temporal lobe bulged through the incision. It was elevated and deeply indented by a large cyst, which was lined by transparent membranes and contained clear colorless fluid. The cyst was situated at the base of the middle fossa below the temporal lobe, its cavity also separated from the basal cisterns by a transparent membrane. To establish a communication between the cyst and the basal cisterns, this membrane was fenestrated. During the subsequent closure of the craniotomy, tachycardia of 140 beats/minute developed, and a few hours after the operation the patient suffered epileptic seizures. He was aphasic for a few days, but subsequently recovered completely.

On SEM of the luminal side of the dome, a dense covering of microvilli (0.1 μm long) and scattered projections of varying length (0.4 to 1.7 μm) were noted (Fig. 6 left). Transmission electron microscopy showed a superficial layer a few cells thick separated by intercellular clefts with local areas of widening, and at some places desmosomal junctions. The cytoplasm of the

Fig. 5. Case 2. Left: Transmission electron micrograph of the luminal lining of the cyst dome. A few layers of light and dark cells are seen, separated by clefts of varying width. Note the desmosomal junctions, microvilli, and basal lamina (B) separating the layer from the glia. Bar = 1 μm. × 11,000. Right: Scanning electron micrograph of the luminal surface of the cyst roof showing scattered stubby microvilli and fenestrations. Bar = 1 μm. × 15,500.

Fig. 6. Case 3. Left: Scanning electron micrograph of the luminal surface of the cyst roof showing a dense covering of small microvilli (0.1 μm long) and scattered projections of varying length (0.4 to 1.7 μm). Bar = 1 μm. × 945. Right: Transmission electron micrograph of the cyst roof showing many layers of light cells separated by tortuous and locally dilated intercellular clefts. Note the tonofilaments in the cytoplasm abutting on the desmosomal junction (arrow), the vacuoles derived from distended endoplasmic reticulum, and the pinocytotic vesicles (pv) along an incomplete basal lamina. Bar = 0.1 μm. × 18,900.
cells varied from light to very dark, and contained mitochondria, tonofilaments converging on the desmosomes, and pinocytotic pits on an incomplete basal lamina. This basement membrane separated the neurothelial layer from the dura with its collagen fibers (Fig. 6 right).

**Case 4**

This 32-year-old man had complained of epileptic seizures for a year and recent episodes of hallucination. Neurological examination showed no abnormalities. On CT scanning a rectangular area with the attenuation of CSF was revealed. This was thought to be a very large arachnoid cyst occupying the left temporal and parietal areas. The cyst was explored through a left frontotemporal craniotomy. The slack dura was opened, exposing the dome of a large cyst. The cyst contained clear colorless fluid and was lined on all sides by thin transparent membranes; it occupied the widely opened Sylvian fissure, exposing the insula, and reached far into the parietal area. Toward the base of the brain, the cyst cavity was separated from the basal cisterns by a thicker semi-opaque membrane. To ensure communication with the basal cisterns, the membrane was opened. The patient's subsequent course was uneventful.

On SEM of the cyst dome, numerous stubby microvilli (0.2 to 0.7 μm high) could be seen on its luminal surface. Fenestrations were also present, the smaller ones 0.1 μm in diameter and the larger ones 1.5 μm in diameter, resembling those in Case 2 (Fig. 7). Transmission electron microscopy (TEM) showed multiple layers of neurothelial cells at the surface of the cyst roof. The cells had short microvilli, interdigitating intercellular clefts, desmosomal junctions, nuclei with margined chromatin, and prominent cytoplasmic vesicles. A basal lamina was regularly observed delimiting the lining neurothelial cells. In between the deeper cellular layers, collagen fibers were observed.

**Results of Ultracytochemistry**

Ultracytochemistry studies were performed on specimens from Case 4. On light microscopy of small samples and 1-μm epoxy-embedded sections, the enzyme activity of both K-NPPase and alkaline phosphatase was difficult to assess, as the staining proved to be rather faint. In TEM, the reaction product (cerium salt) for both enzymes showed a localization in relation to the cell membranes and was sharply demarcated. The tissue blank was consistently negative. The inhibition of K-NPPase with ouabain and the exclusion of K+ greatly reduced the enzyme activity.

For K-NPPase, the localization of reaction product was mainly at the luminal surface of the cells lining the cyst cavity (Fig. 8 upper left and right). Reaction product was also frequently present along the tortuous basolateral cell membranes of these cells. For alkaline phosphatase, the reaction product was consistently present along the cell membranes of the abluminal side of the surface epithelial cells (Fig. 8 lower). Overlap between the areas with K-NPPase and alkaline phosphatase was generally absent, although some overlap in the region where the basolateral area and abluminal areas meet cannot be excluded.

**Discussion**

**Morphology**

Typically, arachnoid cysts occupying the Sylvian fissure compress the frontal lobe forward and the temporal lobe backward, separating and eventually obliterating the frontal, parietal, and temporal opercula, widely opening the Sylvian fissure, and exposing the insula. In pronounced cases, the cyst may extend far toward the parietal convexity (as in our Case 4), or reach into the subtemporal area, truncating the temporal lobe, as in Case 2. In Case 2 the cyst hollowed out the temporal lobe medially and extended occipitally through the tentorium into the posterior fossa (similar to Case 1 of Williams and Guthkelch64). Where the insula is exposed, the branches of the MCA can be seen running on its surface in the subarachnoid space underlying the floor of the cyst. The adjoining brain, although indented, usually has an intact gyral pattern, and is covered by the normal leptomeninges underlying the cyst wall. Presumably due to pressure atrophy, the leptomeningeal covering may be deficient, and minute perforations may occur. This may explain the appearance of air in the cyst a few hours after pneumoencephalography.15,36 In our Case 1, the floor of the cyst was gliotic, with absence (or disappearance) of the neurothelial lining in the ultrastructural studies. In Case 2, the floor of the cyst showed normal insular cortex, but the cyst dome was bordered by white matter of the indented temporal lobe.
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Cyst Ultrastructure and Function

Scanning electron microscopy of the dome of the cyst showed a covering by stubby microvilli (measuring 0.1 to 0.2 μm in height), which were densely and evenly distributed or scattered in patches. In addition, in individual cases there may be larger projections of diverse appearance: inflated balloon-like shapes, intertwining thread-like structures, or elongated processes (Case 4). In some cases, transitions both in size and in shape could be observed between the smaller microvilli and the larger processes. Furthermore, fenestrations in the uppermost cell layer may occur.

By contrast, the inner surface of a traumatic leptomeningeal cyst shows only a smooth surface with slightly bulging fibrous patterns, but lacking microvilli, similar to the appearance of normal arachnoid surfaces or to the external surface of the roof of an arachnoid cyst that has been detached from the dura (Fig. 1 right). Where the cyst wall adjoins brain tissue, the surface may have either microvilli (Case 2) or a smooth profile (Case 1).

Transmission electron microscopy revealed that the cyst lining consisted of a single or multiple layer of cells with a cytoplasm varying from light to very dark, and with nuclei that had marginated chromatin. In the cytoplasm, multivesicular bodies and lysosomal structures could be observed, and pinocytotic vesicles were noted along a complete, or quite often discontinuous, basal lamina separating the layer of cells from the adjoining dura or brain parenchyma. Where the cyst lining comprised a multiple-cellular layer, the intercellular clefts were sometimes tortuous, with interdigitations and sinusoid dilatations. Generally, abundant desmosomal junctions could be seen, but occasionally there were only a few. In the cytoplasm, there may be tonofilaments converging on the junctions.

Fig. 8. Transmission electron micrographs of the ultra-cytochemistry studies in Case 4. Upper Left: K-NPPase reaction. The reaction product (cerium salt) is present along the luminal (apical) membrane of the lining neurothelial cells of the cyst dome. Note widening of the intercellular clefts, with pinocytotic pits (arrows). Bar = 0.1 μm. × 20,700. Upper Right: K-NPPase reaction. The reaction product (cerium salt) is present along the tortuous basolateral cell membranes lining the interdigitating intercellular clefts. Bar = 1 μm. × 20,000. Lower: Alkaline phosphatase reaction. Note the absence of reaction product (cerium salt) along the apical membrane. Reaction product is present, however, along the cell membranes bordering the intercellular clefts. Bar = 1 μm. × 20,000.
These morphological features have been described previously, and conform most closely to those of subdural neurothelium or arachnoid mesothelium (Fig. 9 upper), which forms the outer layer of the arachnoid bordering the dura mater and which lines arachnoid granulations (Fig. 9 lower). Although subdural neurothelial cells normally do not possess microvilli, the neurothelial lining of arachnoid villi displays microvilli, as depicted by Gomez and Potts in Fig. 9 of their study and as described by d'Avella, et al. The microvilli (forming a brush-like border), the tortuous intercellular clefts, and the intercellular desmosomal junctions are among the features indicating a secretory (or absorptive) function, as they constitute the prerequisites for a standing osmotic gradient, which is necessary for fluid secretion (or absorption).

The ultracytochemical results demonstrate a polar distribution of both transport ATPase (K-NPase) and nonspecific alkaline phosphatase, with the reaction product of the former located at the luminal (apical) or basolateral surface of the neurothelial lining cells, and that of the latter located along the basal cell membranes. The location of transport ATPase at the apical cell membrane is an indication of secretory function, and is similar to the situation in choroid plexus epithelium. The location of transport ATPase in the basolateral cell membranes of the intercellular clefts is the more common situation in hypertonic and isotonic transport epithelia, whether of the absorbing or secreting type. Its presence in both locations in the neurothelial lining cells of an arachnoid cyst can therefore be interpreted as evidence for their secretory activity, substantiating the concept of active fluid transport as the basic mechanism involved in expansion of the cysts.

As to the pathogenesis of arachnoid cysts, they may conceivably derive from subdural neurothelium, differentiating toward the arachnoid villus with its fluid-transporting capacities. According to this hypothesis, it may be surmised that such an ectopic villus, failing to make connection with a dural sinus in which it can drain its secretion, may collect its secretory product in a closed cyst in the same way that epidermoid cysts may arise from sequestered epithelium.

**Other Cyst Locations**

Arachnoid cysts have been reported at other locations, which are grouped into convexity, basal or suprasellar, quadrigeminal, and posterior fossa lesions. Their lining was once described as arachnoidal or ependymal on the basis of light microscopic observations, which lacked the resolution required to assess their ultrastructural detail. Some of the cysts, therefore, might have been determined to be neuroepithelial if electron microscopy had been performed. The cysts situated at the convexity of the frontal and parietal lobes may be related to those of the Sylvian area. Among the cysts reported in the basal and suprasellar regions, many may not be true arachnoid cysts, but instead could be leptomeningeal fluid collections that occur secondary to trauma or meningitis. Like the suprasellar cysts, those of the quadrigeminal region usually cause hydrocephalus due to blocking of the aqueduct. Among the arachnoid cysts in children, an appreciable number have been reported in the posterior fossa. Many of these are situated in the midline, while others occur in the cerebellopontine angle or overlie the cerebellar hemispheres. Although a midline posterior fossa cyst and a cyst in the fourth ventricle have been reported with arachnoid mesothelial lining on the basis of TEM, many cysts of the posterior fossa may be of the secondary type.

**Diagnosis**

Symptomatology has been reported as nonspecific, with an appreciable number of cases being asymptomatic. Generally, symptomatology comprises cranial deformity or enlargement, raised intracranial pressure, epileptic seizures, mental retardation, and focal signs.
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In a few series, a preponderance for males and for the left cerebral hemisphere has been reported. 6,17,25,39

Local bulging of the head has been recognized as an indication for the presence of an intracranial arachnoid cyst since 1831. 5 This bulging, associated with thinning of the temporal squama, may also be visualized on plain skull films, in conjunction with elevation of the lesser wing of the sphenoid and enlargement of the middle cranial fossa.

In young children in whom the skull is thin and translucent, the cyst may light up with transillumination. Cerebral angiography and ventriculography may only indicate the presence of a space-occupying lesion by virtue of displacement of either the blood vessels or cerebral ventricles, respectively, without disclosing the true nature of the lesion. Computerized tomography, on the other hand, readily demonstrates the cyst on the basis of its low attenuation value, which resembles that of CSF. In particular, arachnoid cysts of the Sylvian fissure may present as a triangle or a square with straight edges due to shortening of the temporal lobe and flattening of the insula. 2,17 Nuclear magnetic resonance scanning, especially in the saturation-recovery images, allows recognition of arachnoid cysts because of the relaxation properties of their contents, which equal those of CSF and differentiate them from other cysts containing a fluid richer in protein. 24 Cisternography with radiiodinated albumin 23,32,54 and, more recently, metrizamide-enhanced CT cisternography 52 have demonstrated the absence of contrast material within the cysts in spite of filling of the surrounding subarachnoid spaces; however, only 8 to 12 hours later there was evidence of contrast medium within the cysts. This is in accordance with the observations made during operation that the cysts appear as cavities completely segregated from the neighboring subarachnoid spaces. The subsequent appearance of tracer, contrast material, or air (in pneumoencephalography) 17,36 within the cysts may be due to either active or passive transport of tracer or contrast fluid across the membranes, or to leakage through perforations in the surrounding membranes, which may result from pressure atrophy. The delayed appearance of contrast fluid within arachnoid cysts differentiates them from traumatic leptomeningeal cysts, in which the contrast fluid or tracer penetrates at the same time as into the adjacent subarachnoid spaces. 16

Before arachnoid cysts were recognized as a separate entity, they used to be grouped among other fluid collections containing clear colorless CSF-like fluid, under the headings of serous meningitis, external hydrocephalus, porencephaly, and leptomeningeal cysts. The terminology and differential diagnosis of the various entities are shown in Table 2. Robinson 51 described the associated developmental disturbances of the adjoining brain areas, and Starkman, et al. 36 concluded that arachnoid cysts were closed compartments situated within the arachnoid and segregated from the subarachnoid spaces.

On the other hand, leptomeningeal cysts of traumatic origin are pockets of the subarachnoid space, which are more or less separated from the remainder of the subarachnoid space by adhesions. 58 Very often they are associated with old fractures, while the subjacent ventricle may be dilated due to loss of brain tissue. Therefore, traumatic leptomeningeal cysts are bordered by gliotic cortical tissue or white matter, whereas the cortex adjoining arachnoid cysts, although compressed, tends to have a normal configuration.

Neuroepithelial or ependymal cysts may be indistinguishable from arachnoid cysts, as they contain CSF-like fluid and may be located on the brain surface. Their location may range from the cerebral convexity to deep within the brain parenchyma. Ghatak, et al. 33 reported finding a cyst extending from the surface of the temporal lobe to the lateral ventricle, and which had a lining of ependymal cells, with filaments, pinocytotic vesicles along a basal lamina, interdigitations, and a dense covering of microvilli, but which lacked cilia.

Other light microscopy studies 30,31,45 have shown an ependymal lining of such cysts and convincingly demonstrated the presence of cilia. Koto, et al. 33 described a cyst covering the right cerebral hemisphere of an infant and having a lining of choroid epithelial cells, characterized by a covering of microvilli, tight intercellular junctions, and basal interdigitations.

Porencephalic cysts are cavities within the brain which communicate with the ventricular system. These are considered to result from defects of the brain parenchyma following insults in prenatal life. Consequently, they tend to be bordered by gliotic tissue with traversing trabecular processes. 26

Colloid cysts of the third ventricle have been categorized among the neuroepithelial cysts. Their content, however, is usually not clear CSF-like fluid. Their lining has been considered as endodermal, with two types of

<p>| TABLE 2 |</p>
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<th>Classification of cysts</th>
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<td>1. Benign cysts of the brain containing clear colorless CSF-like fluid.</td>
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<tr>
<td>a. True arachnoid cysts or intra-arachnoid cysts, situated upon or beside the subarachnoid spaces, lined by subdural neuroepithelial cells.</td>
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<td>b. Leptomeningeal cysts due to sequestration of subarachnoid space by infection, trauma, or hemorrhage, comprising part of the subarachnoid space.</td>
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<tr>
<td>c. Porencephalic cysts. Cavities resulting from defect of brain parenchyma, communicating with the ventricular system.</td>
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<tr>
<td>d. Neuroepithelial cysts. Lesions similar to arachnoid cysts; their location may range from the brain surface to paraventricular, lined by ependymal or choroid epithelial cells.</td>
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<td>2. Cysts concomitant with tumors (gliomas, neurinomas, meningiomas) containing yellow fluid with a high protein content.</td>
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<td>3. Subdural hematomas of long standing. These may contain yellow fluid, rich in protein, from which heme pigments may have been leached out.</td>
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<td>4. Colloid cysts of the third ventricle.</td>
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<td>5. Hydatid cysts.</td>
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cells, a darker non-ciliated and a lighter ciliated type.8,27,35

Treatment

In view of the secretion of fluid within the cyst cavity and the consequent expansion of the cyst, rational therapy should be directed primarily at establishing a communication with the subarachnoid spaces or the ventricle, preferably with marsupialization of the cyst lining, which is capable of fluid secretion.2,3 When the feasibility of these measures is in doubt, a shunt may be installed.6,57

The therapeutic measures have generally been reported to be effective, as judged from the alleviation of complaints and the reduction of the cyst volume on postoperative CT scans. However, in some cases the reexpansion of the brain may have disappointing results, presumably when the atrophy of the brain parenchyma is too far advanced. The mere evacuation of the cyst through a burr hole has proved to be ineffective as a permanent measure.17,23

The indication for surgical intervention is obvious in those cases with evident intracranial pressure elevation, due to rapid expansion of the cyst, obstruction of CSF pathways causing hydrocephalus, or hemorrhage within the cyst and the development of subdural hematomas. In cases where the cyst is asymptomatic, the indication for operation may be less evident, especially since the operation is not entirely devoid of risk. Nevertheless, treatment should be considered in children, since the capacity for expansion of the cyst might otherwise jeopardize the normal development of the adjacent brain structures (as demonstrated by the truncation or "agenesis" of the temporal lobe, with concomitant disturbances of cortical function).39

Simple as the exploration of an arachnoid cyst may seem, in addition to the well known postoperative complications of hemorrhage, infection, or CSF leakage, unexpected deterioration may occur. This is illustrated by the tachycardia and postoperative seizures, as in our Case 3, or irreversible hypotension followed by death, as in another (infant) patient. Drowsiness progressing to coma and death have also been reported a few hours after the intervention. Where obvious causes can be excluded, it must be assumed that displacement of brain structures following rapid decompression may be responsible in evoking the untoward responses. Therefore, especially in young children with very large cysts, a more gradual decompression by means of a shunt should be considered before undertaking direct exploration.

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