Chapter 55: CSF Leaks

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Leakage of cerebrospinal fluid (CSF) from its intracranial location may present difficulties in diagnosis, localization, and treatment. Physicians may overlook the leakage in a trauma patient with other serious injuries or confuse the clear fluid discharge with other pathologic conditions. Both the recognition and the localization of CSF leaks can be very challenging, and management varies with etiology and site. The development of skull base surgery has resulted in an increased incidence of CSF leaks, and contemporary diagnostic studies have replaced older techniques of diagnosis and localization. This chapter describes the manifestations of CSF leaks in clinical practice and current methods of diagnosis, localization, and management.

Classification

CSF rhinorrhea

We classify CSF rhinorrhea according to a modification of a scheme Ommaya (1976) developed (Fig. 55-1). This classification scheme is based on etiology and clinical presentation and has implications for evaluation and management. CSF rhinorrhea may be *traumatic* or *nontraumatic* in origin. Traumatic fistulas may be due to *accidental* trauma or *surgical* causes. Accidental trauma is the cause in approximately 80% of patients with CSF rhinorrhea, even though the incidence of CSF rhinorrhea in patients who suffer serious head trauma is only 2% to 3% (Loew et al, 1984). Intracranial and extracranial surgical procedures cause approximately 16% of CSF leaks from the nose. The remaining 4% are nontraumatic in origin (Loew et al, 1984), and these may be *high-pressure* or *normal-pressure* fistulas. High-pressure leaks are more common and result from tumors or hydrocephalus. Tumors account for over half of the cases of nontraumatic CSF rhinorrhea and directly cause the leak by erosion of bone and soft tissue or indirectly through elevation of intracranial pressure (Briant and Bird, 1982; Ommaya, 1976). Hydrocephalus, which may be communicating or obstructive, also increases intracranial pressure. Normal-pressure CSF leaks occurs as a result of congenital anomalies or osteomyelitis; occasionally they may occur without any obvious cause.

CSF otorrhea

Although no commonly used classification system exists for CSF otorrhea, this entity may also be *traumatic* or *nontraumatic* in origin (Fig. 55-2). Traumatic fistulas may be caused by *accidental* or *surgical* trauma. The most common cause of CSF otorrhea is accidental trauma, with CSF leaks occurring in approximately 0.4% to 6.7% of patients with demonstrable skull fractures (Besley, 1916; Gurdjian, 1932; Raaf, 1967). Longitudinal temporal bone fractures result in CSF otorrhea more frequently than do transverse fractures. Longitudinal fractures course from the squama, through the posterior portion of the bony external auditory canal wall, and into the tegmen, where they tear the overlying dura. The tympanic membrane is often torn also, resulting in a pathway for egress of fluid out of the ear canal. Transverse fractures are less common and are usually associated with an intact tympanic membrane. This fracture line course perpendicular to the long axis of the temporal

bone and may transect the internal auditory canal. When transverse fractures cause CSF leaks into the middle ear, passage of CSF down the eustachian tube into the nasopharynx results in CSF otorhinorrhea. If the blow tears the tympanic membrane, CSF leaking from the internal auditory canal will produce otorrhea.

Although a much less frequent etiologic agent than trauma, surgery for chronic suppurative ear disease or malignancy is the second most common cause of CSF otorrhea. Finally, rare nontraumatic fistulas occur as a result of congenital anomalies, tumors, or osteitis and are most often congenital in origin. The role of increased intracranial pressure as a precipitating factor of CSF otorrhea is unclear. However, it probably does not play the same role as it oes in CSF rhinorrhea.

Pathophysiology

Leakage of CSF occurs when arachnoid, dura, bone, and epithelium are violated, resulting in extracranial flow of CSF. Rhinorrhea is more common than CSF otorrhea in most reported series (Laun, 1982; von Haacke and Croft, 1983).

CSF rhinorrhea

Traumatic CSF rhinorrhea

The roof of the ethmoid and the cribriform plate are the most frequent sites of CSF rhinorrhea because of the thin bone and tight adherence of dura to bone in this area (Calcaterra, 1985). Trauma to this region may lead to dural tearing with creation of a CSF leak. The appearance of CSF may be apparent immediately after trauma or days to weeks later. Delayed appearance of a CSF leak may be due to delayed elevation of intracranial pressure following trauma, lysis of a clot in an area of bone and dural dehiscence, resolution of soft-tissue edema, maturation and contraction of the wound edges, or loss of vascularity and necrosis of soft tissue and bone around the wound (Loew et al, 1984; Myers and Sataloff, 1984; Park et al, 1983). Also, during or following dural healing, dura may herniate through a fracture line. The constant physiologic changes in CSF pressure may result in progressive dural herniation with its eventual dehiscence and CSF leakage (Calcaterra, 1985; Gagnon et al, 1984).

Nontraumatic CSF rhinorrhea

Rhinorrhea from tumors of hydrocephalus occurs as a result of elevated intracranial pressure leading to continued erosion and weakening of bone with the eventual development of a fracture and a CSF fistula (Park et al, 1983). The cribriform plate and roof of the ethmoid, which are the thinnest areas of bone at the base of the skull, are the most frequent sites of nontraumatic CSF rhinorrhea (Briant and Bird, 1982. Tumors may also cause CSF rhinorrhea through direct erosion of bone. The mechanism by which nontraumatic CSF rhinorrhea occurs in normal pressure systems probably results from normal physiologic elevations of CSF pressure. Pressure elevations of up to 80 mm of water normally occur spontaneously every few seconds (von Haacke and Croft, 1983). This increase in CSF pressure generally is not able to erode or fracture bone. However, the presence of sudden, short-live, marked increases in intracranial pressure due to coughing or straining may be an

important precipitating factor in the development of spontaneous CSF rhinorrhea.

Although CSF rhinorrhea occurring in this situation is classified as spontaneous, CSF rhinorrhea occurring in the presence of congenitally dehiscent areas or anatomic pathways containing dural herniation is classified as congenital. Differentiating between CSF rhinorrhea caused by a congenitally dehiscent area and that which occurs spontaneously may be difficult. Differentiation between these two entitites has little clinical importance, because evaluation and management of both are essentially the same. Similarly, the empty-sella syndrome can be classified as either congenital or spontaneous. The empty-sella syndrome occurring because of an absent portion of the diaphragma sellae is classified as congenital. The empty-sella syndrome occurring because of pituitary gland atrophy or degeneration is classified as spontaneous.

Infrequently, CSF rhinorrhea occurs as a result of thinning and weakening of skull base bone by osteitis or osteomyelitis.

CSF otorrhea

CSF otorrhea is the escape of CSF through the external auditory canal. This condition requires a pathologic communication between the subarachnoid space and the pneumatized areas of the temporal bone. In addition, there must be a perforation of the tympanic membrane or a defect in the external auditory canal wall; otherwise, the CSF leaking into the temporal bone will pass down the eustachian tube and cause symptoms of rhinorrhea or postnasal discharge. If the CSF fistula is small and the fluid flow rate is slow or intermittent, nasal or postnasal discharge may be imperceptible, and the CSF may accumulate in the middle ear space with its true nature undetected until meningitis occurs or a myringotomy is done and continuous otorrhea follows.

Traumatic CSF otorrhea

The pathophysiology of CSF otorrhea resulting from trauma is similar to that of CSF rhinorrhea. Fractures and penetrating injuries associate with CSF leakage pass through the bony labyrinth of the inner ear, internal auditory canal, or tegment (roof) of the middle ear or mastoid. Penetrating injuries usually occur when pointed objects are accidentally pushed into the ear canal and perforate the tympanic membrane. The point then will break through the promontory or stapes footplate, and CSF can then flow to the outside via communication between perilymph spaces of the inner ear and the internal auditory canal or a widely patent cochlear aqueduct.

Nontraumatic CSF otorrhea

The role of increased intracranial pressure as a precipitating factor in nontraumatic CSF otorrhea is unclear. It probably does not play a major role in CSF otorrhea, because the cribriform plate is the weakest point of the skull base, and rhinorrhea is more likely to result from increased CSF pressure. CSF leakage through the cribriform plate may thereby prevent the development of CSF leakage at other areas. Tumors and osteitis or osteomyelitis are infrequent causes of bone erosion leading to CSF leakage. CSF otorrhea occurring as a result of a congenital anomaly is commonly associated with Mondini inner ear dysplasia (Neely,

1985; Park et al, 1982). In this situation CSF otorrhea may occur as a result of a widely patent cochlear aqueduct or a defect in the cribrose plate of the internal auditory canal or the fallopian canal. However, CSF otorrhea may also occur as a result of a congenital dehiscence of the tegmen with no inner ear malformation. These tegmen defects have been reported in approximately 21% of the population (Ahren and Thulin, 1965; Ferguson et al, 1986). Physiologic CSF pressure changes or sudden increases in intracranial pressure may play an important role in herniation of dura through the tegmen defect and eventual CSF leakage.

Diagnosis

Symptoms

CSF rhinorrhea

The major presenting symptom in patients with CSF rhinorrhea is fluid draining from the nose. In trauma cases CSF rhinorrhea develops within 48 hours in approximately 55% of all patients who ultimately develop CSF rhinorrhea as a result of the trauma. This frequency increases to 70% by the end of the first week as edema, which may be temporarily preventing CSF leakage, resolves (Loew et al, 1984). Hyposmia or anosmia is an associated symptom in 60% to 80% of patients with CSF rhinorrhea due to olfactory nerve damage from fracture of the cribriform plate (Lain, 1982; Ommaya, 1976). In addition, headaches caused by intracranial air have been reported in approximately 20% of patients with traumatic CSF leakage (Ommaya, 1976; Park et al, 1983). Approximately 20% of patients with CSF rhinorrhea develop meningitis as their initial manifestation (Loew et al, 1984). Most of these patients belong to the delayed posttraumatic group. The risk of developing meningitis in the first 3 weeks following head trauma and before correction of a CSF leak has been reported to be from 3% to 11% (Loew et al, 1984; Mincy, 1966).

Postoperative CSF rhinorrhea most often results from transphenoidal removal of pituitary tumors and occurs in approximately 3% to 6% of patients undergoing this type of surgery (Ciric an Tarkington, 1974; Loew et al, 1984; VanGilder and Goldenberg, 1975). CSF rhinorrhea may also complicate the removal of less common tumors at the skull base, such as olfactory meningiomas or esthesioneuroblastomas. Fluid drainage from the nose is the most common presenting symptom in these cases.

Nontraumatic CSF rhinorrhea is insidious at onset an may remain uniagnosed for years. These patients may seek medical attention because of nasal drainage or because of complaints consistent with an intracranial space-occupying lesion. The amount of nasal drainage may be more profuse in cases of nontraumatic fistulas than in cases of traumatic ones. Aeroceles are rarely present intracranially, and anosmia and meningitis are also rare. Headaches are common in these patients. Nontraumatic CSF fistulas causing rhinorrhea tend to occur in adults over 30 years of age, with a female preponderance of 2:1 (Ommaya, 1976).

CSF otorrhea

When accidental trauma causes CSF otorrhea, the majority of patients develop signs and symptoms immediately. In one report, 94% of those who ultimately developed CSF otorrhea following trauma had an immediate onset (Laun, 1982). In those patients in whom

the tympanic membrane is intact, fluid will be visible behind the tympanic membrane or will drain into the nasopharynx by way of the eustachian tube, causing rhinorrhea or a sensation of postnasal discharge. Additional symptoms may consist of a hearing loss, pressure sensation in the ear, or dizziness. When surgical procedures are the cause, CSF otorrhea may present in the same manner as a traumatic CSF otorrhea of accidental origin. However, the surgery may also produce CSF leakage from the incision site, a fluctuant mass at the surgical site, or meningitis in the postoperative period (Myers and Sataloff, 1984). CSF otorhinorrhea following surgery for acoustic neuroma occurs in approximately 10% to 20% of patients (Gardner et al, 1983; Glasscock and Dickins, 1982; House and Hitselberger, 1968).

Nontraumatic CSF otorrhea of a congenital origin usually occurs in children but may develop at any time of life. Children who have this type of otorrhea frequently have a form of Mondini inner ear malformation and have a history of recurring episodes of meningitis. A less common presentation occurs in children who only have hearing loss due to middle ear fluid. Myringotomy reveals CSF exuding from the middle ear. These children may have a congenital dehiscence of the fallopian canal or Hyrtl's fissure. Adults who have leakage of CSF fluid into the middle ear may present with hearing loss only, otorrhea, or otorhinorrhea. These patients, who have normal inner ear function, have a CSF fistula as a result of ruptured meningoencephaloceles that present through congenital defects in the tegmen of the temporal bone. Recurrent meningitis is sometimes the only problem in these patients, and its cause may go undetected for years. Although the bone defect in the tegment has probably been present from the time of birth, CSF leakage may develop years later when the meninges finally are worn through by the constant fluctuations in CSF pressure (Neely, 1985; Park et al, 1982).

Diagnostic studies

Demonstrating extracranial CSF

The accurate diagnosis of a CSF fistula depends on unequivocal demonstration of extracranial CSF and precise localization of its site of leakage. In many instances of profuse nasal drainage, the diagnosis of extracranial CSF is obvious; it is rarely confused with vasomotor or allergic rhinitis. However, in situations in which minimal or intermittent drainage occurs, diagnosis can be difficult. Persistence of a clear, nonsticky, nasal discharge should arouse suspicion of a CSF leak. In a comatose, head injury patient, or in anyone suspected of having CSF rhinorrhea, the body should be placed in the lateral position with the face bent downward to increase CSF flow. Pressure applied to the jugular veins may also make latent CSF leakage apparent (Loew et al, 1984). A classic sign of CSF leakage is the presence of a "halo sign" in which a clear fluid area surrounds a blood stain when CSF mixed with blood is absorbed onto paper. In addition, a wet handkerchief that dries without stiffening is also highly suggestive that a clear rhinorrhea is due to CSF. Usually neither test is sufficient to make the diagnosis. Testing for glucose content in the suspicious fluid is also done sometimes, with a value greater than 30 mg/mL being confirmatory for CSF. However, collecting adequate amounts of fluid to analyze for chemical content is often difficult, and the glucose content of tears may be a confounding factor. Glucose oxidase test papers are inaccurate, and their use provides equivocal information. Immunoelectrophoretic identification of transferrin (beta1 and beta2) (Oberaschen and Arrer, 1986) is an accurate, but generally unavailable, test for CSF.

Currently, the most definitive test to establish the diagnosis of extracranial CSF is metrizamide computed tomography cisternography (MCTC) (Loew et al, 1984; Park et al, 1983). Metrizamide is injected into the subarachnoid space and visualized by computed tomography (CT) scan (axial, coronal, and sagittal). CT readily demonstrates extracranial extravasation of CSF (made radiopaque by metrizamide), thereby not only diagnosing a CSF leak, but also localizing its site (Figs. 55-3 and 55-4). In nontraumatic CSF rhinorrhea, plain CT or MCTC is also useful as an initial diagnostic test to differentiate between high-pressure leaks. Conventional skull films, noncomputed tomography studies, pneumoencephalography, and subdural pneumography are outdated studies that CT studies have replaced.

Other techniques used to demonstrate CSF leakage involve the injection of various colored dyes (methylene blue, indigo carmine, toluidine blue) or radioactive isotopes (radioactive sodium, 111In-DTPA (diethylentriamine pentacetic acid), 99mTc-DTPA) into the subarachnoid space. The former technique has been largely discontinued because of the morbidity it produces (Calcaterra, 1985; Chandler, 1983). The latter technique, however, is currently widely used an involves radionuclide cisternography combined with scintigraphy of pledgets placed intranasally. This technique has a reported overall success rate of 25% to 65% and has been useful in patients with intermittent rhinorrhea not diagnosable by other means (Curnes et al, 1985; Glaubitt et al, 1983).

Localizing the site of leakage

Clinical localization of the site of CSF leakage should not be overlooked despite the avances in imaging studies. A leak occurring in only one side of the nose generally correlates with the side of the defect. However, a leak that shifts from one side to the other, or occurs bilaterally, provides no localizing information. Leakage from the nose when the head is upright or tilted backward suggests that the leak is through the cribriform plate, ethmoid roof, or frontal sinus. Leakage only on tilting the head forward suggests that the leakage is coming from the sphenoid sinus or via the middle ear (Chandler, 1983).

As previously mentioned, MCTC is useful not only for the diagnosis of extracranial CSF, but also for localizing the site. The success rate of this technique, however, is highly dependent on the presence of an active leak at the time of the study. Manelfe et al (1982) have reported a success rate of over 80% in localizing the site of leakage in active leaks. However, the presence of an inactive leak at the time of examination has been reported to result in an identification rate of only 20% to 30% (Mamo et al, 1982; Manelfe et al, 1982). Artificially increasing the CSF pressure in order to open an inactive CSF leak has reportedly increased the success rate to 100% (Naidich and Moran, 1980).

The use of raioactive isotopes injected into the subarachnoid space is not
in localizing the site of leakage. A false-positive result is common because of absorption of radioactivity within the bloodstream and redistribution of this activity through nasal mucosa to the pledgets. Passage of radioactivity to nasal mucosa through the olfactory nerves may also result in false-positive results (Calcaterra, 1985; Hasegawa et al, 1983).

Injection of fluorescein into the subarachnoid space has also been used to localize the site of CSF leakage (Calcaterra, 1985; Kirchner and Proud, 1960). This fluorescent dye is commonly used intraoperatively to localize exactly the site of CSF leakage. Following

injection of fluorescein diluted by CSF into the subarachnoid space, the investigator uses a black light to detect fluorescence in the intraoperative field. The demonstration of greenish fluid delineates the site of CSF leakage. Fluorescein has also been used preoperatively in conjunction with nasal endoscopy. This technique has been reported to be useful in the diagnosis of CSF leaks (Reck and Wissen-Siegert, 1984).

Magnetic resonance imaging (MRI) has also been used to localize the site of CSF leakage (Fig. 55-5). Currently it does not seem to be as accurate as MCTC, but further studies are needed. The advantages MRI does have over MCTC are that it is noninvasive (no radiopaque dye is injected) and it does not involve radiation exposure.

A new imaging technique, positron emission tomography (PET), has also been investigated recently to determine its ability to localize the site of CSF leakage. This technique is similar to radioactive cisternography and involves intrathecal injection of 68Ga-EDTA. Superimposition of images obtained from PET over images obtained by CT was helpful in locating sites of CSF leakage (Bergstrand et al, 1982). Further studies, however, are needed to compare PET with currently used methods.

Management

The management of patients with CSF rhinorrhea depends on the etiology of the leak, the location of the fistula, and the temporal relationship of the leak to the inciting factor. The majority of traumatic CSF fistulas heal without surgical intervention. Patients who develop CSF rhinorrhea shortly after trauma generally do not need an operation to close the CSF fistula, provided no indication for intracranial exploration coexists. Those patients who show no evidence of resolution of CSF leakage by the end of 1 week will probably require surgical exploration and closure of the leak. Patients who develop CSF rhinorrhea days or weeks following trauma generally do not heal without surgical intervention. To allow edema to subside, closure of the CSF leak is frequently postponed in acutely traumatized patients for 2 to 3 weeks. Adjacent facial structures should be reduced early, and reduction may result in cessation of the CSF leakage without further therapy. CSF rhinorrhea resulting from a bullet wound requires exploration. Additional criteria for surgical exploration of patients with CSF rhinorrhea include large defects in the skull base with herniation of brain or evidence of a spicule of bone penetrating the brain (Cooper, 1982). A highly controversial indication for surgery is traumatic CSF rhinorrhea of early onset that is associated with meningitis or a pneumocele. If either of these conditions exists immediately after trauma and resolves with conservative therapy, surgical exploration is probably not required (Briant and Bird, 1982; Cooper, 1982). However, if resolution does not occur, surgical exploration is indicated.

When CSF rhinorrhea results from surgery, the dural injury should be repaired when it occurs. In those situations in which injury to the dura is not suspected until postoperatively when CSF rhinorrhea occurs, conservative therapy is indicated, because the majority of these leaks will close. Massive leaks that occur in the immediate postoperative period usually do not close with conservative measures and require surgical closure.

Several additional points need consideration in the management of patients with traumatic CSF rhinorrhea. As mentioned, the risk of developing meningitis in the first 3 weeks following trauma has been reported to be approximately 3% to 11% (Loew et al, 1984;

Mincy, 1966). Prophylactic antibiotics have not been shown to be effective in the prevention of meningitis and are not recommended in posttraumatic patients (Klastersky et al, 1976; MacGee et al, 1970). Conservative management of CSF rhinorrhea includes keeping the patient at bed rest in an upright position that minimizes the leak. Coughing, sneezing, nose blowing, and straining are to be avoided. Laxatives are prescribed to reduce straining with bowel movements. If no resolution of the rhinorrhea occurs in 72 hours, repeated or continuous lumbar drainage of CSF may be tried for the next 4 days with removal of 150 mL/day (Cooper, 1982). This therapy is rarely helpful in nontraumatic or delayed CSF rhinorrhea.

The first step in the treatment of nontraumatic, high-pressure CSF rhinorrhea is to lower the high intracranial pressure. Following this measure, CSF rhinorrhea resolves in most patients. The minority of patients who continue to have CSF rhinorrhea following normalization of intracranial pressure require surgical exploration (Loew et al, 1984). Normal-pressure, nontraumatic CSF leaks rarely close with conservative therapy and thus almost always require surgical exploration and closure (Briant and Bird, 1982).

Operative approaches

The operative management of CSF rhinorrhea can be divided into intracranial and extracranial approaches, each with its advantages and disadvantages. The specific approach selected depends on the site of CSF leakage.

Intracranial approaches require a craniotomy with its attendant morbidity, mortality, and prolonged hospitalization. In addition, anosmia is a frequent complication of craniotomy for CSF rhinorrhea due to unavoidable trauma to the olfactory nerves (Loew et al, 1984). The advantages of intracranial surgery include the ability to achieve a fluid-tight dural closure and to repair multiple areas of leakage. Intracranial approaches can be further subdivided into extradural and intradural repairs. Theoretically the extradural approach allows the maintenance of intact dura that protects the brain during retraction. However, from a practical standpoint, the dura tears frequently when it is elevated (Loew et al, 1984). Intradural approaches allow a better view of the dura and, sometimes, a clearer identification of the fistula site, although entering the dura exposes the brain to potential infection. In extradural or intradural approaches, spinal fluid is removed by lumbar drains for several days postoperatively until the edema resolves.

The advantages of an extracranial approach include minimal morbidity and mortality while still achieving excellent visualization of the dural defect. The major requirement for the successful application of this technique is precise, preoperative localization of the leakage site. If the site of leakage is not well defined, or if multiple sites of leakage are present, all fistulas may not be closed using an extracranial approach.

In both intracranial and extracranial approaches, pedicle flaps shoul be strongly considered to close areas of leakage that have been irradiated previously. These flaps should be rotated in from sites out of the irradiated field or brought in as free flaps (McCarthy and Zide, 1984).

Repair of specific sites

Ethmoid roof-cribriform plate

The extracranial route provides the best approach for the repair of a unilateral CSF leak localized to the roof of the ethmoid or the cribriform plate. This surgery is done using a nasoorbital incision through which a complete ethmoidectomy can be performed. Removal of the middle turbinate aids exposure of the cribriform plate. Using fluorescein enhances accurate visualization of the dural defect. When general anesthesia is administered, 0.5 mL of 5% fluorescein is mixed with 9.5 mL of CSF and slowly injected into the subarachnoid space (Montgomery, 1979). On exposure of the site of leakage, egress of fluorescein-stained spinal fluid will be evident by its striking color. Positive-pressure ventilation by the anesthesiologist will increase the rate of CSF flow and may make a leak evident. Although complications from the use of intrathecal fluorescein have been reported, they are rare and reversible. These complications have consisted of lower extremity weakness, numbness, seizures, opisthotonos, and cranial nerve deficits (Mosely et al, 1979). The dural efect can be sealed with temporalis fascia grafts, fascia lata, or pericranium used alone or bolstered with fat or with mucoperiosteal flaps from the lateral nasal wall or septum (Loew et al, 1984; Montgomery, 1979). If possible, the edges of the fascia are tucked under the bony edges of the dehiscence. If the dura is sealed well, closing a small bony defect at the site of leakage is not necessary (Briant and Bird, 1982). The tissues used to seal the defect are supported by absorbable surgical sponge placed against the graft or flap followed by an intranasally placed antibiotic-impregnated gauze strip. The intranasal packing is removed 1 week after surgery.

An intracranial approach is used if the site of leakage cannot be delineated clearly or if multiple leaks exist. In both the extradural and intradural approaches to the repair, fluid-tight closure is accomplished by suturing temporalis fascia, fascia lata, or pericranium to the dural defect. Pedicle pericranium flaps may also be used to provide further support.

Frontal sinus

Leaks of CSF through the frontal sinus are best repaired through an extracranial approach using an osteoplastic flap (Briant and Bird, 1982; Park et al, 1983). The advantages and disadvantages of this technique are similar to those of the extracranial repair of CSF leaks of the cribriform plate or roof of the ethmoid. A coronal or brow incision provides access to the frontal sinus. An inferiorly based osteoplastic flap is then created. Bone fragments and lining mucosa are removed from the sinus, and the leak is identified. Dura can then be sutured directly, which is usually quite difficult, or fascia inserted through the defect in the posterior wall of the frontal sinus and tucked under the bony edges. To achieve a fluid-tight closure, fascia can also be sutured to the dura, if enough dura is exposed. Fat obliteration of the frontal sinus reinforces the fascia repair and obliterates the nasofrontal ducts, thus eliminating a potential pathway for ascending infection.

Sphenoid sinus

Sphenoid sinus CSF fistulas are best approache through a transseptal route (Chandler, 1983; Loew et al, 1984; Park et al, 1983). The anterior wall of the sphenoid sinus is exposed and resected. Sphenoid sinus mucosa and the septum of the sinus are also removed. The

sphenoid sinus is then packed firmly with a combination of muscle, bone, and fat. Cartilage from the nasal septum may be placed at the sinus opening to provide support for the tissues packed into the sinus. The septal mucosal flaps are then brought together using septal splints and intranasal packing that is remove in 1 week. Removal of the septal splints is usually done in 2 weeks.

Ear

The precise, preoperative localization of the CSF leak site will determine the surgical approach to the ear. Defects in the tegmen can be exposed by a mastoidectomy or atticotomy operation. Leaks from the posterior fossa dura into the mastoid are approached through a mastoidectomy. When the defect and leak are confined to the middle ear, a tympanotomy may be all that is required. Craniotomy is usually reserved for failures of the various extracranial operation and for very large or multiple defects. A small, extradural craniotomy via the temporal squama, in combination with a mastoidectomy, provides the advantages of a limited craniotomy in addition to those of a mastoidectomy (Adkins and Osguthorpe, 1983). This combined approach facilitates accurate and secure placement of the dural graft.

Many materials have been advocated for dural repair, including, most commonly, temporalis fascia, fascia lata, periosteum, perichondrium, homograft ura, and methyl methacrylate. Composite grafts of conchal cartilage with attached perichondrium have been used to seal the dura and at the same time provide a covering for the associated defect in the bone. In most cases direct suture of the graft material to the dura is not possible, so the graft is held in place by abdominal wall adipose tissue or temporalis muscle pedicle flaps.

In mastoidectomy and atticotomy approaches, the leak site is identified and the bone defect surrounding the dural opening is enlarged. The graft material is inserted through the enlarged opening so that it underlies the dura and is supported circumferentially by bone. The posterior ear canal wall is left intact, if possible, so that the mastoid cavity can be filled with fat or a muscle flap to support the repair site. Also, leaving the canal wall intact eliminates the potential source of infection that would exist from an open mastoid cavity. Small, congenital defects in the tegmen associated with adult-onset CSF leakage have been closed successfully by being packed tightly with bone dust accumulated from the mastoidectomy drilling, rather than through enlargement of the defect to place a soft-tissue graft.

The most frequent locations for middle ear CSF leaks are (1) oval winow, (2) round window, and (3) Hyrtl's fissure, in that order (Schuknecht et al, 1982). If the explored ear is nonhearing, the site of leakage is simply plugged with the graft. In hearing ears the CSF leak is sealed with fascia that is packed in place with antibiotic-impregnated ribbon gauze that is removed in 3 to 5 days, at which time the tympanomeatal flap is returned to its original position (Schuknecht et al, 1982).

New materials

Lyophilized dura, alcoholic prolamine solution (Krahling and Konig, 1984), and fibrin or acrylate glues have been used successfully to close dural defects. However, because these substances have only recently been introduced, the long-term success rates with their use compared with those of more conventional methods are unclear. Further studies are needed

to define their role in the surgery of CSF leaks.

Endoscopic techniques

The evolving techniques of endoscopic sinus surgery are now being applied to the diagnosis and treatment of CSF leaks from the nose. These leaks can be identified directly through the use of endoscopes by visualizing a clear fluid, or a greenish-yellow fluid following intrathecal injection of fluorescein (Messerklinger, 1972; Reck and Wissen-Siegert, 1984). Following localization of the leak site, various techniques can be used for closure. Grafts consisting of fat, muscle and fascia, or mucoperiosteum have been placed against the dura at the leakage site (Mattox and Kennedy, 1990; Papay et al, 1989; Stankiewicz, in press). Fibrin glue and microfibrillar collagen have also been used to seal the leak (Mattox and Kennedy, 1990; Stankiewicz, in press). Although initial results appear promising, continued follow-up is needed to assess the long-term results of these endoscopic techniques.

Results

The failure rate for intracranial repair of CSF rhinorrhea has been reported to be from 6% to 27% (Calcaterra, 1985; Cooper, 1982; Laun, 1982; Loew et al, 1984). Of these failures, most will require multiple surgical attempts at closure, with a final failure rate ranging from 1% to 10% (Loew et al, 1984; Ray and Bergland, 1969). The failure rate for extracranial repair of CSF rhinorrhea is similar to that reported for intracranial repair, varying from 6% to 33% (Calcaterra, 1985; Hubbard et al, 1985; Park et al, 1983). Because of the similar failure rates of extracranial and intracranial procedures, extracranial repair is usuallyu attempted first unless extenuating circumstances require an intracranial procedure, thus avoiding the higher morbidity and mortality of an intracranial approach.