

Traumatic Cerebrospinal Fluid Leaks

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KEYWORDS

- Cerebrospinal fluid leak • Skull base trauma
- Skull base surgery • CSF rhinorrhea

Key Points: CSF LEAKS

- Eighty percent of cerebrospinal fluid (CSF) leaks occur following nonsurgical trauma and complicate 2% of all head traumas, and 12% to 30% of all basilar skull fractures
- The most common presentation is unilateral watery rhinorrhea, and β_2 -transferrin is now the preferred method of confirming a fluid as CSF
- High-resolution computed tomography (CT) is the preferred method of localizing the site of skull base defect following craniomaxillofacial trauma, but can be coupled with magnetic resonance imaging (MRI) or cisternography
- Conservative management of CSF leaks includes strict bed rest, elevation of the head, and avoidance of straining, retching, or nose blowing, and results in resolution of the majority of traumatic CSF leaks over a 7-day period
- CSF diversion improves the rates of resolution when added to conservative therapy
- Endoscopic endonasal approaches should be the preferred method of repair with greater than 90% success rates; however, open transcranial and extracranial repairs still have their place in operative management
- Small postsurgical defects can be repaired endoscopically with mucosal grafts, while the pedicled nasal septal flap has emerged as the preferred method of repair for large defects
- Prophylactic antibiotics have not been shown to reduce the risk of meningitis and may select for more virulent organisms
- Data suggest that early surgical repair (<7 days) can reduce the risk of meningitis.

The authors have no relevant disclosures.

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Otolaryngol Clin N Am 44 (2011) 857–873

doi:[10.1016/j.otc.2011.06.007](https://doi.org/10.1016/j.otc.2011.06.007)

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EBM Question	Level of Evidence	Grade of Recommendation
Do spontaneous CSF leaks represent a distinct clinical entity and a variant of idiopathic intracranial hypertension?	2b	B
Does a reduction in CSF pressure alone lead to resolution of CSF leaks?	5	D
Does decreasing ICP, transiently or long-term, improve outcomes of endoscopic closure of spontaneous CSF leaks?	4	C

CSF is an ultrafiltrate of plasma or serum, which contains electrolytes, glucose, and proteins. Located in the cerebral ventricles and cranial/spinal subarachnoid space, this fluid serves in physical support and provides buoyancy for the brain and spinal elements. CSF also serves to maintain homeostasis of neural tissues by removing by-products of metabolism and regulating the chemical environment of the brain.¹ CSF is produced by the choroid plexus in the lateral, third, and fourth ventricles. It circulates from the lateral ventricles through the foramina of Monro into the third ventricle and then to the fourth ventricle through the aqueduct of Sylvius, then communicates with the subarachnoid space through the foramen of Magendie and foramina of Luschka. CSF circulates throughout the meninges between the arachnoid and is reabsorbed into the venous system via arachnoid villi, which project into the dural venous sinuses. Adults average 140 mL of CSF volume, and the body produces 0.33 mL of CSF per minute and thus regenerates its CSF volume 3 times per day.¹ As CSF is an ultrafiltrate of serum, serum abnormalities will be reflected in CSF (ie, hyperglycemia). Fluctuations in the cerebral blood flow related to the cardiac output result in generalized brain volume fluctuations, which gives rise to the vascular pulsations seen in CSF. In addition, major branches of the circle of Willis are located in the subarachnoid space and are likely contribute to this phenomenon.

CSF leaks are rare but are associated with morbidity such as general malaise and headache. More importantly, they can lead to potentially life-threatening complications such as meningitis. As such, they require thorough and timely evaluation and treatment. CSF leaks occur when the bony cranial vault and its underlying dura are breached. Such leaks may be broadly categorized as traumatic or nontraumatic in origin. Traumatic causes can be further subclassified into surgical and nonsurgical, with surgical causes divided into planned (as in failure of reconstruction of a planned dural resection) or unplanned (as a complication following an ethmoidectomy).² Nontraumatic CSF leaks may be subclassified into high or normal pressure leaks, with the recognition that tumors can occupy either subclass by mass effect in the high-pressure group or by direct erosive effect on the skull base in the normal-pressure group. If no cause can be found the leak may be classified as idiopathic; however, with careful history taking, physical examination (including nasal endoscopy), and radiologic evaluation, true idiopathic leaks are rare.²

EPIDEMIOLOGY OF CEREBROSPINAL FLUID LEAKS

Approximately 80% of CSF leaks result from nonsurgical trauma, 16% from surgical procedures (although this number is rising), and the remaining 4% are nontraumatic. Of the traumatic leaks, more than 50% are evident within the first 2 days, 70% within the first week, and almost all present within the first 3 months.^{3,4} Delayed presentation may result from wound contraction or scar formation, necrosis of bony edges or soft

tissue, slow resolution of edema, devascularization of tissues, posttreatment tumor retraction, or progressive increases in intracranial pressure (secondary to brain edema or other process). As with most maxillofacial trauma, traumatic CSF leaks occur most commonly in young males and complicate 2% of all head traumas, and 12% to 30% of all basilar skull fractures.⁵

Anterior skull base leaks are more common than middle or posterior leaks, due to the firm adherence of the dura to the anterior basilar skull. The most common sites of CSF rhinorrhea following accidental trauma are the sphenoid sinus (30%), frontal sinus (30%), and ethmoid/cribriform (23%) (Fig. 1).⁶ Temporal bone fractures with resultant CSF leak can present with CSF otorrhea or rhinorrhea via egress through the Eustachian tube with an intact tympanic membrane. Although a rare complication of functional endoscopic sinus surgery (FESS), the frequency in which this procedure is performed makes it a significant cause of CSF leaks (Fig. 2). When looking at surgical trauma, the most common sites of CSF leak following FESS are ethmoid/cribriform (80%), followed the frontal sinus (8%) and sphenoid sinus (4%). After neurosurgical procedures the most common site of CSF leak is the sphenoid sinus (67%) because of the high number of pituitary tumors that are addressed via transsphenoidal approach.⁶

Regarding nontraumatic leaks, high-pressure leaks comprise approximately half of these, with more than 80% resulting from tumor obstruction.⁷ The remainder is caused by either benign intracranial hypertension (BIH) or hydrocephalus; however, recent publications suggest an increased role of BIH in idiopathic or “spontaneous” CSF leaks.⁸ One hypothesis is that in patients with occult elevated CSF pressure, an intermittent CSF leak may serve as a release valve that decompresses the elevated pressure. Once the leak resolves, either by the normalization of CSF pressure with an intermittent leak or by surgical repair, the CSF pressure will slowly increase and result in recurrence of the leak. It is not surprising that the association between idiopathic

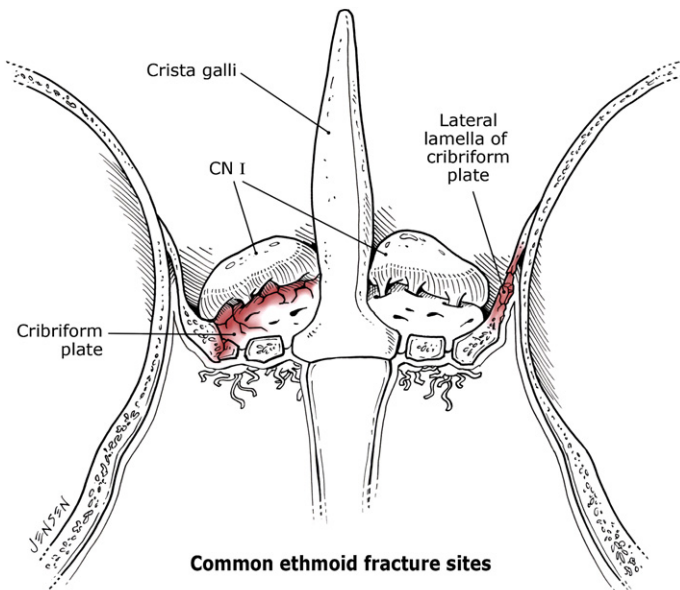


Fig. 1. Magnified view of the ethmoid bone in the coronal plane with the most common sites of fracture labeled. CN, cranial nerve.

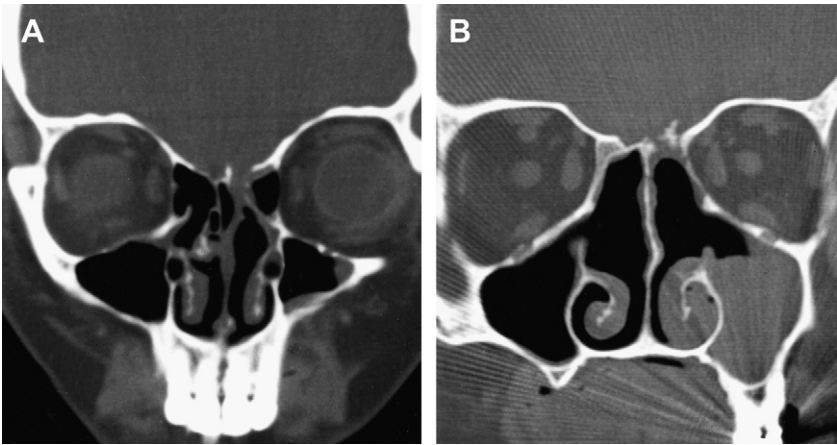


Fig. 2. Coronal CT scan demonstrating a defect in the roof of the left ethmoid sinus (A), and a defect in a separate patient with displacement of bony fragments intracranially (B). Both are the result of surgical trauma, sustained at another institution.

CSF leaks and BIH has been made, as the demographics of the populations are quite similar.⁹ Furthermore, the clinical manifestations and demographic profile of patients with empty sella syndrome are highly similar to those for patients with BIH and patients with nontraumatic CSF leaks.¹⁰ These associations have important clinical implications in the management of idiopathic CSF leaks as well as surgical failures after traumatic CSF leak repair.

DETECTION OF CEREBROSPINAL FLUID LEAKS

Presentation

Presentation of traumatic CSF leaks can be subtle, and diligence is required when one is suspected. Fain and colleagues¹¹ presented an analysis of 80 cases of trauma to the cranial base. From these cases they determined that there are 5 types of frontobasal trauma. Type I involves only the anterior wall of the frontal sinus. Type II involves the face (craniofacial disjunction of the Lefort II type or crush face), extending upward to the cranial base and to the anterior wall of the frontal sinus, because of the facial retusion. Type III involves the frontal part of the skull and extends down to the cranial base. Type IV is a combination of types II and III, whereas type V involves only ethmoid or sphenoid bones. In this study CSF leaks were infrequent in types I and II, but occurred more frequently in types III, IV, and V, which included a dural tear in each case. Therefore, when a patient presents with one of these fractures a CSF leak should be suspected.

The most common presenting sign is unilateral watery rhinorrhea following skull base trauma. Patients may complain of a salty or even sweet taste in their mouth; however, with severe trauma to the skull, history may be unobtainable secondary to the patient's neurologic status. Rhinorrhea is classically positional in nature and is most commonly associated with standing or leaning forward. Although this presentation seems straightforward, establishment of the diagnosis and precise localization of the leak can present major challenges. Other rhinologic pathology, including seasonal allergic rhinitis, perennial nonallergic rhinitis, and vasomotor rhinitis, are relatively common, and may mimic some of the signs and symptoms of CSF rhinorrhea or may occur simultaneously with a CSF leak. Furthermore, CSF rhinorrhea is often intermittent, even after

trauma, which may lead to false-negative results on diagnostic testing if testing is performed during the quiescent phase. Lastly, the subarachnoid cistern is a relatively low-pressure system. Thus, leaks may be of low volume, which can lead to false-negative testing or failure to recognize that a leak even exists. In cases of high clinical suspicion and initially negative diagnostic testing, further follow-up with repeat testing is warranted.

Identification

Traditionally the presence of a halo sign (clear ring surrounding a central bloody spot) on gauze, tissue, or linen has been used to predict CSF leak following trauma. This halo forms as blood and CSF separate; however, this test should only be used to arouse suspicion as tears, saliva, and other non-CSF rhinorrhea can give false-positive results. Historically the components of the rhinorrhea (including glucose, protein, and electrolytes) have been measured to confirm the diagnosis of CSF. These tests, however, should not be relied on, as their sensitivity and specificity are unacceptably low.^{7,12,13}

β_2 -Transferrin has emerged as a highly sensitive and specific way of identifying CSF, and is now the preferred method of confirming a fluid as CSF. The method was initially discovered in 1979 by Meurman and colleagues¹⁴ who, when performing protein electrophoresis of CSF, tears, nasal secretions, and serum, noted a β_2 -transferrin fraction only in the CSF samples. Techniques for isolating this marker have been subsequently simplified and refined, leading to enhanced sensitivity and specificity.^{15,16} As with any test, a reliable result requires an adequate sample. Of note, although quite specific, there are reports of β_2 -transferrin being detected in aqueous humor¹⁷ and in the serum of patients with alcohol-related chronic liver disease.¹⁸

Beta-trace protein (β TP) is another marker that has been used for the detection of CSF. This protein is produced by the meninges and choroid plexus and is released into CSF. It is present in other body fluids, including serum, but at much lower concentrations than in CSF. Detection of β TP has 100% sensitivity and specificity in cases of confirmed CSF rhinorrhea, but cannot be reliably used in patients with renal insufficiency or bacterial meningitis, because serum and CSF levels of β TP substantially increase with reduced glomerular filtration rate and decrease with bacterial meningitis.^{19,20}

LOCALIZATION

Once confirmed, localization of the dural defect is critical to management of CSF leaks, particularly if operative management is considered. Following identification of a traumatic CSF leak, nasal endoscopy should be performed. This procedure may narrow the side/site of the leak. Findings commonly are nonspecific, including glistening of nasal mucosa, but occasionally active leaks can be identified. Although direct visualization plays an important role, imaging of the skull base is critical to localization of CSF leaks, particularly traumatic leaks.

High-Resolution CT Scan

Multiple imaging studies have been used to localize defects, but the most common is high-resolution CT (HRCT) scanning. This technology uses 1- to 2-mm sections in both the coronal and axial planes with bone algorithm (see **Fig. 2**), resulting in localization of the majority of skull base defects that result in CSF leak. However, it is important to recognize that congenital or acquired thinning or absence of portions of the bony skull base may be identified and may not necessarily correspond to the site of CSF leak.²¹

This technology can be used with most surgical image guidance systems. Due to the relative ease of obtaining this study and high degree of accuracy, this method should be used as the primary imaging modality for traumatic CSF leaks. Plain CT scans may lead to false-positive results secondary to volume averaging, and their use should be limited. The use of intrathecal fluorescein in combination with HRCT allows for the identification of most CSF leaks.

Intrathecal Fluorescein

Intrathecal agents have been used both to confirm the presence of and to attempt to localize CSF leaks. These agents are administered via lumbar puncture into the subarachnoid space and, as such, complications can be severe. Visible dyes, radiopaque dyes, and radioactive markers have been used with a positive result being visualization, either directly or radiographically, of the agent within the nose and paranasal sinuses.

Intrathecal fluorescein is the most popular visible agent. Popularized by Messerklinger,²² intrathecal fluorescein has been associated with multiple complications, including grand mal seizures and even death. However, in a study of 420 administrations low-dose (50 mg or less) intrathecal fluorescein was found to be useful in localizing CSF fistulas and was deemed unlikely to be associated with adverse events, as most complications were dose-related.²³ The current recommended dilution is 0.1 mL of 10% intravenous fluorescein (not ophthalmic preparation) in 10 mL of the patient's own CSF, which is infused slowly over 30 minutes. Patients should be extensively counseled about the risks, as this use is not approved by the US Food and Drug Administration (ie, off-label use).

CT Cisternograms

CT cisternography involves the intrathecal administration of radiopaque contrast (metrizamide, iohexol, or iopamidol) followed by CT scanning. Studies have shown that approximately 80% of CSF leaks can be confirmed through this technology.²⁰ Weaknesses of this technology include its invasive nature, which can limit its use particularly in the pediatric population, as well as its low sensitivity in intermittent leaks.²⁴ Positive findings usually reveal pooling of contrast in the frontal or sphenoid sinuses, but may not necessarily locate the actual defect. Furthermore, the density of the dye may obscure bony anatomy, leading to more difficulty in locating the bony defect.

Radionuclide Cisternograms

A variety of radioactive markers have been used to detect CSF leaks, including radioactive iodine (¹³¹I)-labeled serum albumin, technetium (^{99m}Tc)-labeled serum albumin or diethylenetriamine penta-acetic acid (DTPA), and radioactive indium (¹¹¹In)-labeled DTPA. This technique is similar to intrathecal fluorescein and involves administration of the tracer via a lumbar puncture. Intranasal pledgets are placed in defined locations under endoscopic guidance and analyzed for tracer uptake approximately 12 to 24 hours later. A scintillation camera is also used, but has poor resolution and difficulty precisely localizing the leak. Overpressure radionuclide cisternography increases the intrathecal pressure with a constant infusion to improve the sensitivity of radionuclide cisternography²⁵; however, this modality in clinical practice results in a high degree of false-positive findings with sensitivities from 62% to 76%, limiting its utility.²⁴

MRI and MR Cisternograms

In contrast to the previously discussed cisternograms, MR cisternography is a noninvasive method for assessing the presence of intranasal CSF. This technique uses

T2-weighted images with fat suppression and image reversal to highlight CSF. The characteristic signal tracking from the intracranial space to the paranasal sinuses represents a CSF leak. The sensitivity of this test is reported to be 85% to 92%, with 100% specificity.²⁶ MRI and MR cisternography are able to distinguish inflammatory tissue from meningoencephaloceles; however, bony detail is poor (Fig. 3).

Cisternography warrants discussion however, because of the specificity and noninvasive nature of β_2 -transferrin testing, the role of various cisternography studies has been significantly altered. In the authors' practice, after the diagnosis of a CSF leak has been confirmed through β_2 -transferrin testing, high-resolution CT and MRI of the skull base are used to detail the anatomic integrity of the skull base. Endoscopic exploration confirms the location and is used for repair of the defect (discussed later) in the majority of cases. This philosophy has been adopted by other investigators as well.²⁷ Using this approach CT and MRI are complementary studies, with CT providing detailed bony anatomy, particularly skull base dehiscence/fractures, and MRI providing soft-tissue detail, including coincident meningoencephaloceles and incidental intracranial pathology. Modern image-guidance software enables the application of CT-MRI fusion for surgical navigation, and results in accurately identifying and localizing the site of CSF leakage in 90% of cases.²⁸

CONSERVATIVE MANAGEMENT

Once a CSF leak has been confirmed and localized, the optimal management decision depends on a variety of factors. Even if the otolaryngologist is primarily managing the CSF leak, direct input should be obtained from the trauma service, neurosurgical team and, particularly if meningitis is suspected, infectious disease colleagues. Conservative treatment consists of strict bed rest and elevation of the head at least 30°. In addition, patients should be advised to refrain from coughing, sneezing, nose blowing, and straining or Valsalva maneuvers. Stool softeners are recommended, as well as antiemetics to avoid emesis or retching, antitussives to

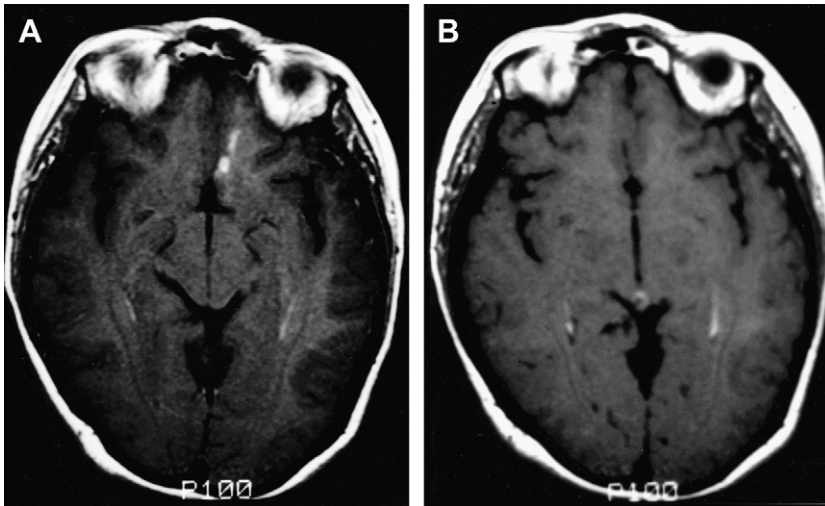


Fig. 3. Axial MRI scan post gadolinium following skull base trauma showing an enhancing lesion in the inferior-medial frontal lobe (A) with follow-up scan showing resolution of the lesion in 3 months (B).

avoid coughing, and strict blood pressure management. The goal of these measures is to reduce active flow through the leak, reduce CSF pressure, and allow healing of the defect to seal the leak, avoiding surgical intervention. In a series of 81 cases of traumatic CSF fistula, the overall rate of cessation with conservative treatment was 39.5% when used for 3 days. Resolution with conservative treatment of CSF fistulas involving temporal bone origin was 60%, whereas anterior skull base defects resolved 26.4% of the time with conservative treatment.²⁹ If conservative management is extended to 7 days, resolution rates improve to 85%, again with leaks of temporal bone origin healing with a significantly higher rate than those of anterior skull base origin.³⁰ The main reason for this discrepancy may be anatomic differences in the skull base bone and dural structures that are damaged with trauma to these subsites (ie, thin bone of the anterior skull base is more likely to cause significant dural lacerations than the thicker temporal bone).

Cerebrospinal Fluid Diversion

If there is persistence of the leak with conservative treatment, CSF diversion (most commonly with a lumbar drain but occasionally serial lumbar punctures) is pursued. Lumbar drains are passive devices yet they require active management, as CSF cell count, protein and glucose measurements, and cultures should be collected frequently to monitor for meningitis, particularly if systemic signs exist. Average drainage rates are around 10 mL per hour. Optimal drainage lowers CSF pressure to decompress the leak; however, if drainage is too high severe headaches and pneumocephalus may result from drawing of air through the skull base defect into the cranial vault. There is also the added risk of meningitis. The benefits are that the addition of CSF diversion to conservative measures raises success rates to 70% to 90% with the average duration of drainage being 6.5 days.^{30,31} Another benefit of this treatment is that it can be performed at the bedside, even if patients are not stable enough to go to the operating room. Lumbar drains can also be used as an adjunctive treatment to increase the success rates following a variety of surgical repairs.³¹

SURGICAL MANAGEMENT

Transcranial Approach

Although CSF rhinorrhea was initially described in the seventeenth century, it was not until 1926 that Dandy³² reported the first successful repair by using a bifrontal craniotomy for access and a fascia lata graft for repair. With this approach, access to the cribriform plate region and roof of the ethmoid is obtained via a frontal craniotomy. An extended craniotomy and skull base dissection are required to access defects in the sphenoid sinus. After craniotomy the brain is retracted and the site of the defect is identified. Multiple tissues can be used for repair including fascia lata grafts, muscle plugs, and pedicled galeal or pericranial flaps. Tissue sealants, such as fibrin glue, can be used to hold the graft in position; however, this will only last a few weeks, leading the authors to prefer suture closure to the dura distal to the defect, which more securely holds the graft in place. Reported success rates vary; however, recurrence rates as high as 27% have been reported.³³ The clear advantage of this approach is that it provides direct access to the defect and allows for repair of multiple sites; however, with high reported failure rates, the morbidity of a craniotomy, and brain retraction (including potential hematoma, seizures, and anosmia), extracranial techniques are now preferred in most circumstances. At present these techniques are mostly used in patients who require a craniotomy and exposure of the skull base to treat associated intracranial pathology.

Extracranial Approach

The first extracranial approach was described by Dohlman³⁴ in 1948, when he used a naso-orbital incision to repair a CSF leak. Dissection is then carried into the sinus cavities through the external incision for trans-sinus access to the skull base defect. The defect is identified and repaired directly using tissue grafts. Success rates with this approach range from 86% to 97%.^{35,36} The benefits of this approach include improved success rates with decreased morbidity, including avoiding anosmia and brain retraction with improved exposure of the posterior wall of the frontal sinus, fovea ethmoidalis, cribriform plate, posterior ethmoids, sphenoid, and parasellar regions. Drawbacks include the necessity of a facial scar, facial numbness, orbital injury, and relative difficulty of dissection. Further intracranial pathology as well as leaks originating from the lateral aspects of the frontal and sphenoid sinuses cannot be addressed.

Transnasal Approach

Further advancements were made in 1952, when Hirsch³⁷ reported the successful closure of two sphenoid sinus CSF leaks via a transnasal approach. In 1964, Vrabec and Hallberg³⁸ repaired a cribriform defect using this approach. In an attempt to improve visualization, Lehrer and Deutsch³⁹ used a microscope; however, access and visualization of the lateral and superior walls of the sphenoid sinus were limited. Inherent to this approach is the risk of facial numbness and septal perforation, and with the advent of endoscopic techniques this approach became rarely used.

Endoscopic Endonasal Approach

Endoscopic techniques have emerged as the preferred approach to the repair of skull base defects since their initial description by Wigand⁴⁰ in 1981. This initial report described repair of a defect encountered during sinus surgery. In 1989 the first report of the use of rigid transnasal endoscopy for the endonasal repair of CSF rhinorrhea was described,⁴¹ followed by a series of cases presented by Mattox and Kennedy⁴² addressing CSF rhinorrhea with the aid of endoscopic visualization. Following identification and localization of the skull base defect, standard endoscopic techniques are used to expose the defect site. This approach provides excellent exposure of the ethmoid roof, cribriform plate, and the sphenoid sinus.

When lateral exposure in the sphenoid is necessary, endoscopic dissection of the pterygomaxillary space can be performed. Exposure of the posterior aspect of the frontal sinus may require an osteoplastic flap or simple trephine. If needed, intrathecal fluorescein can be used to aid in identification of the skull base defect. Associated meningoencephaloceles are then addressed with bipolar electrocautery.

Mucosa that is adherent to dura must be dissected away. It is critical that the mucosa surrounding the skull base defect be removed for approximately half a centimeter. This technique both stimulates osteogenesis (thickening the bone surrounding the defect) and improves graft incorporation. Following site preparation, the decision for graft type can be made.

The choice of graft material has been a source of debate for some time; however, based on a recent meta-analysis it appears graft material does not affect success rate as long as sound surgical technique is used.⁴³ Graft choices include temporalis fascia, fascia lata, muscle plugs, mucosal grafts (with or without bone), autogenous fat, free cartilage grafts (from the nasal septum or auricle), and free bone grafts (from the nasal septum, calvarium, or iliac crest). For small defects, free mucosal or free fascial grafts can be placed in an overlay fashion. Significant overcorrection

should be performed when forming the graft, as one can expect approximately 20% shrinkage in free mucosal graft size, starting in the first few postoperative days. Large defects at risk for secondary encephalocele are sometimes treated by placement of a bone or cartilaginous graft in an underlay fashion, with a fascial or mucosal graft placed as an overlay to form a watertight seal. In the authors' experience, this is usually not necessary. For large skull base defects the use of vascularized pedicled mucosal flaps are preferred, and this method is discussed in the following section.

In general, the authors' technique involves the placement of an inlay dural substitute graft (ie, Duragen; Integra, Plainsboro, NJ, USA) followed by a mucosal graft in the case of small defects or a vascularized pedicle flap for larger defects. Following graft placement, a sealant (such as fibrin glue) can be used to hold the graft in place. Absorbable nasal packing (gel-foam) is usually placed directly against the mucosal surface of the graft for additional support, followed by placement of nonabsorbable packing for further support and hemostasis. Packs are usually removed in 5 to 7 days, and antistaphylococcal antibiotic coverage should be used while nonabsorbable packs are in place.

Postoperatively the patient should be placed on conservative measures (as previously discussed). As dissection occurs through a contaminated operative field, most surgeons elect to use perioperative antibiotics. Antibiotic choice should have good CSF penetration (eg, ceftriaxone). At a minimum an antistaphylococcal antibiotic should be given postoperatively if nonabsorbable nasal packing is used. Admission to the intensive care unit for monitoring and frequent neurologic status checks should be considered for the first 24 hours to monitor for complications such as hematoma or cerebral edema, although this is institution dependent. If a lumbar drain is in place from prior attempts to slow CSF drainage, or if one was placed intraoperatively, it should be actively managed as discussed previously. Following removal of the nasal packing, serial nasal endoscopies should be performed for debridement of any crusting and to inspect the operative site. Care should be taken not to disrupt the graft during debridements, and patients should avoid strenuous activity or straining for approximately 6 weeks following the repair.

The endonasal endoscopic technique offers several advantages, including excellent visualization and identification of the defect as well as graft placement. Reported outcomes are excellent, with success rates of 90% for primary procedures and 96% for secondary endoscopic repair. The approach is remarkably well tolerated and complication rates are low, but present the risk for hemorrhage, infection, and graft failure.

Surgical Defects

CSF leaks after FESS tend to be small in size, less than 5 mm, and amenable to closure using any combination of inlaid bone, overlaid fascia, or free mucosal graft. However, over the past few years advances in endonasal endoscopic tumor removals have resulted in larger dural defects and have necessitated development of newer vascularized techniques to reconstruct the skull base. Although devascularized, layered reconstruction techniques of large skull base defects have been extensively described; they often result in high incidence of CSF leak and risk of meningitis. The use of vascularized mucosal pedicled flaps has been the most significant advancement in skull base reconstruction over the past decade.⁴⁴⁻⁴⁶ The use of the posteriorly based pedicled nasal septal flap has become the primary mode of reconstruction following extensive endoscopic cranial base resections,⁴⁷ and decreased CSF leak rates following extensive endoscopic skull base reconstruction have been documented.⁴⁵ This flap is raised endoscopically (**Fig. 4**) and is based on the posterior septal artery (**Fig. 5**). Correct harvesting of this flap allows rotation of the flap in the

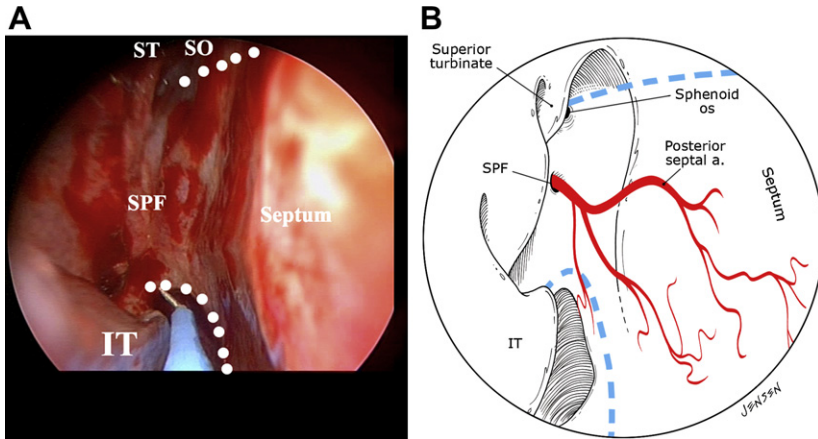


Fig. 4. Endoscopic view of the posterior nasal cavity (A) with an artist's representation of the same view (B). Dashed lines mark the location of cuts for the posteriorly based nasal septal flap. IT, inferior turbinate; SO, sphenoid os; SPF, sphenopalatine fossa; ST, superior turbinate.

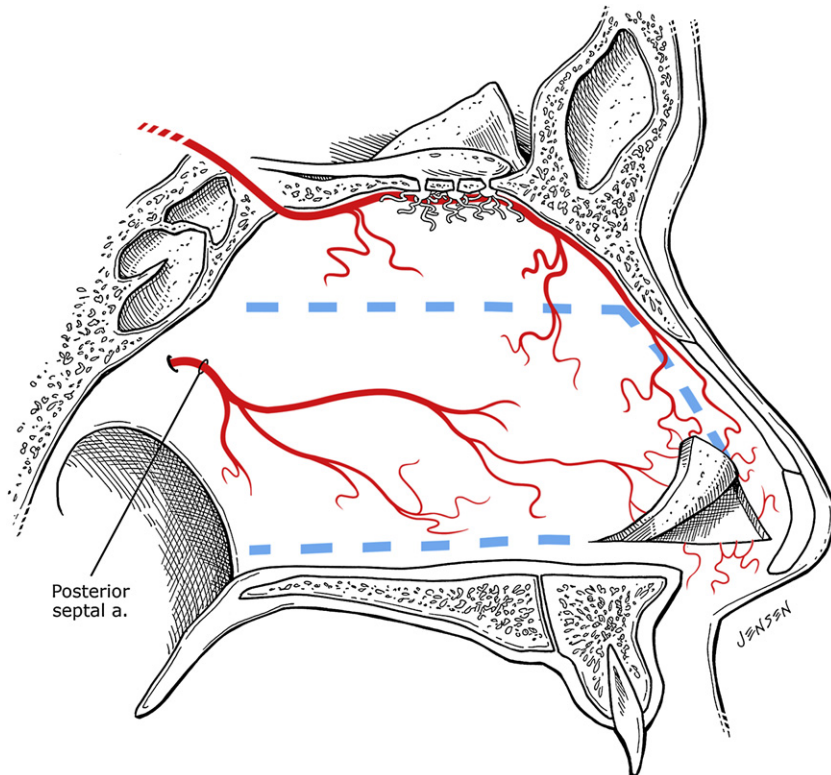


Fig. 5. Axial view of the nasal septum demonstrating the posterior septal branch of the sphenopalatine artery.

posterior, superior, inferior, or lateral directions. This flap is large enough to cover maximal dural defects extending from the frontal sinus to sella and from orbit to orbit (ie, craniofacial resection defect).⁴⁵ This flap can be used successfully in pediatric patients older than 14 years, but caution is recommended in younger patients, and it may not be a viable option in patients younger than 10 years, due to inadequate length.⁴⁸ Following endoscopic skull base reconstruction, it is often difficult to assess the integrity of the repair. Vascularized flaps have a characteristic appearance (mucosal enhancement) on MRI, which can cause some confusion following tumor resection, as radiologists and surgeons unfamiliar with this appearance may confuse the flap with residual tumor because traditional graft material (fat, explanted muscle, and synthetics) do not enhance. However, its appearance to the trained eye makes it ideal for monitoring the integrity of the skull base repair.⁴⁹ When a nasoseptal flap cannot be used, an anteriorly based pericranial flap can be elevated using minimally invasive techniques and placed intranasally without a craniotomy.⁵⁰ Other vascularized flaps available for endonasal skull base reconstruction include middle and inferior turbinate pedicled flaps,^{51–53} temporoparietal fascia flap,⁵⁴ and palatal flap.⁵⁵

CLINICAL QUESTIONS

Do Prophylactic Antibiotics Prevent Meningitis in Posttraumatic CSF Leaks?

Bacterial meningitis is the major cause of morbidity and mortality in patients with CSF leaks, making antibiotic prophylaxis a reasonable suggestion; however, this topic has been the source of significant controversy. The primary concern is that a CSF leak presents a direct route of infection from the contaminated nasal cavity to the intracranial space; however, unwarranted antibiotic use has the potential to select for resistant organisms. The reported incidence of meningitis in patients with posttraumatic CSF fistulas varies widely from 2% to 50%, with 10% being a generally accepted rate.^{56–59}

Most of the controversy stems from two separate meta-analyses published 1 year apart. In 1997 Brodie⁵⁸ published a meta-analysis that showed a statistically significant reduction in the incidence of meningitis with prophylactic antibiotic therapy for patients with traumatic CSF leakage. This article was followed by a meta-analysis published in 1998 by Villalobos and colleagues,⁶⁰ which concluded that antibiotic prophylaxis after basilar skull fracture does not decrease the incidence of meningitis, independent of active CSF leak. However, critiques of these meta-analyses point out that neither of these studies included an extensive review of the literature and that the conclusions drawn were based mainly on retrospective and observational studies.⁶¹ Further, it was suggested that the meta-analysis by Brodie omitted one of the largest case series addressing this question.⁶² Recently a Cochrane Database review was performed to address these deficiencies. The analysis included 208 patients from 4 randomized controlled trials and an additional 2168 patients from 17 nonrandomized controlled trials. The analysis concluded that the evidence does not support the use of prophylactic antibiotics to reduce the risk of meningitis in patients with basilar skull fractures or basilar skull fractures with active CSF leak.⁶¹ Analysis of both randomized and nonrandomized controlled trials failed to show a benefit or adverse effects of prophylactic antibiotic use in patients with active CSF leak; therefore, the current available literature suggests that prophylactic antibiotics do not decrease the risk of meningitis. It should be stated that perioperative antibiotics are indicated for surgical repair of CSF leaks, and in certain circumstances (such as active bacterial rhinosinusitis or grossly contaminated tract leading to the intracranial cavity) antibiotic coverage is reasonable.

Strength of evidence: Grade A

Does Early Surgical Repair Prevent Meningitis in Patients with Traumatic CSF Leaks?

Multiple factors seem to affect the rate of meningitis, including the duration of CSF leakage, the site of the skull base defect, and active sinus infections. Patients with traumatic CSF leaks lasting more than 7 days have been shown to have an estimated eightfold to tenfold increase in the risk of meningitis.^{56,57} This finding has led some investigators to advocate surgical repair of CSF leaks that are persistent after 7 days of conservative treatment.⁶ However, in some series conservative treatment has been associated with a high incidence of meningitis.⁶³ Furthermore, it has been shown that endoscopic closure of defects prevented meningitis in patients with and without a history of CSF infection.⁶⁴ These data lead the authors and many investigators to recommend early (less than 7 days) or urgent (less than 3 days) closure of traumatic CSF leaks as long as the patient is a suitable candidate for endoscopic closure.^{6,63,64} However, in these studies it is difficult to assess the long-term risk of meningitis following surgical repair, as delayed cases of meningitis have been reported. Harvey and colleagues⁶⁵ looked to address this long-term risk and found that surgical repair results in decreased incidence of intracranial complications (including meningitis) over the long term. After assessment of risk factors for development of complications, they concluded that in patients with small nonleaking encephaloceles without adjacent infection, early surgical repair may not be indicated.

Strength of evidence: Grade C

SUMMARY

Indications for surgical intervention on traumatic CSF leaks include failure of conservative measures, identification of a CSF leak during FESS or skull base surgery, large skull base defects unlikely to heal with conservative measures (particularly if associated with pneumocephalus), or indication for associated surgical procedure to address other intracranial pathology. Recognizing that a single management strategy cannot possibly direct the care of each patient, given the varied mechanism of injury and patient presentations; the authors suggest the following evaluations to guide management.

Nonsurgical Trauma

When a patient presents with a nonsurgical traumatic CSF leak, fluid should be sent immediately for β_2 -transferrin testing (as in some institutions this is a send-out test and may take several days to receive the result). If positive, conservative measures should be rapidly initiated and a high-resolution CT scan should be obtained for localization of the skull base defect. If needed, an MRI scan or cisternograms may be obtained for additional information/confirmation as already discussed. If the defect is in the anterior skull base, conservative treatment should be performed for 3 days. At this point, if the leak persists consideration of CSF diversion or endoscopic repair should be discussed in a multidisciplinary fashion. If CSF diversion is initiated and the leak is persistent, definitive repair (preferably endoscopic) should be performed around CSF leak day 7. If the CSF leak originates in the temporal bone, conservative measures should be continued for 5 to 7 days followed by CSF diversion for an additional 7 days, as these are more likely to close spontaneously and less likely to result in meningitis. Further operative repair of these defects cannot be addressed endoscopically, and definitive surgical repair of these defects can carry significant morbidity. If the leak persists for 2 weeks, definitive surgical repair should be performed.

Special consideration should be given to patients with coexistent intracranial pathology. If significant brain edema is present, early repair is likely to fail in the setting of elevated intracranial pressure. However, if the patient requires neurosurgical intervention, it is reasonable to repair coexistent CSF leaks at the same time. If a significant defect exists early definitive repair should be initiated, as conservative therapies are likely to fail. Perioperative antibiotics should be administered for surgical repair. If the patient is too ill to tolerate surgical repair, conservative measures should be continued until the patient is stable enough to undergo an operation.

Surgical Trauma

Intraoperative CSF leaks recognized at the time of surgery (either FESS or skull base procedures) should be immediately repaired. For planned dural resections, reconstruction should be “watertight.” If recognition is delayed by days, weeks, or months, it is reasonable, although not the authors’ practice, to initiate a short period of conservative therapy (a few days to 1 week). The leak should be confirmed and identified (as already discussed), and repaired if the leak persists for 1 week despite conservative measures. One should recognize that the later the CSF leak presents following the procedure, the less likely it is to resolve with conservative measures alone. Moreover, if the leak is massive in nature, early surgical intervention is indicated.

Traumatic CSF leaks are infrequent complications of craniomaxillofacial trauma or sinus/skull base surgery, but carry significant morbidity and mortality if not promptly recognized and appropriately managed. The main complication of CSF leaks is meningitis; however, tension pneumocephalus, meningoencephaloceles, and brain abscesses do occur. Identification and localization are critical to management. Conservative therapies should be initiated immediately, and consideration for operative intervention should involve a multidisciplinary team consisting of otolaryngology, neurosurgery, trauma, craniomaxillofacial surgery and, if meningitis is suspected, infectious disease physicians. The current evidence fails to show any benefit or adverse effects of prophylactic antibiotics, and it appears that endoscopic closure of persistent CSF leaks can prevent meningitis. This procedure has a high success rate in both primary and secondary surgery, and should be the primary approach for repair of posttraumatic anterior skull base CSF leaks.

EBM Question	Author’s reply
Does spontaneous CSF leaks represent a distinct clinical entity and a variant of idiopathic intracranial hypertension?	Spontaneous CSF are both associated with raised ICP and idiopathic intracranial hypertension
Does a reduction in CSF pressure alone lead to resolution of CSF leaks?	Case reports describe resolution of CSF leaks with both diversion and other methods of CSF pressure reduction.
Does decreasing ICP, transiently or long-term, improve outcomes of endoscopic closure of spontaneous CSF leaks?	CSF pressure modifying medications and diversion have been shown to improve the longterm morbidity of IIH patients.

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