Paparella: Volume III: Head and Neck

Section 2: Disorders of the Head and Neck

Part 1: Nose and Paranasal Sinuses

Chapter 8: Cerebrospinal Rhinorrhea

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In my opinion, extracranial techniques should be reserved for sellar and parasellar leaks in the carefully selected patient in whom the etiology of the leak has been clearly understood and the fistula tract demonstrated and in whom the intracranial methods cannot be undertaken for definite reasons. The rationale for this opinion is that CSF rhinorrhea is not simply a matter of closure of the bony defect, but, more importantly, necessitates repair of meningeal defects. Such meningeal repairs are best conducted after adequate inspection intradurally, and rhinologic procedures seldom provide an adequate view within the dura mater, except for the lower part of the sella turcica as exposed through the sphenoid sinus.

Ommaya, 1985

Cerebrospinal fluid rhinorrhea is a rare cause of a common complaint - watery nasal discharge. The fundamental cause, as Ommaya emphasizes, is a *meningeal* fistula; the bony defects are secondary. Contrary to his instruction however, extracranial techniques should be employed for the majority of cases because of their safety and success.

A patient with an unrecognized or untreated dural fistula in the base of the skull has a risk of meningitis approaching 50 per cent. The organisms originate in the nose and sinuses and most commonly include *Pneumococcus pneumoniae,* beta-hemolytic streptococci, or, in children, *Haemophilus influenzae.*

Otolaryngologists are generally called upon to recognize and localize cerebrospinal fluid fistulas before meningitis occurs. They are more and more commonly asked to treat cerebrospinal fluid leaks as well or to collaborate in their repair with a neurosurgeon. The important clinical questions that arise include:

1. Is the rhinorrhea fluid really cerebrospinal fluid?

2. Where is it coming from?

3. Why is the dura dehiscent?

4. How can it be exposed?

5. How should it be repaired?

To understand and begin to answer these questions, some background information will be helpful.

Historical Perspectives

Galen hypothesized that cerebrospinal fluid was excreted into the nose via the pituitary gland! He was not refuted until 1655, and even to this day, some individuals dispute the idea that communications between the cranial cavity and the nose are abnormal. To sustain the notion that cerebrospinal fluid rhinorrhea might sometimes be physiologic, Gordon cited the following observations:

1. Glucose is present in many samples of normal nasal secretions, so it might be an indication of cerebrospinal fluid contamination.

2. Some rhinorrhea, as described by Proetz, was not diminished by vasoconstriction. Thus, Gordon felt it must be independent of mucosal circulation.

3. Headaches associated with raised intracranial pressure could, according to Gordon, be relieved by rhinorrhea.

4. Cerebrospinal fluid in dogs allegedly finds its way into the submucosal lymphatics of the dog's nasal mucosa.

The more conventional view is that cerebrospinal fluid rhinorrhea is distinctly abnormal. The failure of human anatomists to identify a normal nose-brain pathway, the rarity with which nasal organisms invade the cerebrospinal fluid in patients with chronic ethmoiditis, and the paucity of pneumatoceles in patients given to frequent and forceful blowing of the nose seem to support this view.

In 1826, Miller seemed to be the first to describe a high-pressure cerebrospinal fluid leak, which occurred in a hydrocephalic child. Chiari discovered intracranial pneumocephalus at an autopsy in 1884 and related its presence to a defect he discovered in the ethmoidal roof. St. Clair-Thompson reported 20 clinical cases of cerebrospinal fluid rhinorrhea in 1899 and raised the question of the role of the arachnoid sleeves that accompany olfactory nerves down through the cribriform plate. He also coined the misnomer spontaneous cerebrospinal fluid rhinorrhea to indicate cases in which trauma was not known to be the cause of the leak. It is well to remember that "spontaneous" leaks are not spontaneous at all. There are many causes, which include congenital focal bony defects, primary empty sellae, nasal encephalopathy, pituitary tumors, tumors producing hydrocephalus, and previous unremembered traumatic events.

Calvert's study of the outcome of traumatic frontoethmoidal leaks in the preantibiotic era (1942) dispels any notion of the benign nature of an untreated leak. Fifty per cent of the patients he reported on experienced meningitis, and in half of these patients, the disease was fatal. Klastersky and colleagues went on to show that prophylactic antibiotics were no substitute for repair. In his double-blind study, a significant incidence of damaging meningitis ensued despite the use of potent antimicrobial agents. Furthermore, the mortality rate from pneumococcal meningitis in the antibiotic era is still as high as 20 per cent.

The first successful intracranial repair of a cerebrospinal fluid leak was described by Dandy in 1926. This was followed immediately by Cushing's report of three more cases in

1927. Unsuccessful attempts at transnasal cauterization diminished much thought of an extracranial repair technique until Dohlman's report in 1948. He used septal and middle turbinate flaps to seal the leakage and avoided the need for craniotomy. Further descriptions of success with otolaryngologic techniques have been published by Vrabec and Halberg, Chidlow and DeSanto, Montgomery, McCabe, and Calcaterra. Montgomery's flaps were posteriorly based and used upper septal mucosa. The middle turbinate was removed for access. McCabe preferred to use the middle turbinate, including its bone, by rotating it laterally and upward to seal the roof of the ethmoid bone.

Other reports of interest in this field include Ommaya's identification of high-pressure leaks, Calcaterra's excellent review of the success of extracranial repairs, and Hubbard's follow-up on the management of patients with so-called spontaneous rhinorrhea.

Physiology of Cerebrospinal Fluid

Each of us produces approximately 500 mL of CSF each day from the choroid and the brain. The volume of cerebrospinal fluid in a normal adult is between 90 and 150 mL. Apparently one could leak more than 300 mL of cerebrospinal fluid each day and still maintain an adequate volume for normal functions!

The glucose content of cerebrospinal fluid is about 65 per cent that of blood glucose. This is higher than the level of reducing substances present in lacrimal and nasal secretions. However, it is not enough to rely on chemical titers of glucose as a specific test for cerebrospinal fluid.

There are no white blood cells, fibrin, or platelets in cerebrospinal fluid. It is now wonder that some leaks tend to persist; there is really no intrinsic system to manufacture a seal.

The function of cerebrospinal fluid is a matter of conjecture. It probably acts as a mechanical cushion for the brain, and it also seems to serve as a metabolic sink. The brain weight is 1400 g in air, but it "weighs" only 50 g in cerebrospinal fluid. There are no lymphatics in the brain, but we probably diffuse metabolites into the cerebrospinal fluid. From here they would circulate to the arachnoid villae and ultimately into the blood stream.

The cranial contents are conceptualized as three compartments: the brain, vascular spaces, and cerebrospinal fluid. The cranium is rigid, so the sum of the volumes remains constant. Therefore, they must be reciprocals of each other. The vascular spaces are the most rapidly elastic. Cerebrospinal fluid is incompressible, of course, but can be slowly displaced by an expansion of one of the other two factors. The brain has no meaningful way of compressing, and the consequences of brain tissue displacement, including coning, are well known. The descriptive model relating the compartments, volumes, and pressure dynamics is known as the Monroe-Kelly hypothesis.

These concepts are important in the context of cerebrospinal leaks for two reasons. Air can enter the cranium through the same fistula used by the cerebrospinal fluid to escape. Variations in the air pressure gradient between the cranial cavity occur with respiration, sneezing, and nose blowing. Furthermore, vascular pulsations in the cranium, combined with

the tamponading effects of brain tissue near the fistula, may set up a pump action and a ball and valve effect. The air that is admitted becomes an expanding intracranial mass (a tension pneumocephalus). The volume of blood and cerebrospinal fluid in the head can diminish, but once *their* capacities to adapt are exhausted, minute increases in the air mass can have disastrous effects on the brain.

Some cerebrospinal fluid leaks are associated with, and may even be a consequence of, localized or generalized elevations of intracranial pressure. High pressure, along with nontraumatic cerebrospinal fluid leaks, is a special case. The egress of fluid may be acting as a protective release. In such case, repairing the leak without correcting the cause of the pressure may worsen the patient's condition. The presence of hydrocephalus or a tumor may do this, and these conditions need to be considered and excluded.

Anatomy of the Anterior Portion of the Skull Base

When the separate components of the anterior portion of the skull base are viewed from below, the most notable feature of the frontal bone is a large central gap between the right and left orbital plates that is known as the ethmoidal notch. The cribriform plate of the ethmoid bone normally fills in this gap, so together the orbital plates of the frontal bone and the central ethmoidal complex form the floor of the anterior portion of the cranium. The lesser wings of the sphenoid bone, which are united across the midline behind the cribriform plate, in the jugum, form the posterior margins of the anterior cranial fossa.

The dura is thinnest and most adherent to the cribriform plate and to those most medial segments of the ethmoidal sinuses, which are exposed directly to the dura by the fact that the ethmoidal notch is actually wider than the cribriform plate, that is, the orbital plates of the frontal bone fail to slide all the way over the ethmoidal cells. They fall short as they approach the cribriform plate. The ethmoidal sinuses are roofed over incompletely by the orbital plates of the frontal bone.

The favorite site for a dural fistula to form is through the ethmoidal notch. Thus, it might just as well enter an ethmoidal sinus as the cribriform plate. The dura is more intimately attached, and the bone is delicate. The ethmoidal laminae are easily split by the forces transmitted to them by the quite thick outer calvarium. The calvarium, the supraorbital ridges of the frontal bone, and the buttresses of the zygomatic and maxillary bones may protect the brain and eyes, but they transmit their blows to the ethmoid bone. The dura is not only thin and adherent, but in the cribriform plate it is tethered by the passage of the olfactory nerve fibers through the 20 or 30 olfactory foramina and elsewhere by the periorbita interweaving into the frontoethmoidal suture lines.

The anterior ethmoidal arteries are very important landmarks in the ethmoidal region. They lie clamped between the orbital plates of the frontal bone and the open-roofed ethmoidal labyrinths. They run from the orbit, forward and medially, across the sometimes dehiscent sinus roofs, to a brief intracranial exposure at the anterior end of the cribriform plate. Here, they give off the tiny anterior meningeal arteries, then they dive forward under cover of the nasal bones to accompany the anterior ethmoidal nerves to their intranasal location.

There is one normal potential connection between the cerebrospinal fluid and the nose. At the front of the cribriform plate, anterior to the crista galli, a small pore marks the passage of the primitive venous connections between intranasal mucosa and the anterior end of the sagittal sinus. Hopefully, this has been obliterated before birth without enclosing any glial remnants, for these remnants can later give rise to the congenital communication between the nose and the brain that a nasal glioma or an intranasal meningomyelocele represent.

The level of the ethmoidal roof and cribriform plate is a subject of interest when designing any extracranial approach to these situations. The roofs of the ethmoidal sinuses may be envisioned as coming straight forward from the planum sphenoidal to the anterior ethmoidal arteries before they turn up to become "frontoethmoidal". The artery serves as a valuable landmark in approaches to the ethmoidal sinuses from the orbital side. Behind the artery, the roof of the ethmoidal sinus is relatively level and continuous with the roof of the sphenoidal sinus. Anterior to the artery, it swoops up, eventually, to become the posterior wall of the frontal sinuses. The roof of the nasal cavity, the cribriform plate, is quite narrow, and is always on a horizontal plane that is slightly lower than the roof of the ethmoidal sinus. This relationship is fundamental. An instrument placed in the nose and laid up as high as the cribriform plate, if directed laterally, will penetrate an ethmoidal cell. However, an instrument applied to the ethmoidal roof, then pressed medially will *not* enter the nasal cavity. The cribriform plate is lower than the instrument; the instrument will penetrate into the anterior cranial fossa, in the region of the olfactory groove.

Some mention should be made of the olfactory filaments that penetrate the cribriform plate. About 15 to 20 fibers are found on each side. They carry the afferent axons of the bipolar cells of the olfactory mucosa upward to their synapses in the glomeruli of the olfactory bulbs. Each filament is accompanied by an arachnoid sleeve. The distance between the nasal roof and the cerebrospinal fluid at the foramina of the cribriform plate is only the thickness of the olfactory mucosa. In a sense, the dura is functionally perforated at these points.

Clinical Evaluation of Suspected Cerebrospinal Fluid Rhinorrhea

Is Fluid From the Nose Cerebrospinal Fluid?

With a history that is at all compatible with a breach in the skull base dura, any persistent watery nasal discharge should be suspect. Certain features help distinguish it from the watery nasal discharge of chronic allergic rhinitis or sinusitis. One of the most characteristic is the uncontrollable nature of the fluid. Having virtually no mucoprotein component, and hence no viscosity, cerebrospinal fluid cannot be sniffed back into the nasal cavity readily. Patients with intermittent leaks will frequently report embarrassing dripping that appears suddenly and without warning. A patient might lean forward over a counter, for example, and find that fluid has dripped onto the countertop before he or she could either sense or conceal the event. There is no sneezing, no congestion, no lacrimation, and no response to antihistamines. Some patients can produce this fluid in the office by leaning forward and performing a Valsalva maneuver. This raises the intracranial pressure and may produce a drop or two that can be observed and collected or touched with a glucose oxidase-peroxidase Tes-Tape. Leaning forward or rolling over may also produce fluid because of the "teapot effect", which refers to the situation in which a sinus, typically the sphenoidal sinus,

is slowly filled with fluid to the level of its ostium. The fluid then comes out with a gush when the head is tipped forward to an ostium-dependent position.

Patients who cannot produce cerebrospinal fluid for examination, but in whom the history is suggestive, or their anxiety requires an answer, can be given a Tes-Tape and asked to check the next drop they notice by absorbing it on the paper. They can report back later and bring the tape. Their inability to wet the Tes-Tape over a suitable period or to demonstrate the presence of glucose (by observing and recording the corresponding color change) argue against an active fistula. If fluid *is* produced, but the test results are repeatedly negative (even after meals), the chance that the fluid is cerebrospinal is even further reduced. A negative test result can occasionally occur in the presence of cerebrospinal fluid with a low glucose content. The lack of fluid does not exclude a dural fistula, which is temporarily plugged by arachnoidea. Tears (via the nasolacrimal duct) can sometimes produce a false-positive result.

Of the numerous imaging studies reported on the literature, none is exclusive and none is infallible. Sinus x-ray films, or better yet, anteroposterior (AP) sinus tomograms, will occasionally show a fluid level on the brow-up lateral plain film (in the sphenoidal) region or in an individual ethmoidal cell or concha bullosa. Plain films usually are not very helpful. Normal tomograms are often marked by the radiologist to indicate a cribriform dehiscence. However, there is a tendency to "overcall" suspected bony defects in the cribriform region, which after all is small, multiperforated, and grooved by the ethmoidal arteries. A bony deformity and a fluid density and opacity in one of the ethmoidal cells is helpful in localization, but this can follow trauma without a leak too.

Computed tomography (CT) scans provide a better image of the intracranial spaces and soft tissues, but sometimes patients cannot be positioned with enough extension, particularly after trauma, to obtain primary images in the coronal plane. Reconstructed coronal images (from the axial cuts) are usually too coarse to draw definite conclusions. Metrizamide CT scans are helpful if the dye leaks down into the nose or an ethmoidal cell and is retained there for imaging before being blown out or drained. However, to distinguish a wisp of metrizamide from one of the fine bony laminae of the ethmoidal complex can be challenging.

Radioisotopes, like technetium pertechnetate (T 99m), have long been recommended in the identification of cerebrospinal fluid fistulas. When we employed it, our technique was to place tiny cotton pledgets in seven locations in the nose. Printed labels were made ahead of time and tied to the pledgets to reflect the locations, which included the cribriform roof, the middle meatus, and the sphenoethmoid recess, in both nasal cavities. The seventh pledget was a lacrimal control, carefully placed under one inferior turbinate. Insertion was performed under meticulous topical cocaine administration, using fine alligator forceps and taking great care to avoid abrading the nasal mucosa. The pledget strings and tags were taped out on the face, like cat whiskers. Before the pledgets were placed, patients were routed to nuclear medicine, at which point the radioisotope was instilled through a lumbar puncture. About 2 hours after the lumbar puncture, the pledgets were removed and placed in special glass tubes that were returned, with the patient, to the nuclear medicine lab for counting. If the counts were normal, a head scan was taken to prove that the isotope had risen into the basal cisterns. The patient should be referred back to rhinology again for a second set of pledgets to be inserted, which were removed after about 6 hours, for a second pass, or the patient could even

be deferred overnight and checked for activity in the morning.

In our hands, the isotope test was often defeated by the scheduling logistics and the wide variations in timing. Pledgets removed too early of course gave falsely negative results. Pledgets removed too late were of questionable value. Often they all gave positive results, and it was evident on removal that a continuous layer of mucus, stimulated by the presence of these foreign bodies, had spread the isotope throughout the nasal cavities. We have abandoned the radioisotope tests in favor of fluorescein dye, but we admit that institutions may vary in their preferences.

As indicated, our best test is probably the instillation of fluorescein dye. This procedure avoids having to send the patient or the pledgets, or both, back and forth to different departments. It allows the rhinologist to make a direct determination. If an early reading is obtained and is shown to be negative, the pledgets can be immediately reinserted and the test rescheduled for later in the day.

The safety of fluorescein dye has been questioned by some. Those who have reported it to be safe, however, have stressed a method of dilution and instillation that we have followed and have found to be satisfactory. One-half milliliter of a 10 per cent mixture is slowly instilled through the spinal catheter over at least 1 minute. The placement of the nasal pledgets and the use of an inferior meatus control are similar to the procedure described for the isotope test. An ultraviolet light is used to illuminate the pledgets in a darkened room when they are inspected. When fluorescence is present, it is generally easy to distinguish as a soft green glow in the cotton fibers. Bear in mind that it takes intrathecal dyes about 6 hours to circulate up to the skull base and about 24 hours to completely cover the hemisphere. In older patients, it may take even longer.

Why is the Dura Broken?

Once cerebrospinal fluid is positively identified, it is prudent to stop and think what this means. There is a break in the *dura.* In most instances, the reasons are obvious from the history - it might be trauma, surgery, or a tumor. In so-called spontaneous cerebrospinal fluid rhinorrhea, a search must be made for a cause, especially an unsuspected intracranial cause. An intracranial lesion, such as a previously undetected neurologic tumor, can be the cause of the breech in the dura. Raised intracranial pressure, which can have many causes and can be generalized or localized, can also be responsible. Cerebrospinal fluid pulsations transmitted against thins meninges and congenitally hypoplastic anterior cranial fossa bone can, over sufficient periods, result in multiple spontaneous leaks. The diagnosis of raised intracranial pressure is difficult, of course, in the presence of a leak, since the leak decompresses the pressure. If the fistula were to be successfully sealed, the pressure elevation would then become manifested. The best way to avoid missing an unsuspected intracranial problem is to make sure that all patients under consideration for extra-cranial repair of cerebrospinal fluid fistulas have been examined by a neurosurgeon.

Where does the Cerebrospinal Fluid Come From?

Numerous descriptions of cerebrospinal fluid localizing studies are available in the literature, but in practice, pinpointing the site of a leak before repair is often a challenging

exercise. We have learned to live with the following rather pragmatic approach.

First, exclude the ear. Obviously, an extracranial approach directed at the sinuses will be of no service to the patient whose source of a cerebrospinal leak is the posterior or middle fossa. Drainage reaches the ear via the temporomastoid air cells and the nose via the eustachian tube. Dye tests sometimes stain fluid that is present in the middle ear. If the eustachian transport is slow enough, a pseudo-serous otitis media can be produced, which can be suspected by the history and confirmed by tuning fork tests and otoscopy. Since otogenic serous otitis media can accompany posttraumatic or postsurgical nose and sinus alterations, the problem can sometimes be one of proving that a clear fluid observed in the middle ear is *not* cerebrospinal fluid. In serous otitis media, the eustachian tube is functionally occluded, so middle ear pressure drops. This vacuum can be measured by impedance audiometry and it produces a so-called negative tympanogram, type B. Fluid leaking into the cranium, however, would leave the middle ear pressure unchanged. A tympanogram may generate a type B waveform but at a normal atmospheric pressure.

Second, exclude the frontal sinus, since a high frontal sinus leak will be above the field of exposure in a trans-ethmoidal repair. In this case, the history of the injury and careful review of the frontal sinus imaging studies are most helpful.

Finally, consider midline sellar leaks (virtually all follow hypophysectomy), which constitute a separate category. They are accessible through the nasal septum, but by the same token many other regions of the sphenoid bone are not, so sellar leaks are a special case. Once the ear, the frontal sinus, and the sella are excluded, all we really care about is which *side* the leak is on. The most frequently used and successful approach is an extended external ethmoidectomy, and through it one can expose the cribriform plate, the ethmoidal roof, and the sphenoid sinus. The exact preoperative differentiation of which of these sites is involved is less important. The decision as to which side to explore may be made on no more than the knowledge of which side drips. The pledgets may suggest the ethmoid roof (positive test results for the middle meatus), the sphenoid sinus or the cribriform plate, but the critical data is the side. One cannot be sure by the x-ray films. We have seen instances in which the obvious traumatic bony disruption was on the side opposite the leak, and others in which the leak was bilateral, with a defect in the upper septum and cribriform plate. In the final analysis, some leaks will not be localized with complete accuracy until the time of exploration.

High Frontal Sinus Leakage

Known frontal sinus leaks are probably best explored through a coronal scalp incision and frontal sinus osteoplastic flap. This approach is certainly the most useful in identifying leaks involving the upper two thirds of the sinuses, and it lets the surgeon get at both sides at once. Posterior table bone can be removed at the site of the leak, and the dura can be repaired directly. Free fat graft frontal sinus obliteration should be undertaken only if it is clear that all of the mucosa has been removed. A pericranial-periosteal flap can be used to be certain that the obliterated sinus is sealed off from contamination from within the nasal cavity. Frontal sinus obliteration is harder to achieve lower down. In our institution, leaks of the lower third of the sinuses are usually approached in the same fashion as ethmoid bone and cribriform plate leaks; that is, through the external ethmoidectomy approach. In this way,

intranasal flaps can be used for coverage, and the concept of obliteration can be set aside.

External Ethmoidectomy and the Extracranial Repair of Cerebrospinal Fluid Leaks

Once we have shown that the central nervous system holds no surprises; that the fluid is cerebrospinal fluid; that it does not come from the ear, the sella, or the upper frontal sinus; and that a particular side is the most likely source, we usually advise extracranial repair by the transethmoidal route. The usual sequence is as follows: At a suitable time before the operation, the patient is subjected to a spinal tap, and fluorescein is instilled in the cerebrospinal fluid, as it was for the confirmation and localization study. In the operating room, general anesthesia is administered and an *external* ethmoidectomy is initiated. Since the facial and orbital bones may have been displaced by the previous trauma, and the dura and arachnoid can hang very low (almost like a meningoencephalocele) in the nasal and ethmoidal roofs, the ethmoidectomy should be carried out in a very cautious and individualized manner. The primary goal is to clean the mucosa away from the entire roof of the ethmoidal sinus and to extend the exploration into the sphenoid bone so that its roof, the sella, and the ipsilateral wall can also be visualized. It is, of course, crucial not to *create* a leak in the attempt to find one. If the bony dehiscence cannot be found, an entry should be made from the nasal cavity, immediately anterior to the middle turbinate into the ethmoidectomy cavity. The anterior end of the middle turbinate and the level of the anterior end of the cribriform plate can then be visualized. The turbinate is incised, and the bony core is dissected out of its mucosal envelope. This should be done before the turbinate is mobile. A communicating incision is then established between the cribriform plate and the ethmoidal cavity by separating the entire line of attachment of the lateral nasal wall from the cribriform plate.

These maneuvers produce a good view of the cribriform plate, and they preserve a posteriorly based middle turbinate mucosal flap that has excellent mobility and vascularity. By getting the middle turbinate out of the way so that the cribriform place can be seen, the upper septum can also be clearly visualized and used to donate a second flap. The exploration is complete when the opposite side of the sphenoid bone has been examined in a "cross court" fashion. This is accomplished by removing the sphenoidal face and rostrum and lightly displacing the posterior septum laterally. There are few instances in which this exploration does not reveal the fistula. Nonetheless, we have usually obtained permission preoperatively to explore the other side in an identical manner if the first exploration is disappointing.

Identification of the Leak

Once the exposure is complete, and atraumatically accomplished, the operator needs to keep in mind the appearance that must be identified. Although most of the operation is done with a head light, the final touches and the search are carried out with the aid of the operating microscope. A careful look for anything that pulsates is essential. Cerebrospinal fluid, or anything else communicating with the cranial cavity, such as an arachnoid protrusion, transmits pulsations of the brain. The first evidence of cerebrospinal fluid in some cases is a fine, clear streaming in the blood that accumulates in the ethmoidectomy cavity. Using a suction and close observation, the stream can be traced to its source. Sometimes, the only clue is a dark hole, which is free of blood yet distinctly darker than any communication into a sinus or air-filled cavity. Of course, if the fluorescein dye has circulated up into the cranial

cavity, any cerebrospinal fluid will have a greenish yellow fluorescence. It resembles diluted antifreeze. The intensity of the microscope light is sufficient to recognize this appearance. It should be appreciated that removing the epithelium from the cribriform plate will produce little cerebrospinal fluid leaks from several of the arachnoid sleeves accompanying the olfactory nerves. The nerves are strong and obvious, and they restrict the dissection of the mucosa from the olfactory region. Occasionally, an arachnoid diverticulum protrudes through a bony defect. The arachnoid is usually thin and pulsatile at the point at which it herniates through the dural dehiscence, and the fluorescein-stained cerebrospinal fluid can sometimes be seen within it. Arachnoid diverticuli of this type should especially be suspected in cases of recurrent meningitis in which actual dripping of cerebrospinal fluid has been very difficult to demonstrate.

Repair of the Leak

There is nothing that will reduce the chances of successful extracranial repair more than an imprecise and inconclusive exploration. However, an exploration that produces abnormal findings is also doomed to failure unless the surgeon arrives at the point of the leak with a clear plan for carrying out the repair. Stuffing in free tissue grafts is not reliable by itself, and flaps can also fail for a variety of reasons.

A helpful concept we use in creating our repair was derived from a simple consideration of the normal anatomy. This is the concept of the four-layered closure. The purpose of the first layer is to mechanically stop the flow of fluid so that the rest of the repair can proceed and the overlying layers will not be disrupted in the early postoperative period. The second layer may or may not be necessary in a given case. This layer provides the bony rigid support of the intracranial contents. If the bony defect is small, this is already sufficient. If the bony defect is large (a centimeter or more), mechanical support with a thin bony plate is usually advisable. The third layer is the healing layer, and it needs to be living tissue. The first two patches are really free grafts. Something pedicled is needed to bring in a blood supply, fibrin, and the cellular elements of wound healing. There is no good substitute for a healthy flap of pedicled intranasal mucosa. The turbinates, septum, and nasal atrium are all acceptable sources. The fourth layer is the temporary supportive dressing, the layer that keeps the first three layers in place. The dressing should be something antiseptic, soft, and atraumatically removable. A simple 1/2 inch Cortisporin-impregnated gauze strip can be layered into the nasal cavity to perform this role in most cases, and an absorbable gelatin sponge (Gelfoam) can be placed in direct contact with the flaps to aid atraumatic separation when the nasal pack is removed.

With a single, small, discrete fistula, a typical application of this four-layered strategy would go as follows: A small, free muscle plug would be inserted through the dural defect to stop the flow of cerebrospinal fluid. It is best to push the plug through the fistula so that it acts as a one-way valve and is not simply flushed away with the first cough or strain. The second layer, if necessary, would be a thin strut of ethmoidal plate or vomer from the septum. The third layer is made from unfolded middle turbinate mucoperiosteum, which is thick and of excellent vascularity. The intranasal packing, of 1/2-inch gauze impregnated with petroleum jelly, is stripped of its jelly and impregnated with Cortisporin ointment before insertion to support the flap. It is placed through the nasal cavity under direct vision via the ethmoidectomy. A small piece of compressed absorbable gelatin sponge placed directly

against the flap will help the packing separate from the flap later at the time of removal.

The Third Layer: The Intranasal Flaps

The third layer of the repair deserves further comment. Anyone who has witnessed the adhesions and synechiae that can form after major intranasal trauma - such as aggressive polypectomy, or the abrasions following inexpert packing - can appreciate the ease with which tissue from one wall of the nasal cavity can be transposed to another. The pattern of blood flow through the nasal mucosa is rich, and almost any random flap will survive. If we pedicle an intranasal flap (eg the middle turbinate flap on the sphenopalatine vessel), we improve the viability and minimize the need to cauterize to control bleeding from several arteries and veins. The reach of our flap is limited. The posteriorly pedicled middle turbinate flap, for example, reaches only as far forward on the ethmoidal roof as the anterior ethmoidal artery and has difficulty stretching to the front of the cribriform plate. The Boyden flap, which was originally described to reconstruct the nasofrontal duct in chronic sinusitis surgery, is the one we prefer to seal cerebrospinal fluid leaks anterior to these demarcations.

Both the Boyden and middle turbinate flaps are created through the exposure provided by an external ethmoidectomy. It is well to appreciate that a large area of well-vascularized nasal mucosa lies in front of the middle turbinate on the agger nasi cells and the large concave intranasal surfaces (the atrium of the nose) supported by the frontal process of the maxilla and the underside of the nasal bones themselves. A low puncture through the lacrimal fossa will enter the nose at the anterior extremity of the middle turbinate. The mucosa of the Boyden flap lies anterior to this and is thus preserved. Resection of the anterior ethmoidal air cells underlying the flap provides exposure without depriving the flap of its blood supply. The posterior edge of this flap is easily released by the entry into the nose. The tip of the flap is created by making a cut along the top of the crest for the inferior turbinate at the lower edge of the nasal atrium. The anterior margin of the flap roughly parallels the free edge of the piriform aperture of the nose. This incision rises up in front of the mucosa covering the nasal atrium and ends near the vault of the nasal dorsum up and under the nasal bone. These incisions generate a superiorly based anterior nasal mucosal flap approximately 3 cm long and 2 cm wide. The base lies above the anterior end of the middle turbinate, in front of the plane of the cribriform plate, and is well vascularized but narrow. Once the anterior ethmoidal cells are resected, and the nasofrontal region is opened, the flap can be turned upward so that its raw surface is applied to the repair site in the roof of the frontoethmoidal sinuses or in the lower portion of the posterior wall of the frontal sinus. An absorbable gelatin sponge can help in supporting the flap, and a temporary "French roll" of thin polymeric silicone (Silastic) sheeting will maintain drainage of the frontal sinus while the flap heals.

When the site of the leak is on the cribriform, ethmoidal, or sphenoidal roof, and it has been identified, plugged, and denuded of surrounding mucosa, a middle turbinate flap is created. A vertical incision in the anterior end of the middle turbinate is used to gain access to the skeletal support, liberating the turbinate bone from within. The temptation to enter the nose from the ethmoid sinus, to view the cribriform plate before making the flap, should probably be resisted. The middle turbinate flap ought to be created before the bone supporting the middle turbinate is mobilized; otherwise, separating the mucosa from the unstable bone is difficult. The bone is resected piecemeal from within the middle turbinate's mucosal envelope. If a middle turbinate flap later proves unnecessary, nothing has really been lost. In

the course of this resection, the detachment of the lateral nasal wall from the cribriform region will occur almost automatically. The posterior attachment of the flap will come to lie over the sphenopalatine foramen and it will include the posterior nasal branches of the sphenopalatine artery and veins in its base. The flap is simply unfurled so that it provides a large raw surface that can be applied to the denuded cribriform plate or to the ethmoid roof, or both.

Actually, the dependent free edge of the middle turbinate usually does not unfold completely. Intranasal examination later will show what appears to be an ethmoidectomy cavity, with a hypoplastic middle turbinate lying on its roof! The loss of a middle turbinate would, of course, constitute an important physiologic deficit and an especially undesirable one if the leak were to continue and nasal crusting were to develop to harbor pathogenic bacteria. However, since the turbinal mucosa is preserved, the consequences in terms of crusting and desiccation are generally unrecognizable. Also, since the posterior attachment of the mucosa was preserved, no scar crosses the posterior pathway of ciliary activity. Some minor scarring and synechiae form in the cribriform area, but they seem inconsequential to air flow. Olfaction, of course, is sacrificed on the side of a full-length cribriform repair, because the olfactory mucosa is destroyed.

A middle turbinate flap can be turned posteriorly into the sphenoidal sinus, if necessary. This makes it suitable for repair of some ipsilateral intrasphenoidal leaks. The familiar transseptal route has been less effective at exposing lateralized sphenoidal leaks than has the ethmoidal approach. It does, however, allow a two-layered repair in the midline sellar defect that might follow a transsphenoidal hypophysectomy. In the early postoperative period, this is usually quite satisfactory, but for the long-standing, persistent sphenoidal leak, the added exposure (particularly of the off-midline portions of the sphenoid sinus) offered by the transethmoidal route, and the advantage of allowing the middle turbinate flap to be developed, favor this approach over the transseptal one.

Healing of the donor site after the development of the two lateral nasal wall flaps deserves mention. In the case of the middle turbinate flap, healing of the donor site is hardly a real issue, since the entire structure is transposed, the bone was removed, and the nasal and ethmoidal cavities simply communicate. With the Boyden flap, a large area of the lateral wall of the anterior nasal cavity is exposed. Fortunately, secondary reepithelialization tends to proceed without significant intranasal obstruction or deformity in this area. Nasal saline irrigation can reduce crusting during this period, and the frontal sinus polymeric silicone roll drain may help to avoid adhesions across the nasofrontal duct.

Sometimes the middle turbinate is not available (eg a leak can be produced by aggressive intranasal polyp surgery, which may have destroyed the turbinate). In this case, an upper septal mucosal flap may be used - one that extends from a base near the sphenoidal rostrum to a tip on the anterior end of the ethmoidal plate. Transethmoidal exposure of the upper nasal cavity, once the middle turbinate is gone or once its remnants have been mobilized and displaced inferiorly, is excellent. A posteriorly based flap can be cut from the septum with a Freer knife and readily transposed to the cribriform plate. Like the middle turbinate flap, the reach anteriorly is limited. However, the septal flap does provide well-vascularized mucoperiosteal mucosa and is a valuable third layer in cases in which the middle turbinate flap cannot be used.

When both middle turbinates *and* the upper septum are gone, frontal pericranium should be considered. This tissue can be mobilized through a midline forehead incision extended from or discontinuous with the medial canthus incision. The pericranium is pedicled, inferiorly on the glabella and is transposed into the nasal cavity through a window created by partial resection of the nasal bone on the side of the leak. I have used this technique successfully in a single case. Both turbinates had been removed for severe nasal polyposis; then, the surgeon had inadvertently removed the upper septum and a fragment of cribriform plate in the course of performing an associated septal operation. Although it is hard to know on the strength of one case whether the flap of frontal pericranium was responsible for the seal, it is at least some indication that even after severe disruption of the nose, a layered extracranial repair can be successful in avoiding a frontal craniotomy.

Specific Leaks

Acute Posttraumatic Cerebrospinal Fluid Rhinorrhea

The initial treatment of acute traumatic cerebrospinal fluid leaks is to treat the associated facial fractures; 85 per cent of leaks will stop in the first week after anatomic reduction and stabilization. A formal extracranial repair is probably unwarranted until sufficient time has passed for the leak to prove it will not seal spontaneously and for the bones from which one wishes to elevate mucosa to become stabilized. In cases of brain damage, it is probably better to operate, with the presumption of recovery, than to allow prolonged cerebrospinal fluid leakage (and the attendant danger of meningitis) just because the patient is comatose and the prognosis for mental recovery is guarded.

Intraoperative Accidents

Inadvertent intraoperative leaks during rhinologic surgery should be recognized and dealt with at the time of the surgery. Wishful thinking is only an invitation to trouble. If only the arachnoid sheaths of the olfactory nerves are transected, it is true that nasal packing alone will probably suffice. The break in the dura is minuscule after all. Defects greater than this should be carefully illuminated and studied, and a local immediate repair should be carried out, bearing in mind the layered concept. A small fragment of locally harvested homograft muscle should be passed up through the perforation until it appears sealed. Structural support for the muscle plug should be provided with local septal bone or cartilaginous tissues, if necessary. Usually the defect is small enough and the access poor enough that transposition of an intranasal flap is questionable. Fortunately, it is usually unnecessary. Individual circumstances will dictate. It is vitally important to realize that an inadvertent intraoperative leak attending sinus or nasal surgery should not be simply regretted and ignored. To do so is to invite early postoperative tension pneumocephalus, which cannot be ignored, or to face months of uncertainty about the meaning of postnasal drainage or headaches.

Cerebrospinal Fluid Rhinorrhea after Transsphenoidal Hypophysectomy

Delayed post-traumatic cerebrospinal fluid leakage, appearing after transsphenoidal surgery or cranial base operations in which the surgeon has already attempted a primary repair, is a different specific entity than the accidental leak. Some seal has already been used. If a leak appears after a transsphenoidal hypophysectomy, we have found it valuable to go

back through the sublabial transseptal route immediately. In the first 2 weeks, it is relatively easy to separate the nasal flaps via the sublabial route, and the duplication of the upward slanting midline sellar exposure is very likely to reveal the source. An autogenous muscle graft, supported by a septal bony strut across the opening in the face of the sella, is usually successful in stopping this complication. Free *intrasphenoidal* grafts are not used. They will only form an intrasinus foreign body, which is later the source of infection. We do not rely on spinal taps to stop these leaks because by the time it is evident they have failed, a good deal of time and money will have been expended, and the septal flaps will be adherent to each other. The rapidity of production of cerebrospinal fluid is such that spinal taps would have to be very frequent to prevent outward flow, and over-withdrawal of fluid would be expected to reverse the flow gradient and encourage intranasal to intracranial contamination.

Postoperative Cerebrospinal Fluid Rhinorrhea after Major Skull Base Resections

With respect to resection of the cranial base - for ethmoidal cancer, for example - or to any situation in which a large, deliberate surgical meningeal defect will be created, the principal goal is to avoid a delayed leak by correcting it properly at the time of the resection. This comes down to careful planning of the approach incisions so that they do not overlay the dural defect, as well as releasing all tension on the skin and soft tissue closure, using homograft dura to form the initial sealing layer, performing a watertight dural layer closure, and avoiding dead space. Split calvarial bone grafts can be helpful in achieving a supporting layer. Bony stabilization has sometimes been assisted with cyanoacrylate glue or a cranioplastic acrylic. The critical living-tissue third layer in the anterior cranial fossa is usually created with a pericranial or galeal flap. When the orbit is resected, the temporalis muscle can also be rotated into the anterior cranial floor defect. Drains should not be placed near these closure lines. For middle and posterior fossa procedures, such as glomus jugulare resections, middle ear obliteration is probably advisable. Temporalis flaps work well in big defects, but it ought to be borne in mind that the cavities into which they must reach are often quite deep. Releasing incisions to minimize dead space and tension need to be planned with great care so as not to devascularize the flaps or the overlying soft tissue closure.

Conclusions

With a carefully planned approach, the rhinologic surgeon, working closely with his colleagues in radiology and neurosurgery, can expect to identify, localize, and successfully repair most cases in which cerebrospinal fluid rhinorrhea occurs. There are no shortcuts, however, and every case is an individual challenge in planning and execution. Fluorescein dye techniques and the concept of a four-layered repair have proved to be quite helpful. With the availability of the extracranial approaches to cerebrospinal fluid rhinorrhea, a frontal craniotomy in these cases should be quite exceptional.