Chapter 130 – Microvascular Decompression of the Seventh Cranial Nerve

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Hemifacial spasm (HFS) is a relatively uncommon affliction with an estimated yearly incidence of approximately 1 per 10,000.[1] The disorder is characterized by involuntary unilateral contractions and twitching of the eye and face. The spasms are painless, yet they often cause significant social distress. In addition, many patients with HFS have difficulty reading, driving, or working. Medications generally offer no relief of symptoms. Botulinus toxin (Botox, Allergan, Inc.) injections have become increasingly popular as a temporizing measure for HFS, but do not have long-lasting efficacy. As a result of the lack of response of HFS to these conservative therapies, surgical interventions for HFS have emerged over the past half century.

The hypothesis that vascular compression of the seventh cranial nerve (CN VII) causes HFS opened the door for these surgical therapies. The theory was first proposed by Campbell and Keedy in 1947 as well as Laine and Nayrac in 1948.[2,3] Their proposal was based on Dandy's hypothesis that vascular compression of the dorsal root of the trigeminal nerve causes trigeminal neuralgia.[4] Subsequently, Gardner and Sava published their initial experience with decompression of the seventh nerve for HFS in 1962.[5,6] However, despite these early reports, decades would pass before Jannetta popularized microvascular decompression (MVD) of CN VII as the definitive treatment for hemifacial spasm.[7,8] Today, microvascular decompression of the seventh nerve stands alone as the single most effective and long-lasting treatment for HFS.

PATIENT SELECTION

HFS is a purely clinical diagnosis and therefore a careful history and physical examination must be obtained before consideration of craniotomy and MVD. Typically, HFS manifests as unilateral intermittent twitching of the facial muscles, usually beginning with orbicularis oculi. Subsequently, spasms spread contiguously to the other facial muscles. In atypical HFS, twitching begins in the lower facial muscles and subsequently spreads upward. The disease generally takes a relentless course and over a variable period of time progresses to all of the major muscles of one side of the face, including the frontalis, platysma, and stapedius muscles. The latter creates an odd clicking sound in the ipsilateral ear. Severe spasms may lead to tonic facial contractures (i.e., “tonus” phenomenon), which can be associated with facial muscle paresis. The cosmetic deformity of HFS is the most obvious concern for most patients, prohibiting normal social interactions. Moreover, HFS usually becomes a functional disability. Repetitive closure of the eyelids, especially when associated with tonus phenomenon, can impair vision and prohibit the pursuit of both occupational and leisure activities, including driving and reading.

HFS is slightly more common in women, and the left side of the face is more often affected than the right.[8] Symptoms may be exacerbated by fatigue and stressful activities. Although the spasms are involuntary, voluntary movements such as talking and smiling can trigger contractions. Spasms continue during sleep, and no known medical conditions are associated with increased incidence of HFS. There is no apparent genetic transmission of HFS, although rare familial cases have been reported. No known toxin exposures appear to increase the incidence of this condition.

The differential diagnosis of hemifacial spasm includes postparalytic synkinesis, which occurs after facial nerve trauma or Bell's palsy. This phenomenon is usually brought on by facial movement and rarely occurs at rest. Other conditions that can be confused with hemifacial spasm include blepharospasm, tics or habit spasms, facial myokymia, Meige syndrome (combination of oromandibular dystonia and blepharospasm), and focal cortical seizures. Blepharospasm and myokymia typically occur bilaterally, whereas bilateral HFS is exceedingly rare. The distinguishing features of HFS and similar conditions are outlined in Table 130-1.

Table 130-1 -- DISTINGUISHING FEATURES OF HEMIFACIAL SPASM AND SIMILAR CONDITIONS

Condition	Spasm Location	Spasm Pattern	Spasm Laterality	Pain
Hemifacial spasm	Orbicularis, with spread to other facial muscles	Arrhythmic and intermittent, may persist during sleep	Unilateral	Usually none

Condition	Spasm Location	Spasm Pattern	Spasm Laterality	Pain
Blepharospasm	Orbicularis, may spread to the rest of the face	Symmetrical and forceful	Bilateral	Usually none, but may have photophobia and/or ocular dysesthesias
Post-paralytic synkinesis	Anywhere in facial nerve distribution	Associated with movement	Usually unilateral	Varies
Facial myokymia	All facial muscles	Movements exacerbated by fatigue; fine and continuous	Varies	Usually none
Tics and habit spasms	Usually outside the facial nerve distribution	Stereotyped and simple	Often bilateral	Usually none
Focal seizure activity	Anywhere in facial nerve distribution	Clonic, gross movements	Unilateral unless seizure generalizes	None

PREOPERATIVE EVALUATION

Although preoperative imaging cannot make or support the diagnosis of HFS, mass lesions such as neoplasms of the cerebellopontine angle, posterior circulation aneurysms, or arteriovenous malformations may be associated with HFS in 1% to 2% of cases. Therefore an essential part of the patient's preoperative evaluation is magnetic resonance imaging (MRI) scan with and without contrast to rule out the existence of such a mass lesion. If a mass lesion is discovered, both the mass lesion and the vascular compression source must be treated in order to achieve a cure. However, MRI and computed tomography (CT) scans should not be relied upon to identify vessels responsible for compression. A vessel (arterial or venous) of any size can be responsible for the compression syndrome. If the MRI scan does demonstrate a large vessel near the CN VII root exit zone, the finding does not necessarily mean that this vessel is the primary source of compression or the only source of the compression. Thus, the role of preoperative imaging is to rule out other sources of vascular compression rather than guide the operative approach in most cases.

Once a patient is diagnosed with HFS and obtains adequate imaging, it is reasonable to refer the patient to a neurologist or neurophysiologist for electroneurophysiologic testing. The routine battery of tests that are performed includes baseline brain stem auditory evoked responses (BAERs), facial electromyography (EMG), and baseline lateral spread (LS) testing (see later text). Abnormalities are noted before any operations. Such testing is most critical in patients who have received Botox injections before seeking surgical treatment, because their baseline responses are often abnormal.

Patients with HFS have a characteristic electrophysiologic anomaly termed the *lateral spread response*. An understanding of the importance of this abnormal muscle response is critical, both to the diagnosis of HFS as well as the operation to cure it.[9] The LS can be evoked via percutaneous stimulation of one branch of the facial nerve while recording stimulus-evoked EMG from the various muscles of the face innervated by the facial nerve. In patients with HFS, when one branch of the facial nerve is electrically stimulated (e.g., zygomatic branch), not only do the muscles innervated by that branch (e.g., orbicularis oculi) display an evoked EMG response, but other facial muscles (e.g., mentalis) not innervated by that branch also demonstrate evoked EMG activity. This paradoxical EMG response can be evoked in the operating room and consists of a triphasic EMG potential followed by a series of after-discharge potentials most typically recorded at a latency of 10 to 12 milliseconds. This response is obliterated following an adequate decompression of the seventh nerve (Fig. 130-1).

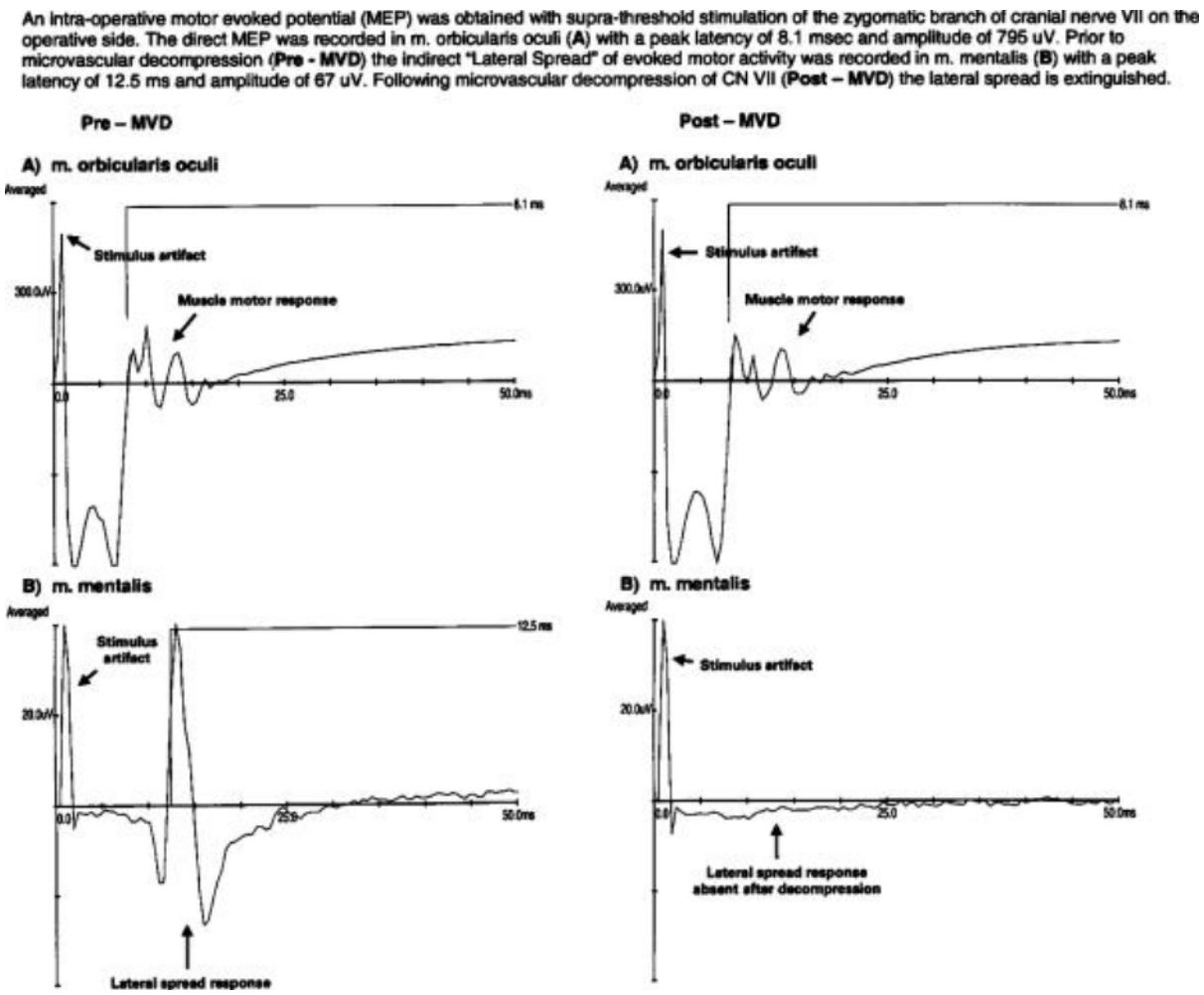

Figure 130-1 Demonstration of the "lateral spread" phenomenon as visualized intraoperatively during an MVD for HFS.

Most patients with hemifacial spasm are between the fourth and sixth decades of life, and are reasonable candidates for general anesthesia. Nonetheless, all of these patients require a formal perioperative risk assessment before any operation. Preoperative laboratory evaluations including electrolytes, complete blood counts, and coagulation profiles should be assessed. Electrocardiography and chest roentgenography help rule out occult cardiovascular disease. It is not necessary to keep blood products on hold in most cases. Any patient with a significant cardiac or other medical risk should have it addressed before craniotomy for MVD.

SURGICAL APPROACH

Patient Positioning and Preparation

The retrosigmoid or retromastoid approach is the standard surgical approach for MVD of the seventh nerve. Once anesthetized, the patient is placed in three-point head fixation with pins in the ipsilateral frontotemporal region and the contralateral mastoid. This approach maximizes access to the mastoid region on the treated side. When placing the pin fixation, it is important to keep the operative side pins anterior to a line running superiorly from the earlobe. If the pin is placed too far posteriorly, it will be difficult to insert the self-retaining retractor, because the handles will contact the head holder. In addition, the squamosal portion of the temporal bone is to be avoided with the fixation pins, because this area of the skull is particularly thin and pinning in this area can lead to an epidural hematoma. Sixty pounds of pressure is usually adequate for rigid head fixation.

After pinning, the patient is rotated with the treated side up into the lateral decubitus position. All pressure points are padded, and an axillary roll is placed. The patient is taped to the table securely at the hip and underneath the axilla. In addition, the ipsilateral shoulder is pulled caudally with care not to stretch the brachial plexus. The neck is gently flexed and elevated from the lateral position such that two fingerbreadths are maintained between the chin and the sternum. This maneuver is designed to minimize the risk of jugular venous thrombosis. Proper patient positioning is demonstrated in Figure 130-2.

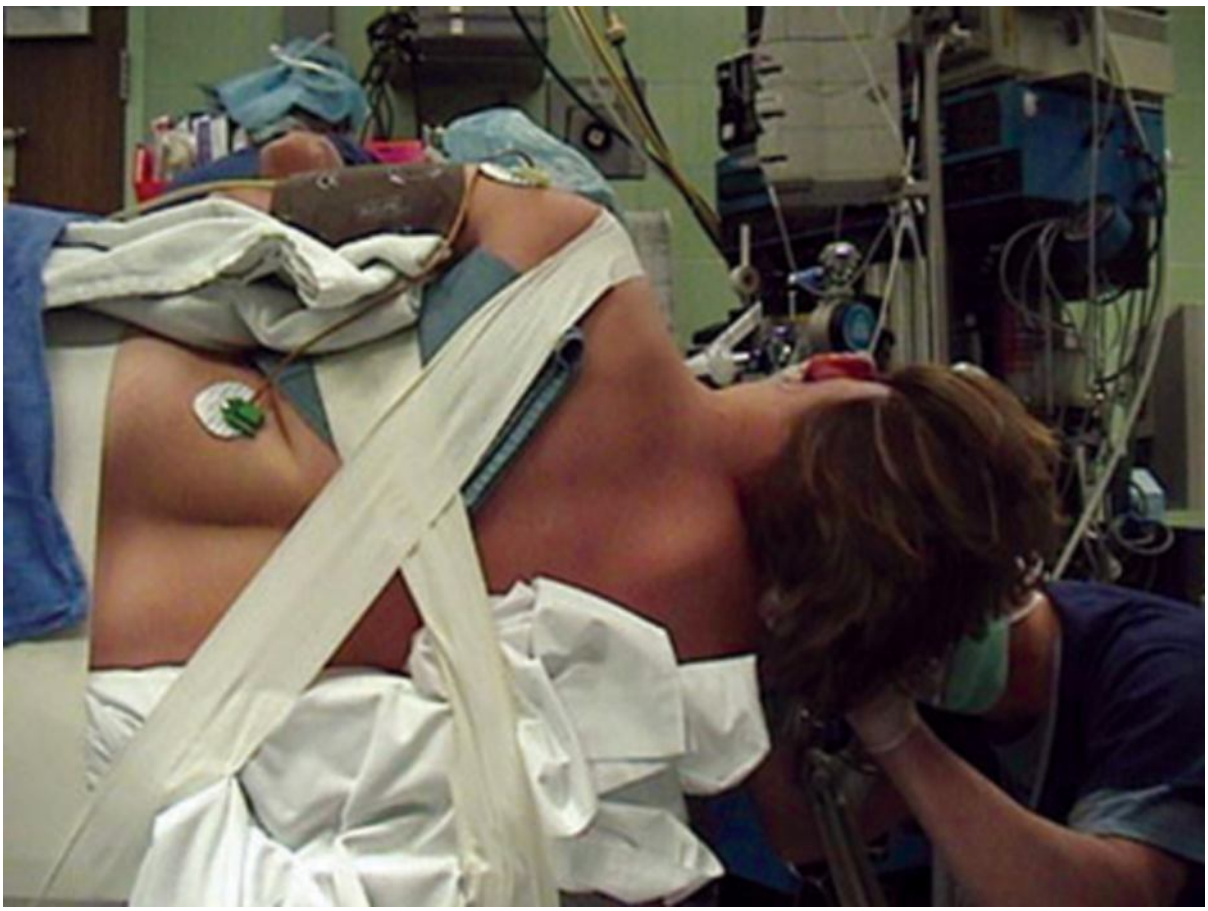

Figure 130-2 Proper lateral decubitus positioning for a seventh nerve decompression. This photograph and all subsequent operative photographs depict a left-sided operation.

A Doppler monitor should be secured to the anterior chest wall in order to detect the occurrence of air embolism during venous sinus exposure. While lumbar cerebrospinal fluid (CSF) drainage is not routinely used, we do consider the use of CSF drainage if the patient is especially young (in which case the posterior fossa is fuller) or if the operation is a revision procedure (in which case the cerebellum may be adherent to the overlying dura). Furthermore, a lumbar drain is of additional value if the surgeon is less experienced at the procedure: lumbar drainage creates early cerebellar relaxation and minimizes the CSF pulsations that can make microsurgical dissection in this region more difficult.

Intraoperative Monitoring

Intraoperative BAERs are monitored, as are stimulus-evoked EMG responses from the orbicularis and mentalis muscle groups in response to stimulation of the zygomatic branch of the facial nerve. In addition, spontaneous EMG from cranial nerves IX and X is recorded to detect any injury potentials. The anesthesiologist should use only short-acting muscle relaxation for induction, thereby allowing for EMG monitoring and assessment of the presence of a lateral spread response before incision. The surgeon must ensure that an adequate recording of the lateral spread response is present before skin incision.

Surgical Exposure

A 3- × 5-cm area behind the ear is shaved. The mastoid eminence, digastric groove, and inion should be identified externally. The line between the inion and the external auditory meatus approximates the position of the transverse sinus. The digastric groove overlies the sigmoid sinus, and the junction of these two lines defines the transverse-sigmoid junction. A vertical incision is drawn 3 to 5 cm long, approximately 0.5 cm medial to the shaved hairline and parallel to it. A standard preoperative sterile skin preparation is applied. During draping of the operative field, the patient is administered 12.5 to 25 g of intravenous mannitol to facilitate cerebellar relaxation. Skin is opened with a scalpel. Deeper dissection uses monopolar electrocautery and bipolar sacrifice of the occipital artery. The initial periosteal dissection should always proceed medially before completing the lateral dissection, because the lateral periosteum is more loosely attached to the skull than is the medial periosteum, and the muscle thickness is greater medially than laterally. If the surgeon performs the muscle and periosteal dissection laterally (toward the ear) first, there is usually not enough tissue resistance to allow the Weitlaner retractor to force the medial tissue out of the

surgical field. This situation makes visualization of the intracranial contents more difficult. If one should make the mistake of dissecting laterally first, the problem can be overcome in two ways: (1) by extending the incision superiorly and inferiorly to allow for more tissue mobilization or (2) by substituting skin hook and spring retractors for the Weitlaner self-retaining retractor.

Once the muscle and periosteum are mobilized and retracted, the underlying bony anatomy should be clearly identified. The mastoid emissary vein, which often marks the transverse-sigmoid sinus junction, will almost always be encountered and will need to be filled with wax for adequate hemostasis. The digastric groove must also be visualized; drilling along its medial border creates the dural exposure necessary for an adequate brain stem view.

Following periosteal dissection, the overlying bone may be removed via a craniectomy or craniotomy. Even though bone removal can be minimized to only a few centimeters, the authors prefer bone removal sufficient to expose the inferior edge of the transverse-sigmoid junction and the medial edge of the sigmoid sinus down to the level of the lower digastric groove. This 4- × 3-cm ovoid opening provides adequate inferolateral exposure to access the lateral cerebellomedullary cistern without significant cerebellar retraction. It is necessary to remove a portion of the posterior mastoid air cells in order to expose the edge of the sigmoid sinus. All the air cells are thoroughly waxed in order to avoid a postoperative CSF leak.[10]

Before dural opening, the Greenberg brain retractor system is attached to the three-point head holder in case fixed spatulas are required to control unforeseen intracranial bleeding. The system is not used in the vast majority of cases. The dura mater is then opened in a curvilinear fashion and reflected laterally with 4-0 monofilament suture. The dural reflection must lie immediately parallel to the descending sigmoid sinus so as to avoid a shelf of dura reducing subsequent lateral visualization. The medial dural cuff is maintained to allow for a watertight closure. A 3- × 0.5-cm rubber dam cottonoid sponge is placed along the lateral aspect of the cerebellar lobe to begin navigating around the cerebellum (Fig. 130-3).

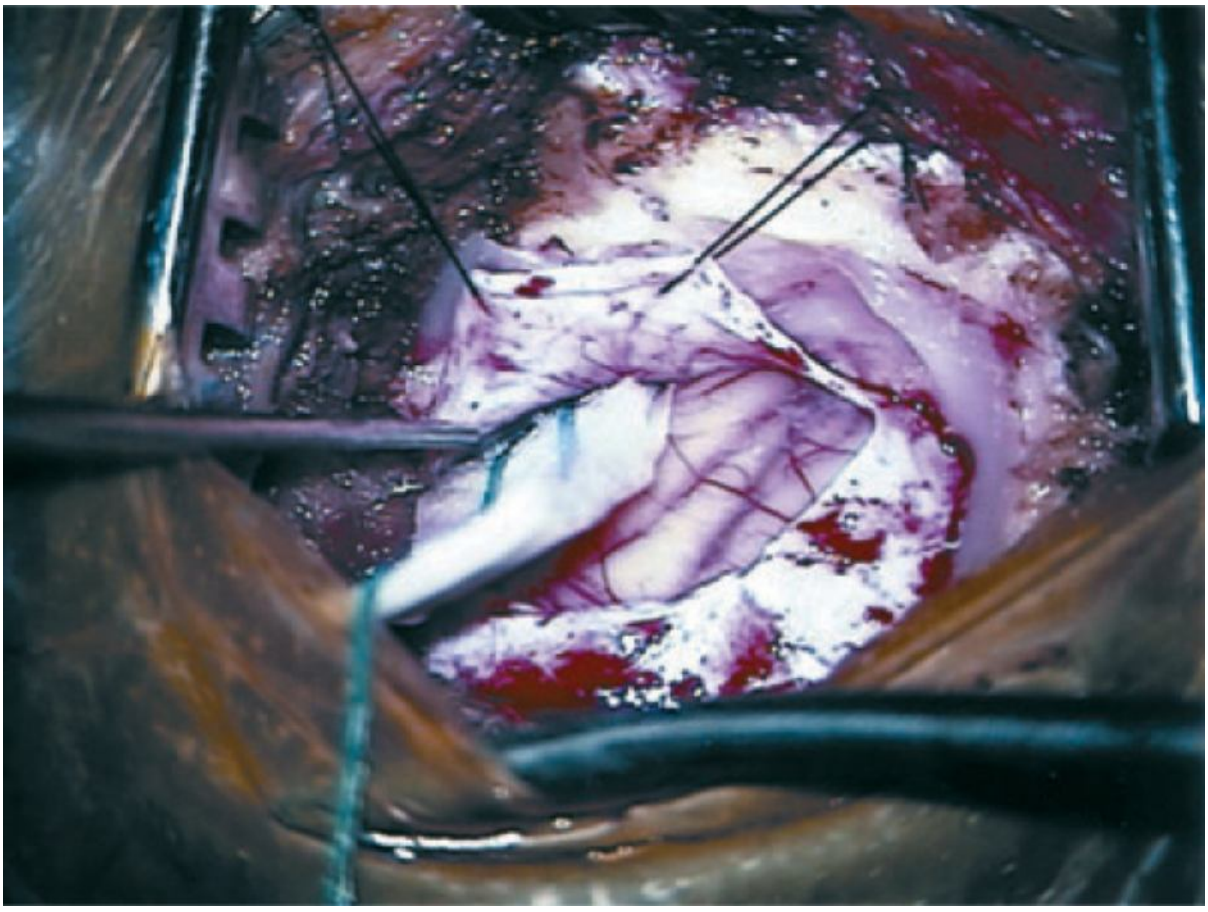

Figure 130-3 Rubber dam cottonoid placed on the lateral cerebellum in preparation for navigation around the cerebellar hemisphere.

Nerve Exposure

At this point, the operating microscope with a 325-mm objective lens is brought into the field. Dissection proceeds laterally around the edge of the cerebellum toward the lateral cerebellomedullary cistern, which contains the 11th cranial nerve. The brain is gently retracted with a cottonoid sponge. All cottonoids are moistened with saline and

placed over an appropriately sized (0.5 × 3.0 cm) piece of latex (rubber dam) cut from a sterile surgical glove. The latex prevents trauma to the cerebellum and allows for easier advancement of the cottonoids. A rubber dam cottonoid and Teflon pledgets are pictured in Figure 130-4. Whereas we favor not using a retractor during the case, but rather retract-ing with a controlled sucker (no. 3-4F), a 60-degree tapered retractor can be placed over the rubber dam cottonoid against the inferolateral aspect of the cerebellum. Following gentle navigation around the inferolateral cerebellar edge, the petrous dura overlying the 11th cranial nerve is visualized. Safe exposure of this landmark requires a dynamic dissection process with intermittent adjustments of both the patient's position and the operating microscope. Dissection around the lateral border of the cerebellum without adequate adjustment of the patient's position and/or the microscope's position may lead to cerebellar injury. This injury can be profound enough to cause lateral cerebellar infarction and brain herniation.

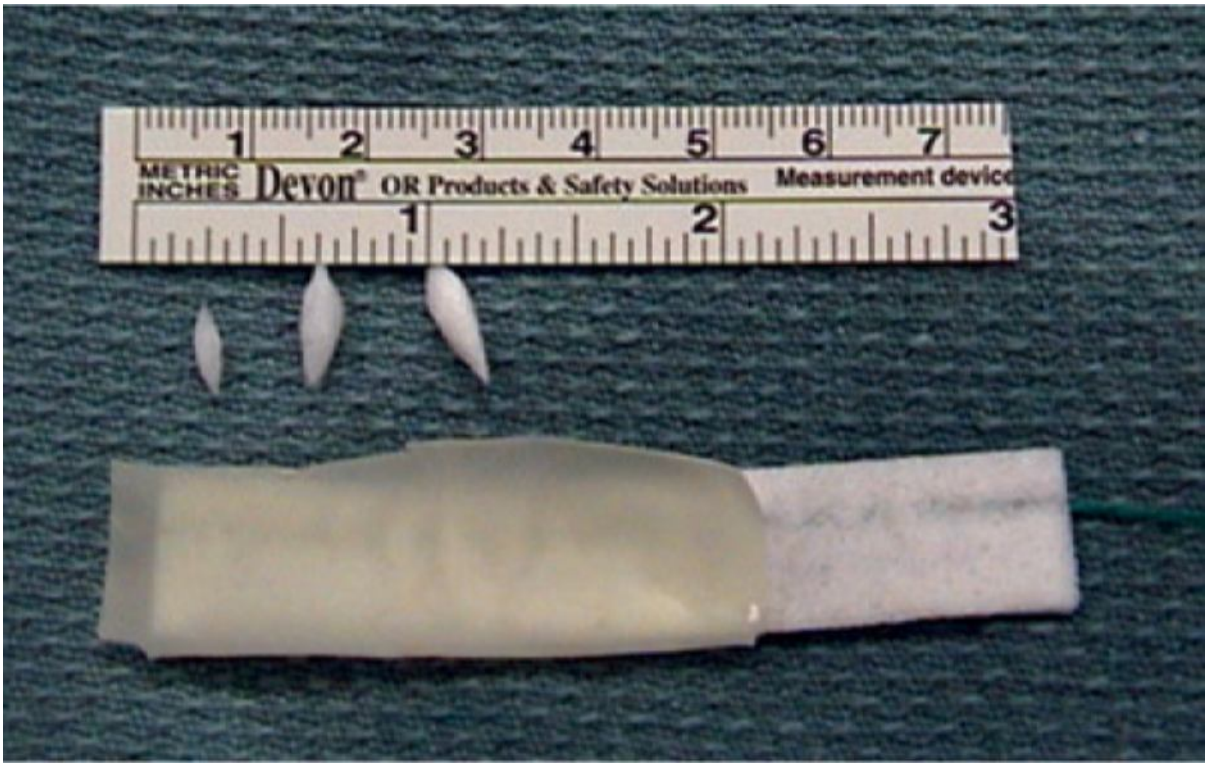

Figure 130-4 Patties and pledgets for microvascular decompression. Teflon pledgets are visualized superiorly, and a rubber dam cottonoid inferiorly.

Beneath the petrous dura, the 11th nerve is visualized. If necessary, a portion of the overlying bone can be drilled off to facilitate cisternal visualization. The arachnoid between the nerve and the cerebellar flocculus is opened with an arachnoid knife. At this point the operator holds still, patiently allowing CSF egress and cerebellar relaxation. The working zone of the posterior fossa is markedly increased by this maneuver. In addition, this maneuver allows for initial inspection of the lower cranial nerves (CN IX through XI). An overly superior approach to this cistern exposes the seventh and eighth nerves prematurely without adequate CSF drainage. An overly inferior approach to this cistern leads to the cerebellar tonsils near their termination at the foramen magnum.

Once CSF is drained and the cerebellum relaxes, the arachnoid over the lower cranial nerves is opened sharply with an arachnoid knife and microscissors (Fig. 130-5). The flocculus of the cerebellum and the choroid plexus protruding through the foramen of Luschka are exposed and gently retracted. Retraction at this point may result in changes in the BAER. If significant BAER changes occur (i.e., greater than 50% amplitude reduction or greater than 1 millisecond latency), retraction should be relaxed and the surgeon should pause until all responses return to their baseline configuration. Additional arachnoid around CN VIII may need to be cut to reduce transmitted retraction forces from the cerebellum as it is mobilized medially. It is important to watch the retractor (if used) or cottonoid frequently throughout the operative period because it is common for one of them to move against the lower cranial nerves and cause potential irreversible damage. Thorough, sharp arachnoidal dissection helps prevent cranial nerve injury during this portion of the procedure. In particular, removal of arachnoidal adhesions around the eighth nerve minimizes retraction injury to the cochlear nerve and cochlear nucleus. Any injury potentials are immediately reported to the surgeon during this critical portion of the dissection.

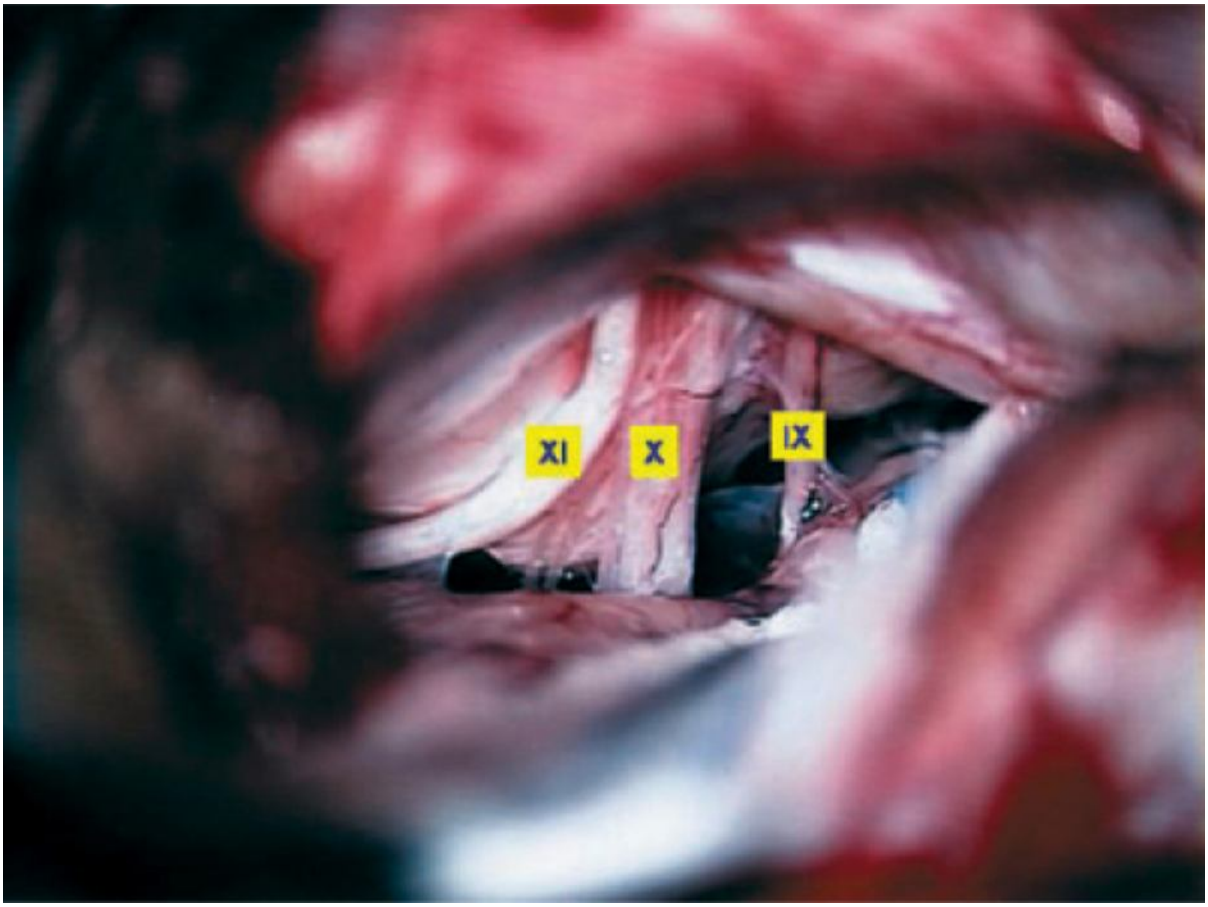

Figure 130-5 Visualization of the ninth (IX), tenth (X), and eleventh (XI) cranial nerves following arachnoidal dissection.

The facial nerve must be exposed along its entire course, especially at the brain stem root exit zone, the most common site of vascular compression. To visualize the root exit zone, it is important to completely dissect the flocculus and choroid plexus from the base of cranial nerves VIII, IX, and X. Looking toward the brain stem (achieved by angling the microscope inward and rotating the patient toward the surgeon), the surgeon should focus just medial to the ninth and tenth nerves. It is from this area that the facial nerve arises as it runs rostrally to follow the course of CN VIII, deep to it (Fig. 130-6). It is often necessary to work between CN IX and X and between CN VIII and IX to get the best view of the brain stem origin of the seventh nerve.

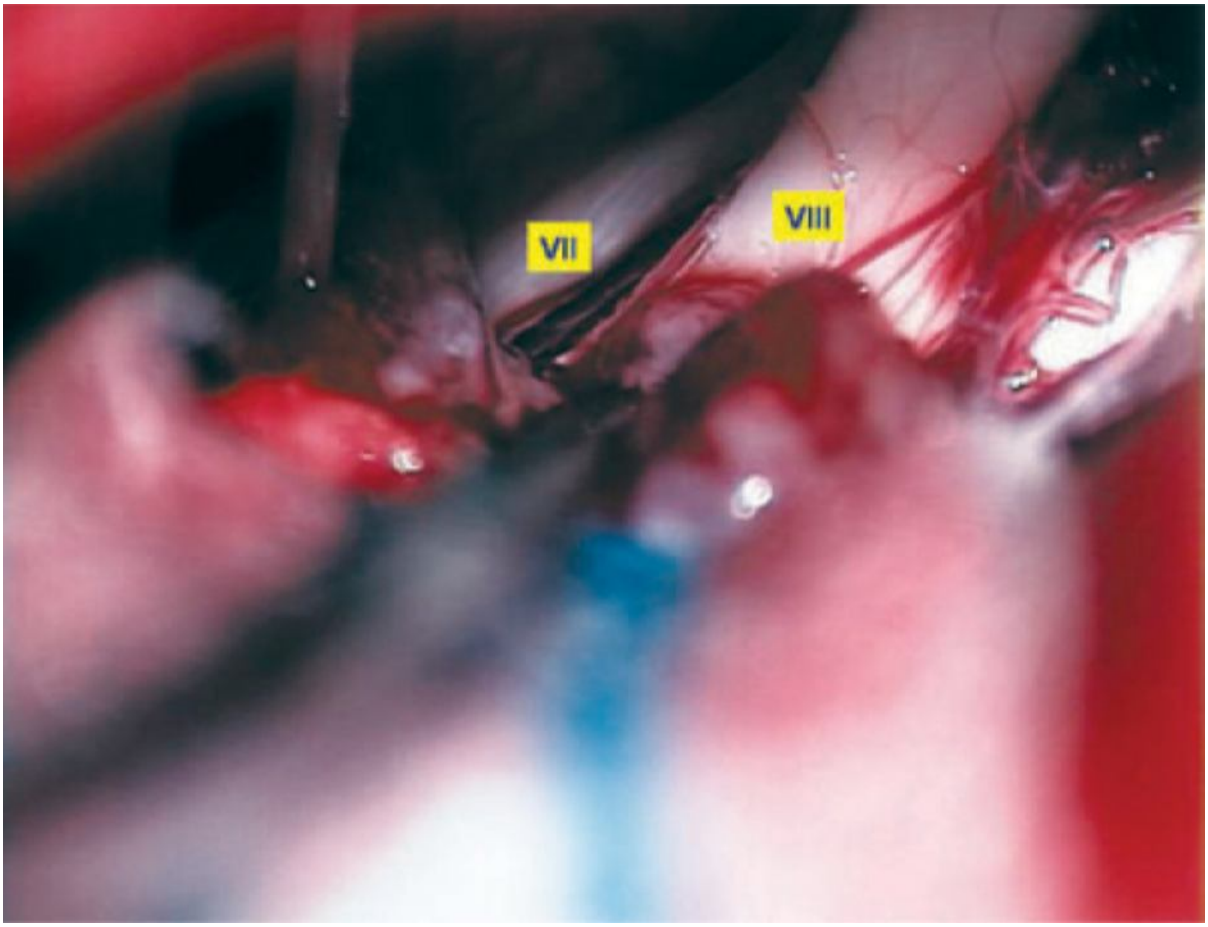

Figure 130-6 Visualization of the seventh nerve (VII) exiting the brain stem anterior and deep to the superficial eighth nerve (VIII). A small artery is compressing the nerve at its root exit zone.

Microvascular Decompression

The most common vessels compressing the facial nerve are the posterior inferior cerebellar artery (PICA) and anterior inferior cerebellar artery (AICA).[8,11,12] Because of the proximity of the PICA origin from the vertebral artery (VA) to the compression site, the authors often first decompress the VA from the brain stem before decompression of PICA from the seventh nerve root exit zone. The VA is reliably located just medial to CN XI. The VA is elevated from the brain stem and large pieces of Teflon felt are placed between the vessel and the medulla so as to move the entire vascular complex laterally. This maneuver shifts the PICA and other VA perforators away from the brain stem as well as the CN VII exit zone. Early mobilization shifts PICA distally, reducing the need for neural tissue retraction during subsequent decompression. Once the VA is mobilized laterally, attention is turned to the CN VII root exit zone. Any arteries compressing this site are mobilized away from the brain stem and decompressed using Teflon pledgets (Fig. 130-7). At this point, the lateral spreads often begin to change morphology and amplitude or may even suddenly disappear. If they do not fully disappear, additional arterial sources of compression are sought. Some may be quite small and may be considered inconsequential to the spasm; however, by decompressing them the lateral spreads often completely resolve. If the spreads remain, a search for veins contacting the CN VII exit zone is carried out. Often these veins can also be decompressed from the brain stem using sharp dissection and small pieces of Teflon. In other instances they need to be divided using a microknife, scissors, or hook. At no time should bipolar cautery be used in the region of CN VII or the brain stem; even small energy surges can lead to permanent CN VII and VIII dysfunction. Hemostasis from divided veins is readily achieved using gentle Teflon tamponade. When veins are found and eliminated, it often takes several minutes for the lateral spreads to completely disappear or fall to negligible levels. The reason for this delayed change is not clear; however, it is a reliable enough finding that the authors will wait several minutes for the lateral spreads to abate before searching for additional pathology. Such patience eliminates unnecessary dissection, which puts the cranial nerves and the brain stem at risk for iatrogenic injury.

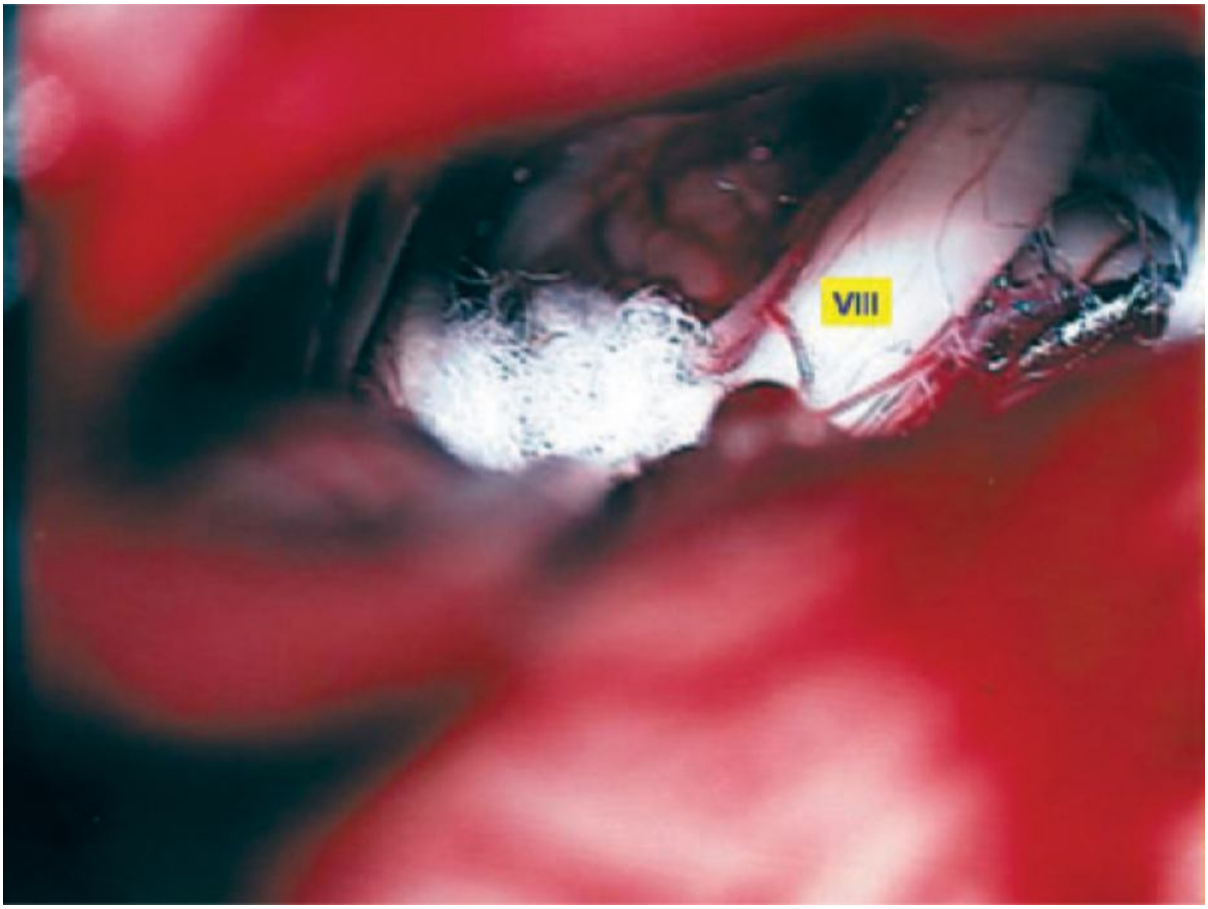

Figure 130-7 Adequate decompression of the seventh nerve with Teflon pledgets.

Throughout the operation, close communication is maintained with the neurophysiology team. Any change in the BAER morphology mandates immediate action, because the eighth cranial nerve is especially sensitive to manipulation. If the amplitude drops by more than 50% or if the latency is increased by more than 1 millisecond, surgical manipulation ceases, and the BAER should be allowed to return to baseline. In less favorable circumstances, the surgeon should always wait until the latency drops below a 0.5-millisecond delay and the wave amplitude increases to at least 50% of baseline before resuming manipulation. Occasionally, the BAER will change after placement of a Teflon felt, necessitating removal. Sudden loss of BAER usually denotes a vascular injury to CN VIII either from vasospasm or vessel loss. Small perforator injury can lead to such changes, especially branches of AICA or the labyrinthine artery. Inadvertent spread of bipolar current can also lead to irreversible injury. The authors have frequently witnessed improvement of BAER recordings following irrigation of the eighth nerve with papaverine, implying that improvement of local microcirculation can improve nerve function.

The neurophysiology team is essential for successful performance of a microvascular decompression of the seventh nerve. Accurate intraoperative monitoring prevents iatrogenic injury. In addition, tracking the lateral spread of impulses along the seventh nerve can lead the surgeon to the critical pathology, and lateral spread obliteration increases the surgeon's confidence that the patient will be cured. In certain cases, decompression of large vessels that appear to be clear culprits may in fact have no effect on lateral spreads. Furthermore, final decompression of small venules or arterioles can lead to ultimate resolution of abnormal electrical crosstalk and irritation. Therefore, neurophysiology input is critical to both novice and experienced microsurgeons.

Closure

At the conclusion of the decompression procedure, several Valsalva maneuvers to 40 mm Hg for 10 seconds are performed to ensure hemostasis. It is important to be sure the patient is pharmacologically paralyzed before Valsalva maneuvers are requested. The region is gently irrigated with warm saline bulb irrigation, and the dura is closed. Dural closure must be watertight. If the dura cannot be easily reapproximated, a small piece of muscle is sewn over the opening to seal off the leak. Larger defects are closed with a dural graft. Valsalva maneuvers are again performed to ensure the adequacy of the dural closure. If there is an inadvertent injury to a dural venous sinus during closure, then the area of the injury should be packed with hemostatic agents, such as Avitene, and allowed to stop spontaneously. Under no circumstances should such an injury be directly repaired with suture or vascular clips, which may worsen such an injury.

After dural closure, the bone edges of the mastoid air cells are thoroughly waxed once again. A pad of Gelfoam is placed over the dura to fill in any dead space. Sometimes, fibrin sealant glue can be applied to the dura as an additional barrier to CSF leakage. A wire mesh with titanium screws (KLS Martin, Jacksonville, FL) is fastened to the bony edges to reapproximate the shape of the skull. Care must be taken to avoid plunging into the subdural space with a slipped screw while attaching the cranioplasty plate. The authors have found that this technique prevents the adhesion of nuchal muscles to the dura, which may cause chronic postoperative headaches. The muscles are approximated with interrupted 2-0 absorbable sutures, and the fascia is closed in a similar fashion. The subcutaneous tissue and skin are closed with 3-0 absorbable sutures and a running 4-0 nylon suture. A sterile bandage is affixed with paper tape.

POSTOPERATIVE MANAGEMENT

As with any other patient status postcraniotomy with intradural manipulation, postoperative cardiac telemetry and respiratory monitoring is essential. Following a postoperative neurologic examination and a few hours of surveillance in the postanesthesia care unit, patients are transferred to the neurologic stepdown unit for overnight observation. Postoperative hypertension is to be avoided, because it entails an increased risk of late hemorrhage. The authors use intravenous short-acting antihypertensive agents (e.g., labetalol or hydralazine) to prevent elevation of the systolic blood pressure above 160 mm Hg for 24 hours. Postoperative nausea is treated with intravenous ondansetron, and other agents are added if necessary. Any patient with a severe headache undergoes an immediate non–contrast-enhanced head CT scan to rule out any postoperative hematoma. Patients with persistent headache not alleviated by medication and a negative scan undergo high-volume lumbar puncture. Approximately 15% to 20% of patients benefit from this method of treatment for mild postoperative intracranial hypertension. The cause of the intracranial hypertension is unclear, but it may be related to blood products and bone dust within the subarachnoid space, impairing CSF reabsorption.

Most patients are transferred from the stepdown unit to a regular floor on postoperative day 1. Diet is advanced, as well as activity. Most patients are ready to leave the hospital within an average of 3 days. Following discharge home, all patients are brought back to clinic within 10 to 14 days of the procedure for a wound check, neurologic examination, and audiogram. There is no role for routine postoperative imaging. The majority of patients are spasm-free at the time of discharge; in addition, patients with residual postoperative spasm tend to achieve delayed symptom resolution if the intraoperative decompression was adequate.[13–15] Cure rates generally range from 80% to 90% in experienced hands.

Complications include CSF leaks, wound infections, and cranial nerve injuries. Postoperative CSF leaks can present with rhinorrhea, otorrhea, or leakage directly through the incision. A lumbar drain with aggressive CSF drainage can eliminate an early postoperative leak, but if this therapy fails, then the wound needs to be revised in the operating room. Wound infections are uncommon, but they also necessitate operative washout and wound revision, along with appropriate antibiotics. Cranial nerve injuries are usually managed expectantly with two major exceptions: (1) patients with significant orbicularis weakness may require a gold weight for corneal protection, and (2) patients with significant vocal cord dysfunction may require a vocal cord injection.

MVD of the seventh nerve remains the gold standard therapy for hemifacial spasm. No other treatment has comparable long-term results. However, the procedure should not be taken lightly, because the complications of the operation can be serious. Results are best in experienced hands. Careful patient selection, meticulous operative technique, and reliable neurophysiologic monitoring are the three keys to successful treatment.

PEARLS

- The preoperative decision to perform an MVD for HFS is guided by clinical presentation, not by MRI.
- Correct preoperative patient positioning is critical to adequate exposure of the CN VII-VIII complex and minimization of brain retraction.
- The copious application of bone wax to the mastoid air cells both before dural opening and after dural closure prevents postoperative CSF leaks.
- Following dural opening, navigation around the cerebellar lobe toward the seventh nerve exit zone is a dynamic process with multiple adjustments of both microscope and patient position. Patience and sharp arachnoidal dissection facilitate safe CSF egress to minimize retraction injury.
- Entry into the cerebellomedullary cistern (i.e., the cistern of the 11th nerve) from below with subsequent dissection upward is the safest way to identify the eighth nerve (which runs quite superficially) and thus prevent injury to it. Cerebellar relaxation afforded by this maneuver facilitates subsequent dissection.

PITFALLS

- Tearing of a venous sinus during dural opening or closure can lead to significant blood loss, and hemostasis is difficult to obtain in this situation.
- Aggressive retraction of the lateral cerebellar lobe can lead to a cerebellar infarction postoperatively and, in rare cases, mass effect upon the brain stem.
- The eighth cranial nerve is extraordinarily sensitive to manipulation; attempts to adequately decompress the seventh nerve at the expense of eighth nerve manipulation rather than extensive arachnoidal dissection can lead to deafness.
- Attempts to directly coagulate small arterioles or venules at the root exit zone of the seventh nerve from the brain stem are prone to causing irreversible neural injury.
- Failure to keep the hand steady while affixing the cranioplasty plate can cause the screw to plunge into the subdural space, causing significant bleeding and necessitating redo wound closure.