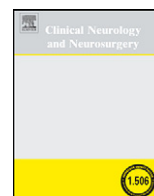




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Microvascular decompression surgery for trigeminal neuralgia and hemifacial spasm: Nuances of the technique based on experiences with 100 patients and review of the literature

Aaron A. Cohen-Gadol*

Goodman Campbell Brain and Spine, Indiana University Department of Neurological Surgery, 1801 North Senate Blvd #610, Indianapolis, IN 46202, USA

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ABSTRACT

Background: Microvascular decompression (MVD) surgery for trigeminal neuralgia and hemifacial spasm offers a relatively low-risk opportunity to treat cranial nerve hyperactivity-compression syndromes, which are associated with severe, disabling facial pain and spasm. Although a number of publications have described the technique in detail, combining the technical nuances from different schools of thought or neurosurgical training in an effort to increase the safety and efficacy of this procedure would be beneficial to the surgeon.

Methods: The nuances of technique and operative findings from performing this procedure for the last 100 cases have been reviewed and combined. The author has reflected on his experience performing microvascular decompression operation.

Findings: The specifics of operating room set-up, positioning, craniotomy, and intradural microsurgical methods are provided, including managing postoperative care and complications.

Conclusion: In the presence of alternative methods of therapy, microvascular decompression operations should be performed with low risk to the patient. There is a learning curve involved with this operation and the surgeon should remain always critical of his/her performance and aspire for a “perfect” result.

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Trigeminal neuralgia is one of the “worst pains humans have been afflicted with.” The severity of this disabling pain and the high rate of its successful surgical treatment have made microvascular decompression surgery (MVD) one of the most satisfying operations in neurosurgery. The typical pain of trigeminal neuralgia is relatively easy to diagnose. Surgical neuroanatomy exposed during MVD surgery is “pristine” and pleasing to the surgeon.

Hemifacial spasm is another syndrome among the cranial nerve hyperactivity-compression disorders. It is characterized as involuntary intermittent twitching of the muscles of the face (usually unilateral). The spasms typically originate around the eye and later spread to other muscles of the ipsilateral face, including the platysma. Postparalytic hemifacial spasm is the most likely condition to be mistaken for the neurovascular conflict phenomenon, hemifacial spasm. Post-paralytic hemifacial spasm follows Bell's palsy or trauma to the nerve (i.e. acoustic neuroma surgery) [1].

The pathogenesis of cranial nerve hyperactivity syndromes such as trigeminal neuralgia, hemifacial spasm, geniculate neuralgia, paroxysmal positional vertigo and glossopharyngeal neuralgia has remained elusive. Although vascular compression of the respective

nerves at the brainstem (neurovascular conflict-peripheral hypothesis) has been proposed as the principle factor involved in genesis of cranial nerve hyperactivity through demyelination of the root entry/exit zone of the nerve (ephaptic transmission) [2,3], there are some patients who harbor no compressive artery on detailed exploratory posterior fossa surgery. In addition, cadaveric studies have revealed an intimate relationship between the cranial nerves in the posterior fossa and the neighboring vessels; these cadavers had no history of cranial hyperactivity syndrome prior to their death [4]. Recently, investigators have implicated other factors (central hypothesis: hyperactivity of the trigeminal and facial nuclei) in addition to peripheral vascular conflict as a cause for the disorder [5].

Irrespective of what is the responsible etiology in the pathogenesis of pain and spasms, microvascular decompression surgery, through mobilizing the offending vessel or performing a rhizotomy, provides an effective and durable palliative option for symptomatic relief. Although percutaneous procedures offer a less invasive route to the Gasserian ganglion for the completion of the rhizotomy, posterior fossa exploration offers the *only opportunity* to identify an offending vessel through a non-destructive procedure with a more durable result [6]. The patient's age and medical co-morbidities remain important factors which could limit the application of a posterior fossa exploratory operation.

* Corresponding author. Tel.: +1 317 396 1275; fax: +1 317 924 8472.
E-mail address: acohenmd@gmail.com

We have offered an MVD operation to our patients who are younger than 70 years of age without prohibitive medical comorbidities. Herein, the details and key steps to maximize safety and efficiency and minimize complications during MVD operations for trigeminal neuralgia and hemifacial spasm are described. An attempt has been made to combine the pearls from multiple schools of neurosurgical teaching. The details of the techniques may have been discretely described previously; however, it is worthwhile to re-emphasize these nuances in one collection, especially for a novice surgeon. Finally, we attempt to reflect our experience with the performance of microvascular decompression surgery for the 100 most recent patients within the past 2 years.

1. Diagnosis

The character of the pain in trigeminal neuralgia is typically unilateral, episodic, severe, stabbing, shock-like, or lancinating and exacerbated by cutaneous stimuli such as tactile pressure, chewing, brushing, breeze of air, or shaving [7]. The tentative desirable response to neuropathic pain medications such as Carbamazepine or Gabapentin often supports the diagnosis of typical trigeminal neuralgia and is potentially a predictor of a good response to MVD surgery. The character of the pain may change with the use of neuropathic pain medications or previous percutaneous procedures and the pain may become more constant. Therefore, the clinician should interview the patient regarding the character of the pain at its inception before any treatment was rendered. The patients who complain of only constant or burning pain and have facial numbness without triggering stimuli are suffering from atypical facial pain and are not good candidates for a posterior fossa exploratory operation. Detailed neurological examination is usually normal.

Patients with hemifacial spasm often have ipsilateral facial weakness due to the use of botulinum injections, facial neuropathy related to neurovascular conflict, or facial muscle weakness related to continued repetitive spasm. Any finding on exam other than the ones mentioned above should persuade the clinician to suspect an underlying structural lesion.

2. Imaging

All patients who are planning to undergo an MVD operation should have a brain magnetic resonance imaging (MRI) or computed tomography (CT) scan to exclude a structural pathology such as a meningioma, acoustic neuroma, or an epidermoid tumor. Epidermoid tumors may present with hemifacial spasm, due to facial nerve irritation, as their only dominant presenting symptom and should be excluded [8]. Even if a high resolution MRI does not identify an offending vascular loop, the consideration for a posterior fossa exploration is appropriate. We have routinely offered MVD surgery to the patients who did not harbor an “MRI evident” vascular loop and have found compressive arterial loops during their posterior fossa exploratory surgery.

3. Operating room set-up and patient positioning

We place the patient in a lateral decubitus (lateral) position. The surgical technician who hands the instruments to the surgeon stands on one side of the patient and the surgeon either stands (during the craniotomy) or sits (during microsurgical portion of the operation) on the other side across from the technician (Fig. 1A); this position of the surgeon relative to the technician allows an easy transfer of surgical instruments between them.

The lateral position has several advantages over the supine position (Fig. 1B). It allows us to perform a lumbar puncture at the start of the procedure while the suboccipital area is being prepared for

surgery. The needle is left in place for a short interval while a urine cup, taped to the back of the patient, collects the cerebrospinal fluid (CSF). During this interval, the patient is draped. By the time the patient's lower torso is draped, the needle is removed by one of the attending nursing staff. This time interval allows approximately 30–40 cm³ of CSF drainage. This CSF drainage will significantly decrease the tension in the infratentorial space and allow a smooth entry into the cerebellopontine angle (CP) by going around the cerebellum during the later stages of surgery. We believe that entry into the posterior fossa by retraction of the cerebellar hemisphere and access into the CP angle is one of the most critical and potentially dangerous parts of an MVD operation. Significant relaxation of the posterior fossa contents through CSF release using a lumbar puncture at the beginning of the procedure prevents initial injury to the lateral cerebellar hemisphere from aggressive retraction. Since cerebellar retraction is minimized, the risk of avulsion of the bridging veins, including the superior petrosal and supracerebellar veins, is also minimized. Alternatively, the surgeon may microscopically open the basolateral cisterns by elevating cerebellum carefully immediately after dural opening to release CSF and achieve brain relaxation.

The lateral position also obviates the need for turning the patient's head into an uncomfortable position (Fig. 1C) during the surgery and may decrease the risk of neck pain postoperatively. This factor is especially important for overweight or obese patients with generous supraclavicular fat pads that would prevent an adequate turn of their head to the contralateral side. The lateral position also allows us to let the ipsilateral shoulder fall forward, away from the incision; this maneuver facilitates movement of the instruments in and out of the surgical field. The shoulder of the patient should be moved to the edge of the bed and the patient's neck and head placed in a “military position”; this maneuver will place the surgical field closer to the surgeon and provides an unhindered draping of the suboccipital area. The head of the patient is slightly flexed and slightly turned contralaterally and held in a pinion (Fig. 1C).

We do not routinely monitor cranial nerve VIII during MVD operations aimed at trigeminal neuralgia; although, this mode of monitoring may be routinely considered by a novice surgeon. On the other hand, the use of intraoperative monitoring for cranial nerve VIII and lateral spread reflex (LSR) is strongly recommended for the patients who undergo an MVD operation for hemifacial spasm. LSR is a measure of hyperactivity of the facial nerve/nucleus. This reflex involves electrical stimulation of the zygomatic or temporal branch of the facial nerve leading to a response recorded from the mentalis muscle (lateral spread). Intraoperative disappearance of this reflex upon mobilization of the offending vessel confirms the identification of the pathological entity.

The retromastoid surgical corridors for trigeminal neuralgia and hemifacial spasm aim to reach along the supramedial and inframedial corners of the posterior fossa. Fig. 1D demonstrates the roadmap for these exposures.

4. The incision

We have recently employed a modified reverse “U” incision—originally described year ago by Walter Dandy—and have enjoyed its advantages over the more commonly used linear incision (Fig. 1E). The Dandy incision typically avoids the neurovascular bundle (occipital nerve and artery) that is usually injured by the linear incision and potentially decreases the risk of postoperative occipital neuralgia. The Dandy incision also obviates the need for deep muscle dissection caudal to the floor of the posterior fossa and, in our experience, has decreased the rate of postoperative suboccipital pain. Most importantly, the Dandy incision reflects and mobilizes the musculocutaneous scalp flap

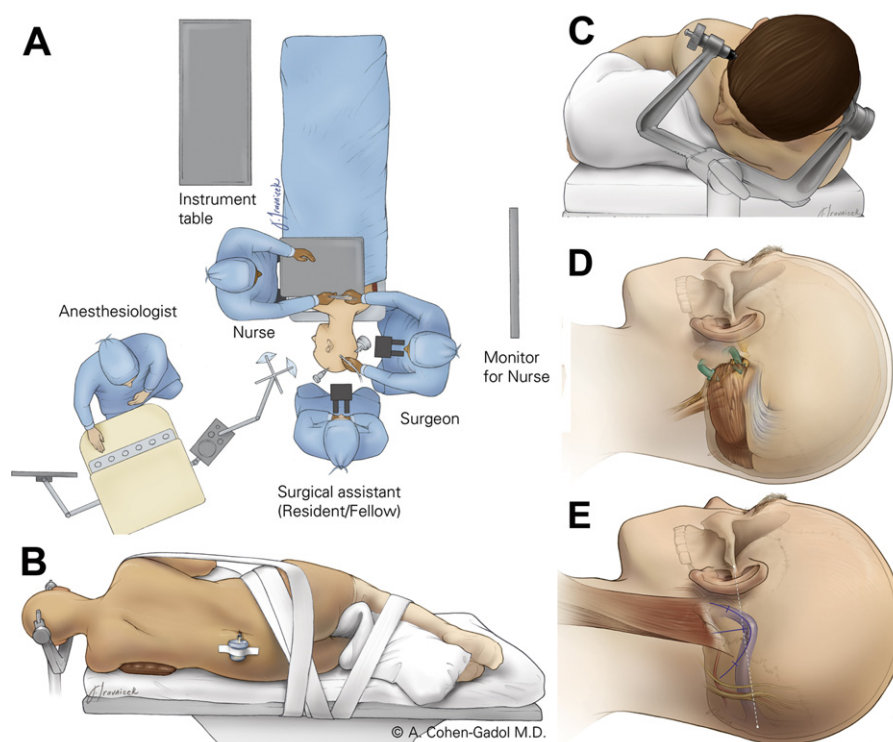


Fig. 1. (A) The surgical technician who hands the instruments to the surgeon stands on one side of the patient and the surgeon stands (during the craniotomy) or sits (during microsurgical portion of the operation) on the other side across from the technician. This position of the surgeon relative to the technician allows an easy transfer of surgical instruments between them. The operating room set-up is illustrated. (B) While the suboccipital area is prepared for surgery, a lumbar puncture can be performed that significantly decreases the tension in the infratentorial space and allows smooth entry into the cerebellopontine angle (CP) around the cerebellum during the later stages of surgery. The need to turn the patient's head into an uncomfortable position during the surgery is obviated and may decrease neck pain postoperatively. The ipsilateral shoulder falls forward away from the incision, allowing movement of the instruments in and out of the surgical field while avoiding the bulk of the shoulder, especially among obese patients. (C) The head of the patient is slightly flexed and slightly turned contralaterally, held in a pinion. This position of pinion placement on the head of the patient facilitates anchoring the retractor systems to the pinion later in the procedure. This slight contralateral turn of the head will assist the surgeon with the intradural portion of the operation when the operator attempt to go "around the cerebellum" to enter the CP angle cisterns. (D) The surgical corridors used for microvascular decompression for trigeminal neuralgia (supralateral cerebellar approach) and hemifacial spasm (infralateral cerebellar approach) are shown. (E) The modified reverse "U" incision typically avoids the neurovascular bundle (occipital nerve and artery); obviates the need for muscle dissection caudal to the posterior fossa floor; and reflects and mobilizes the musculocutaneous scalp flap inferiorly, out of the way of the cerebellar retractor. The relationship of the incision relative to theinion-zygomatic line and the mastoid groove is shown. ©Aaron Afshin Cohen-Gadol.

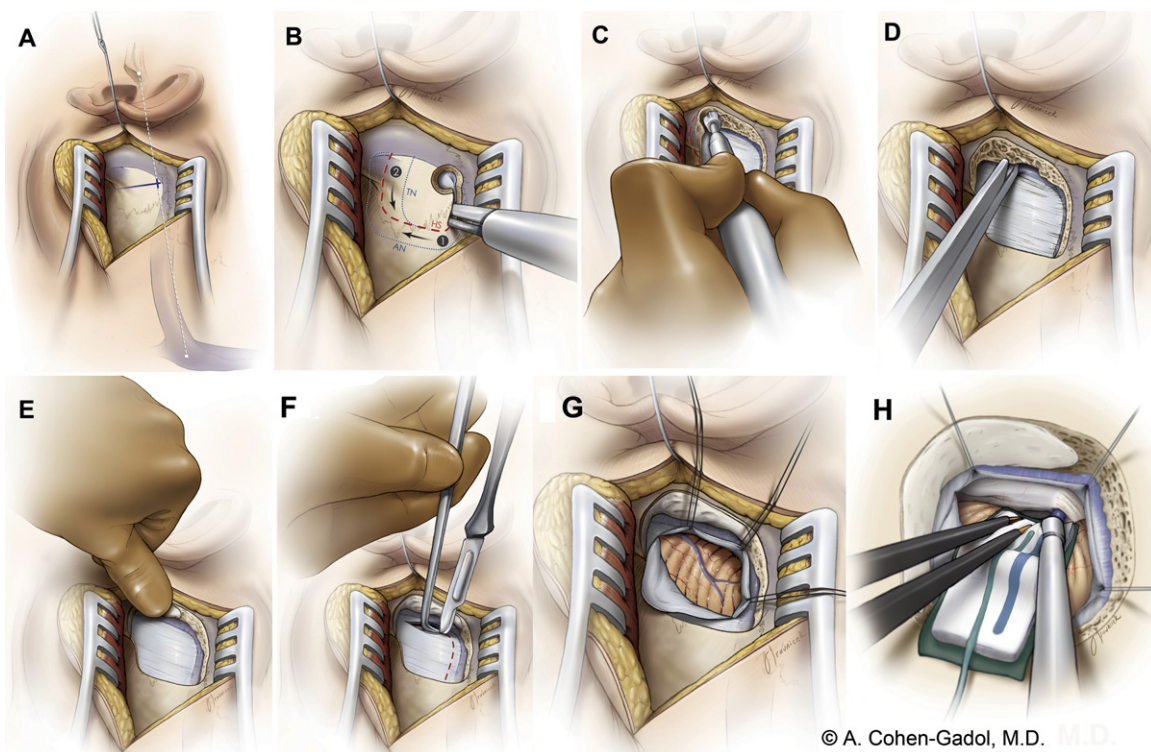
inferiorly, out of the way of the retractor blade that holds the cerebellar hemisphere medially during the later stages of the operation. The linear incision, however, requires the musculocutaneous flap to be retracted, compressed, and folded underneath the retractor blade. This mass of retracted musculocutaneous flap can hang over the craniotomy defect and interfere, especially in obese patients with thick scalps, with the ability of the retractor to provide adequate field of view through the posterior fossa. This disability is often remedied through additional medial retraction of the flap through extension of the linear incision further into the suboccipital muscles, potentially increasing the risk of postoperative suboccipital headaches. This enfolded musculocutaneous flap also increases the working distance between the hand of the surgeon and the target—the trigeminal or facial nerve.

5. The craniotomy or craniectomy

Craniotomy (or craniectomy) is bounded superiorly by the transverse sinus and laterally by the sigmoid sinus. In older patients, the suboccipital dura and the edges of the sinuses may be especially adherent to the inner cortex of the bone, and craniectomy may be beneficial to avoid the risk of dural sinus injury. Placement of the initial burr hole at the junction of the transverse and sigmoid junction would facilitate the later steps in the craniotomy. We have referred to this burr hole as the "strategic burr-hole." Our study, which included a review of 100 dry skulls, revealed that this

junction is variable. However, a line connecting theinion to the posterior root of the zygoma defines the transverse sinus. This line transects the vertical line defined by the mastoid groove. A burr hole placed just inferior and medial to the junction of these two lines often barely exposes the medial and inferior aspects of the junction of the dural sinuses (Fig. 2A) [9]. The asterion is not a constant finding especially in older crania, and a burr hole placed over the asterion often exposes the entire width of the transverse sinus and may place this structure at risk of injury. An emissary vein should not be mistaken for the sigmoid sinus.

The size of the craniotomy or craniectomy is often small and about twice the size of a quarter coin. If an MVD is performed for trigeminal neuralgia, bony removal exposes the inferior edge of the transverse sinus. If the patient is suffering from hemifacial spasm, bony removal is extended inferiorly and exposure of the transverse sinus is not necessary (Fig. 2B). For either diagnosis, the medial edge of the sigmoid sinus is exposed by drilling the mastoid bone and the thin inner cortex of this bone is removed using Kerrison rongeurs (Fig. 2C and D). The sigmoid sinus is often very adherent or embedded in the inner cortex of the bone, and the "mouth" of the Kerrison rongeur should be pointed away (superiorly) from the sinus (Fig. 2D). The bone over the sigmoid sinus should be removed in small pieces using the rongeur to avoid any injury to the sinus. If a small tear in the wall of the sinus is detected, bone wax may be used to seal the hole against the edge of the mastoid bone. Impaction of hemostatic material into the sinus should be avoided to prevent



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Fig. 2. (A) Craniotomy (or craniectomy) is bounded superiorly by the transverse sinus and laterally by the sigmoid sinus. Initial “strategic” burr hole placement at the transverse and sigmoid sinus junction is variable. However, a burr hole placed just inferior and medial to the juncture of a horizontal line (dotted white) that connects theinion to the posterior root of the zygoma, and a vertical line (blue), defined by the mastoid groove, often barely exposes the medial and inferior aspects of the junction of the dural sinuses. The asterion is not a constant finding (especially in older crania), and a burr hole over the asterion often exposes the entire width of the sinus, placing this structure at risk of injury. (B) The size of the craniotomy or craniectomy is often small and about twice the size of a quarter coin. If the MVD is performed for trigeminal neuralgia, bony removal exposes the inferior edge of the transverse sinus. If the patient is suffering from hemifacial spasm, bony removal is extended inferior, and an exposure of the transverse sinus is not necessary. (C) The medial edge of the sigmoid sinus is exposed by drilling the mastoid bone, and the thin inner cortex of this bone is removed using Kerrison rongeurs. (D) The sigmoid sinus is often very adherent or embedded in the inner cortex of the bone. The bone may be removed in small pieces using a small Kerrison rongeur. The Kerrison’s mouth should be pointed away from the sinus to avoid injury to the sinus. If a small tear in the wall of the sinus is detected, bone wax may be used to seal the hole against the edge of the mastoid bone. Impaction of hemostatic material into the sinus should be avoided to prevent sinus thrombosis. (E) Mastoid air cells should be well waxed both after completing the craniotomy and later in the procedure at the end of dural closure. (F) Leaving an edge to avoid sinus injury, the dura may be opened along the dural sinuses. During opening, if a dural sinus tear is evident, this laceration in the sinus may be sutured primarily or over a piece of muscle. Bipolar coagulation usually retracts the dural edges further, increasing the size of the laceration and compounding the problem. (G) The edges of the dura and the venous sinuses are tacked up against the craniotomy edges using three silk sutures. This pattern of dural opening leaves the majority of the dura on the cerebellar hemisphere, which is then covered by pieces of cottonoids. This method avoids shrinkage of the dura due to the heat generated by the operating microscope’s lamp aimed at the surgical field during the intradural portion of the operation. (H) A piece of glove (cut slightly larger than the cottonoid), acts as a rubber dam and protects the cerebellar hemisphere from potential injury caused by friction from the cottonoid’s surface. The cottonoid is advanced parallel to the junction (groove) of the tentorium and petrous apex, slightly toward the petrous side. Identification of these landmarks prevents (1) the unintentional exposure (and resultant tear and bleeding) of the supracerebellar bridging veins superiorly or (2) the placement of retraction directly against the VII/VIII cranial nerve complex inferiorly, which would potentially place hearing at risk. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.) ©Aaron Afshin Cohen-Gadol.

sinus thrombosis. The emissary vein may be found and coagulated. Mastoid air cell should be well waxed both after completing the craniotomy and later in the procedure, at the end of dural closure (Fig. 2E). In older patients, an exposure of the sigmoid sinus may not be necessary if the wall of the sinus is very adherent to the bone; cerebellar atrophy in this patient population obviates the need for a lateral trajectory and significant cerebellar retraction.

The dura may be opened along the venous sinuses for the patients with trigeminal neuralgia, leaving an edge of dura around the sinuses (Fig. 2F). The edges of the dura and the venous sinuses are tacked up against the craniotomy edges using three silk sutures (Fig. 2G). This pattern of dural opening leaves the majority of the dura, which is then covered by pieces of cottonoids, on the cerebellar hemisphere. This method avoids shrinkage of the dura due to the heat generated by the operating microscope’s lamp aimed at the surgical field during the intradural portion of the operation.

If there is evidence of a dural sinus tear during opening of the dura, this laceration in the sinus may be sutured primarily or over a piece of muscle. Bipolar coagulation usually retracts the dural edges

further, increasing the size of the laceration and compounding the problem.

5.1. Entry into the cerebellopontine angle

The operating microscope is brought into the field for the rest of the procedure. Brain relaxation at this time (provided by the lumbar puncture performed at the initial stages of the operation) makes the next operative steps smooth, efficient, and trouble free. As mentioned previously, in our opinion, entry into the posterior fossa and exposure of the CP angle cisterns can be challenging and associated with risks inherent in a “tight” posterior fossa space.

Jannetta has suggested a method for protecting the cerebellar cortex during the advancement of the cottonoid around the cerebellum [10]: the cottonoid used to cover and go around the cerebellar hemisphere is lined with a piece of glove (called rubber dam) cut in the shape of the cottonoid. This rubber dam is first placed on the cerebellum, and then the corresponding cottonoid is placed over it. As the cottonoid is advanced around the cere-

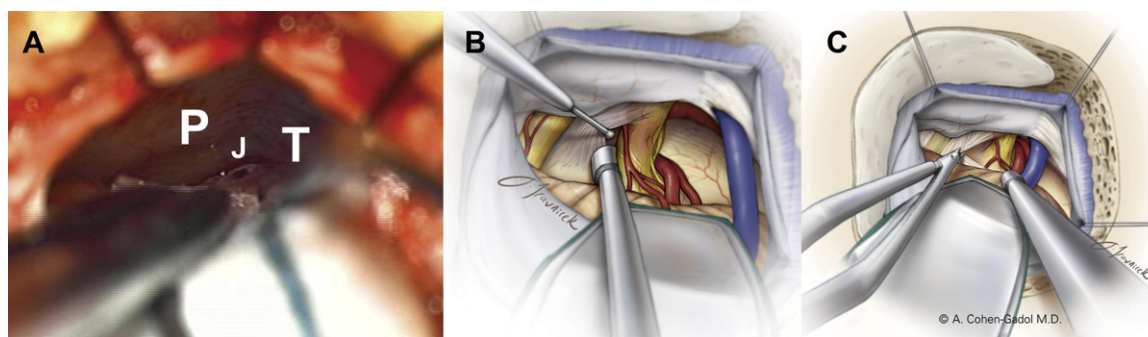


Fig. 3. (A) Gentle inferomedial retraction of the cerebellar hemisphere exposes the superior petrosal vein (also shown in Fig. 2H). The arachnoid membrane just inferior to the vein is sharply opened, and an additional amount of CSF is released. The arachnoid membrane over the VII/VII cranial nerves complex is left intact. The retractor blade is placed just inferior to the superior petrosal vein, possibly placing this vein under slight tension. Often, this maneuver provides all the working space needed caudal to the vein. Abbreviations: P, petrous dura; J, junction of petrous bone and tentorium; T, tentorium. (B and C) Care must be taken to preserve all of the vessels around the brainstem. Deeper arachnoid membranes should be cut with caution and first dissected free of their entangled vessels using a fine ball-tip probe. The trigeminal nerve is located deeper and more medial than the VII/VIII complex in the CP angle cisterns. The VII/VIII complex should not be mistaken for the trigeminal nerve. This would result in decompression of the wrong nerve. One of the most common patterns of vascular compression by the superior cerebellar artery, along the shoulder of the root entry zone, is shown. ©Aaron Afshin Cohen-Gadol.

bellar hemisphere, the cottonoid “glides” over the cerebellum over the rubber dam, and the cerebellar cortex is protected from potential injury caused by the friction from the surface of the cottonoid (Figs. 2H and 3A).

5.2. The intradural portion of the MVD operation for patients with trigeminal neuralgia

The junction of the tentorium and petrous apex is identified and the cottonoid is advanced parallel to this junction (groove) and slightly toward the petrous side over the lateral cerebellum (Figs. 2H and 3A). Identification of these landmarks along the corridor of entry prevents the surgeon from unintentionally moving too superiorly along a plane, exposing the supracerebellar bridging veins with their resultant tear and bleeding, or moving too inferiorly, placing traction directly against the seventh/eighth cranial nerve complex and potentially placing hearing at risk.

Gentle inferomedial retraction of the cerebellar hemisphere will expose the superior petrosal vein. We have exceedingly rarely sacrificed this vein and have rarely sacrificed one of its branches. The arachnoid membranes just inferior to the vein is sharply opened and an additional amount of CSF released. The arachnoid over the VII/VII cranial nerves complex is usually left intact. The retractor blade is placed just inferior to the superior petrosal vein; this vein may be under slight tension but often this maneuver is all that is needed to provide enough working space caudal to the vein to work through without a need to sacrifice the vein (Fig. 3B). The surgeon may often find a small artery usually entangled with the arachnoid layers just superior to the VII/VIII cranial nerve complex that feeds the petrous meninges and may be sacrificed if necessary. This artery should not be mistaken with the labyrinthine artery which enters the internal auditory canal along with the VII/VIII cranial nerves.

Care must be taken to preserve all the vessels around the brainstem. Deeper arachnoid membranes should be cut with caution and first dissected free of their entangled vessels using a fine ball-tip probe (Fig. 3B and C). The trigeminal nerve is located deeper and more medial than the VII/VIII complex in the CP angle cisterns. The latter should not be mistaken with the former, leading the surgeon to decompress the wrong nerve.

Advancement of the retractor blade deeper along the cerebellum and slight increase in cerebellar retraction will bring the root entry zone of the trigeminal nerve in view. As mentioned previously, the use of a “reverse U” musculocutaneous flap prevents the scalp flap from blocking the retractor blade during medial retraction.

6. Technical pearls for a thorough microvascular decompression for trigeminal neuralgia

It is important to remember that the presumed site of neurovascular conflict is typically at the root entry zone of the nerve as it enters the brainstem and usually not along the nerve in the CP angle cisterns, although the central myelin may extend distally along the nerve. Detailed, patient, and careful inspection (360°) of the space around the root entry zone and the nerve in the CP cisterns is important. Generous opening of the arachnoid layers in the region around the nerve and gentle handling of the nerve allows a thorough inspection. Some clinicians argue that this manipulation leads to a rhizotomy of the nerve and is responsible for some of the pain control afforded by MVD operations. Arterial compression by the superior cerebellar artery along the shoulder of the root entry zone is one of most common sites of compression [10]. Mobilization of the arterial loop often discloses a site of discoloration along the root entry zone and the nerve. Discovery of this discoloration confirms that the intended pathology is found and is predictive of a good outcome after surgery. The possibility of multiple offending vessels (arterial and/or venous loops) should be excluded with careful inspection (Fig. 4A). The use of microsurgical instruments with fine dissecting tips that are slightly bent or bayoneted is helpful in the small working space afforded during this portion of the operation.

We consider neurovascular conflict due only to venous loops a controversial phenomenon. We do mobilize the larger veins if possible without sacrificing them. Small veins may be sacrificed. We routinely perform a rhizotomy as well when arterial compression and/or a discoloration of the nerve is not found. This is the case also if only potential offending veins are found. We complete a rhizotomy by gentle bipolar coagulation (at low levels of current) of the root entry zone at the site that corresponds to the patient's preoperative pain distribution (middle third of the root entry zone for V2 and the lower third for V3). We have avoided partial root transaction because of the potential risk of disabling anesthesia dolorosa.

Teflon implants should be shredded into a variety of different ball-shaped sizes by the assistant. Each implant may be soaked in saline solution just before its use as it is held in fine forceps and handled by the surgeon. This preparation allows the implant to be molded to the shape of the space between the artery and the nerve. This maneuver will also provide an adequate decompression of the nerve along the length of the nerve and prevents a delayed displacement of the implant. Please refer to Fig. 4B–D for the tech-

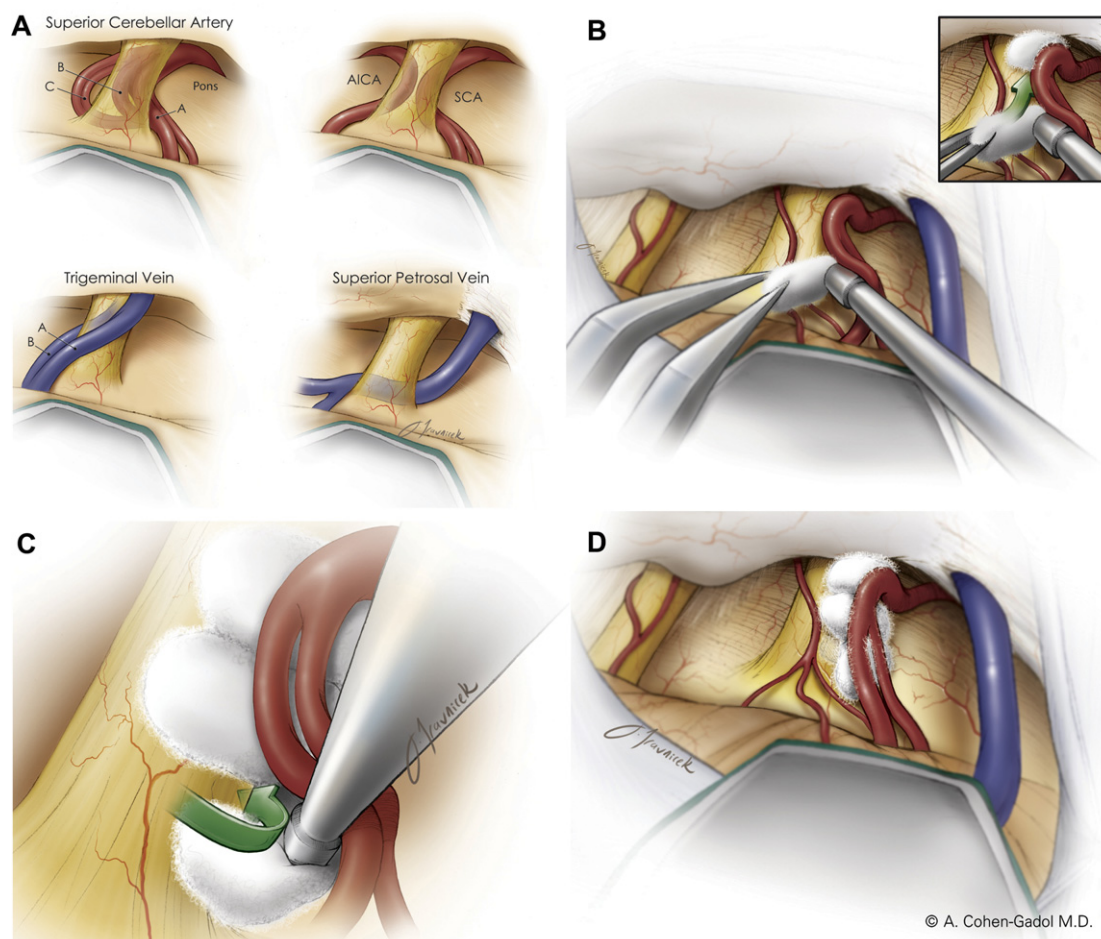


Fig. 4. (A) The most common patterns of arterial or venous offending vessels are depicted in the accompanying sketch. (B and C) Pieces of shredded Teflon implant are used to prevent any contact between the nerve and surrounding vessels. The use of unshredded Teflon should be avoided because of the risk of displacement of the implant later. Pieces of Teflon are inserted and pushed ahead along the nerve (inset in B). At the brainstem and along the root entry zone of the nerve, a piece of shredded Teflon is inserted along the shoulder and medial axilla of the nerve to prevent any contact of the vessel with the brainstem or nerve (C). The vessel is elevated and generously padded away from the nerve to prevent any contact (D). ©Aaron Afshin Cohen-Gadol.

nical details related to placement of the implant and variations of the compressive vessels. Operative findings are summarized in Fig. 5A–J.

Based on our experience during re-exploratory surgeries, we have identified the following factors, which may have prevented the initial surgery from achieving an adequate decompression:

- (1) Inadequate exposure of the root entry zone is the number one reason for inadequate decompression. This factor disables the surgeon from finding all the offending vessel(s), which may be hidden deep in the arachnoid membranes. More often than realized, the offending superior cerebellar artery is concealed underneath a vein and thick veil of arachnoid at the shoulder or axilla of the nerve. Careful opening of the arachnoid membranes using microsurgical methods around the entry zone is critical (Fig. 5F). For unknown reasons, these arachnoid membranes are usually thicker and more fibrotic among the patients with trigeminal neuralgia.
- (2) The offending vessel may be hidden at the axilla of the nerve, on the medial side of the root entry zone. Aggressive manipulation of the nerve should be avoided. The surgeon may work superior and inferior to the nerve to partially mobilize the artery. Small pieces of *shredded* Teflon may be inserted from the inferior

aspect of the nerve and pushed superiorly between the nerve and artery in a semi-blinded fashion. Identification of the Teflon patch superior to the nerve confirms adequate mobilization of the vessel. Endoscopic techniques may assist with enhanced visualization in the situations where the artery is located deep in the axilla of the entry zone, hidden from the surgeon's field of view.

- (3) The Teflon patch should be shredded and inserted piecemeal to conform to the area between the artery and the nerve. The use of unshredded Teflon patch, which may be easily dislodged, should be avoided.
- (4) The petrous bone over the distal part of the nerve into the Meckel's nerve may be hypertrophic. Bony removal in this area may expose a compressive vessel over the distal part of the nerve (Fig. 5G and H).
- (5) Overzealous use of the Teflon implant should be avoided. Teflon granuloma may cause pain recurrence (Fig. 5I and J).

Overall, the most common reason for inadequate decompression is an inadequate exposure of the root entry zone and an inadequate inspection of this site. Decompression of the wrong nerve (VII/VII complex) has been reported, but is rare [11].

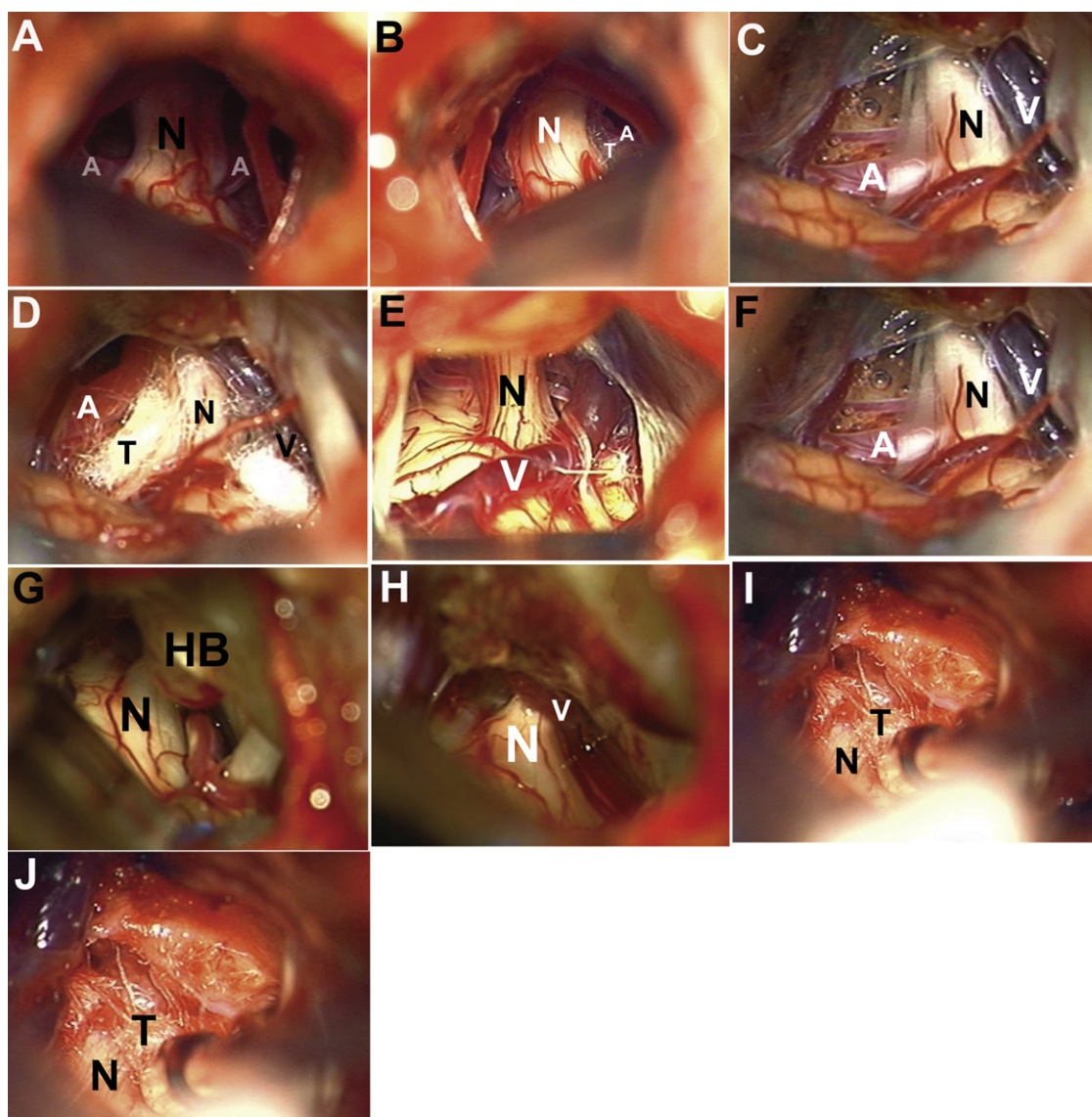


Fig. 5. (A) Intraoperative photos through surgical microscope: one of the most common patterns of arterial compression along the axial of the nerve (A) and after vessel mobilization and insertion of Teflon implant (B). Both arterial and venous neurovascular conflict (C) and after Teflon implantation (D). Venous conflict (E). Hard-to-reach vessel hidden underneath a vein and motor roots of the nerve (F). Hyperostotic petrous bone along the distal part of the nerve (G) hiding a distal compressive vessel after bony removal (H). Overzealous Teflon implantation from the first surgery causing Teflon granuloma and recurrent trigeminal pain (I) and the exposure of the nerve after removal of the Teflon implant revealing no neighboring compressive vessel (J). *Abbreviations:* A, offending artery; N, trigeminal nerve; T, Teflon implant; V, offending vein; MR, motor roots off the trigeminal nerves (Portio minor); HB, hypertrophied bone. ©Aaron Afshin Cohen-Gadol.

7. The intradural portion of the MVD operation for patients with hemifacial spasm

For patients with hemifacial spasm, the dural closure is depicted in Fig. 6A. For patients with trigeminal neuralgia, the junction of the tentorium and petrous apex is identified and the cottonoid is advanced parallel to this junction (groove) and toward the petrous side. For the patients with hemifacial spasm, the petrous bone (as it turns slightly to join the floor of the posterior fossa) is identified, and the cottonoid is advanced over the rubber dam toward the lower cranial nerves (Fig. 6B and C). While superomedial retraction is applied, purely medial retraction of the cerebellum, parallel to the VII/VIII cranial nerves, is avoided to prevent direct transmission of retraction vector to these nerves and potential hearing loss.

The arachnoid over the lower cranial nerves is sharply opened and cranial nerve IX is identified. Cerebellar retraction in the supra-medial direction allows the operator to follow cranial nerve IX along its length to its exit zone from the lower brainstem. Arach-

noid membranes over the VII/VIII nerve complex and lower cranial nerves are opened sharply close to the brainstem to prevent undo traction on these nerves (Fig. 6D–F). Intraoperative monitoring of cranial nerve VIII is important in increasing the safety of this operation. A loss or decrease in the patient's hearing is this operation's most common adverse effect.

The vector of retraction is parallel to the pathway of cranial nerve IX; therefore, the retractor blade is placed on the cerebellum just superficial to the most medial visible part of cranial nerve IX. Any change in brainstem auditory evoked potential requires repositioning of the retractor or its temporary release. A small portion of the cerebellar flocculus overlying the root exit zone of cranial nerve VII is removed to assist with the exposure of the root exit zone of the nerve while minimizing the amount of retraction (Fig. 7A). The root exit zone of nerve VII (grayish in color) is anterior and slightly inferior to nerve VIII (more whitish in color) and may be directly visualized upon gentle elevation of nerve VIII using a fine dissector (Fig. 7B). The offending vessel (most commonly the posterior

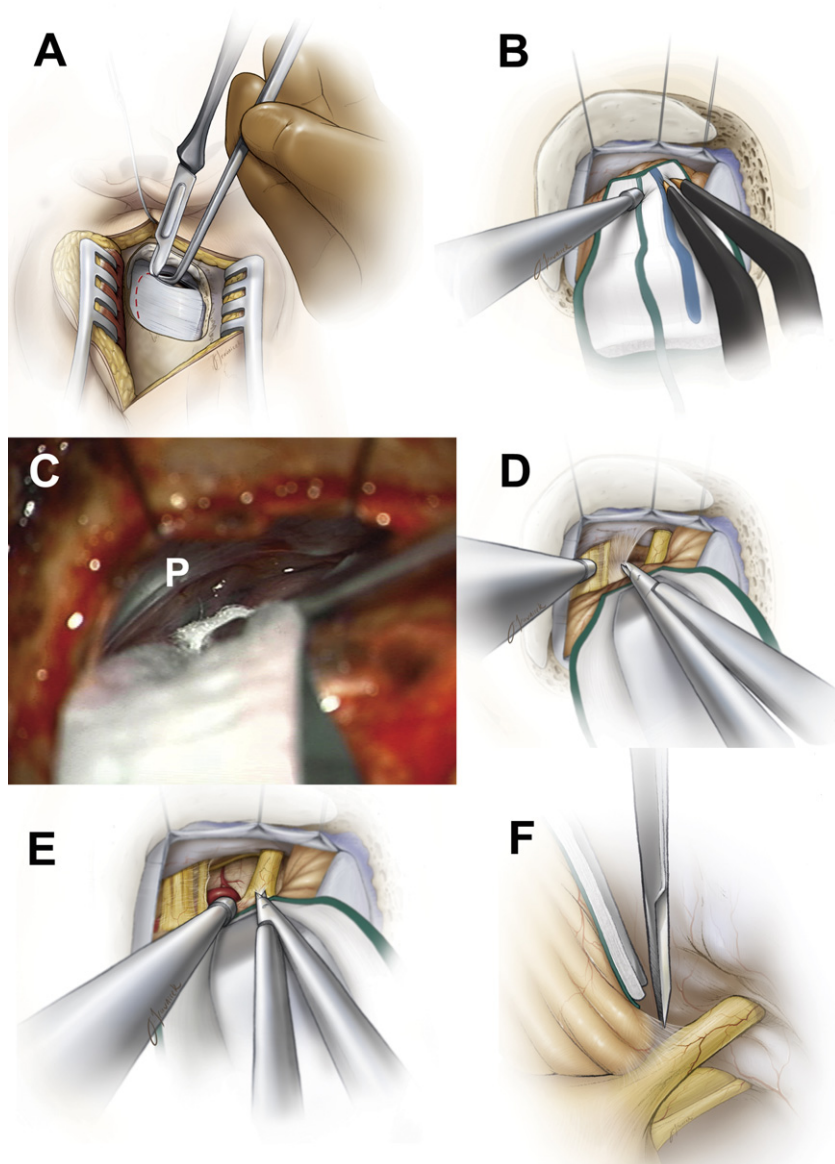


Fig. 6. (A) The dural opening for the patients with hemifacial spasm is shown. (B and C) The junction of the petrous bone and floor of the posterior fossa is identified and the cottonoid is advanced over the rubber dam along the inferior petrous bone toward the lower cranial nerves. Medial retraction of the cerebellum parallel to the VII/VIII cranial nerves is avoided to prevent transmission of retraction to these nerves and potential resultant hearing loss. *Abbreviation:* P, petrous dura. (D–F) Arachnoid membranes over the VII/VIII complex and lower cranial nerves are opened sharply (D), close to the brainstem to prevent undo traction on these nerves. The vector of retraction is parallel to the pathway of cranial nerve IX. The retractor blade is then placed on the cerebellum just superficial to the visible part of cranial nerve IX (E). Again, direct retraction parallel to the VII and VIII cranial nerve complex is avoided, and any change in brainstem auditory evoked potential requires repositioning of the retractor or its temporary release. Microscissors are used to sharply open the arachnoid layers adhering the VII/VIII cranial nerve complex to the flocculus (F). ©Aaron Afshin Cohen-Gadol.

inferior cerebellar artery, anterior inferior cerebellar artery, or vertebral artery) [8] is hidden in the axilla of the nerve and may be mobilized and padded away using different size shredded Teflon implants (Fig. 7C–F). It has been our experience that the length of the artery along the exit zone of nerve VII and over the brainstem inferior to nerve VII needs to be padded away with the implant to achieve a desirable result. The lateral spread reflex (LSR), which is present in most patients, disappears upon mobilization of the artery and is another confirmatory test to assure that the pathological entity is found and managed appropriately.

8. Technical pearls for a thorough microvascular decompression for hemifacial spasm

It is important to remember that the presumed site of neurovascular conflict is at the root exit zone of the nerve as it

enters the brainstem, and not along the nerve in the CP angle cisterns. Detailed and careful inspection of the space around the root exit zone is important. Gentle handling of the surrounding neurovascular structures allows safe and thorough inspection. Arterial compression by an offending vessel along the shoulder of the root exit zone has rarely been reported [12]. Mobilization of the arterial loop often discloses a site of discoloration along the nerve (Fig. 7B and C). Discovery of this discoloration confirms that the intended pathology is found and is predictive of good outcome after surgery.

Based on our experience during re-exploratory surgeries, we have identified the following factors which may have disabled the initial surgeon to complete an adequate decompression:

- (1) Just as in the case of the patients with trigeminal neuralgia, inadequate exposure of the root exit zone is the number one

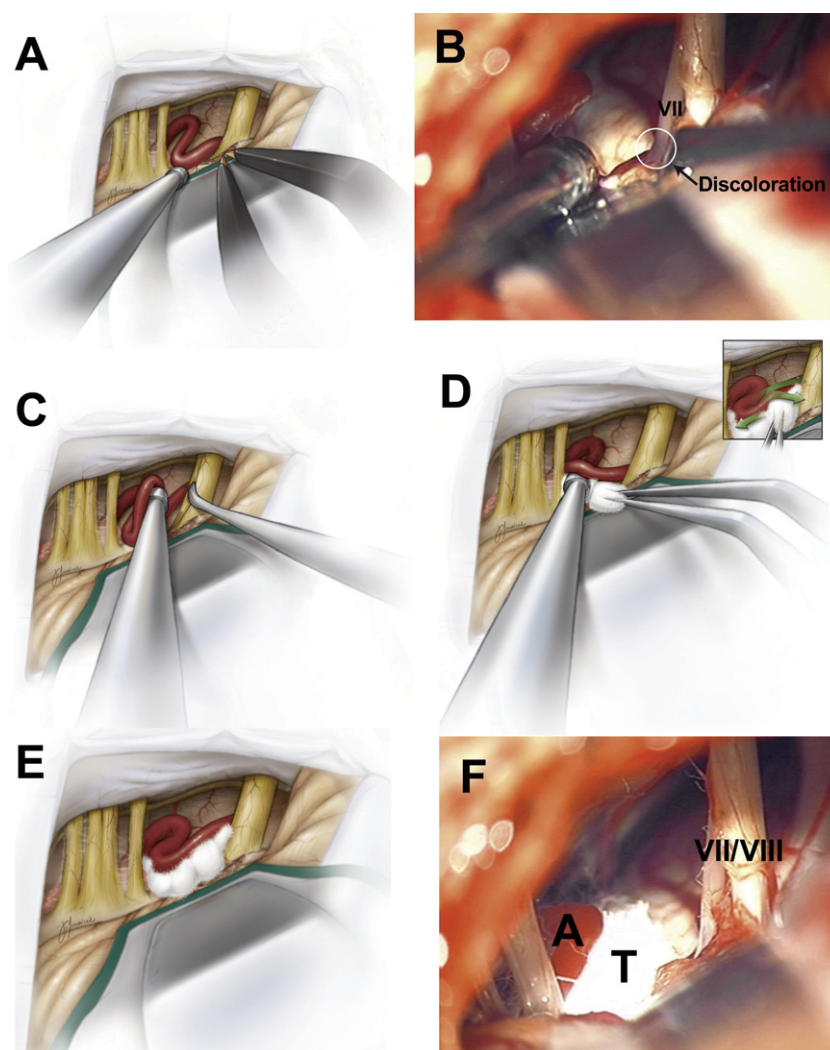


Fig. 7. (A) A small portion of the cerebellar flocculus overlying the root exit zone of the VII cranial nerve is removed to assist with the exposure of the root exit zone of the nerve while minimizing the amount of retraction. (B and C) The nerve VII root exit zone is anterior and slightly inferior to nerve VIII and may be directly visualized when nerve VIII is gently elevated with a fine dissector. The offending vessel (most commonly, the posterior inferior cerebellar artery, causing discoloration at the site of compression) is hidden in the nerve's inferior axilla and may be mobilized and padded away from the nerve using a shredded Teflon implant. Our experience has shown that the artery's entire length along nerve VII's root exit zone and the area over the brainstem need to be padded. The lateral spread reflex (LSR), present in most patients, disappears upon mobilization of the artery and is another confirmatory test to assure that the pathological entity is found and managed appropriately. (D–F) Inadequate exposure of the root entry zone is the primary reason for inadequate decompression. The offending vessel(s) may be hidden deep in the arachnoid membranes or at the depth of the cleft formed by the cerebellum and brainstem. Shredded Teflon implant should be inserted piecemeal between the artery and nerve. Unshredded Teflon patch, which may be easily dislodged, should not be used. Pieces of the prosthesis are pushed superiorly and inferiorly (inset) to mobilize the artery along its entire length (D and E). The offending vessel is kept away from the brainstem and nerve (F). *Abbreviations:* T, Teflon; A, offending artery. ©Aaron Afshin Cohen-Gadol.

reason for inadequate decompression. This factor disables the surgeon from finding all the offending vessel(s), which may be hidden deep in the arachnoid membranes or at the depth of the cleft formed by the cerebellum and the brainstem. More often than realized, the surgeon mobilizes the vessel along the nerve and not along the root exit zone located more medially and deeply (than realized) along the brainstem. Careful retraction of the cerebellum is necessary to expose the hard-to-reach root exit zone of the nerve. A change in the brainstem auditory evoked potentials is not a reason for inadequate exposure of the root exit zone of the nerve. Rather, release of the arachnoid adhesions along the VII/VIII nerve complex to relieve traction on these nerves is necessary.

- (2) The Teflon implant should be shredded and inserted piecemeal to conform to the area between the artery and the nerve. Use of unshredded Teflon which may be easily dislodged should be avoided.

9. Closure

Before closure of the dura, the CP angle is well irrigated with saline solution to assure that there is no bleeding, to clear the field, and, importantly, to make sure the implanted Teflon pieces are not becoming mobile and displaced because of the flow of the fluid and CSF. The dura is then approximated primarily. We do not perform a “watertight” dural closure and have had a very low rate of CSF leakage through the incision or the nose. Mastoid air cells are rewaxed thoroughly and the bone flap replaced, or a methylmethacrylate cranioplasty is performed. The muscle and scalp are closed in the anatomical layers. Staples are used for skin closure.

10. Postoperative care

Patients are watched overnight in the Intensive Care Unit and then transferred to the floor. We do not routinely perform a head CT

postoperatively. Dexamethasone taper for 1 week is administered prophylactically against aseptic meningitis. Nausea and pain are controlled through appropriated medications. Preoperative pain medications for trigeminal neuralgia are weaned off starting 1 week after surgery if the patient remains pain free.

The effect of surgery may be delayed especially for the patients with hemifacial spasm. Patients should be assured of this delayed effect in order to avoid their disappointment with the result of surgery. Occasional delayed facial palsy may occur after MVD for hemifacial spasm. This palsy is temporary and responds well to a Dexamethasone taper of 1 week in duration.

10.1. Management of postoperative complications

Sensorineural hearing loss is a rare complication from an MVD for trigeminal neuralgia and a more serious risk from an MVD for hemifacial spasm. This hearing loss should be distinguished from middle ear effusion, which is identified as a sense of “fullness in the ear” and is temporary. If the patient is suffering from CSF rhinorrhea, we return the patient to the operating room immediately for repacking of the mastoid air cells with additional bone wax and fat graft (1% in our series of last 100 patients). If CSF leakage is from the wound, we place the lumbar drain for 72 h. If drainage continues when the lumbar drain is discontinued, we return the patient to the operating room for a “watertight” dural closure and wound revision.

We have not experienced any incidence of hearing loss with our patients who underwent MVD for trigeminal neuralgia but have had two patients who had decreased hearing after MVD for hemifacial spasm (2%). Other complications included CSF leakage through the incision in one patient who required placement of additional stitches in the wound and local wound care. There was no other incidence of cranial nerve palsy except one delayed facial nerve palsy 2 weeks after surgery for hemifacial spasm, which resolved with administration of oral steroids. No patient suffered from stroke or hemorrhagic infarction of the cerebellum or brainstem. No patient was sent to rehabilitation after his/her surgery; all returned home, and all patients returned to work within 3 months of their surgery. One patient suffered from aseptic meningitis after surgery without any sequela. One patient had a moderate-size subdural hematoma 10 days after surgery and presented with intractable headaches (patients with intractable headaches after surgery should undergo a CT scan). She required burr-hole evacuation of her subdural hematoma. No patient suffered from a postoperative infratentorial hematoma. The overall complication rate of the procedure (CSF leak, meningitis subdural hematoma, hearing loss) in our series was 6%. The incidence of major morbidity or mortality has been 0%. The rate of facial pain and spasm control without the use of medications after surgery has been 90% among our patients. All patients were followed for one year postoperatively and the above outcomes were measured through follow-up patient visits and phone calls. No postoperative hearing test was completed unless the patient complained of hearing difficulty.

The above data compare favorably to what has been reported previously. Although the details and nuances mentioned in the present paper have been discussed in different reports, the present offering combines, in one place, the pearls of the previous studies.

When compared with the largest recent series of patients who underwent microvascular decompression surgery by Jannetta [13]. McLaughlin et al. [13] reported approximately 1% risk of hearing loss, 2% risk of CSF leakage, and 0.5% chance of cerebellar injury. These findings are similar to the ones reported in our series.

Jannetta has employed the linear incision without difficulty; the “U” incision has proven more practical for the author for the purposes mentioned above. Our bony removal and dural opening is very similar to the ones recommended by others [13]. Jan-

netta reported minimal consequences with sacrifice of the superior petrosal vein; we have avoided sacrificing this vein as it rarely interferes with our dissection field as long as the retractor is placed judiciously. Jannetta has also emphasized the value of using a shredded implant. Venous compression has usually been handled with mobilization of the nerve and implant insertion if the vein is on the pial surface of the brainstem for any segment of its route. However the offending veins that are in the subarachnoid space can be coagulated and cut (Jannetta, personal communication). Although the sling retraction method has been described previously [14] we have not routinely employed this method because of the risk of arterial kinking and vascular injury/infarct. Others have found the sling method effective [15].

The short-term followup results reported here are consistent with those reported in the literature [15]. Long-term studies (>10 years) are important to evaluate the ultimate outcome of microvascular decompression surgery.

11. Final thoughts

As mentioned previously, MVD surgery is an effective and gratifying surgery, both for the patient and the surgeon. The surgeon should take advantage of microsurgical techniques in performing the operation. In the presence of alternative methods of therapy, including percutaneous procedures that carry relatively minimal risk, MVD operations should be performed with low risk to the patient. There is a learning curve involved with this operation and the surgeon should remain always critical of his/her performance and aspire for a “perfect” result.

References

- [1] Martinelli P, Gabellini AS, Lugaesi E. Facial nucleus involvement in post-paralytic hemifacial spasm? *J Neurol Neurosurg Psychiatry* 1983;46:586.
- [2] Granit R, Leksell L, Skoglund CR. Fibre interaction in injured or compressed region of nerve. *Brain* 1944;67:125–40.
- [3] Howe JF, Calvin WH, Loeser JD. Impulses reflected from dorsal root ganglia and from focal nerve injuries. *Brain Res* 1976;116:139–44.
- [4] Matsushima T, Inoue T, Fukui M. Arteries in contact with the cisternal portion of the facial nerve in autopsy cases: microsurgical anatomy for neurovascular decompression surgery of hemifacial spasm. *Surg Neurol* 1990;34:87–93.
- [5] Möller AR. Vascular compression of cranial nerves. II: Pathophysiology. *Neurol Res* 1999;21:439–43.
- [6] Barker 2nd FG, Jannetta PJ, Bissonette DJ, Larkins MV, Jho HD. The long-term outcome of microvascular decompression for trigeminal neuralgia. *N Engl J Med* 1996;334:1077–83.
- [7] Miller JP, Acar F, Burchiel KJ. Classification of trigeminal neuralgia: clinical, therapeutic, and prognostic implications in a series of 144 patients undergoing microvascular decompression. *J Neurosurg* 2009;111:1231–4.
- [8] Wilkins RH. Facial nerve decompression for hemifacial spasm. In: Apuzzo ML, editor. *Brain surgery: complication avoidance and management*. New York: Churchill Livingstone; 1993. p. 2115–40.
- [9] Tubbs RS, Loukas M, Shojja MM, Bellevue MP, Cohen-Gadol AA. Surface landmarks for the junction between the transverse and sigmoid sinuses: application of the “strategic” burr hole for suboccipital craniotomy. *Neurosurgery* 2009;65:37–41, discussion 41.
- [10] Jannetta PJ. Supralateral exposure of the trigeminal nerve in the cerebello-pontine angle for microvascular decompression. In: Apuzzo ML, editor. *Brain surgery: complication avoidance and management*. New York: Churchill Livingstone; 1993. p. 2085–96.
- [11] Amador N, Pollock BE. Repeat posterior fossa exploration for patients with persistent or recurrent idiopathic trigeminal neuralgia. *J Neurosurg* 2008;108:916–20.
- [12] Jannetta PJ. Hemifacial spasm. In: Samii M, Jannetta PJ, editors. *The cranial nerves: anatomy, pathology, pathophysiology, diagnosis, treatment*. Berlin: Springer-Verlag; 1981.
- [13] McLaughlin MR, Jannetta PJ, Clyde BL, Subach BR, Comey CH, Resnick DK. Microvascular decompression of cranial nerves: lessons learned after 4400 operations. *J Neurosurg* 1999;90:1–8.
- [14] Masuoka J, Matsushima T, Kawashima M, Nakahara Y, Funaki T, Mineta T. Stitched sling retraction technique for microvascular decompression: procedures and techniques based on an anatomical viewpoint. *Neurosurg Rev* 2011;34:373–80.
- [15] Sindou M, Leston J, Howeydi T, Decullier E, Chapuis F. Micro-vascular decompression for primary trigeminal neuralgia (typical or atypical). Long-term effectiveness on pain; prospective study with survival analysis in a consecutive series of 362 patients. *Acta Neurochir* 2006;148:1235–45.