Chapter 42
Microvascular Decompression for Trigeminal Neuralgia and Other Neurovascular Compression Syndromes

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The most accepted etiology of trigeminal neuralgia is neurovascular compression of the trigeminal nerve root; hence the most definitive cure for trigeminal neuralgia is with microvascular decompression. In this chapter, we describe in detail the anatomy and pathophysiology of trigeminal neuralgia, describe our technique for microvascular decompression, and provide a brief overview of other cranial nerve neurovascular compression syndromes.

Anatomy of the Trigeminal Nerve

The trigeminal nerve (CN V) is the largest cranial nerve, with nuclei extending from the midbrain to the upper cervical spinal cord. It comprises three divisions or branches, one sensory ganglion ("Gasserian" or "semilunar" ganglia), one large sensory root (*portio major*), one motor root (*portio minor*), and four brainstem nuclei. The trigeminal nerve has mixed sensory and motor functions, innervating extensively the skin and mucosae of the head (general somatic afferent), and the muscles derived from the first branchial arch, including the muscles of mastication.

The CN V has four major nuclei in the brainstem. Three sensory nuclei comprise the mesencephalic nucleus (proprioception), the principal sensory nucleus in the pons, and the spinal nuclei (pain and temperature) in the medulla oblongata. The small motor nuclei of CN V are in the rostral pons.

The apparent nerve origin is in the ventrolateral pons. The nerve then occupies the prepontine cistern (this segment is called *plexus triangularis*) and travels anteriorly, and slightly laterally, to pierce the dura (*porus trigeminus*) over the anterior petrous temporal bone and enter Meckel's cave in the middle cranial fossa. Meckel's

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cave is a space lined by the dura mater that connects the middle and posterior cranial fossae and contains CSF (trigeminal cistern), the sensory and motor roots, ganglia, and proximal portions of the three main divisions of the nerve: the ophthalmic (V1), maxillary (V2), and mandibular (V3).

The spatial somatotopic relationship of the three divisions is maintained in its sensory root or *portio major* [1]; therefore, fibers coming from V1 are rostromedial and from V3 are caudolateral. From the brainstem nuclei, 2nd-order neurons ascend to the ventral posteromedial nucleus of the thalamus (3rd-order neuron) as the trigeminal lemniscus (ventral [crossed] and dorsal [uncrossed] trigeminothalamic tract), and then, via the thalamic radiation, terminate in layers 2 and 4 of the somatosensory cortex (4th-order neuron) in the parietal lobe.

Pathophysiology of Trigeminal Neuralgia

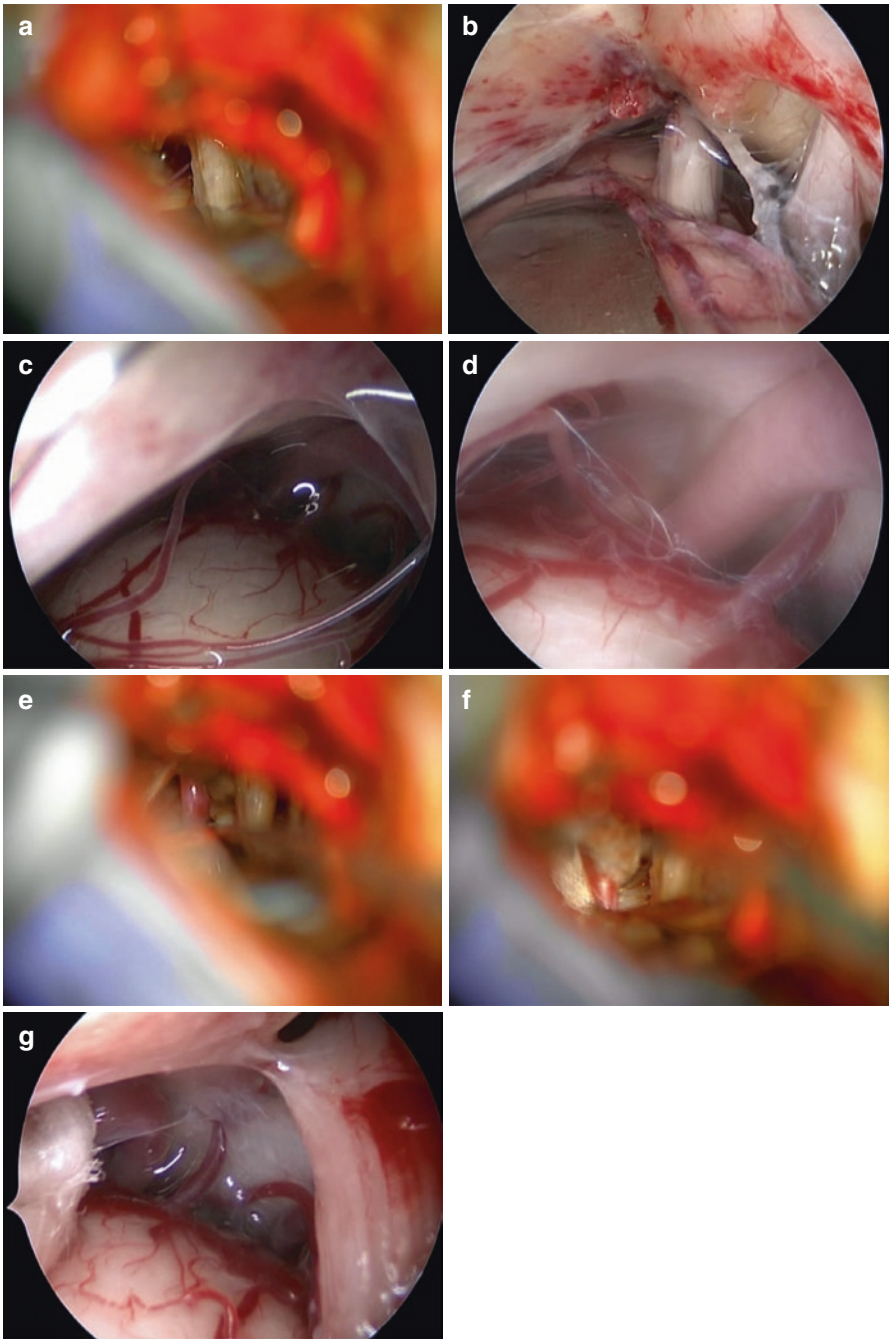
Trigeminal neuralgia (TGN), also known as *tic douloureux*, is an excruciating and disabling facial pain syndrome. The pain in TGN is characterized by recurring bouts of brief unilateral electric shock-like discharges in the distribution of one or more branches of the trigeminal nerve.

TGN is the most common facial pain syndrome and its incidence increases with age. Its annual incidence in the USA is 3.4/100,000 in men and 5.9/100,000 in women [2], and the right side tends to be involved more frequently.

Multiple theories attempt to explain the etiology of trigeminal neuralgia; in general, they can be divided into two main groups: (1) peripheral and (2) central mechanisms.

1. Peripheral: The most widely accepted peripheral mechanism is the neurovascular compression theory. A compressive arterial or venous loop is found in more than 96% of patients with typical trigeminal neuralgia [3, 4]. Compression can occur at any point in the cisternal segment of the trigeminal nerve, between the brainstem and Meckel's cave. The root entry zone or Obersteiner-Redlich zone – where central myelin transitions into peripheral myelin – is particularly vulnerable. The superior cerebellar artery (SCA) is responsible for 88% and the AICA for <25% [4] of cases. The most common intraoperative finding is a rostroventral loop of the superior cerebellar artery (SCA) (Figs. 42.1 and 42.2). As expected

Fig. 42.1 Right-sided microvascular decompression in a patient with trigeminal neuralgia. (**a**) Intraoperative photograph of the right trigeminal nerve. (**b**) Same view with the 0-degree neuroendoscope showing better illumination, wider field, and a more detailed anatomy. The cerebellum is being retracted inferiorly on its superolateral surface to avoid traction injury on cranial nerves VII and VIII. The internal acoustic meatus and VII–VIII cranial nerve complex are seen. It is important to preserve the arachnoidal trabeculae surrounding these nerves. Note the entry of the trigeminal nerve in Meckel's cave. This same corridor can be used to carefully visualize from the foramen magnum to the tentorium. (**c** and **d**) The endoscope is advanced between the tentorium and the trigeminal nerve to reveal the neurovascular conflict, in this case a rostral loop of the superior cerebellar artery. This view is facilitated by the endoscope. (**e**) The SCA loop is removed from under the trigeminal nerve. (**f**) A piece of Teflon felt is placed around the SCA, and the vessel is kept away from the nerve. (**g**) View with the neuro-endoscope showing successful decompression from the root entry zone to Meckel's cave (arrow)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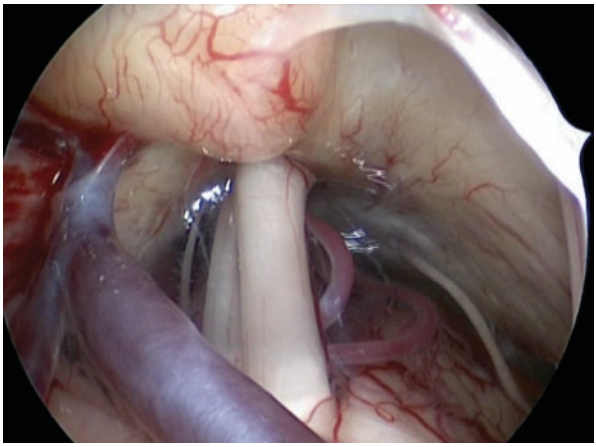

Fig. 42.2 Intraoperative endoscopic view showing a ventral-rostral loop of the superior cerebellar artery under the ipsilateral trigeminal nerve. This is the most commonly encountered neurovascular conflict in trigeminal neuralgia. Note the superior petrosal vein on the left

by the somatotopic arrangement of the fibers in portio major, in most cases (55%), symptoms are transmitted in the territory of V3, and in 32% both V2 and V3 are involved [5]. Venous compression is seen in 8–10% [6] – usually by the transverse pontine vein or the superior petrosal vein or its tributaries, usually near the porus trigeminus. However, it is important to note that not all vascular compression appears to be pathologic. A study using a 3 Tesla MRI found that neurovascular compression of the trigeminal nerve is a common finding, even in asymptomatic individuals [7].

This chronic vascular compression and the pulsatile effect of the vessels on the nerve cause focal demyelination and abnormal remyelination, which result in symptomatic ephaptic neurotransmission. Sensory signals coming from large myelinated sensory Type A fibers are aberrantly transmitted to small nociceptive type C and delta fibers. Therefore, innoxious stimuli such as chewing, teeth brushing, cold, etc. precipitate severe pain attacks.

2. Central: On the other hand, pain from central mechanisms arises from cells in the intra-axial trigeminal nerve pathway secondary to neuronal hyperactivity or focal epileptogenic discharges from these injured cells [8]. These injuries include compression, demyelination, micro-infarction, or infection.

Diagnosis and Classification of Trigeminal Neuralgia

The different treatment modalities for TGN include antiepileptic medications, minimally invasive percutaneous rhizotomies, radiation, and open microneurosurgery. The diagnosis of TGN is clinical, i.e., based on a stereotypical history. According to the International Headache Society [9], the diagnosis of TGN is made if at least three attacks of unilateral pain fulfill the following criteria:

1. No radiation beyond the territory of innervation of the trigeminal nerve
2. Pain with at least three of the following characteristics: (a) recurring pain paroxysms lasting from a fraction of a second to 2 min; (b) severe in intensity; (c) electric shock-like, shooting, stabbing, or sharp quality; and (d) triggered by innocuous stimuli

Burchiel [10] classified patients with trigeminal neuralgia into two types based on predominance of episodic (type I) or constant (type II) pain. Type I patients have a symptomatology more consistent with the "classical" descriptions of trigeminal neuralgia. Most of these patients have an identifiable vascular compression, and, therefore, surgery is very effective, with reported >84% excellent-to-good pain relief [11].

The natural history of trigeminal neuralgia has not been clearly described. The most common progression of symptomatology includes pain attacks becoming more frequent with fewer periods of remission, with a concomitant development of sensory disturbances or dysesthesias. For this reason, it has been postulated that typical trigeminal neuralgia, atypical trigeminal neuralgia, and trigeminal neuropathic pain may represent different degrees of injury to the trigeminal nerve [12]. This might explain why severe nerve damage can often be demonstrated upon exploration of trigeminal nerve in patients with an atypical syndrome.

Differential Diagnosis

Secondary causes of trigeminal neuralgia should be investigated in patients with associated focal neurological deficit, bilateral symptoms, young age, and absence of refractory period in-between attacks. The differential diagnosis of facial pain includes, but is not limited to, temporomandibular joint disease, post-traumatic (invasive dental procedures or fractures) or postherpetic neuralgia, short-lasting unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT), short-lasting unilateral neuralgiform attacks with cranial autonomic symptoms (SUNA), paroxysmal hemicrania, atypical facial pain syndrome, and occipital neuralgia.

Treatment of Trigeminal Neuralgia

Most neurovascular compression syndromes are initially treated medically with anticonvulsant medications, and patients usually respond well. First-line medical treatment is generally considered to be a trial of carbamazepine, gabapentin, or both. Clonazepam, baclofen (typically used in combination with a second drug), and phenytoin or fosphenytoin (in acute attacks) can all be tried in patients refractory or intolerant to carbamazepine or gabapentin.

Surgical treatment is considered when pain relief is inadequate from medications or when doses needed for pain relief are causing significant drug toxicity [13]. Surgical modalities are categorized as "destructive" (ablative) or "physiologic" (microvascular decompression). Ablative options are offered in patients who are not good surgical candidates and involve delivering an injury to the nerve's sensory fibers extracranially (peripheral neurectomy) or intracranially (rhizotomies). Rhizotomies of the preganglionic fibers intracranially can be achieved with one of the following: stereotactic radiosurgery of the nerve root or percutaneous procedures through the foramen ovale (radiofrequency ablation, glycerol injection, or balloon compression). Immediate pain relief as high as 87% has been reported in patients undergoing rhizotomies with 50% incidence of recurrence in 6 months in patients receiving their first balloon rhizotomy and 24 months in patients receiving their first glycerol rhizotomy [14]. Other modalities of treatment include motor cortex and peripheral nerve stimulators. Details on the different treatment modalities for trigeminal neuralgia and other neurovascular compression syndromes are not within the scope of this chapter.

Microvascular decompression has become the gold standard treatment for trigeminal neuralgia and other neurovascular compression syndromes. This approach is the least likely to require additional treatment [15]. Excellent surgical results are reported in the neurosurgical literature, with Barker and Jannetta first reporting a 70% rate of pain relief without medication 10 years after microvascular decompression [3]. In that study, the annual risk of pain recurrence was less than 2% at 5 years and less than 1% at 10 years after MVD. More recent series report long-term significant pain relief in 80–100% of patients if properly selected for surgery [16–18].

Other Vascular Compression Syndromes

These include hemifacial spam, glossopharyngeal neuralgia, intermediate neuralgia, disabling tinnitus, disabling positional vertigo, and medically refractory hypertension. A short overview of these syndromes is given in Table 42.1.

Microvascular Decompression

The surgical management of neurovascular compression syndromes, especially trigeminal neuralgia, has evolved tremendously. Initially, surgery for trigeminal neuralgia consisted of either performing a Gasserian ganglionectomy (Hartley and Krausse in 1892) or sectioning the sensory root "portio major" (Cushing and others in 1928) via a middle fossa subtemporal approach [19]. Years later, Dandy described a "cerebellar" posterior fossa suboccipital approach for partial nerve root sectioning, an approach he pioneered to reduce overall complication rates. Employing this

Table 42.1 Other neurovascular compression syndromes

	Hemifacial spasm	Vestibular paroxysmia	Geniculate neuralgia (*nervus intermedius* or nerve of Wrisberg)	Vagoglossopharyngeal neuralgia
Definition and clinical presentation	Involuntary spasms of the muscles of facial expression. Commonly around the orbit	Acute episodes of vertigo with or without tinnitus and hearing loss	Unilateral paroxysmal otalgia and prosopalgia (referred pain to deep viscero-cranial structures such as nasal cavity, palatal region, and orbits)	Severe and sharp pain in the sensory (GSA) distribution of the glossopharyngeal (CN IX) and vagus (CN X) nerves. The CN IX receives the posterior EAM, tragus, posterior 1/3 of the tongue, soft nasopharynx, tonsillar fossa, mastoid, tympanic membrane, and Eustachian tube. The CN X receives the posterior fossa dura; posterior pinna, external acoustic meatus, and external surface of the tympanic membrane (Arnold's nerve); and larynx. Deadly arrhythmias, cardiac arrest, and syncope can also occur
Differential diagnosis [2]	Cerebellopontine angle tumors and trauma	Meniere's, vestibular migraine, benign paroxysmal positional vertigo, and superior semicircular canal dehiscence	Herpetic ganglionitis (Ramsay-Hunt) and *tic convulsive* (geniculate neuralgia + hemifacial spasm)	Trigeminal neuralgia, superior laryngeal and geniculate neuralgia, Eagle's syndrome, and peritonsillar trauma
Compressive vessel [2]	AICA 43%, PICA 31%, VA 23%, and venous 3%. Multivessel 38%	AICA 75% and venous 10%	AICA [3] but also PICA or branches of the VA [4]	PICA 68%, VA 2%, and venous 6%. Multivessel 23%
Relief from microvascular decompression	>90%	One case reported [5]	72–90% [6, 7]	80–90% [8]
Postoperative complications	Facial palsy, hearing loss, dysgeusia, diplopia (CN IV), lower cranial nerves palsy, middle ear effusion, CSF leak, supratentorial SDH, and posterior circulation infarcts [9]	Imbalance, hearing loss, facial palsy, diplopia, etc.	Facial numbness, facial paresis, sensorineural hearing loss, vertigo, lower cranial nerve palsy, chemical meningitis, CSF leak, and wound infection [4]	Lower cranial nerve palsies, dysphagia, hoarseness, transient hypertension, and CSF leak

"relatively bloodless" operation, Dandy also inadvertently performed the first microvascular decompression by mobilizing arterial loops in contiguity to the nerve that was obstructing his view. He later observed alterations in the shape of the nerve at the point of vascular compression. Based on these direct intraoperative observations, he is credited for being the first to theorize that vascular compression of the trigeminal nerve could be the cause of trigeminal neuralgia. In the 1950s, Gardner at the Cleveland Clinic also hypothesized that gentle compression on the nerve is the pathophysiological mechanism responsible for the pain paroxysms in trigeminal neuralgia [19]. Finally, the microsurgical technique utilized today was developed and perfected by Jannetta, who confirmed this previously mentioned vascular compression in most cases of trigeminal neuralgia with the aid of magnification provided by the neurosurgical microscope.

Preoperative Workup

Neuroimaging is obtained in all patients. Thin-cut high-resolution 3D T2-weighted steady-state free precession sequences are obtained to assess the cisternal segment of the nerve and characterize the neurovascular conflict. In patients with classic medically refractory neuralgia, we advocate for posterior fossa exploration even if no apparent neurovascular conflict is noted in imaging. Very importantly, a volumetric T1-weighted sequence in combination with a TOF MR angiography helps rule out other pathologies, such as extra-axial tumors, vascular malformations, and aneurysms, and also demyelinating plaques [6]. The secondary causes of trigeminal neuralgia should be thoroughly investigated in all patients but especially in those with any associated neurological deficit, bilateral symptoms, young age, and absence of refractory or pain-free period.

Microvascular Decompression Technique

After general anesthesia is induced and the patient is intubated, the body is securely positioned in lateral decubitus using an inflatable bean bag with all pressure points padded in the customary way including the use of an axillary roll. Prior to securing the head to the table with a head holder (we use the Sugita head holder, Mizuho), the head of the patient is slightly rotated to the contralateral shoulder, and the neck is slightly flexed so that the mastoid process is parallel to the floor. It is important to position and tape the ipsilateral shoulder in such a fashion as not to obstruct the movements of the surgeon's hands. Preoperative lumbar drainage is not typically used in our institution but is an acceptable option. Others have described a supine position of the body, back elevated 30 degrees, with the head turned to the contralateral side, and secured in slightly flexed position.

Intraoperative Monitoring

Intraoperative neurophysiologic monitoring is useful in some of the procedures and not utilized for all of the syndromes.

Brainstem auditory evoked responses (BAER) can be used in all of the procedures to monitor for stretch injury to the cochlear nerve from direct manipulation or retraction of the cerebellum.

Vocal cord EMGs are useful when decompressing the lower cranial nerves and medulla. We monitor the vocal cord electromyogram with a nerve integrity monitor system (NIMS, Medtronics).

Facial EMGs are used primarily in cases of hemifacial spasm. The zygomatic branch is stimulated at baseline to look for pathologic responses – after-potentials (from the orbicularis oculi) or lateral spread (from the orbicularis oris). Post-decompression recordings are used to determine the need for further decompression if the pathologic responses do not resolve. Discussion with anesthesiology is important to ensure no long-acting muscle relaxation is used at the time of induction.

Any change in neuromonitoring parameters should prompt a quick response from the surgeon by unlocking and loosening the cerebellar retraction, repositioning the blades, draining CSF, irrigating, or decompressing an expanding hematoma.

Procedure

A point 3 cm posterior to the external acoustic meatus (EAM) in females and 3.5 cm in males is marked (Fig. 42.3). We center our incision at this point in all cases. For aesthetic purposes, we shave only what is necessary for the incision (usually no more than 1 cm behind the hairline). Additionally, the individual osseous and muscular anatomy is studied and major landmarks identified and marked. These include the tip of the mastoid and two very important lines (Fig. 42.3). The first line is measured from the root of the zygomatic process to the inion, across the EAM. The second line is measured perpendicular to the previous line and extends upward from the digastric notch. The point of intersection of these two lines generally correlates to the junction of the transverse and sigmoid sinuses. Our craniectomy is centered slightly caudal and posterior to this point for trigeminal root exposures. For the lower cranial nerves and facial and vestibulocochlear root exposures, the incision is made along the perpendicular line going up from the palpable digastric grove. Frameless neuronavigation can also be used to confirm the location of the major dural sinuses. The superior nuchal line, which usually corresponds to the transverse sinus, and the asterion, which usually corresponds to the transverse-sigmoid sinus junction, can also be used as landmarks.

A small incision is made, oriented obliquely down for decompressing the trigeminal nerve and vertically for decompressing the facial and lower cranial nerves. The scalp and musculature are dissected down using monopolar electrocautery, and

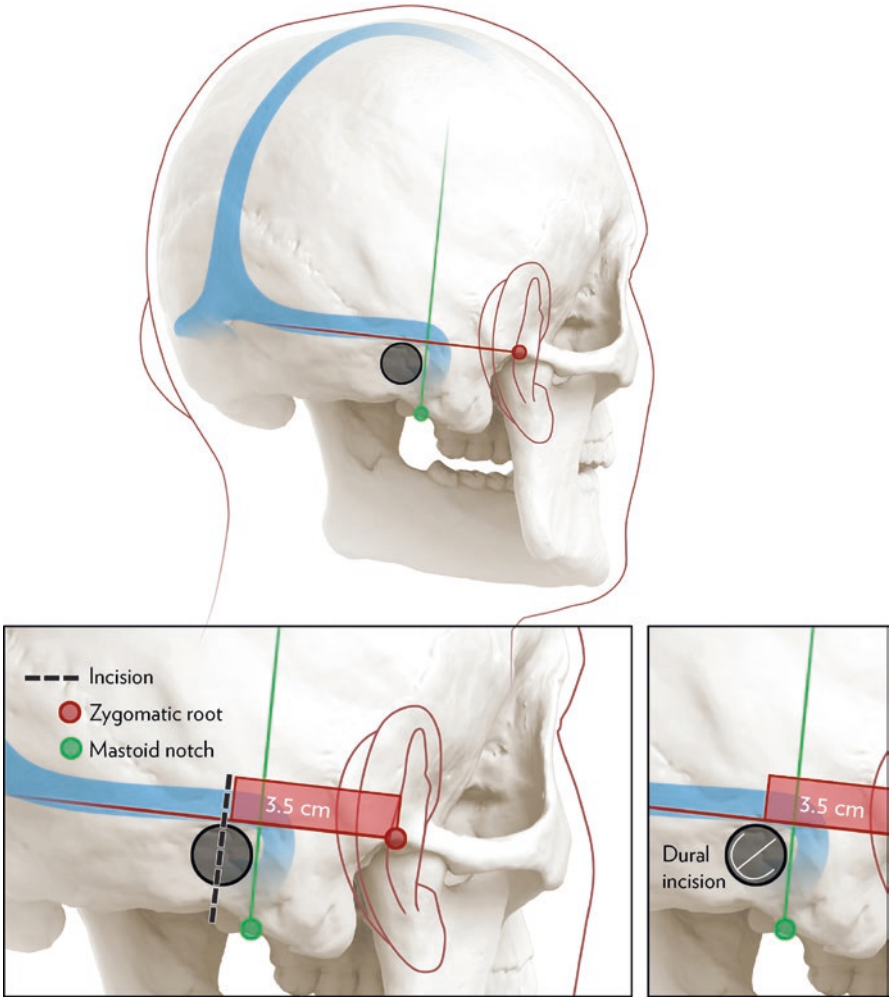

Fig. 42.3 *Upper*: The incision is made 3 cm posterior to the external acoustic meatus (EAM) in females and 3.5 cm in males. The transverse-sigmoid sinus junction is usually located at the point of intersection of two important reference lines. The first line (in red) is measured from the root of the zygomatic process to the inion, across the EAM. The second line (in green) is measured perpendicular to the previous line and extends upward from the digastric notch of the mastoid process. The craniectomy (*bottom left*) is made just caudal and posterior to this intersection when decompressing the trigeminal, facial, and vestibulocochlear nerves and more inferiorly when decompressing the lower cranial nerves and medulla. Once complete hemostasis is achieved, the dura is opened in "C" fashion inferior to the transverse sinus and posterior to the sigmoid sinus, and a T incision made across toward the transverse-sigmoid junction (*bottom right*)

a self-retaining retractor is placed. Care is taken to preserve the greater occipital, lesser occipital, and greater auricular nerves, given that injury to these nerves is the major cause for postoperative pain [20]. These nerves are not always easy to preserve and sometimes require proper recognition and transection to avoid partial

injury and a neuritis. The craniectomy is done slightly posteriorly and inferiorly to the junction of the sigmoid and transverse sinuses for trigeminal neuralgia and more inferiorly for decompression of more lower cranial nerves. A high-speed drill is used initially. A dissector is used to carefully separate the endosteal layer of the dura and dural sinuses from the bone, and the craniectomy is expanded using a Kerrison rongeur. For the trigeminal nerve, the craniectomy is extended as far as the margins of the junction of the transverse and sigmoid sinuses prior to opening the dura. For the lower cranial nerve explorations, the craniectomy is extended anteriorly to the posterior margin of the sigmoid sinus. All exposed mastoid air cells are completely filled with bone wax. Occasionally exposed emissary veins may be either cauterized with bipolar electrocautery or sealed with wax or oxidized cellulose or absorbable gelatin sponge. Profuse bleeding may occur with exposure of an emissary vein, and the surgeon should be prepared for this possibility. Complete hemostasis must be obtained before the dura is opened.

Prior to any dural opening, the anesthesiologist should be reminded during this approach to be sure that the end-tidal PCO2 is less than 35 mm Hg, with the head elevated above the heart.

The dura is opened in "C" fashion inferior to the transverse sinus and medial to the sigmoid sinus, and a T incision made across toward the transverse-sigmoid junction (Fig. 42.3). The dural edges are tacked up, and cerebrospinal fluid is slowly drained from under tentorium with very gentle traction on the cerebellum to achieve cerebellar relaxation. It is essential to patiently release cerebrospinal fluid early on to allow for easier retraction of the cerebellum. Sterilely draped operative microscope is then used for the rest of the operation. A malleable retractor is used to very gently inferiorly retract the superior surface of the cerebellum to visualize the tentorial edge and arachnoid. This arachnoid is then carefully opened sharply under increased magnification being mindful of cranial nerve IV – which in some cases could be mistaken for a thick arachnoid trabecula. Additional release of CSF allows for further relaxation of the cerebellum and better visualization of the petrosal vein and tributaries and ultimately the trigeminal nerve root. Patience and careful use of retraction must be exercised to avoid causing CN VIII traction or avulsion of the petrosal vein and tributaries (usually located in the path of the dissection at the tentorial-petrosal angle) or occasional veins draining into the tentorium. The superior petrosal vein and its tributaries (Figs. 42.4 and 42.5) must be visualized carefully as early as possible and most often may need to be cauterized and transected for best visualization of the medially located trigeminal root. Its tributaries often include the trigeminal vein, vein of the cerebellopontine fissure, pontotrigeminal vein, anterolateral marginal vein, and vein of the middle cerebellar peduncle. These veins can sometimes independently drain into the superior petrosal sinus in the petro-tentorial angle. It is for this reason also that the blade of the retractor should be placed initially laterally along the superior cerebellar surface parallel the tentorium. Then the blade of the retractor is repositioned more obliquely on the superolateral surface of the cerebellum which creates a nice corridor toward the trigeminal nerve. The arachnoid here is also dissected exposing the entire root of the trigeminal nerve. Once the trigeminal root is exposed and in view (Fig. 42.1a), further dissection of the arachnoid over the VII/VIII nerve complex should be avoided as much as

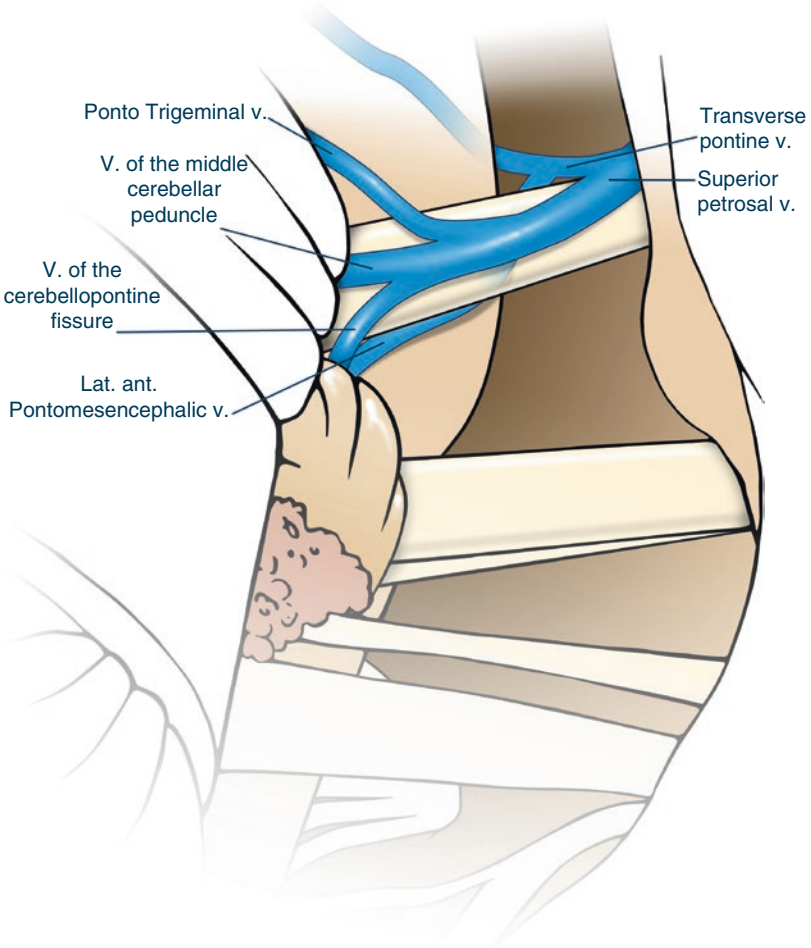

Fig. 42.4 This is an illustration of the right superior petrosal vein and its main tributaries in relationship with the trigeminal nerve. The tentorium was removed

possible to avoid either direct injury or traction on them. Frequently fixed retraction is no longer needed at this point often with the cerebellum relaxed with additional CSF removal.

Neurovascular compression can occur at any point along the nerve – from its root entry zone to Meckel's cave. The superior cerebellar artery (SCA) is the most common cause of neurovascular compression in trigeminal neuralgia (Fig. 42.2). The second segment of the SCA (lateral pontomesencephalic segment) bifurcates in the ambient cistern under the trochlear nerve (CN IV) and loops down near the trigeminal nerve root entry zone [21]. In trigeminal neuralgia, this vessel is usually found looped under the nerve (Figs. 42.1 and 42.2), and treatment consists of carefully mobilizing the SCA loop from under the nerve and

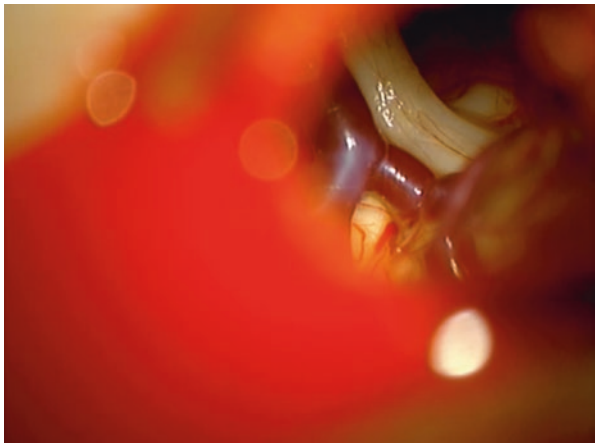

Fig. 42.5 Intraoperative picture illustrating a case of TGN from venous compression. Note the right trigeminal nerve being compressed by the superior petrosal vein and the vein of the cerebellopontine fissure

placing a piece of shredded Teflon felt to tuck the artery away from the nerve (Fig. 42.1). It is our experience that it is best to avoid any contact of the Teflon wool with the nerve root itself (Fig. 42.1g). The offending vessel can also be transpositioned away, in a sling fashion, from the nerve by using sutures [22] or surgical adhesive. The nerve is again inspected thoroughly to make sure there are no other compressive arteries or veins and that the offending vessel has been successfully mobilized.

Another vessel that could be found compressing the trigeminal nerve is the anterior inferior cerebellar artery (AICA). The lateral pontine segment of the AICA travels through the cerebellopontine angle above, below, or in-between the facial and vestibulocochlear nerves. This vessel can loop up and compress the nerve inferiorly (caudally). Less common vascular root compression can be from elongated dolichoectatic vertebral arteries, basilar artery, or both –that typically requires transposition of the arteries toward the clivus. Rarely observed vessels compressing the trigeminal root include pontine arterioles, aneurysms, or vascular malformations. At the time of surgery, it is important to carefully inspect the entire length of the nerve for any of these offending entities under microscopic magnification or using the neuro-endoscope.

A 0 or 30 degrees neuro-endoscope can be used to inspect the trigeminal nerve from the root entry zone to Meckel's cave for compressive vessels when the neurovascular conflict is not obvious (Fig. 42.1) and, at the end of the case, to verify that the nerve has been successfully decompressed.

Venous compression of the trigeminal nerve may also be encountered especially from one of the branches of the petrosal. Classically, the anatomical nomination of posterior fossa veins has been particularly complex. Matsushima and Rhoton [23] divided these veins in four groups: (1) superficial, (2) deep, (3) brainstem veins, and (4) bridging veins or major draining groups. Cerebellopontine angle and rostral pontine veins relevant for this procedure include mostly veins draining into the superior petrosal vein (Fig. 42.4), which then drains into the superior petrosal sinus. Compressive veins can either be bipolar-electrocoagulated and divided if small or

transpositioned if large or if in opposition to the brainstem to avoid complications associated with venous sacrifice – which are rare but can occur in ~ 4.8% [24]. Also keep in mind that it is very important to minimize nerve manipulation to reduce postoperative dysesthesias.

The technique for decompressing the lower cranial nerves is similar. The only variation is the direction of the incision, which is done vertically to allow easier muscle retraction, and obviously, the craniectomy is done more inferiorly.

Once the nerve is decompressed, the subarachnoid cistern is thoroughly irrigated with antibiotic solution and complete hemostasis achieved. The dura is closed in watertight fashion whenever possible. Synthetic dural substitute is layed on top of the durotomy and fibrin glue is applied. Any exposed mastoid air cells should be re-waxed. Bone dust and chips, preserved during the craniectomy portion of the procedure, are packed in a sheet of Surgicel and packed in the craniectomy defect. Burr hole cover is placed on top. The wound is closed anatomically, with attention to watertight musculofascial and skin closure.

Postoperatively, patients are monitored in the stepdown unit overnight and discharged home the next day. If the patient is on carbamazepine or other pharmacotherapy, these are slowly weaned off depending on symptom relief.

Outcomes and Complications

In experienced hands, microvascular decompression surgery is safe and effective, with mortality rates of <0.5% [25] and recurrence rates of 3–15.9% [15, 18] for trigeminal neuralgia. Despite being a safe and effective neurosurgical intervention, there are risks associated with all microvascular decompression surgeries for cranial nerve and brainstem compression hyperactivity syndromes. Procedure-associated risks comprise the standard complications of posterior fossa craniotomies such as cerebrospinal fluid leakage in up to 7% of cases, meningitis and wound infection, pontocerebellar edema, and supratentorial or posterior fossa hematoma and complications directly associated with neurovascular manipulation such as arterial or venous infarction and cranial neuropathies. The most frequent cranial neuropathies include facial numbness in around 4% of the patients, hearing deficits from cochlear nerve retraction while retracting the cerebellum in ~ 10% [26], and diplopia from trochlear nerve injury (usually transient). See Table 42.1 for complications of MVD in the other compression neuropathies.

Conclusions

Trigeminal neuralgia is, and has been historically, one of the most painful illnesses in medicine. Microvascular decompression surgery, done by experienced and caring hands, has proven to be safe and effective in attaining a complete and

definitive relief. Given its good outcomes, this elegant procedure is one of the most rewarding neurosurgical operations. As emphasized here, microvascular decompression is also highly effective for other neurovascular compression syndromes. As a final note, careful patient selection, profound anatomical knowledge, and diligent review of the patient's neuroimaging in preparation for the case are key.

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