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Treatment of trigeminal neuralgia

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Trigeminal neuralgia, also known as tic douloureux, is a clinical syndrome distinguished by brief, repetitive, extremely intense paroxysms of unilateral lancinating facial pain. These paroxysms are confined entirely to one or more divisions of the trigeminal nerve [1,2], with the second and third divisions, either alone or in combination, affected most often. The pain can be triggered by cutaneous stimuli, including those resulting from daily activities such as chewing, talking, brushing the teeth, shaving, or washing the face. A breeze on the face can also trigger severe pain. Patients may experience many attacks daily and, although they are pain-free between attacks, they live in fear of impending pain. Despite their repeated occurrence, the painful spasms occur infrequently at night, and periods of spontaneous remission are common. An accurate history is paramount for proper diagnosis, because physical findings are minimal or absent; few syndromes are as consistent and no other condition has this history. Nonconforming features or presentations should alert the clinician to question the diagnosis. The condition may begin at any age, although it tends to occur more frequently with advancing age and it afflicts women somewhat more often than men.

Many physicians have sought to understand the nature of trigeminal neuralgia and to devise methods to control the pain. Thus, many diverse techniques have been advocated. Historically, however, effective persistent relief has been achieved only by destructive lesions placed within the trigeminal system [3]. Such lesions, which usually produce significant sensory loss, can be associated with bothersome dysesthesia, anesthesia dolorosa, corneal anesthesia, and neuroparalytic keratitis. The latter can result in loss of vision, whereas patients who develop anesthesia dolorosa are in such misery that they are scarcely better than they were with trigeminal neuralgia [1,2,4–8].

The primary treatment for patients with trigeminal neuralgia is medical therapy [9]. Several surgical therapies can be offered to patients when medical therapy is ineffective or associated with significant side effects. These procedures include percutaneous glycerol rhizotomy, radiofrequency rhizotomy, mechanical balloon compression, peripheral nerve section, and microvascular decompression. Most procedures have high rates of initial pain relief and obviate the need for medications. Recently, radiosurgery has been added to the armamentarium of therapies to treat patients with trigeminal neuralgia [10–15]. Surgeons should have the expertise to use different surgical procedures depending on the patient's age, medical condition, pain location, and preference.

Medical therapy

Medical therapy is to be considered the initial treatment of choice before resorting to any surgical alternatives in patients with trigeminal neuralgia. Because of the extreme intensity and brief duration of pain, narcotic analgesics are seldom useful. Carbamazepine (Tegretol, Carbatrol) and oxcarbazepine (Trileptal) are the most effective therapeutic agents. Several other medications, most of which are anticonvulsants, have been used as ancillary drugs and may, on rare occasions, provide an additional measure of control, either alone or in conjunction with carbamazepine or phenytoin [16,17]. These include clonazepam (Klonopin),

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lamotrigine (Lamictal), valproic acid, gabapentin, and baclofen [18–25]. In general, they are not as effective as carbamazepine, oxcarbazepine, or phenytoin but serve as minor agents that may benefit some patients occasionally.

Currently, carbamazepine is the initial drug of choice for the management of trigeminal neuralgia [26–28] because it controls the pain in approximately 90% of patients. Our recommended dosage is 100 mg twice daily with meals, increasing by 100 mg every other day until pain control is achieved or toxicity develops. The gradual increase in dosage allows many patients to tolerate large doses of this medication. Although an average controlling dose is 400 to 800 mg/d, some patients may require, and tolerate, twice this dosage. Response to medication and clinical side effects are the most useful dosing indicators; blood levels do not correlate well with clinical response.

Twenty percent to 40% of patients treated with carbamazepine experience drug-related side effects, including somnolence, dizziness, nausea, and nystagmus. They occur more commonly in the elderly and when the drug dose is increased rapidly. Five percent to 10% percent of patients can experience a rash, erythema multiforme, or, rarely, Stevens-Johnson syndrome. The most common idiosyncratic side effects are hematologic, including neutropenia, thrombocytopenia, and, rarely, aplastic anemia. Less common side effects include hepatotoxicity, hyponatremia, and congestive heart failure.

A baseline complete blood cell count and liver and renal function tests should be obtained before initiating carbamazepine therapy. These studies should be repeated at 2-week intervals initially and then periodically thereafter. Carbamazepine should be discontinued if the peripheral white blood cell count drops below 3000 cells/μL or if side effects become intolerable.

Oxcarbazepine, a derivative of carbamazepine, is a newer drug that is reported to have similar clinical effectiveness but fewer side effects than carbamazepine [29,30]. Because there are fewer side effects, higher doses of oxcarbazepine are often tolerated. Patients with trigeminal neuralgia refractory to carbamazepine have demonstrated a good response when they were switched to oxcarbazepine.

Phenytoin (Dilantin), although somewhat less effective than carbamazepine, may be useful in many patients because it has lower toxicity [31]. Patients who have obtained effective pain relief while on carbamazepine but can no longer tolerate it seem to be the best candidates for phenytoin. Phenytoin can also be useful when used in conjunction with carbamazepine. The dose required to achieve pain control is usually 5 to 7 mg/kg/d. Therapeutic levels of phenytoin (plasma concentration of 10–20 μg/mL) can usually be achieved with the administration of 100 mg three to four times per day after an initial loading dose. Plasma levels are useful for dosage regulation and to avoid toxicity. Because only 25% to 60% of patients achieve satisfactory control [31], phenytoin has not typically been the initial drug of choice.

Phenytoin toxicity may be manifested by nystagmus, ataxia, slurred speech, or mental confusion. A morbilliform rash can commonly occur. Other common side effects include gingival hyperplasia, acne, hirsutism, gastrointestinal upset, and hematopoietic complications. Manifestations of systemic hypersensitivity include Stevens-Johnson syndrome, hepatitis, a lupus-like syndrome, and folate-responsive megaloblastic anemia [9].

Baclofen, a gamma-aminobutyric acid (GABA) agonist, has some efficacy in the treatment of trigeminal neuralgia [24,25,32]. There seems to be a synergism between baclofen and either carbamazepine or phenytoin; therefore, combination therapy in specific cases is a reasonable option [33]. The initial dose is 10 mg three times daily. The dose should be increased incrementally until pain relief is achieved or toxicity is encountered. The typical maintenance dose required in trigeminal neuralgia is 50 to 60 mg/d. Common dose-dependent side effects include somnolence, dizziness, and gastrointestinal distress. Baclofen is typically well tolerated and does not have the potentially life-threatening side effects of carbamazepine or phenytoin.

Clonazepam, a benzodiazepine derivative, has been used in the treatment of trigeminal neuralgia since 1975 [19]. Several clinical trials have demonstrated clinical efficacy in 60% to 70% of patients with trigeminal neuralgia [17,18]. A typical maintenance dose of clonazepam for trigeminal neuralgia is 6 to 8 mg/d. Sedation, which is the major dose-related side effect, limits its usefulness.

Gabapentin (Neurontin) has apparently been widely promoted for the treatment of trigeminal neuralgia, although it is has not been approved by the US Food and Drug Administration for this indication. It has fewer clinical side effects than some of the other anticonvulsants; however, there are no good published studies documenting efficacy. In our experience, its effectiveness in classical trigeminal neuralgia is low, probably less than

10%. It is more useful for deinnervation and dysesthetic pain syndromes.

Surgical therapy

There are two main types of surgical procedures that have proven to be clinically useful: selective percutaneous lesioning of the trigeminal nerve and microvascular decompression of the trigeminal nerve via a posterior fossa craniectomy [4–7,34]. Because of the success of most medical therapies, patients who have become refractory to medical therapy, whose symptoms are incompletely controlled, or who have developed toxicity necessitating discontinuation of these drugs are primarily candidates for surgical therapy.

Percutaneous neurolysis techniques

The percutaneous techniques for the treatment of trigeminal neuralgia produce a partial destructive lesion in the preganglionic trigeminal rootlets (Figs. 1 and 2). Such lesions have been shown to relieve the pain of trigeminal neuralgia for a variable period while usually sparing some trigeminal sensory function. The percutaneous approach to the foramen ovale is a useful and well-tolerated option for elderly patients. These techniques have replaced peripheral nerve sections or injections and intracranial sections, because a more controlled selective partial destruction of the nerve can be produced. The useful percutaneous approaches to lesioning the nerve include radiofrequency thermocoagulation [8,35–39], glycerol chemoneurolysis [5,40–44], and mechanical injury of the nerve using balloon compression [45–48].

Percutaneous procedures, by intent, damage the nerve and produce numbness, which may result in corneal anesthesia and anesthesia dolorosa. In addition, they are temporizing procedures by nature. Recurrence is to be expected, although these procedures can provide years of relief in some patients. They are also safer because they avoid an open craniotomy. Although quite rare, however, lethal complications have also occurred with percutaneous procedures.

Recurrence of trigeminal neuralgia can be treated with a repeat percutaneous procedure, but repetition can cause increased numbness and dysesthesia. Therefore, microvascular decompression is recommended for the initial treatment of younger healthy patients (usually before the age of 65–70 years) with trigeminal neuralgia, and percutaneous procedures are recommended for older patients and those with significant medical problems.

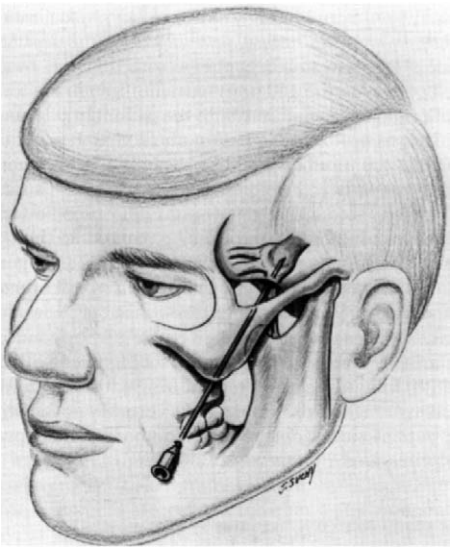

Fig. 1. Placement of the needle through the lateral cheek and into the foramen ovale for percutaneous lesioning of the gasserian ganglion. (*From* Hardy RW. Percutaneous gasserian thermocoagulation in the treatment of trigeminal neuralgia. Cleve Clin Q 1977;44:113–7. Copyright The Cleveland Clinic Foundation.

Radiofrequency lesioning has been used for a longer time than other techniques. It tends to produce a dense lesion that causes more numbness, corneal anesthesia, and anesthesia dolorosa but has a longer duration of effectiveness [8,35,36,38,49]. If physiologic testing is used to try to limit the degree of facial numbness, an awake and cooperative patient is required for the procedure, which may be painful for the patient.

Balloon microcompression reportedly causes less facial numbness, but it often results in temporary trigeminal motor loss. This procedure requires insertion of a larger needle and often evokes a significant trigeminocardiac reflex with bradycardia and profound blood pressure changes. For the latter reason, some advocates of balloon microcompression recommend placing a temporary pacemaker in the patient and using endotracheal anesthesia while performing the procedure [45–48].

In contrast, glycerol chemoneurolysis is a simple easily performed procedure that is well tolerated by the patient. Patients readily accept and request repeat procedures if trigeminal pain recurs. This procedure is less destructive than radiofrequency thermocoagulation and balloon microcompression [4,5,41]. Usually, it produces

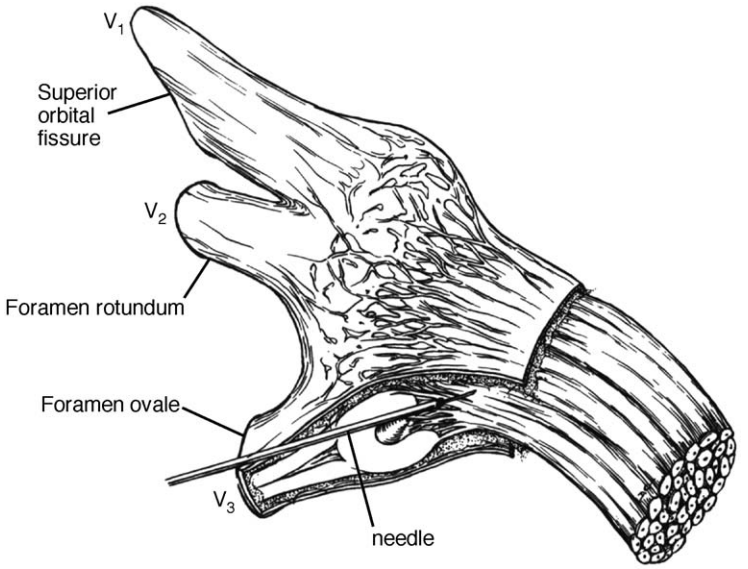

Fig. 2. Trigeminal ganglion, preganglionic rootlets, and postganglionic divisions are shown. The needle is placed through the foramen ovale, through the ganglion, and into the trigeminal cistern containing the preganglionic rootlets. (*From* Ferner H. Die Trigeminuszisterne und ihre praktische Bedeutung fur die Alkoholinjektion in das Gasser'sche Ganglion. Nervenarzt 1949;20:26–9; with permission.)

only mild circumoral numbness, although (as with all these procedures) some patients can experience more numbness, which can be a problem. Corneal anesthesia, although rare, can also occur, but keratitis has not been seen in any of the senior author's patients. Anesthesia dolorosa is also extremely rare. Glycerol chemoneurolysis is the senior author's percutaneous procedure of choice. Although it has a slightly lower long-term success rate than radiofrequency lesioning, it has fewer side effects of trigeminal dysfunction and better patient tolerance.

Percutaneous radiofrequency thermocoagulation

Percutaneous radiofrequency thermal lesioning of the trigeminal nerve was repopularized by Sweet and Wepsic [50] in 1974. They made modifications to earlier gross electrocoagulation techniques of the gasserian ganglion by using short-acting anesthetic agents, electrical stimulation for precise localization, reliable radiofrequency current for precise lesion production, and temperature monitoring to control lesion configuration precisely. This technique is based on the findings that the compound action potentials of nociceptive fibers (A-δ and C fibers) in nerves are blocked at lower temperatures than those of larger A-α and A-β fibers carrying tactile sensations [51].

This procedure can be performed either in the operating room or in the radiology suite. Short-acting sedatives are given intravenously during the procedure. Neuroleptic analgesia can be provided with fentanyl combined with droperidol. Alternatively, methohexital, propofol, or remifentanil can be given. The patient's heart rate, blood pressure, and oxygen saturation are monitored continuously during the procedure.

The patient is placed in the supine position. Using the Hartel technique [52], a standard 100-mm length 18- to 20-gauge needle or cannula with a stylet is inserted in the cheek approximately 2.5 cm lateral to the oral commissure and through the foramen ovale under fluoroscopic guidance (Figs. 3 and 4) [8]. Once the needle is in place, the stylet is withdrawn to check for free flow of cerebrospinal fluid (CSF). Proper placement in the trigeminal cistern usually results in egress of CSF in most patients; however, CSF may not be obtained in patients who have previously undergone a percutaneous procedure.

After the stylet is removed, an electrode is inserted through the cannula. One technique uses a curved electrode tip, which is a coil spring that carries a thermocouple and is used for stimulation and lesion generation [35]. It can be rotated through an axis of 360° for stimulation and lesion

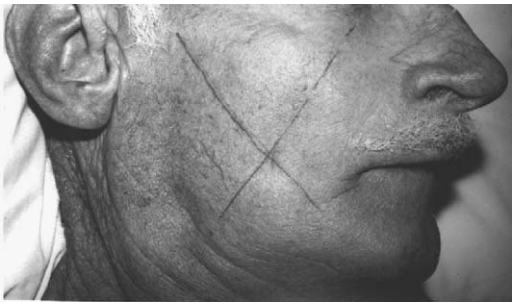

Fig. 3. Guide lines on the patient's face help to orient the needle close to the foramen ovale. The entry site is 2.5 to 3 mm lateral to the corner of the mouth. Two guide lines through this point are drawn, one to a point one third of the way from the external auditory canal to the lateral canthus of the eye and another toward the medial side of the iris of the eye. A needle kept perpendicular to both of these lines will arrive at the skull base in close proximity to the foramen ovale. Final placement through the medial end of the foramen is performed using a fluoroscope. (*From* Apfelbaum RI. Glycerol trigeminal neurolysis. Tech Neurosurg 1999;5:225–31; with permission.)

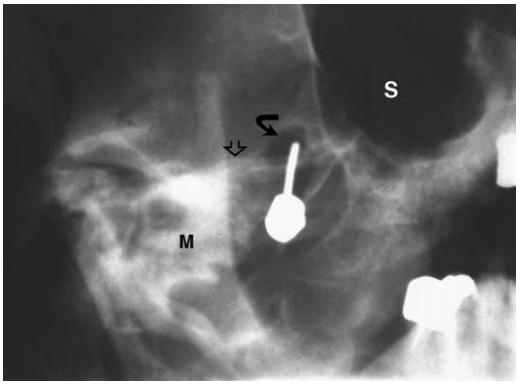

Fig. 4. Fluoroscopic view of a right-sided percutaneous approach to the foramen ovale. The patient's neck is hyperextended, and the head is rotated to the contralateral side about 15° to 20°. This allows fluoroscopic visualization directly along the needle pathway, with the foramen ovale (*curved arrow*) seen projecting over the petrous ridge (*open arrow*). Note that the foramen ovale is punctured at its medial end to enter the trigeminal cistern properly. S, maxillary sinus; M, mandible. (*From* Apfelbaum RI. Glycerol trigeminal neurolysis. Tech Neurosurg 1999;5:225–31; with permission.)

production. Stimulation is used to localize the appropriate divisions of the trigeminal nerve, adjusting the position of the electrode as necessary. Proper localization is achieved when the patient perceives a nonpainful vibratory or paresthetic sensation in the appropriate division at a threshold of less than 0.4 V (50 Hz, 2.5-millisecond continuous pulse train). The radiofrequency current is then placed on the electrode, which raises the temperature of the electrode tip to a predetermined level, producing some thermocoagulation of the preganglionic trigeminal nerve fibers. Using this technique, heating to a predefined temperature for 45 to 60 seconds is performed after transiently deepening the anesthesia. Alternatively, some surgeons prefer to use a thin wire electrode without temperature monitoring (see Fig. 2). The exposed portion of the electrode can be bent to localize the electrode position within the preganglionic fibers, using stimulation as described previously. Using small, carefully applied, graded increments of heating and repeatedly testing the patient, the surgeon can then usually remove pain perception, while some useful touch is preserved in the treated area, because the thin unmyelinated pain fibers are more sensitive to thermal destruction than the larger myelinated touch fibers.

With either technique, the lesion occasionally spreads to adjacent divisions of the trigeminal nerve, producing a larger area of numbness than desired. After each incremental lesion, the patient is allowed to awaken from the anesthesia and is re-examined. The procedure is terminated when the patient develops dense hypalgesia but not anesthesia in the primarily affected division, especially over the trigger zone, and when touching the trigger zone cannot reproduce trigeminal pain [35,49]. After recovery from anesthesia, patients may resume full activity and a regular diet. They are usually discharged after an overnight hospital stay.

This procedure is well tolerated by elderly or medically debilitated patients. Pain is immediately relieved in 99% of patients [35]. The rate of pain recurrence is approximately 15% to 20% over 10 to 15 years [36]. Patients must be aware that this procedure permanently alters facial sensation, producing significant numbness in 90% of cases, and that it may produce corneal anesthesia if the first division is affected or the lesion spreads to involve that division. In a review of 500 patients by Taha and Tew [49], 9% of patients described an intermittent crawling, burning, or itching sensation that did not require treatment, 2% complained of numbness that required treatment, 0.2% developed anesthesia dolorosa, and less than 1% developed neurogenic keratitis or

corneal abrasions. Other complications, such as ocular nerve injury, carotid artery injury, seizures, meningitis, stroke, intracranial hemorrhage, and death, have been reported but are rare [53].

Postoperative dysesthesias are the major adverse effects experienced by patients who have undergone percutaneous radiofrequency thermocoagulation [54]. Patients who suffer from analgesia dolorosa or anesthesia dolorosa are bothered by constant and severe burning, itching, or crawling sensations, which they may find as intolerable as their initial trigeminal neuralgic pain. Unfortunately, these sensations are often refractory to treatment, although some patients respond to a combination of perphenazine and amitriptyline.

The incidence of postoperative dysesthesias has largely declined after the technique modifications described by Tew and Taha [55]. Some of these modifications include using the curved electrode, which allows close contact with the involved sensory fibers enabling selective lesioning; continuous sensory examinations during lesion making; asking the patient whether facial numbness is tolerable during the procedure; and quantitating the numbness by asking the patient to compare the pinprick sensation on the treated side with that on the untreated contralateral side [35].

Percutaneous glycerol chemoneurolysis

Various chemical agents have historically been used as neurolytic agents to treat trigeminal neuralgia. Most chemical agents, such as phenol and ethanol, are strong neurolytics and result in a dense lesion with significant deafferentation [5]. Percutaneous chemoneurolysis with glycerol was introduced in 1981 by Hakanson [56]. Glycerol, a mild neurolytic, provides excellent pain relief while largely sparing trigeminal nerve function in most patients. It is not known whether the glycerol works by direct chemical action or by hyperosmotic damage to the nerve. For successful lesioning, pure anhydrous (99.5%) glycerol is instilled into the trigeminal cistern. Although this form is not currently commercially available as a pharmaceutic product, most hospital pharmacies can package pure laboratory reagent grade anhydrous glycerol in small rubber-stoppered bottles and sterilize them for use.

The procedure is performed in the radiology suite using the same technique for puncturing the foramen ovale under direct fluoroscopic guidance that is used for radiofrequency lesioning [5,52]. The entry site, approximately 2 to 3 cm from the corner of the mouth (see Fig. 3), is selected fluoroscopically by placing the patient with the neck hyperextended and the head rotated to the contralateral side about 15° to 20°. This allows fluoroscopic visualization directly along the needle pathway, with the foramen ovale seen projecting over the petrous ridge.

Needle puncture and advancement through the foramen ovale are accomplished with the patient briefly anesthetized using methohexital, 40 to 60 mg. Other short-acting agents, such as propofol, fentanyl, or remifentanil, can also be used. It is important that the foramen ovale be punctured at its medial end to enter the trigeminal cistern properly (see Fig. 4). Once the needle is in place and a free flow of CSF is obtained after removing the stylet, the patient is allowed to awaken. The patient is then placed in the vertical sitting position by putting the floorboard under the patient's buttocks and then tilting the x-ray table 90° (Fig. 5). A cisternogram is performed by slowly injecting iohexol (Omnipaque, Amersham Health, Princeton, New Jersey, 300 mg%) contrast agent into the needle. If the needle is properly placed, the contrast fills the small cup-shaped trigeminal cistern and then overflows into the posterior fossa under fluoroscopic visualization in the anteroposterior projection (Fig. 6). The cisternogram confirms the correct placement and allows quantification of the size of the trigeminal cistern.

The patient is then tilted back into the supine position to allow the contrast agent to flow out of the cistern, which is confirmed fluoroscopically. A gentle flush with preservative-free saline can be used to wash out the remainder of the contrast. The patient is transferred to a hospital bed and placed back in the sitting upright position. A quantity of glycerol equal to the volume of the cistern is instilled slowly (Fig. 7). This maneuver may produce trigeminal pain, so it is best to premedicate the patient with analgesics. The patient must then be kept sitting up at all times for about 2 hours to keep the glycerol in the trigeminal cistern. Most patients are pain-free within a few hours and may be discharged the following morning.

Ninety percent of patients experience good relief from a single injection. Some may experience a lesser degree of trigeminal pain for 7 to 10 days before the onset of complete pain relief. If a patient's pain continues beyond 7 days, a repeat procedure is recommended. This strategy is effective in most patients. For those who experience

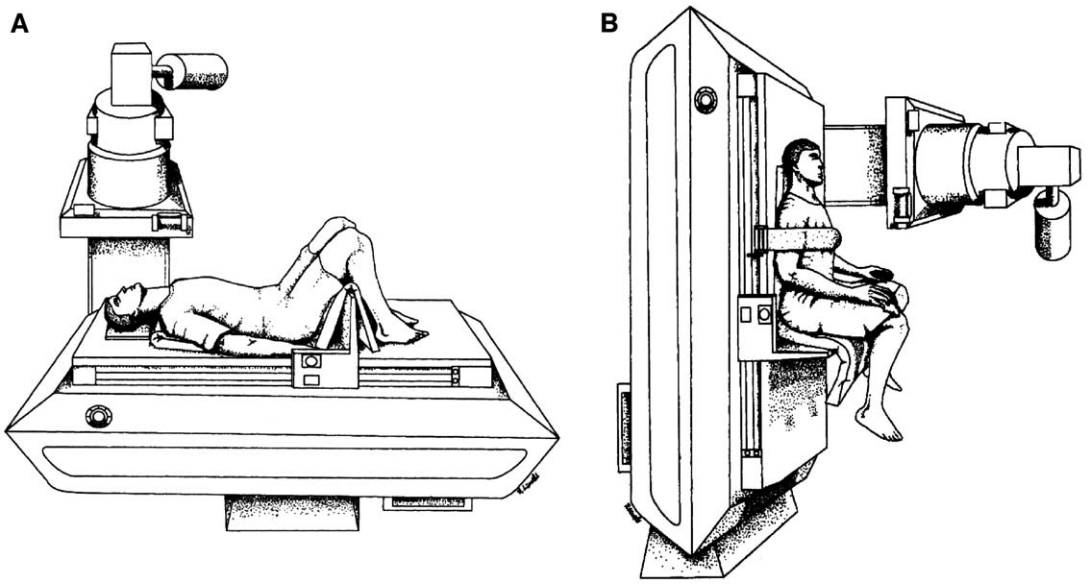

Fig. 5. (*A*) The patient is initially in the supine position with the neck hyperextended and turned to the contralateral side for insertion of the needle under fluoroscopic visualization. (*B*) After the needle is in place, the patient is then placed in the vertical sitting position by placing the footboard under the buttocks and then tilting the x-ray table 90°. (*From* Apfelbaum RI. Glycerol trigeminal neurolysis. Tech Neurosurg 1999;5:225–31; with permission.)

recurrence, the procedure can be readily repeated with good tolerance.

Sensory loss is variable. Most patients report mild numbness, usually in the circumoral region. They can distinguish even light touch and appreciate a difference in sensation between the two sides of the face. This degree of sensory deficit is well tolerated and patients quickly adapt to this change. Some may achieve good pain relief without detectable facial numbness. Others may experience a more profound sensory loss, but this is infrequent. Any numbness that occurs tends to

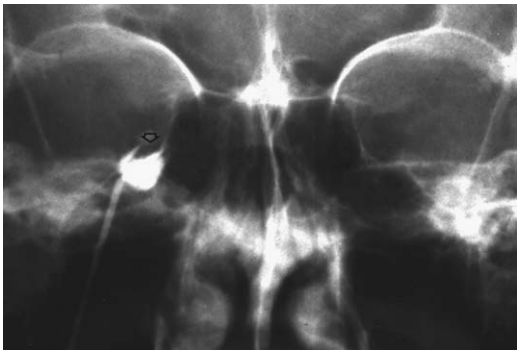

Fig. 6. While the patient is in the upright position, a cisternogram is performed outlining the trigeminal cistern. Note that even with the needle at the medial end of the foramen ovale, the needle is near the lateral extent of the cistern. The filling defect dorsally (*arrow*) in the cistern is the trigeminal root. (*From* Apfelbaum RI. Glycerol trigeminal neurolysis. Tech Neurosurg 1999;5:225–31; with permission.)

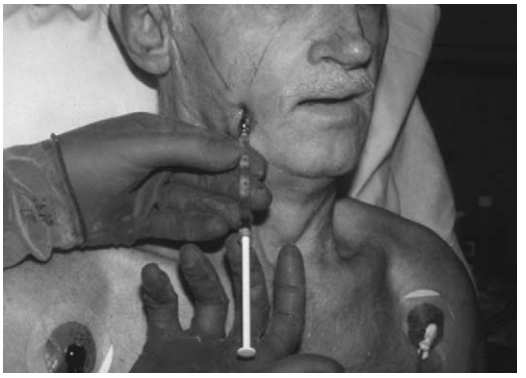

Fig. 7. After the patient is transferred to a hospital bed and placed in the sitting upright position, a quantity of glycerol equal to the volume of the trigeminal cistern is instilled slowly. The patient must then be kept sitting up at all times for about 2 hours to keep the glycerol in the trigeminal cistern.

fade slowly during a period of several months to several years. In a small number of patients, corneal anesthesia may occur with or without dense facial numbness. These patients are cautioned to check their eyes daily for signs of irritation and are urged to make this a routine along with other daily hygiene care, such as dental cleaning. If patients note irritation, they are advised to seek immediate ophthalmologic evaluation.

In the senior author's experience with 303 patients who underwent percutaneous glycerol chemoneurolysis for trigeminal neuralgia, 181 patients received one injection, 83 received two, 19 received three, and 20 received four or more injections (average, 1.55 injections per patient). The average time to recurrence after the first treatment was 21 months, whereas the average time to recurrence was 16 months with subsequent treatments. Seventy-two percent of the patients experienced at least 3 years of relief, and 60% remained pain-free after one injection for more than 10 years. The success rate for a second injection is identical, but effectiveness does subside somewhat for subsequent injections. For many patients, however, this treatment is quite effective.

About 3% of patients experience dysesthesias, which are usually mild and often self-limited. Anesthesia dolorosa is extremely rare and has occurred in only one patient in the senior author's series.

Complications other than trigeminal sensory loss and its associated sequelae are fortunately rare. Two patients had meningeal infections recognized by fever within 16 hours of the procedure and diagnosed by lumbar puncture. Proper treatment prevented any permanent sequelae in those two patients. Because of this potentially serious complication, patients are kept overnight in the hospital after glycerol chemoneurolysis.

Percutaneous balloon compression

Percutaneous balloon compression of the gasserian ganglion with a balloon catheter was introduced by Mullan and Lichtor [57] in 1983 as a technique to traumatize the trigeminal ganglion and preganglionic rootlets mechanically using a percutaneously inserted balloon-tipped catheter. This requires a larger needle (14-gauge), which is placed at the external end of the foramen ovale but not within the foramen. The catheter is threaded through the foramen ovale and into the trigeminal cistern. It is then inflated using a radiopaque contrast agent to a predetermined pressure to compress the neural structures. The technique is based on the observation that mechanical trauma could relieve the pain of trigeminal neuralgia, often for a significant period. Histologic studies have shown that compression selectively injures the large myelinated fibers that mediate light touch and preserves the small unmyelinated fibers that mediate pain sensation [45]. This probably reduces sensory input to the nerve, turning off the trigger to trigeminal pain.

Percutaneous balloon compression is particularly effective for first-division trigeminal neuralgia because of its low risk of corneal anesthesia and because unmyelinated fibers that control the corneal reflex are not injured by compression. It is less likely to cause corneal anesthesia because it does not selectively impair A-δ and C fibers, as does the radiofrequency thermocoagulation technique. This procedure is also good for patients with second- or first-division pain who have not responded to previous percutaneous procedures and want to pursue another percutaneous technique.

The procedure is performed in the radiology suite with the patient under general endotracheal anesthesia. An external pacemaker is used to control the bradyarrhythmias that sometimes occur during balloon compression. This also provides an additional monitor for successful injury of the nerve. Digital pressure monitoring provides additional control of the extent of nerve injury. The goal is to compress the nerve at 1100 mm Hg or 1.3 to 1.5 atmospheres. Corneal injury is unlikely at this pressure, as is severe numbness.

In a review of 183 patients who underwent percutaneous balloon compression for trigeminal neuralgia, 93% achieved pain relief, 61% had facial numbness (80% mild, 14% moderate, and 6% severe), and 19% had minor jaw muscle weakness that resolved within 3 to 12 months. Anesthesia dolorosa did not occur in any patient in this series. The overall occurrence rate was 25%, and 68% of patients who underwent a repeat compression for recurrence achieved lasting pain relief [45].

Microvascular decompression

Microvascular decompression is an effective treatment for trigeminal neuralgia [6,7,34,58]. This operation is based on the observation made by Dandy [59] that the cause of trigeminal neuralgia is compression of the trigeminal nerve at its root entry zone adjacent to the brain stem (except in patients with multiple sclerosis [MS],

who have a demyelinating plaque in the same area). The usual cause of this compression is an aberrantly located and elongated arterial loop; however, venous channels and tumors have also been encountered (Fig. 8). Jannetta and his colleagues [34,58,60,61] devised an operative procedure that involves a limited retromastoid craniectomy and microsurgical techniques. This approach allows dissection at the root entry zone of the trigeminal nerve and displacement of the offending vascular structure, usually by the insertion of a small synthetic sponge prosthesis interposed between the nerve and artery.

The advantages of microvascular decompression over the other percutaneous treatments are that it treats the primary etiology of the disease; the trigeminal nerve is preserved and not damaged; deinnervation sequelae, such as facial numbness and dysesthesia, are avoided; and it has a lower rate of recurrence over long-term follow-up. Nevertheless, microvascular decompression incurs the risk of an open surgical procedure.

The choice of treatment modality should be made by an informed patient and the ability to tolerate an open surgical procedure under general endotracheal anesthesia. The key to this decision should involve consideration of the patient's age, associated illnesses, and assessment of the risks that the patient is willing to assume. Younger patients have a better chance of tolerating an open procedure without complications. Because they have a longer life expectancy, they have a higher risk of recurrence from percutaneous procedures and thus have increased cumulative effects with regard to deinnervation. Older patients have increased risks of complications from open surgical procedures and have a shorter remaining life expectancy. Therefore, they are likely to require fewer repetitions of percutaneous procedures with less cumulative trigeminal deinnervation. In our practice, we recommend microvascular decompression for younger patients (generally, age less than 65–70 years) who are in good medical condition.

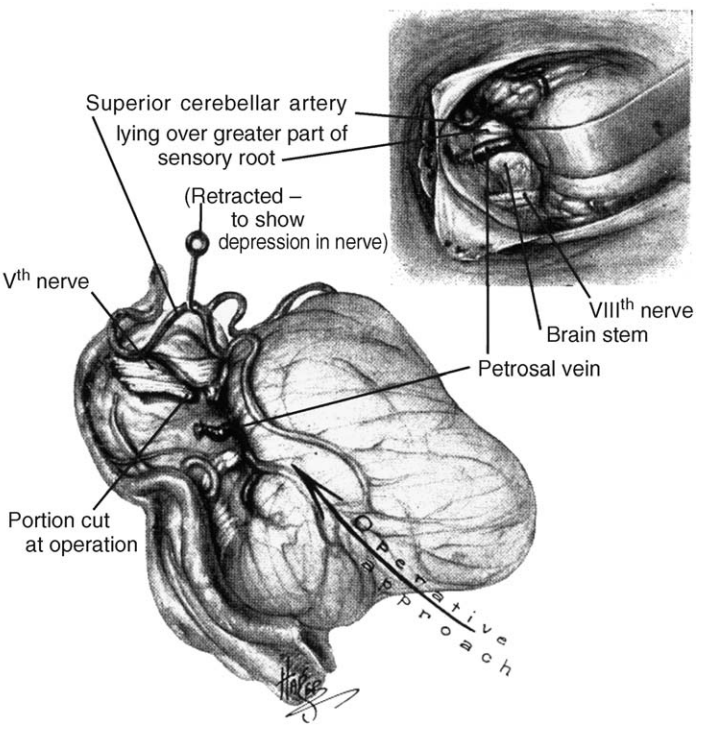

Fig. 8. Illustration demonstrating Dandy's operative approach for partial sectioning of the trigeminal sensory root. Note the vascular compression of the trigeminal nerve adjacent to the brain stem by the superior cerebellar artery. (*From* Dandy WE. The brain. In: Walters W, Ellis FH, Jr, editors. Lewis-Walters practice of surgery. Hagerstown, MD: WF Prior Co.; 1963. p. 1–671.)

The procedure involves a limited suboccipital retromastoid craniectomy performed under general anesthesia [6]. In the senior author's practice, a sitting position is preferred for this operation (Fig. 9); however, equally satisfactory results can be achieved using a lateral position. Because of the risk of air embolization when the sitting position is used, we employ a Doppler precordial detector and an end-tidal carbon dioxide monitor to detect minute amounts of air. This early detection allows the anesthesiologist to raise the venous pressure and prevent the further entrainment of air, avoiding the serious complication of massive air embolization.

Access to the trigeminal nerve is achieved by placing the craniectomy just below the transverse sinus and just medial to the sigmoid sinus (Figs. 10–12). Opening the dura close to these venous sinuses allows exposure of the cerebellopontine angle along the superior lateral margin of the cerebellum. The petrosal vein is usually coagulated and divided to gain access to the region of the trigeminal nerve, and the arachnoid around the nerve is opened widely to inspect this area fully.

Elongated arterial loops impinging on and cross-compressing the root entry zone of the trigeminal nerve are the most common findings in patients undergoing this operation [61]. Occasionally, venous channels impinging on the nerve in a similar manner are found. They can be coagulated and divided to decompress the root entry zone of the nerve. Arterial channels are dissected completely free of the root entry zone and secured with a small plastic sponge prosthesis, usually Ivalon or a shredded Teflon sponge (Figs. 13 and 14). The goal is to redirect the arterial pulsation away from the root entry zone.

After satisfactory decompression, the dura is closed in a watertight fashion and the wound is closed in layers. We routinely place patients on steroids before surgery and for 24 hours after surgery. Most patients tolerate this procedure well and are able to begin oral intake and to get out of bed on the first postoperative day. If patients are operated on in the sitting position, they usually have moderate postoperative headache, which can be controlled with oral analgesics. Most patients can be discharged 3 to 5 days after surgery.

In the senior author's personal series of more than 500 patients treated with microvascular decompression, the pain of trigeminal neuralgia was fully relieved in 91% and reduced in another 6% of the patients [7]. Recurrences of severe pain refractory to treatment occurred in approximately 1% of our patients per year. At a 14-year follow-up, 81% remained with good pain control. Thus, unlike percutaneous lesioning, a steady increase in the frequency of recurrences with time has not been observed with microvascular decompression. Significant complications have included cerebellar hematomas (1.2%), supratentorial strokes (0.6%), transient cranial nerve palsies (up to 3%), and unilateral hearing loss (3%). Five deaths have occurred in this series.

Microvascular decompression thus should not be undertaken lightly and requires special micro-

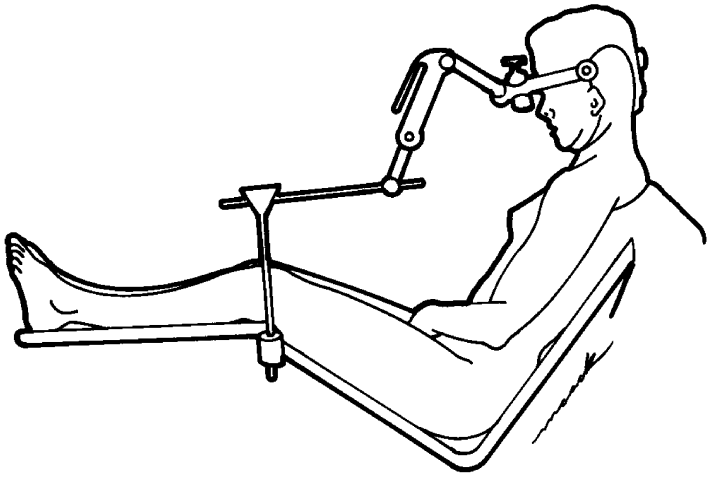

Fig. 9. Illustration of the sitting position used for the retromastoid microvascular decompression procedure. (*From* Apfelbaum RI. Microvascular decompression of the trigeminal nerve. In: Wilson CB, editor. Neurosurgical procedures: personal approaches to classic operations. Baltimore: Williams & Wilkins; 1992. p. 137–53; with permission.)

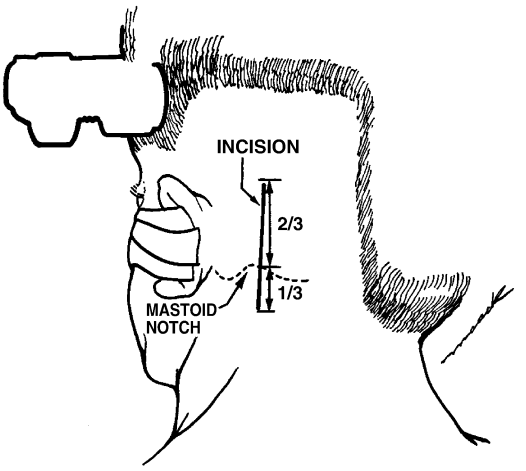

Fig. 10. Location of the surgical incision. The external ear is taped forward, and a vertical paramedian incision is made 3 to 5 mm medial to the mastoid notch with two thirds of the incision above the notch and one third below. (*From* Apfelbaum RI. Microvascular decompression of the trigeminal nerve. In: Wilson CB, editor. Neurosurgical procedures: personal approaches to classic operations. Baltimore: Williams & Wilkins; 1992. p. 137–53; with permission.)

surgical skill and training. Although it offers major advantages by relieving the pain without sacrifice of trigeminal function (no numbness, dysesthetic sequelae, or corneal anesthesia occurs), it does carry with it a small chance of serious or even lethal complications.

Trigeminal rhizotomy

Sectioning of the trigeminal root between the brain stem and gasserian ganglion through a subtemporal approach was first reported by Horsley et al [62] in 1891. Dandy [63] later modified this to a posterior fossa approach with sectioning of the sensory root of the trigeminal nerve at the pons (Fig. 15). As his experience grew, he resorted to partial sectioning of the nerve. Trigeminal rhizotomy fell out of favor with the popularization of percutaneous destructive approaches, however.

In a small number of patients who have undergone microvascular decompression (3% in the senior author's experience), a compressing vascular loop may not be found [2]. On encountering a negative exploration, one can either close and subsequently do a percutaneous lesioning procedure or do a partial section of the nerve. This, of course, requires prior discussion of the possibilities with the patient. We have adopted Dandy's approach in the belief that it is most prudent to try to salvage a benefit from the procedure. In other patients with known demyelinating diseases, posterior fossa trigeminal rhizotomy should be considered when percutaneous techniques fail to give adequate relief.

Partial sectioning of the posterior half of the nerve can provide excellent long-lasting relief of trigeminal pain while preserving most of the touch sensation, sparing corneal sensation, and avoiding neuroparalytic keratitis. It should be considered

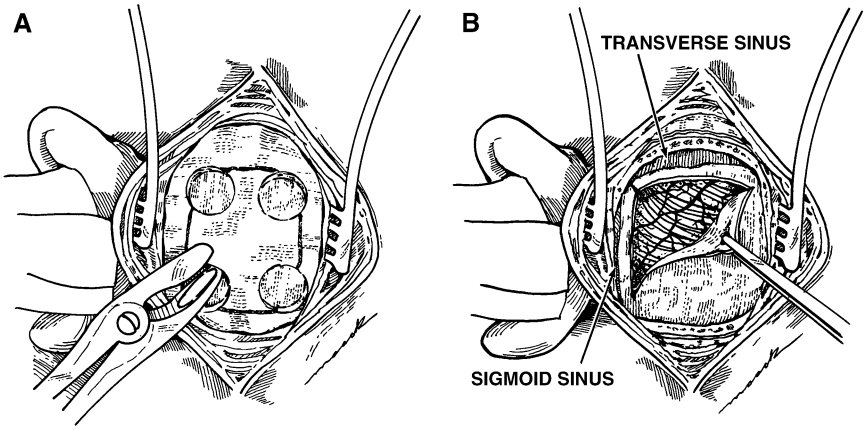

Fig. 11. (*A*) A retromastoid craniectomy is created by placing burr holes, which are then enlarged with a rongeur to create a circular craniectomy. (*B*) Inverted L-shaped dural incision for a left-sided approach. Note the location of the transverse and sigmoid sinus at the superior and lateral margins of the craniectomy. Tenting sutures are placed in the superior and lateral margins of the dura to expand the exposure. (*From* Apfelbaum RI. Microvascular decompression of the trigeminal nerve. In: Wilson CB, editor. Neurosurgical procedures: personal approaches to classic operations. Baltimore: Williams & Wilkins; 1992. p. 137–53; with permission.)

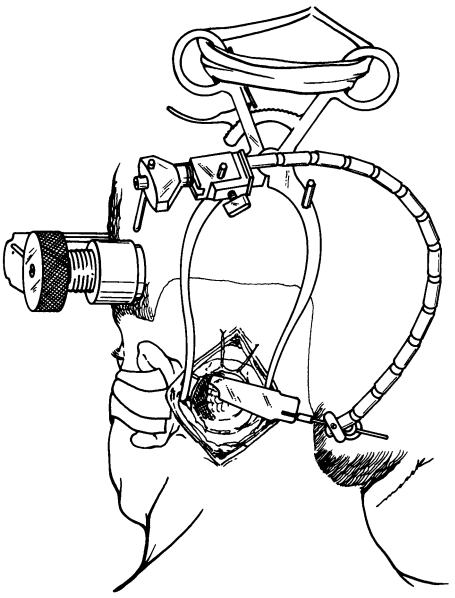

Fig. 12. Position of the self-retaining retractor. Note fixation of the retractor base to the drapes via an encircling gauze sponge to provide firm three-point fixation for the retractor base. The notched blade of the retractor serves to protect the seventh and eighth cranial nerves. (*From* Apfelbaum RI. Microvascular decompression of the trigeminal nerve. In: Wilson CB, editor. Neurosurgical procedures: personal approaches to classic operations. Baltimore: Williams & Wilkins; 1992. p. 137–53; with permission.)

an appropriate strategy in patients in whom a negative or equivocal exploration is encountered during a microvascular decompression. It is equally effective in patients who have demyelinating or neurodegenerative disease causing their trigeminal neuralgia and in whom less invasive procedures (percutaneous lesioning or radiosurgery) have failed to give adequate relief. It may serve as a last line of defense for the patient who has proven to have a recalcitrant form of trigeminal neuralgia and has failed treatment with other more commonly used modalities.

In the senior author's experience, 40 patients who had a negative exploration during microvascular decompression underwent partial sectioning of the trigeminal nerve [2]. Approximately one third to one half of the nerve adjacent to the brain stem was sectioned, starting at its posterior inferior margin. After initially incising the pia with sharp microscissors, the resection can be deepened with a small microhook in a fashion similar to that used by Dandy.

Eighty percent of patients achieved excellent (complete) relief of trigeminal pain, and 5% achieved good reduction of pain to medically controllable levels. Five of the other six patients had additional percutaneous lesioning, two by radiofrequency and three by glycerol; four obtained excellent relief, and one required medication to control the pain. Others have also reported

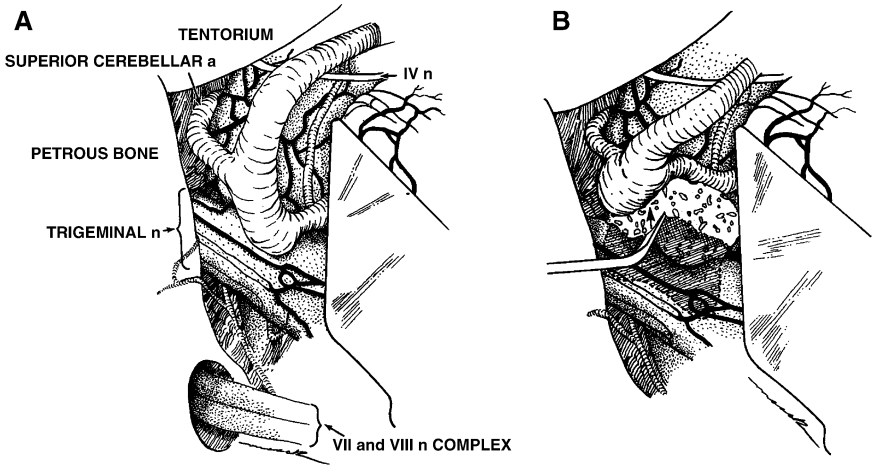

Fig. 13. Illustration of a left trigeminal nerve decompression. (*A*) The superior cerebellar artery is shown indenting the superior aspect of the trigeminal nerve adjacent to the brain stem. (*B*) Teflon felt is interposed between the artery and the nerve to elevate the superior cerebellar artery to a horizontal course. The thrust of the arterial force vector is redirected away from the trigeminal nerve to decompress the root entry zone. (*From* Apfelbaum RI. Microvascular decompression of the trigeminal nerve. In: Wilson CB, editor. Neurosurgical procedures: personal approaches to classic operations. Baltimore: Williams & Wilkins; 1992. p. 137–53; with permission.)

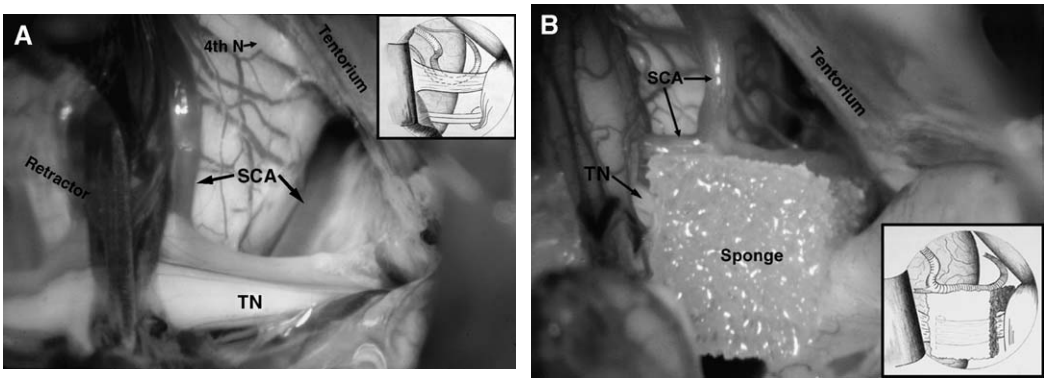

Fig. 14. (*A*) Intraoperative photograph showing an elongated loop of superior cerebellar artery compressing the trigeminal nerve at the root entry zone. (*B*) An Ivalon prosthesis has been inserted between the artery and vein. It sits as a saddle over the nerve to secure it in place. Note the redirection of the artery to a horizontal course.

similar results with posterior fossa exploration with partial trigeminal rhizotomy [64].

In rare cases, we have encountered vascular structures that could not be displaced to decompress the nerve; thus, partial section of the nerve was performed. In one case, an artery intrinsic to the nerve was found, and in another, a persistent trigeminal artery perforated through the nerve.

These results support Dandy's observations and suggest a role for partial trigeminal rhizotomy when intrinsic neural compression is not found during an attempted microvascular decompression.

Stereotactic radiosurgery

Stereotactic radiosurgery is another treatment option for patients with trigeminal neuralgia [10–14,65–73]. It has been used as the first procedure in selected patients of advanced age or poor clinical condition, in those receiving anticoagulation therapy, and in those who refuse or are poor candidates for a surgical procedure [70]. Because radiosurgery does not reliably relieve trigeminal neuralgia immediately, patients with acute severe trigeminal pain are not good candidates for the procedure. In this situation, percutaneous procedures should be considered if the patient is not a candidate for microvascular decompression. Patients should be informed of the potential risk of delayed facial numbness after radiosurgical treatment.

In 1971, Leksell [74] used stereotactically focused radiation to injure the trigeminal ganglion or sensory root partially to treat trigeminal neuralgia in a small number of patients. Since then, several groups have demonstrated the efficacy and safety of gamma knife stereotactic radiosurgery for trigeminal neuralgia [11–13,65,66]. Their target has been the root of the nerve adjacent to the brain stem in most cases. In 1996, Kondziolka et al [73] reported a nonrandomized multicenter study of 50 patients treated with gamma knife radiosurgery to the proximal trigeminal nerve near the pons. The target dose varied from 60 to 90 Gy, and the median time to pain relief was 1 month. After an 18-month median follow-up (range: 11–36

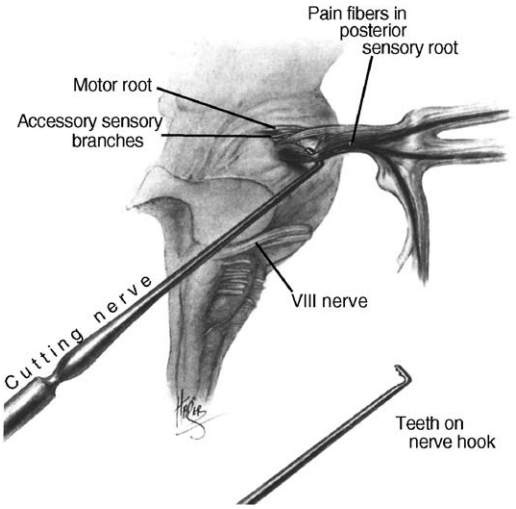

Fig. 15. Illustration demonstrating Dandy's concept of distribution of sensation within the trigeminal nerve and site of sectioning, which is still used today. (*From* Dandy WE. The brain. In: Walters W, Ellis FH, Jr, editors. Lewis-Walters practice of surgery. Hagerstown, MD: WF Prior Co.; 1963. p. 1–671.)

months), 58% of patients were pain free, 36% had obtained good pain control, and 6% failed this therapy. Three patients developed decreased facial sensation and increased paresthesia after radiosurgery, which resolved completely after 6 weeks in 1 patient and improved in another. No other morbidity of treatment occurred. This study demonstrated that a maximum dose greater than 70 Gy (range: 70–90 Gy) was associated with a greater chance of complete pain relief and duration of relief from trigeminal neuralgia than a dose less than 70 Gy.

Several authors have reported their experience treating trigeminal neuralgia with 90 Gy [67,68,70]. With this higher dose, a high degree of pain relief is achieved; however, there is a higher incidence of trigeminal nerve sensory deficit [67,68]. Goss et al [70] reported a series of 25 patients treated with 90 Gy directed to the nerve root entry zone. Seventy-six percent of the patients achieved excellent pain relief, 24% achieved good pain relief, 32% developed facial numbness and paresthesias, 12% had decreased corneal sensation requiring eye drop treatment, and 32% experienced relapse. Pollock et al [68] compared patients with trigeminal neuralgia who received 70 Gy versus those who received 90 Gy. Those patients who received 90 Gy had significantly greater permanent trigeminal nerve dysfunction versus those who received 70 Gy (54% versus 15%), a higher incidence of dysesthesias, and corneal numbness. Although pain relief was greater in the 90-Gy treatment group, the difference was not statistically significant.

The optimal radiosurgical target for the treatment of trigeminal neuralgia remains controversial. Three main targets have been reported in the literature: the gasserian ganglion, the anterior portion of the cisternal nerve just proximal to Meckel's cave (also known as the plexus triangularis region), and the nerve root entry zone near the pons. Because the plexus triangularis region target reduces significant radiation to the brain stem, some favor this target when 90 Gy is used. The proponents of this target report greater safety and a lower incidence of facial numbness [71]. Further studies are required to determine the optimal dose and anatomic target.

Summary

When medical treatment for trigeminal neuralgia fails or is limited by significant side effects, neurosurgeons need to inform their patients of all the available treatment options. The best treatment for the patient depends on the age of the patient, medical comorbidities, and the risks the patient is willing to assume. We recommend microvascular decompression for younger healthy patients with a longer life expectancy. Percutaneous trigeminal neurolysis remains a useful minimally invasive approach for the older patient and for the patient with medical comorbidities and a shorter life expectancy. The role of stereotactic radiosurgery in the treatment of trigeminal neuralgia will be better defined in the future. Partial sectioning of the trigeminal nerve may be considered in patients who have negative explorations during a microvascular decompression or when other less invasive procedures have failed to provide adequate relief.

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