Radiofrequency Gasserian Rhizotomy: The Role of RF Lesioning in the Management of Facial Pain

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Pathophysiology of Facial Pain

Chronic facial pain may result from a myriad assortment of pathologic causes including trauma of structural abnormalities (eg, temporomandibular joint), bacterial or viral infections, tumors (benign or malignant) or other masses, diseases of the central nervous system, idiopathic causes, or reflect pain referred from structural pathology or injuries in the face or neck. The overwhelming majority of causes for pain presenting in the face involve the 5th cranial nerve, and pathology may impact the trigeminal nerve anywhere from the trigeminocervical nucleus at the cervicomedullary junction, through the middle fossa, and extending to the peripheral termini of the 1st through 3rd divisions (V1 through V3). Evaluation of headache and facial pain should first rule out "red flag" conditions (eg, tumor, infection, intracranial hemorrhage, vascular dissection, and fractures) and identify the presence of autoimmune, metabolic, neurovascular, or demyelinating conditions. Facial pain may represent referred pain of cervical origin, and cervicogenic sources for facial pain should be excluded.[1] Numerous syndromes resulting from various lesions of the peripheral branches of trigeminal nerve have been described and should be distinguished from the syndromes of trigeminal neuralgia and atypical facial pain.[2]

Trigeminal Neuralgia: Pathophysiology, Prevalence, and Presentation

The precise pathophysiologic causes of trigeminal neuralgia ("tic douloureux") remain unknown. Presentation is typically in older patients (generally in the 7th decade of life), and is characterized by paroxysms of excruciating lancinating pain affecting 1 or more divisions of the trigeminal nerve. Symptoms may wax and wane over a period of weeks to months, and are characterized by episodic lightning-like spasms of pain. Specific mechanical or environmental triggers may be present (eg, chewing, brushing teeth, cold air, auditory stimuli, and touching a particular region of the face). Functional debilitation may be extreme, as patients may avoid eating, chewing, or oral hygiene for fear of precipitating an attack. The pain is so severe that patients may consider suicide. Bilateral symptoms are extremely rare. The most common distribution involves V3 (60-70%), followed by V2, and least frequently, V1 (less than 5%).

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A correlation with multiple sclerosis has been claimed in approximately 2% to 3% of patients, but no satisfactory unifying pathophysiology has yet been advanced. Operative observations and postmortem examinations suggest that the majority of patients with trigeminal neuralgia have compression of the trigeminal root adjacent to the pons (arterial, venous, or occasionally, neoplasm),[3-6] yet the causal relationship of cross-compression of the trigeminal nerve adjacent to the root entry zone to symptoms is less clear.[7]

Trigeminal neuralgia should be clinically differentiated from atypical facial pain, as the presentation, time course, and prognosis of these distinct clinical entities are quite different.[7-11]

Although among the most debilitating of chronic pain conditions, trigeminal neuralgia is fortunately successfully treated in the majority of patients. Surgical intervention should be reserved for those patients with debilitating pain refractive to pharmacological therapies.

Atypical Facial Pain

In contradistinction to trigeminal neuralgia, atypical facial pain usually involves complaints of a generally constant (as opposed to episodic or paroxysmal) nature. The character of pain may be described as aching, pressure, or burning (as opposed to shooting or electrical), and typically is not isolated to a specific trigeminal distribution and may be bilateral. No particular distribution appears to be predominant. Atypical facial pain patients tend to be younger than those with tic douloureux, and significant psychological dysfunction may precede or accompany the onset of symptoms.[12] The varied presentations for atypical facial pain likely represent a "hodge-podge" of etiologies that simply happen to involve the terminal sensory distribution of the trigeminal nerve or sphenopalatine ganglion, including referred pain of cervical origin, dental pain, cluster headache, or injury to the peripheral branches of the trigeminal nerve following surgery or trauma.

The consistent differences in surgical outcomes between trigeminal neuralgia and atypical facial pain also support the contention that atypical facial pain is more diverse with regard to structural etiology.[13]

Radiofrequency Thermal Neurolysis for Trigeminal Neuralgia

Successful surgical management of trigeminal neuralgia may be broken down into minimally invasive methods (radiofrequency [RF], glycerol, compressive ganglionolysis, and gamma knife) and open surgical methods (suboccipital craniotomy with microvascular decompression). This monograph will focus on percutaneous RF thermal lesioning of the gasserian ganglion.

Alternating current thermal lesions created by modern RF

lesion generators allow the creation of precise, well-defined thermal lesions dependent on electrode configuration and set temperature. As the complication and adverse side effect rates following RF lesioning are generally dependent on the size of the thermal lesion, optimization of lesion size with proper electrode configuration and lesioning parameters may be tailored to any specific indicated clinical scenario.

Stimulation capabilities of modern RF generators also allow the practitioner to precisely identify the nerve tissue selected for thermocoagulation; stimulation of the motor, as well as sensory distribution of target nerves, permits extremely precise electrode positioning before thermal lesioning. This functional "stereotactic" approach represents the hallmark of structure-specific selective nerve lesioning, and has found wide application in the treatment of various central and peripheral pain problems.[14-19]

Percutaneous RF Gasserian Rhizotomy Efficacy Versus Chemical Rhizotomy and Microvacular Decompression

Of the minimally invasive methods devised to treat trigeminal neuralgia, the largest case experience involves percutaneous stereotactic RF gasserian rhizotomy. Large case series have been compiled, and the first-pass success rate of RF gasserian lesioning approaches 80%.[11,16,20-25] Advances in technique have reduced the rate of operative complication, and recent series have reported low complication rates with no mortality.[24-26] Some contemporary controversy remains over the relative efficacy of glycerol versus RF gasserian rhizotomy; the preponderance of current literature suggests that the RF method is more successful, associated with fewer side effects, and is associated with better long-term efficacy.[26]

Although the first- (and if necessary) second-pass efficacy of percutaneous RF gasserian rhizotomy is high, recurrences do occur. Microvascular decompression remains the most effective method for treating trigeminal neuralgia, although the relative degree of invasiveness is clearly higher than for percutaneous gasserian rhizotomy.[5,13,27,28]

Indications/Contraindications

Percutaneous stereotactic RF gasserian rhizotomy should only be considered in patients with intractable trigeminal neuralgia who have failed, or cannot tolerate, pharmacologic therapies. As intraoperative stimulation is the cornerstone of successful localization of the particular division of the trigeminal nerve before thermocoagulation, the patient must possess adequate cognitive faculties. Trigeminal neuralgia primarily affects older patients who often suffer coexisting medical diseases. As percutaneous gasserian rhizotomy, even in skilled hands, is physiologically stressful and performed under intermittent deep intravenous sedation rather than general anesthesia, appropriate screening for coexisting disease states is essential in the preoperative preparation of the patient.

Radiofrequency gasserian rhizotomy should be considered with caution in patients with atypical facial pain. In this subset of patients, long-term prognosis has not been optimistic, and significant psychosocial comorbidity may be present.

As with any potential percutaneous surgery, specific contraindications to percutaneous gasserian rhizotomy include, but are not limited to, uncontrolled bleeding diatheses or ongoing anticoagulant therapy, infection, patient refusal to undergo the procedure or the inability to obtain informed consent, nontrigeminal causes for facial pain, airway contraindications to intermittent deep sedation, an inability by the patient to determine a cause and effect relationship to symptoms, and anatomic abnormalities or variations that would preclude safe fluoroscopic visualization and access to the foramen ovale and gasserian ganglion.

Complications

Reported complications associated with percutaneous gasserian RF rhizotomy include death, infection, meningitis, bleeding (intra- and extracranial), anesthesia dolorosa (deafferentation pain), persistent corneal hypoesthesia leading to ulcerative keratitis, masseter weakness or paralysis, blindness, bradycardia or coronary vasospasm, the development of cavernous-carotid fistula, facial hypoesthesia or anesthesia, and persistent dysesthesiae.[23-26,29-34] However, in skilled hands, the incidence of severe complications or death is extremely low, and the most common complications include return of pain within 1 year, persistent hypoesthesiae or dysesthesiae, masseter weakness, and loss of corneal sensation leading to ulcerative keratitis.[8,21,23-25,35]

Technique

Preoperative preparation of the patient includes obtaining detailed informed consent. Even with perfectly performed procedures, side effects, such as masseter weakness, facial hypoesthesiae, and dysesthesiae, are common, and the possibility of corneal hypoesthesia leading to ulcerative keratitis must be discussed. Any coexisting comorbidities must be medically optimized. A detailed description of the procedure, including intraoperative "wake-up testing," will facilitate the smooth performance of the surgery.

Appropriate preoperative patient fasting should be verified. Preoperative prophylactic antibiotics should be administered 1 hour before the procedure. The choice of antibiotics should cover common skin and intraoral flora. A mild opioid preoperative injection (eg, meperidine 50 mg IM) may be considered. Some advocate the use of prophylactic sympathetic agonist medications (eg, atropine, glycopyrrolate, and ephedrine) to decrease the likelihood of severe bradycardia. The observations of this author suggest that although profound bradycardia and vasovagal episodes do occur, they are rare, and routine premedication with sympathetic agonists are not necessary. Vasovagal responses typically occur on introduction of the electrode into the foramen ovale; if bradycardia is encountered, the electrode may be partially withdrawn and appropriate sympathetic agonists administered.

The patient is place in the supine position, with the head neutral. Supplemental oxygen is administered via nasal cannulae, and the forehead secured to the operating room table with adhesive tape, padded and wrapped around the forehead. Tilting of the operating room table in reverse-Trendelenberg facilitates submental visualization of the foramen ovale, and, as the airway is not secured during the procedure, decreases the likelihood of passive gastroesophageal reflux and subsequent aspiration. Patent intravenous access is mandatory throughout the procedure. Physiologic monitoring, including electrocardiogram, noninvasive blood pressure monitoring, and pulse oximetry are mandatory.

The skin of the face is gently prepared with topical antiseptic, and draped in a sterile fashion. Steep submental fluoroscopic images, with slight ipsilateral angulation, are obtained to image the foramen ovale, typically just medial to the mandibular arch (Fig 1). A skin entry site is marked and anesthetized with buffered lidocaine. The skin entry site will usually lie 2 to 3 cm lateral to the lateral angle of the mouth. A 22-gauge, 4-inch, 2- to 5-mm active tip RF electrode, with a slight curve place in the distal 1 cm, is ideal. Although specialty electrodes have been developed (eg, Tew), equivalent success can be obtained with smaller profile electrodes and with much less patient discomfort. The electrode is introduced parallel to the radiograph beam, and advanced in a coaxial manner to the radiograph beam. Before reaching the petrous portion of the temporal bone, it is common for the patient to experience a V3 distribution paresthesiae. Before advancing the electrode, an intraoral examination is performed, making sure the buccal mucosa has not been perforated.

At this point, the patient is asked to hyperventilate, then deeply sedated with intravenous isopropyl phenol (1.25-2 mg/kg) administered by the anesthesia provider. On reaching the apneic threshold, the electrode is advanced to enter the foramen ovale at its midpoint (Fig 2A). The fluoroscope is repositioned to obtain a lateral image of the midface, and the electrode is advanced approximately 2 to 4 mm further through the canal of the foramen ovale such that the tip of the electrode reaches the junction of the petrous mass and the clivus (Fig 2B).

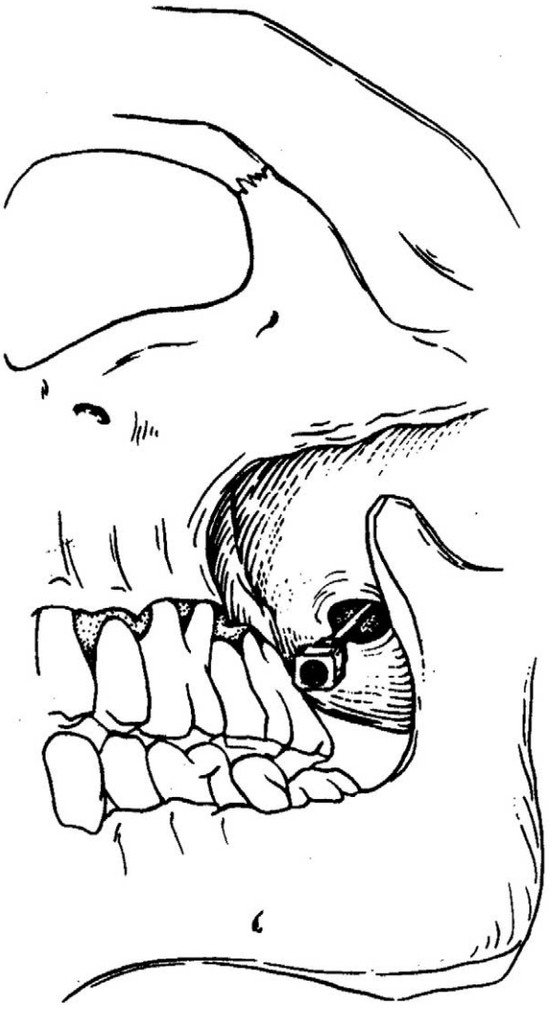

Fig 1. Submental oblique image of foramen ovale.

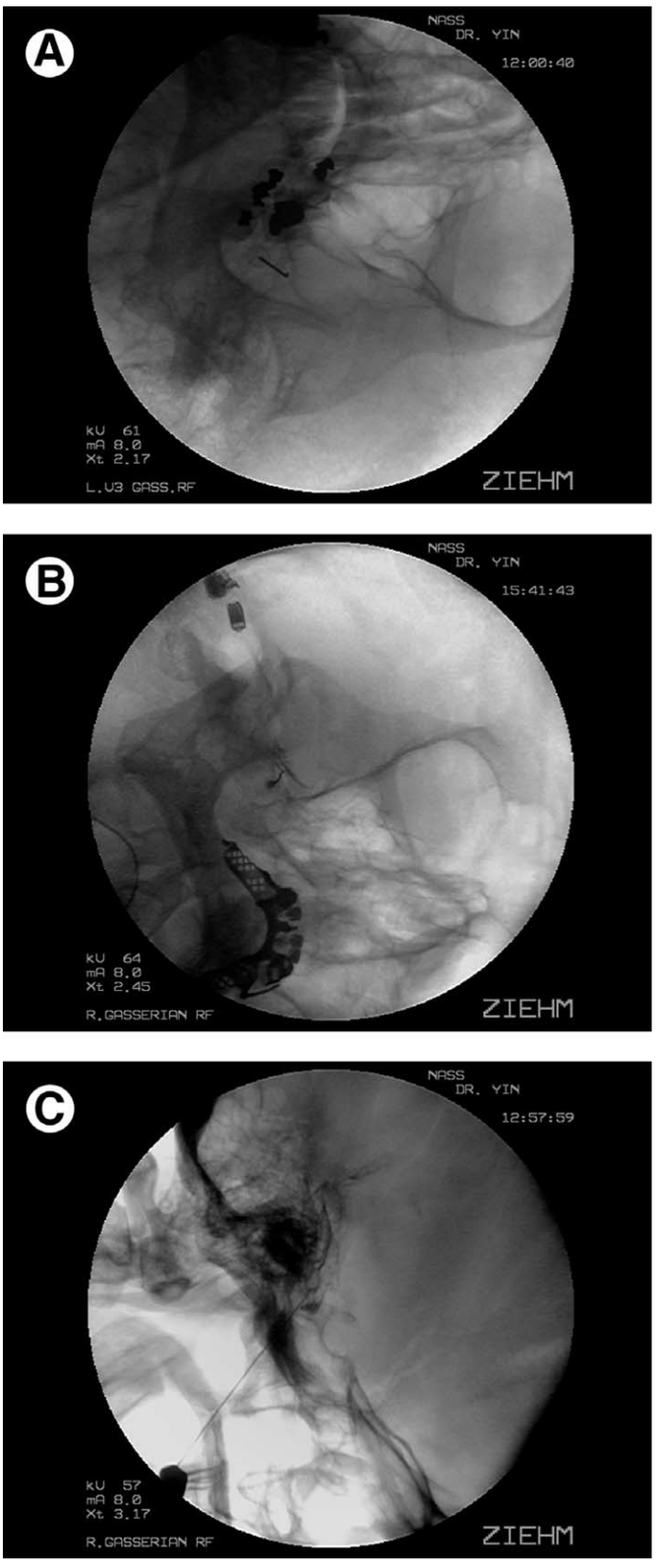

Fig 2. A-C. Coaxial electrode approach toward the foramen ovale, submental oblique view. A. Before entrance into foramen ovale. B. Submental oblique image of contrast in middle fossa. C. Lateral image of contrast in middle fossa.

The stylet of the electrode is removed, and a slow drip of CSF should be returned, indicating successful dural penetration

within Meckel's cavern. If contrast is administered at this point, opacification of the CSF in the middle fossa at the level of the lateral pons will be demonstrated (Fig 2C).

The patient is allowed to emerge from the intravenous anesthetic, and stimulation is performed to verify correct localization of the electrode in the appropriate segmental division of the trigeminal ganglion of interest. Reproduction of concordant symptoms isolated to the trigeminal distribution of the patient's usual symptoms (V1, V2, V3) at 50-Hz, 1-millisecond pulse duration should be reproducible at 0.05 to 0.1 V. The arrangement of the trigeminal ganglion is such that localization of the third division typically occurs closest to the foramen ovale, and the first division more proximally toward the pons. Typically, the third division is also located more laterally on submental imaging of the foramen ovale, and the first division more medially (Fig 3A-C).

If repositioning of the electrode is necessary, additional doses of isopropyl phenol should be given, as any movement of the electrode within the trigeminal ganglion is excruciatingly painful.

Once proper stereotactic localization of the electrode has been confirmed, a thermal lesion is created. The operating physician must understand that the incidence of postoperative sensory complications (including corneal anesthesia) and masseter weakness is in direct proportion to the size of the lesion. As lesion size is directly proportional to the temperature of the lesion, the physician can never be criticized for creating a lesion that is too cool. If properly performed, an excellent clinical outcome may be obtained without persistent sensory abnormalities or masseter weakness.

Once appropriate stimulation parameters have been verified, the patient is again anesthetized with intravenous isopropyl phenol. As the apneic threshold is reached, thermal lesioning is initiated. An initial target temperature of 58°C to 60°C is recommended, with two 60- to 120-second lesions created. The use of advanced high-verniation lesioning techniques allow the use of pulsed RF energy, resulting in minimal chances of temperature overshoot and extremely stable temperature lesions. The author prefers the lesion generator set to deliver 4 pulses per second, with each pulse of 30 milliseconds' duration. Typical voltages necessary for lesion temperatures in the 58°C to 60°C range from 45 to 80 V.

Following the conclusion of lesioning, the electrode is removed and the patient allowed to awaken from the anesthetic. Prior practice had involved verifying hypoesthesia in the distribution of the target pain; however, with the creation of lower temperature lesions, dense immediate postprocedure hypoesthesia or anesthesia is not necessary or even desirable. Once the patient has emerged from anesthesia, corneal reflexes must be examined. If corneal sensation is decreased, protection of the cornea should include a regimen of saline eye drops, or the use of viscous lubricant (eg, Lacrilube or equivalent) and an ophthalmologic eye patch. Advancements in minimally invasive surgical technique and short-acting intravenous anesthetics have permitted this procedure to be performed in an outpatient setting, provided responsible adult supervision and monitoring of the patient in the early postoperative period is available. Alternatively, patients may be admitted for inpatient observation for 24 to 48 hours.

Although many patients will notice complete relief of their trigeminal neuralgia pain immediately following surgery, some will experience postoperative pain for 2 to 3 weeks. Preoperative pharmacologic modalities, especially neuromodulator agents (eg, carbamazepine, gabapentin, and so on) should be continued until the patient has returned for postoperative follow-up. Additional analgesic medications may be required during the early postoperative period.

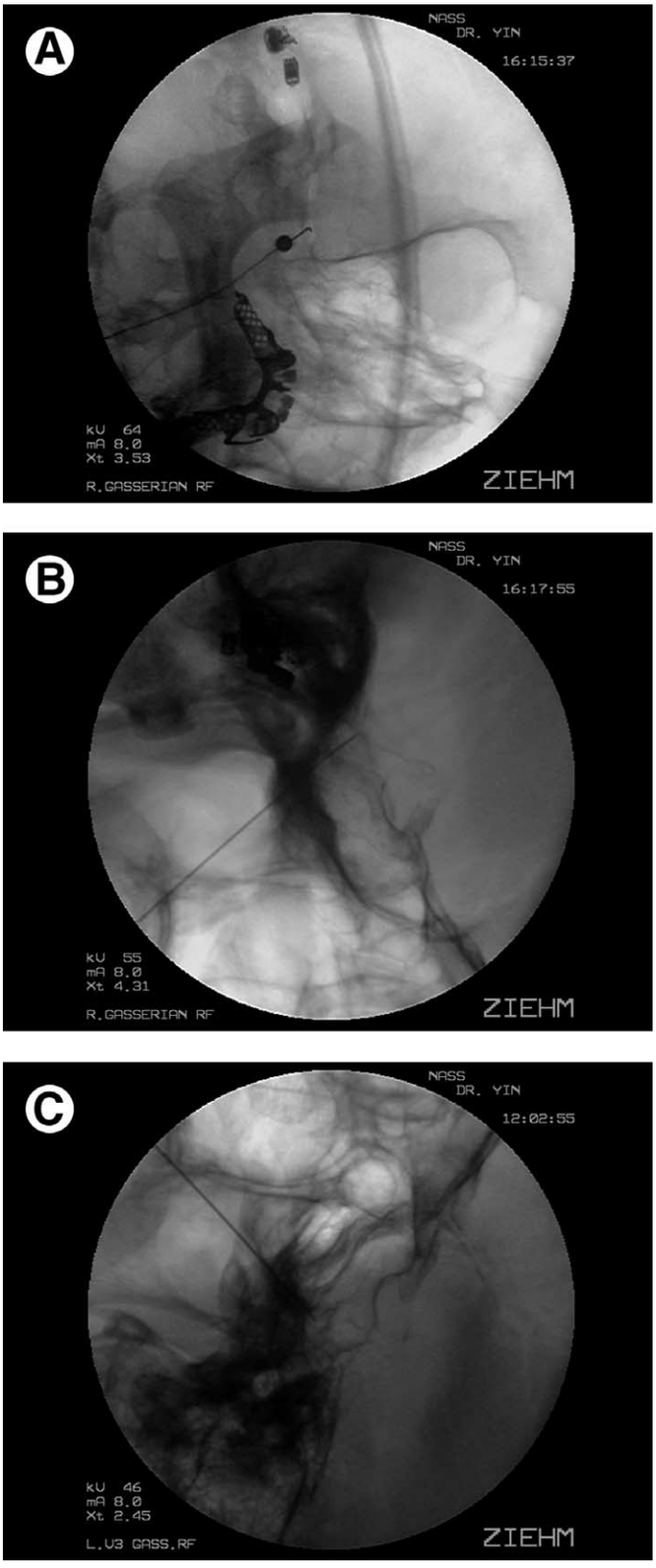

Fig 3. A-C. Final electrode placement for trigeminal ganglion lesion. A. Submental oblique. B. Lateral image, V2 lesion. C. Lateral image, V3 lesion.

Prognosis

The prognosis of trigeminal neuralgia following percutaneous RF gasserian rhizotomy is good. Approximately 80% of patients can be expected to experience high-grade to complete relief of their symptoms; however, a recurrence rate of 15% to 20% can also be expected within the 1st year. If symptoms recur, repeat rhizotomy may be considered and has been demonstrated to be effective. In patients with recalcitrant trigeminal neuralgia who have failed repeat RF neurotomy, microvascular decompression may be considered.

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