

Percutaneous Stereotactic Radiofrequency Thermal Rhizotomy for the Treatment of Trigeminal Neuralgia

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Abstract

Background: Trigeminal neuralgia (TN) is the most common cephalic neuralgia in people over the age of fifty, with a mean incidence of 4 per 100,000. Percutaneous stereotactic differential radiofrequency thermal rhizotomy (RTR) is a well-recognized surgical treatment for TN. The purpose of this study was to evaluate a management algorithm for TN and to evaluate the effectiveness of RTR for TN after failure of pharmacologic management.

Methods: Two hundred and fifty-eight (258) patients underwent RTR from 1992–1996 and were prospectively evaluated. These patients were characterized by age, sex, side of the face and division(s) involved. Patients were evaluated for pain relief, recurrence requiring or not requiring re-operation, and the type and rate of complications. They were followed by serial clinical evaluation and telephone interview. Patients were grouped according to results: (A) Successful Result — excellent or good pain relief; (B) Unsuccessful Result — fair, poor or no pain relief. The RTR group was compared to historical controls. Follow-up ranged from 12–80 months (mean = 38 months).

Results: At early follow-up (defined as median postoperative period up to 6 months), pain relief that was excellent or good (successful results) occurred in 224/258 (87%). At long-term follow-up (> 6 months) recurrence of tic pain required re-operation in 31 patients (12%). In 37 patients (14%), recurrence of tic pain did not require re-operation. Dysesthesia developed in 20 patients (8%); corneal analgesia developed in 8 patients (3%). “Anesthesia dolorosa” developed in 5 patients (2%) and was medically managed. At the conclusion of the long-term follow-up period, 214/258 patients (83%) had excellent to good pain relief (successful result). There were no mortalities, no significant morbidity and a low rate of minor complications.

Conclusion: With the use of this specific diagnostic and management algorithm, patients with TN can be successfully managed with RTR. **Key Words:** Radiofrequency thermal rhizotomy, surgical treatment, facial pain.

Introduction

TRIGEMINAL NEURALGIA (TN) is one of the most painful and debilitating diseases known. A review of the history of TN illustrates the devastating impact that the disease has on afflicted individuals. The cause(s) of TN, as well as its medical and surgical treatments, have undergone considerable evolution (1, 2).

Trigeminal neuralgia was not well described in ancient medicine, and not until the eleventh century did the first description of this problem reach the medical literature (1). These early accounts described “severe spasms of facial pain, without loss of power or sensation.” The relationship of the pain to “the jaws” and “the roots of the teeth” was also noted. At that time, one of the recommended treatments was “wine and rest in a darkened room,” a home remedy not uncommon today.

The first narrative of this disease dates back to the 1671 description by a German physician, Johannes Laurentius Bausch, who suffered from a lightning-like pain in the right face (1). He became unable to speak or eat properly and finally succumbed to malnutrition. A French physician, Nicolaus André (1756), is credited

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with providing the first comprehensive description of TN as a clinical entity and first applied the name “*tic douloureux*” to the disorder (3). His first patient had previously had several teeth extracted without relief of pain, in an attempt to treat an infection of the maxilla. Following the last extraction, André wrote:

. . . what had been regarded as the end of a mild and tolerable ailment, became the source of the sharpest and most uncomfortable pains, I would say the start of a *tic douloureux* that assailed her night and day, deprived her of sleep, and forbade her some of the bodily functions necessary for life. In fact these periodic agitations became so frequent that they rarely allowed five or six minutes of peace during an entire hour; the patient could not eat, drink, cough, spit or wipe her face without renewing her pains.

In 1773, the English physician John Fothergill, in search of treatments for TN, was the first to describe the complete clinical features of the disease, including the fact that light touch was the most common trigger. He noted that TN was more common in women and the elderly (4). In 1787, Pujol stressed the point that TN is often confused with toothache, and healthy teeth are extracted unnecessarily (5). Subsequently, Chapman in 1834 observed that the pain of TN exhibits spontaneous remissions and exacerbations, and has a lancinating quality (6). In 1896, Tiffany observed and recorded, in numerous patients, the preferential involvement of the right side of the face and of the mandibular and maxillary divisions of the trigeminal nerve (7). Oppenheim and Patrick (8, 9), in 1911 and 1914 respectively, commented on the association of TN with multiple sclerosis and its occasional familial pattern. Harris, in 1940, in a study of 1,433 cases of TN, noted that bilateral TN was more common in patients who had multiple sclerosis and/or a positive family history (10).

In the 1950s, the investigations of Kugelberg and Lindblom added significantly to our historical understanding of the clinical features of TN and began a more comprehensive approach to the study of the etiology of this disease (11). They documented that the trigger zone is usually located in the perioral region and that light tactile and vibratory stimuli are the most effective triggers, while painful stimuli are generally ineffective triggers. In addition, they measured a specific latent period between the onset of a stimulus and the onset of a painful paroxysm. They also

found that the attack is self-sustained once it has been initiated and that it is followed by a refractory period lasting up to several minutes.

Sir Charles Symonds, in 1949, first called attention to the fact that the presentation of TN can be somewhat “atypical” in some patients (12). These patients may start out with a diffuse, poorly localized pain which they describe as dull, aching, burning, throbbing, or crawling in nature. This pain tends to become chronic and may be associated with other historical and physical findings that differ from TN. Over the subsequent decades, numerous authors have described patients with similar clinical phenomenology and have coined equally numerous descriptive terms for craniofacial pain syndromes (13, 14).

Many craniofacial pain syndromes have long and very complex histories. Often, eliciting the correct information to make the diagnosis is time consuming and difficult, yet it is critical if the treating doctor is going to make the correct diagnosis. Trigeminal neuralgia has one of the most consistent historical presentations and physical findings of all the craniofacial pain syndromes. The age of onset, sex, location, referral pattern, duration, quality, quantity, triggering stimuli and relieving factors, as well as any associated physical phenomena, are essential parts of the history for diagnosing a craniofacial pain syndrome.

The initial attacks of pain are usually unprovoked and start spontaneously, yet some patients will experience a dull, continuous, aching pain, usually in the upper or lower jaw or teeth, that may last for days to months before the paroxysms of pain start. This has been described in some studies and termed “pre-trigeminal neuralgia,” although there is still some debate as to whether this is truly trigeminal neuralgia. The paroxysms of pain are often characterized as “sharp,” “electrical-like,” “stabbing,” “lancinating,” “bolts” of pain, which last from seconds to minutes. The pain is described as the most severe pain that the patient has ever experienced. It is on one side of the face in approximately 90% of patients, but can occur bilaterally. Only in rare cases is it on both sides at the same time. It is more common in women over the age of fifty, more common on the right side of the face, and most common in the second and third divisions, but always limited to the distribution of the trigeminal system. Very often there are tactile triggers associated with the pain attacks, yet the initial onset is usually spontaneous and the subsequent bouts of pain can also be spontaneous without any tactile triggering stimulus. This phenomenon can become so profound that patients will not be able to touch their

face, brush their teeth, eat, drink, talk, shave or go out on a windy day. Often patients stay at home, change their social and eating habits, lose weight and become depressed. Strangely, the pain does not usually wake patients up from sleep.

The attacks of pain are usually of very short duration, yet they often come in bouts that may last for days or weeks, with patients having a few to hundreds of severe attacks per day. These bouts of pain are often self-limiting, and remission periods frequently may be of long duration. The bouts will recur without warning and without any inciting event or injury. These episodes of pain are often seasonal and may be precipitated by temperature changes, humidity changes, pollen or other climatic conditions.

Many patients exhibit tactile trigger areas, yet these are not universal. The trigger areas of the face are more commonly found in the perioral and perinasal area, which may be due to the somatotopic organization pattern in the trigeminal brainstem complex, as well as the large cortical representation areas that the face and mouth have in the somatosensory cortex (15). The trigger areas are usually affected by very light touch and cold, which elicits the pain. Sharp stimuli or pressure may not elicit pain. As yet, the relationship(s) between the phenomena of hyperalgesia, cold hyperalgesia and allodynia, all common findings in trigeminal neuralgia, and the underlying responsible pathophysiologic mechanisms have not been established (16).

Patients with idiopathic trigeminal neuralgia do not complain of "numbness" or paresthesia of the face, or other associated phenomena, such as headache, diffuse bilateral pain, photophobia, tinnitus, anosmia, dysgeusia, neck pain, autonomic signs, or other cranial nerve abnormalities. If these or other systemic complaints are present, then further evaluation and work-up are essential.

The physical examination entails a thorough evaluation of the head and neck, with special emphasis on the neurological examination. Cranial nerve examination should be performed, with special attention to hearing abnormalities and facial nerve abnormalities. In addition, neurosensory testing of the trigeminal system should include light touch, sharp touch, various temperatures and directions, and two-point discrimination. Note should be taken of any trigger areas, and they should be appropriately mapped out. Aside from the trigger points, when present, and minimal hypoalgesia or hypoesthesia in some patients, the neurological examination is essentially normal. Therefore, in summary, the clinical criteria for a diagnosis of trigeminal neuralgia are:

1. Severe, lancinating, paroxysmal pain
2. Unilateral pain
3. Area limited to the distribution of the trigeminal nerve
4. Trigger areas
5. No sensory deficit

The diagnosis of craniofacial pain is often quite difficult due to the complexity and similarity of presenting symptoms, and the large and diverse classification schemes and categories of disease processes. Numerous classifications have been developed by different organizations in order to aid the clinician, yet these classifications are often cumbersome and confusing. In many patients, the disease presentation does not fit neatly into the classification scheme (17–19). A starting point in the differential diagnosis of craniofacial pain is separating neuralgic from non-neuralgic pain. The cephalic neuralgias are often divided into trigeminal nerve involvement and other cephalic nerve involvement, either of cranial nerve branches or of cervical plexus branches. Trigeminal neuralgia, as discussed above, has a very specific group of findings, and the diagnostic dilemma is whether it is primary (idiopathic/classical) or secondary (due to a structural lesion). The constellation of signs and symptoms with which the patient presents will dictate what further diagnostic studies are necessary before therapy is begun. These studies can include: imaging, neurophysiologic testing, psychologic testing, nerve block or muscle injections, intravenous drug administrations, lumbar puncture for cerebrospinal fluid analysis, hematologic testing and biopsy when necessary.

Our Craniofacial Pain Group has developed an algorithm for the differential diagnosis and management of craniofacial pain disorders. This algorithm incorporates historical data, physical examination data and some diagnostic testing to guide medical and surgical management strategies for patients with craniofacial pain. The current treatment of TN consists of medical and surgical therapies. Medical management consists of pharmacologic and non-pharmacologic approaches, while surgical management consists of numerous peripheral and intracranial procedures. The first line of treatment is usually medical therapy, with drugs such as carbamazepine, baclofen, gabapentin, phenytoin or clonazepam in single or combination regimens. Pharmacologic therapy is effective for many patients; however, for some these medications either fail to relieve the pain and/or produce intolerable side effects with significant medical and functional morbidity. If

medical therapy is unsuccessful or not tolerated, surgical treatment should be considered.

Of the surgical procedures, Percutaneous Stereotactic Differential Radiofrequency Thermal Rhizotomy (RTR) is a well-recognized treatment for TN (20, 21). At the Massachusetts General Hospital (MGH), where this procedure was pioneered (22), RTR is the procedure of choice for patients undergoing initial surgical management. RTR is a technique of controlled thermal ablation of nerve fibers in the trigeminal ganglion or nerve root, producing loss of pain with relative preservation of touch and more complex facial sensations (23–25). The radiofrequency generator and micro-electrodes allow for precise localization with nerve stimulation testing, and provide a finite and restricted thermal nerve lesion (20). RTR produces some sensory loss in the affected distribution, which is usually well tolerated. It does not affect facial nerve function, and therefore facial muscle paresis is not a concern. Pain relief is generally immediate, complication rates and side effects are minimal (the latter usually well tolerated), and patient satisfaction is high (26).

The specific aims of this study were: (A) to evaluate the effectiveness of our management algorithm for TN, (B) to compare medical and surgical treatment outcomes, and (C) to evaluate RTR in patients who did not benefit from or could not tolerate medical management.

Methods

Patient Management Protocol

Patients who presented to the MGH Craniofacial Pain Center between 1991–1996, with a complaint of facial pain, were considered for participation in this study. They were evaluated with a comprehensive interview, history and physical examination. Physical examination of the head and neck consisted of examination of the cranial nerves, with special attention to the neurosensory examination of the trigeminal nerve. Patients with the following five findings were diagnosed with TN: (A) paroxysmal, lacinating, electric-like pain, (B) tactile trigger areas, (C) unilateral symptoms, (D) restricted to the distribution of the trigeminal nerve, and (E) no neurosensory deficit. These patients were entered into our management algorithm (Fig. 1).

All patients with this diagnosis were evaluated by magnetic resonance imaging (MRI) of the brain and brainstem, with special attention to the posterior cranial fossa and trigeminal system, for evidence of tumor, vascular abnormality or

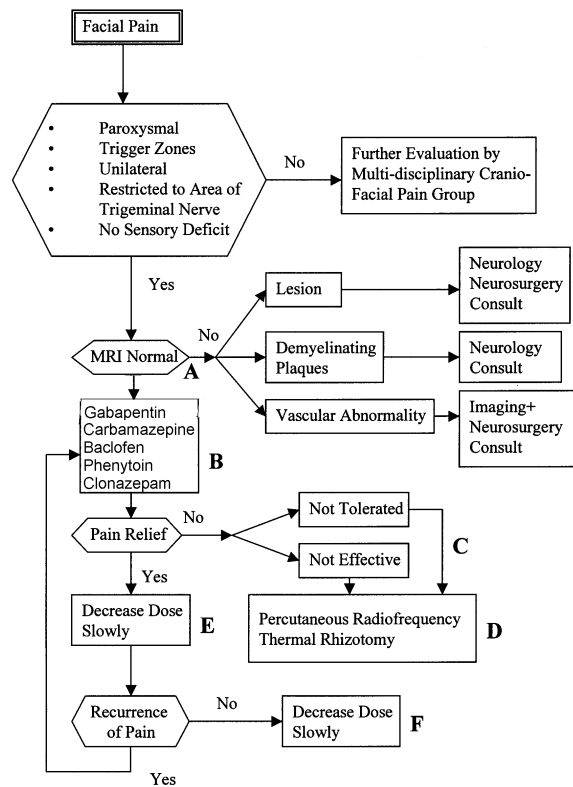


Fig. 1. Algorithm for the management of trigeminal neuralgia (TN).

- A.** MRIs of the brain, brainstem, and base of the skull are obtained. Further evaluations and studies are only performed when physical examination or MRI findings are abnormal.
- B.** Medication is titrated progressively until pain is relieved or adverse effect(s) occur.
 1. Carbamazepine 300–1200 mg per day or
 2. Baclofen 20–80 mg per day or
 3. Gabapentin 300–1200 mg per day or
 4. Phenytoin 300–1200 mg per day or
 5. Carbamazepine + Baclofen or
 6. Gabapentin + Baclofen
 7. Clonazepam 0.5–1.0 mg per day is often added to the above regimens.
- C.** Drug allergy or idiosyncratic reaction, laboratory abnormalities (CBC, Liver function tests, drug levels), significant side effects, patient preference.
- D.** Prior to radiofrequency thermal rhizotomy, local anesthetic blocks are performed in some patients as part of a further evaluation of individual nerve divisions and to permit the patient to experience the feeling of altered sensation.
- E.** Dosages of medications are decreased slowly in a stepwise fashion depending on the prior regimen providing pain relief. After the patient has been pain free for 4–6 weeks, medication can be gradually tapered and eliminated.
- F.** Further decrease in dosage of medication is predicated on the patient's pain history. Many patients are maintained on pharmacologic therapy.

demyelination (Fig. 1A). Patients with a clinical diagnosis of TN and a normal MRI were started

on pharmacologic therapy (Fig. 1B). Those patients who did not conform to the clinical diagnosis of TN, or who had abnormal MRI findings were further evaluated, and appropriate consultations and further diagnostic studies were ordered. These patients were not included in this study.

Patients were prescribed pharmacologic regimens based upon past drug history and responsiveness to specific pharmacologic agents. Pharmacologic therapy was continued and adjusted based upon pain relief and patient tolerance. If pharmacologic therapy was effective in providing adequate pain relief, it was continued and the patients were monitored closely for 4–6 weeks. The dosage(s) of medications were slowly tapered in a stepwise fashion (one medication at a time over 2–4 weeks), monitoring for return of pain (Fig. 1E). With a decreasing dose of medications, if there was a recurrence of pain, the prior drug regimen was re-instituted. Patients who remained pain free were tapered off all medications. However, some patients were maintained on low-dose, prophylactic pharmacologic programs because of intermittent spontaneous or triggered bouts of pain (Fig 1F). Patients for whom pharmacologic therapy was either not effective in providing adequate pain relief and/or was not well tolerated, were considered for surgical treatment with RTR (Fig. 1C); this was 39% of the total study group.

All patients considered appropriate candidates for RTR were interviewed and counseled. The procedure was thoroughly explained and the risks and benefits outlined and discussed. The potential complications and side effects were described and explained in terms that the patients could fully understand. All patients were given a written description of the procedure to facilitate their review of it. Additionally, patients were advised of the surgical alternatives to RTR, namely glycerol rhizolysis, percutaneous balloon compression of the trigeminal ganglion, and posterior fossa exploration / microvascular decompression. The risks and benefits of these other procedures, and similarities and differences to RTR, were explained and discussed. Any patient who wished further surgical consultation was promptly referred to the appropriate individuals at our institution. Patients who finally consented to undergo RTR were included in this study.

RTR Protocol

RTR is a surgical procedure on the trigeminal (Gasserian) ganglion, performed by a percutaneous approach with local anesthesia, intravenous sedation and intermittent general anesthesia

administered by a senior neuroanesthesiologist. The surgical approach utilizes specific anatomic landmarks and radiologic guidance. In 1974, Cosman and Cosman (27) described the history of the development of the modern radiofrequency generator, many basic fundamentals of utilizing the machine, and the various technical parameters, critical for effective treatment.

The modern radiofrequency generator has a built-in impedance monitor, a stimulator, temperature monitor, and a display of voltage and milliamperage. It has a complex timing device that automatically turns off radiofrequency heating at a desired time interval. Cosman states that the radiofrequency lesion electrode is minimally invasive with an electrical shaft that can communicate impedance, stimulation, recording temperature and lesion parameters that go back to the operator for exquisite target identification and lesion control (27). The voltage meter essentially records electrical energy. The milliamperage is the delivered power or current flowing through the tissue. At a given temperature, an inappropriately increase in the milliamperage may reflect dissipating energy secondary to breaks in the circuitry. The impedance ohmmeter measures the resistance to flow of energy. Resistance of 250–260 ohms is seen in nerve root and rootlets, and may drop to 190–200 ohms when in spinal fluid, while an increase of resistance to 400 ohms may imply bone. This auxiliary electronic aid is certainly most helpful for the improvement of lesion making as well as safety of the patient. Finally, the stimulator is most important in localization for final lesion making.

RTR is performed in the Ambulatory Surgical Suite, with the patient in the supine position on a mobile X-ray table. All patients are likely to be apprehensive, and reviewing each step of the surgical procedure at this time is helpful and important. The head and shoulders are placed on a headboard which extends the head over the end of the X-ray table, allowing for an easy sweep of the C-Arm videofluoroscopy unit. The head may then be extended over a foam sponge so that the view of the foramen ovale is obtained. Extension may be up to 20 degrees and rotation of 15–20 degrees away from the side of the pain (28), for optimal visualization of the foramen ovale. The foramen ovale is seen through the pterygomandibular and infratemporal space as an oval structure at the top of the petrous pyramid. Once the foramen is adequately visualized, the image is stored in the fluoroscopy computer and can be reproduced at any point during the procedure.

Following satisfactory imaging of the foramen ovale, the face is surgically prepared and local anesthesia is infiltrated into the skin and subcutaneous tissues of the ipsilateral cheek. A large bore (#15) needle is then placed through the skin in this area to allow for easier passage of the cannula and its stilette. The surgical instruments and electrode enter the skin 2.5–3.0 cm lateral to, and 0.5–1.0 cm below, the labial commissure (Fig. 2) (20, 21, 29). The direction of the instrument is determined by two planes: a lateral plane at a point one-third of the distance from the external auditory meatus to the lateral canthus of the eye, and a medial plane directed from the puncture site to the medial aspect of the pupil. Intersection of these planes identifies the site of the foramen ovale (Fig. 2).

Once the foramen has clearly been identified, a 19-gauge insulated cannula with a sharp stilette is introduced through the puncture site. The neuroanesthesiologist then anesthetizes the patient with an intravenous agent (propofol) which is rapid in onset and ultra-short in duration, to allow the surgeon to proceed with advancing the cannula. The cannula is directed slightly downward and laterally for approximately 2.5 cm. This trajectory is then changed back into Hartel's projection (30) to avoid entrance into the oral cavity. The cannula may be in the perioral tissues, and a quick look will assure the surgeon that the cannula has not penetrated the oral cavity. The cannula and stilette are now directed toward the

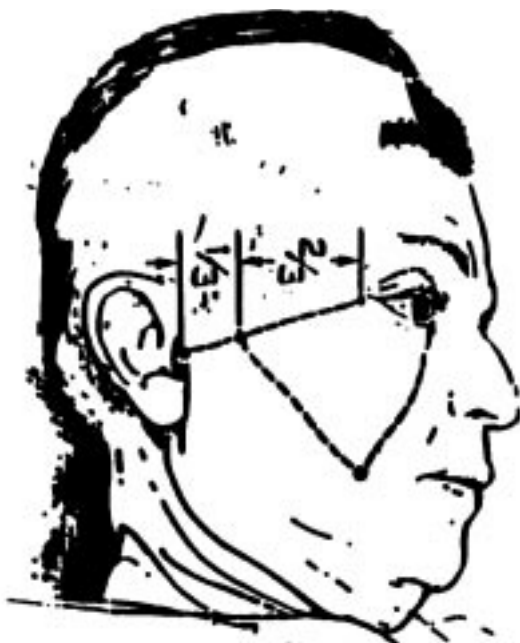


Fig. 2. Skin gridlines for penetration of the foramen ovale and Gasserian ganglion.

medial portion of the foramen ovale in the AP projection. The suggested trajectory through the foramen ovale is determined by the division(s) involved and to be lesioned (Fig. 3) (20, 21). The initial trajectory should aim for a point at the junction of the clivus and petral-clinoid ligament and never approach the floor of the sella. From that point to the petrous pyramid, a line is then divided into three points, the uppermost being the first division trajectory, approximately 50% from the petrous pyramid to the junction of the petral-clinoid ligament and clivus for the second division, and just above the petrous pyramid at the junction of the clivus for the third division. The cannula and stilette are advanced to a point 1 mm in front of the clivus. The stilette is now removed, and in most instances there is free-flow cerebrospinal fluid; however, this is not always mandatory for a successful procedure. Often just rotating the hub of the cannula or placing the patient in Trendelenburg position may allow fluid to egress.

Once a satisfactory position has been obtained, the stilette is removed and replaced with a straight or curved "Tew" electrode (21). The patient is now fully awakened, and once again, the surgeon reviews the previously prepared steps with regard to the patient assisting in localizing the square-wave stimulus. The square-wave (pulsed at 50 cycles per second for 1 millisecond and 0.1–0.28 volts) will allow the patient to identify the location of the stimulus on the face. If it requires more than 0.25–0.28 volts, the electrode will have to be readjusted. Initial temperature on the generator should be approximately 37°C. The impedance ohmmeter should read approximately 100–260 ohms.

The patient is instructed to signal, the moment that he or she feels a nonpainful stimulus (square-wave pulse). The third division is often felt in the lower lip, teeth and chin, the side of the

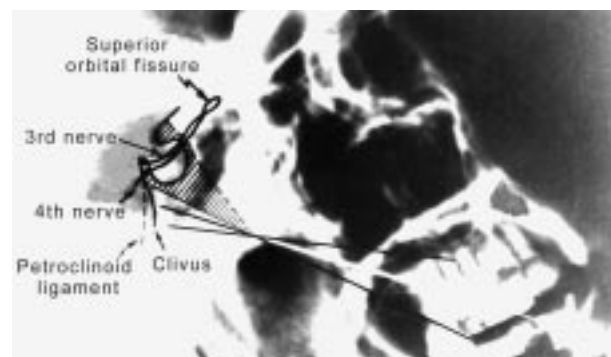


Fig. 3. Lateral projection for electrode placement into appropriate divisions of the trigeminal nerve.

temple or even the ear. The second division stimulus may include the upper lip, teeth, the side of the nose and the middle of the cheek. Stimulation in the first division can give you a reference to the eye, the eyebrow or the forehead. When the patient has identified the area of stimulation and the voltage required is below 0.2 volts, the position is reconfirmed with a mild heating stimulus. The initial response will be between 40–48°C, characteristic for patients undergoing an initial radiofrequency procedure. If there is no satisfactory response or the patient does not experience tingling or heat, the electrode position must be changed. We have found that the curved “Tew” electrode with a coiled spring at its tip may be useful. In the lateral projection, the tip of the electrode is never more than 2–3 mm behind the clival profile. The patient will often tell you that the place of your stimulus is where he feels his pain. The position of the electrode is constantly checked by fluoroscopy to prevent inadvertent moving during stimulation and/or heating.

Once this satisfactory location has been achieved, the neuroanesthesiologist renders the patient unconscious for lesioning. If the threshold for heat was 48°C, the first lesion is made at 60°C for 60 seconds. During the heating, a facial flush has often been observed; it has been described by others as a secondary release of vasodilatory neuropeptides such as substance P (28). This facial flushing confirms the position of the electrode as well as localizing the division that is undergoing neuronal changes secondary to radiofrequency heat. Modern radiofrequency lesion generators are equipped with a timer that turns off the heating element, making it almost foolproof for the surgeon.

The patient is now allowed to awaken completely, following which stimulation by pinprick across the midline may be followed by an aversive response, suggesting an early lesion as one passes the midline to the normal side. If there is no appreciable analgesia or change in sensation, the procedure and lesions are repeated by increasing the temperature by 5–8°C in an incremental way for 60 seconds, until a satisfactory and desired degree of analgesia has been achieved. Sensory testing should be carried out when the patient has fully recovered from intravenous sedation; often, in elderly patients, this may be a problem. All patients are always tested for the corneal sensation, and if there is any change whatsoever, the procedure is promptly discontinued. We have tried to have the patient assist in determining when the endpoint has been reached. We asked those patients who have recurrent trigeminal neu-

ralgia if the degree of numbness is tolerable or whether they wish it to be more numb. In an unanesthetized and responsive patient, when heating evokes little or no reaction, the presence of a thermal lesion can be assured at the completion of the procedure. After the patients have adequately recovered, they are usually discharged home with a companion later that day. If any complications are encountered during or after the procedure, the patient is admitted to the hospital for further observation.

All patients were followed in person, or by telephone interview, at one week, one month, six months, and then yearly. Patients were questioned and examined for facial hypesthesia/hypalgesia, dysesthesia, side effects and complications. All patients were asked to give a satisfaction rating for the management of their pain after RTR, using the published “description of early results” criteria: excellent, good, fair, poor, failure (Table 1) (21). Excellent or good results are considered successful, with no tic pain and no, or only minor dysesthesia. Fair and poor results are considered unsuccessful, with some tic pain and moderate to major dysesthesia. Patients with a recurrence of pain were re-examined and re-entered into the management algorithm on their prior pharmacologic regimen. Patients with recurrence of pain that did not respond to additional pharmacologic therapy were considered for re-operation.

Results

Between 1992 and 1996, a total of 662 patients were seen in the clinic, of whom 258 (39%) were ultimately diagnosed with TN and underwent surgical treatment with RTR. These patients were followed prospectively for a period of 12 to 80 months, the mean follow-up time being 38 months.

The patients were characterized by age, sex, side of the face and division(s) involved. Comparison was made between this patient group and previously published historical controls (Table 2). The average age of patients in this study was 61

TABLE 1
Description of Results

Result	Description
Excellent	No tic pain, dysesthesia, or troublesome paresthesia
Good	No tic pain, minor dysesthesia / paresthesia
Fair	No tic pain, moderate dysesthesia / paresthesia
Poor	Tic pain, major dysesthesia / paresthesia
Failure	Immediate

TABLE 2
Characteristics of Patients

	Present Study	Historic Control**
Average Age	61.5 years (range=41–95)	65 years
Sex	69% female	62% female
Side of the face	58% right	60% right
Division involved		
V-1	0	1%
V-2	13%	16%
V-3	38%	15%
V-1, V-2	8%	15%
V-2, V-3	33%	40%
V-1, V-2, V-3	4%	13%

** Tew JM, van Loveren H. In: Schmidek HH, Sweet WH, editors. Operative neurosurgical techniques: Indications, methods and results, Vol. 2. Philadelphia (PA): W.B. Saunders Co.; 1988. (Reference 57)

years. Females, the right side of the face, and the third division were most commonly affected. There were no patients with isolated first division involvement, and only 8% had first division involvement at all.

Postoperatively, we evaluated patients for early results (immediately postoperative to 6 months) and long-term results (>6 months to 68 months). The grading scale, “description of early results” described in 1995 by Tew and Taha (Table 1) (21) was used for evaluation of outcomes. Patients were also evaluated for complications and side effects of the procedure. Our complications were classified and compared to published combined series data (Table 3) (21).

TABLE 3
Complications

	Present Study	Historic Control**
Masticatory muscle weakness	28.8%	23%
Dysesthesia	8%	20%
Anesthesia dolorosa	2%	1%
Corneal analgesia	3%	7%
Keratitis	0.8%	2%
Aseptic (chemical) meningitis	0.8%	2%
Bacterial meningitis	0	0.2%
Carotid-cavernous fistula	0	0.1%
Intracranial hemorrhage	0	0
Diplopia	0	1.2%
Other cranial nerve deficits	0	0
Death	0	0

** Tew JM, Taha JM. Percutaneous rhizotomy in the treatment of intractable facial pain (trigeminal, glossopharyngeal and vagal). In: Schmidek HH, Sweet WH, editors. Operative neurosurgical techniques, Philadelphia (PA): WB Saunders; 1995. (Reference 21)

Early pain relief (immediately postoperative to 6 months), classified as excellent or good (successful), was obtained in 224/258 patients (87%). The remaining 34 patients experienced only fair or poor pain relief, which was considered to be an unsuccessful outcome. At long-term follow-up, tic pain recurred in 68 patients (26%). Of this group of patients, 31 (12%) required re-operation and 37 (14%) were managed with medical therapy alone (Table 4). Long-term excellent or good (successful) pain relief ultimately was achieved in 214/258 patients (83%).

There was no mortality or major morbidity. Two patients developed postoperative symptoms that were considered consistent with aseptic (chemical) meningitis; they were hospitalized and appropriately evaluated and treated. Blood cultures for both patients were negative and neither patient became septic. The cerebrospinal fluid analysis was normal. Both patients' symptoms resolved uneventfully, and they were discharged from the hospital in 2–3 days.

In our study, 92% of the patients who underwent RTR had early complete pain relief. Overall, late recurrence rate was 26%. Dysesthesia developed in 21 patients (8%); 8 patients (3%) had dysesthesia alone (medically managed) and 13 patients (5%) had dysesthesia with recurrence of tic pain (medically and/or surgically managed). “Anesthesia/analgesia dolorosa” developed in 5 patients (2%). Corneal analgesia developed in 8 patients (3%), with 2 of these patients developing transient keratitis that required ophthalmologic evaluation and treatment. No patient developed ocular muscle paresis or visual loss (see Table 5). Approximately 29% of the patients developed transient ipsilateral masticatory muscle weakness, but no patient developed motor branch involvement that resulted in masticatory muscle palsy or permanent functional impairment. No other cranial nerve deficits were encountered.

Discussion

Both dental and medical specialists are involved with the evaluation and treatment of

TABLE 4
Results

Early pain relief (0–6 months)	224 / 258 (87%)
Unsuccessful (0–6 months)	34 / 258 (13%)
Recurrences requiring re-operation	31 / 258 (12%)
Recurrences not requiring re-operation	37 / 258 (14%)
Long-term pain relief (> 6 months)	214 / 258 (83%)
Mean follow-up = 38 months (range: 12–80 months)	

TABLE 5
Comparison of Different Procedures in the Treatment of Trigeminal Neuralgia
 (Values represent per cent of the number of patients studied)

Technique	Immediate relief	Recurrence	Dysesthesia	Anesthesia dolorosa	Weakness	Morbidity	Mortality
Radiofrequency Rhizotomy (Present Study)	92	26	8.4	1.8	29	1.8	0
Radiofrequency Rhizotomy ⁽⁴⁶⁾	98	23	10	1.5	24	1.2	0
Glycerol Rhizotomy ⁽⁴⁶⁾	90	54	55	1.8	1.8	1.0	0
Balloon Compression ⁽⁴⁶⁾	93	22	5	0.1	66	1.7	0
MVD ⁽⁴⁶⁾	95	17	0.4	0.1	0.1	10	0.6

MVD = microvascular decompression.

⁽⁴⁶⁾ = see reference 46.

patients with craniofacial pain disorders. It has been shown that many patients with these types of problems can go undiagnosed or misdiagnosed, with multiple diagnostic and therapeutic interventions prior to being given the correct diagnosis (8, 32, 33). While the diagnostic criteria for TN are quite specific, not all patients give a "classic" history, and often the physical examination gives information that can be confused with other neurogenic and non-neurogenic pain disorders of the head and neck. To avoid these problems and provide appropriate disease-specific treatment that is evidence-based and effective, an algorithm for the diagnosis and management of TN developed by our clinical group, is presented in this study. This study uses the algorithm to correctly diagnose TN, which is a prerequisite for a successful outcome with RTR.

In the treatment algorithm used for this study, an MRI of the brain, brainstem and cranial base was performed in those patients in whom the clinical diagnosis of TN was made. Reviews of TN by Sweet and others have shown that pathology in any of these regions can produce signs and symptoms that could be confused with primary TN. Multiple sclerosis (MS) (35–37), Chiari malformation (38), pontine hemorrhages (39), midbrain lesions (40), cerebellopontine angle tumors (41), other tumors (42), and most notably, vascular compression (43–45), have all been implicated in causing TN. While the purpose of this study was not to document pathology causing secondary TN, numerous patients in our study were found to have pathology thought to be the cause of their TN; meningiomas, schwannomas, MS plaques, ectatic basilar artery, pitu-

itary tumor and metastatic disease. With this algorithm, these patients were able to be correctly diagnosed and were referred for the most appropriate treatment.

In the present study, 39% of the patients underwent surgical treatment with RTR, while the majority were managed with pharmacologic therapy. This is quite different from other studies, in which 60–75% or more had surgical treatment (14, 21, 46). Regardless of the risks of drug therapy, many patients will benefit from aggressive pharmacologic management with newer agents which have less debilitating and potentially dangerous side effects. Multiple drug regimens, given as initial treatment, may also provide better pain control, and even remission. While the results of this study show that RTR is very effective and is associated with only limited side effects, it must be noted that surgical intervention may cause significant debility from dysesthesia, keratitis and anesthesia dolorosa. Medical therapy prior to surgical consideration is necessary and can be effective in many patients.

Overall comparison of our results with those published (see Table 5) indicates that our results were slightly less successful. The reason for this difference is possibly related to patient selection based on clinical considerations and indications for RTR. The relevant issues of the procedure and the decision-making process were thoroughly discussed with each person and his/her family individually. Nonetheless, pain control is the ultimate goal of RTR, a selective neuroablative procedure which can be performed in a differentially graded fashion. Thus pain relief may be achieved and the more troublesome side effects avoided or minimized.

This conceptual approach to performing RTR may have had an impact on the initial success rate.

Our recurrence rate (26%) was similar to that of other series, as was our rate of anesthesia dolorosa (Table 5). Our rate of dysesthesia was significantly less than that of other series (44, 46, 47). Pain recurrence following a successful RTR may not be related to the technique of the procedure, but is most likely a property of the still unknown disease process. Once a successful thermal lesion is made, the long-term anatomic, histologic and neurophysiologic changes that occur are not known. This probably has some clinical relevance to long-term pain relief and recurrence rates, along with possibly altering the pathophysiologic process that is responsible for producing TN.

Anesthesia dolorosa is an infrequent, but unfortunate complication of RTR as well as other neuroablative procedures. The mechanism for loss of sensation with continued pain is presently not fully understood. It is thought that this represents a central process of abnormal modulation and processing of afferent signals and is not solely the result of peripheral nerve damage (2). Prevention of this problem from occurring at all following RTR probably lies in careful patient selection for RTR, and not in the surgical technique.

It has been shown that the level of sensory loss was correlated to the rate of dysesthesia and the length of time the patient was pain free (47). Mild hypalgesia was correlated with less dysesthesia, but less time pain free, whereas, analgesia was correlated with more dysesthesia, but greater time pain free. This balance between pain relief and the probability of producing dysesthesia following RTR, is difficult to predict and difficult to control. We postulate that our rate of dysesthesia is lower than other series due to careful patient evaluation, careful selection of patients for RTR, and most important, the production of small, precise lesions producing hypalgesia and not analgesia or anesthesia.

Our rates of additional complications (muscle weakness, corneal analgesia, keratitis and aseptic meningitis) are consistent with other series. These problems, while not insignificant, do not produce major morbidity or functional deficits. Masticatory muscle weakness progressively improves spontaneously and can be additionally helped with physical therapy and passive mandibular motion exercises. Corneal analgesia is also usually transient and can be managed successfully with topical preparations and artificial tears. Our one case of keratitis was promptly diagnosed and the patient referred to an ophthalmologist;

treatment prevented corneal scarring or loss of visual acuity. Aseptic (chemical) meningitis in some patients may have been due to an inflammatory reaction to the intracranial manipulation. Our two patients with postoperative symptoms consistent with meningitis were referred for prompt medical evaluation. They were found not to have infectious meningitis, and were successfully managed on an outpatient basis without complications. More serious complications, as indicated in Table 3, did not occur in our study group.

When considering surgical treatment for TN, there are several procedures that are commonly utilized. These were recently compared with respect to efficacy, side effects and complications (47). Of the percutaneous procedures, RTR has the best overall, long-term outcome data. The initial pain relief is equal to or better than that gained from other procedures, the recurrence rate is lower, and the side effects and complications are less frequent and less morbid. RTR allows for pre-lesion testing for localization in order to produce a lesion in only the division(s) involved. RTR also affords the ability to clinically test after a lesion(s), in order to grade the level of hypalgesia/paresthesia, and possibly avoid side effects, while still providing pain relief.

Glycerol rhizolysis is not division specific, and has a very high recurrence rate (approximately 50%) and an equally high incidence of dysesthesia. Balloon compression of the ganglion is also not division specific, can produce significant bradycardia and hypotension during the procedure, has a very high incidence of masticatory motor dysfunction and other cranial nerve abnormalities.

The long-term follow-up data after microvascular decompression (MVD) indicates that it is very effective, and typically does not produce a sensory deficit. However, it has several significant disadvantages when compared to RTR. Those who perform MVD routinely argue that the procedure is safe and effective (44, 47). Yet MVD requires a craniotomy with retraction of the cerebellum and brainstem. Cerebellar dysfunction, hearing loss and facial palsy may result. While these complications are infrequent, the risk may be viewed by the patient as inappropriate or unacceptable.

Stereotactic radiosurgery using the gamma knife has been reported to have a role in the surgical treatment of TN (48, 49). Recently, a multi-institutional study and a follow-up single institution study have found gamma knife radiosurgery to be safe, effective, non-invasive and to have very minimal side effects (50, 51). While these

results are encouraging, there are only very short-term follow-up data available. Numerous additional questions regarding safety and justification remain (52).

Other open surgical procedures involving the root entry zone and the trigeminal brainstem nuclear complex are still being performed, with limited success and the potential for significant complications (53–55). The overall success rate for treating TN is approximately 50% and the complications are often significant and catastrophic; limb and leg ataxia, weakness, sensory loss and death (55, 56).

Those who treat TN, whether surgeon or not, must know all the benefits, risks and complications of all the surgical procedures used to treat this problem. Physicians need to take whatever time is necessary to counsel patients and family about these treatments and to be assured that they fully understand all their options before undergoing any treatment regimen, regardless of the physicians' personal bias. With any surgical procedure there are potential risks and complications, but under no circumstance should the potential cure be worse than the disease.

Conclusion

The management algorithm presented in this study is an effective way to evaluate, diagnose and treat patients with TN. Surgical treatment with RTR is a safe and effective way to manage patients with TN in whom pharmacologic therapy is either ineffective or not tolerated. The side effects of RTR are low and are well tolerated. We believe that RTR should be the procedure of choice for initial surgical management of TN.

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