

Treatment of Cranio-Facial Pain with Radiofrequency Procedures

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INTRODUCTION

Chronic pain conditions arising in the head, face, or neck have been traditionally a confusing area with a lack of a universally accepted classification. Despite the little progress in the understanding of the pathophysiologic processes in the origin of cranio-facial pain, new drugs and surgical procedures have improved the treatment.

When the pain is limited to the distribution of a specific cranial nerve or its branch (the trigeminal, facial, glossopharyngeal and vagus), the term “typical neuralgia” is used, being the “classic or major trigeminal neuralgia, the most common.”

The term “atypical neuralgia” labels a group of facial pain conditions where the pain is not limited to a cranial nerve distribution and the borders of the various clinical conditions are not precise.

The diagnosis of a head or cranio-facial pain requires a comprehensive medical history and physical examination of the patient.

The pain characteristics—location, sensorial features, severity or intensity—associated factors that aggravate or relieve it and accompanying signs: sensory changes—hyper or hypoalgesia—motor disturbances and autonomic dysfunction—hyperhidrosis, lacrimation—or any other nervous system dysfunction, must be assessed and registered in detail by the clinician.

The examination of the patient must include the inspection of every external part of the head as well as the facial expression and position of the head. Palpation

must be oriented to determine trigger points, tenderness areas, tumors or tumefaction. The cervical area, including the muscles of the neck must be examined as it can be closely related to certain cranio-facial or head pain conditions, and a careful evaluation of the cranial nerves, the upper 3 cervical nerves and sympathetic innervation of the head is mandatory.¹

TRIGEMINAL NEURALGIA

Trigeminal neuralgia (TN) or Tic Douloureux is the most common cephalic neuralgia in people over the age of 50, with a mean incidence per annum of 4 per 100,000. Current data suggest that 60% of the patients are female, with a peak incidence between ages 50 and 70.² Onset of pain before age 40 is relatively uncommon and should at least suggest the possibility of multiple sclerosis.³

Trigeminal neuralgia can be divided into 3 categories: so called “idiopathic” trigeminal neuralgia, “atypical” trigeminal neuralgia, and “symptomatic” trigeminal neuralgia in patients with tumor involvement of the base of the skull or multiple sclerosis.

While in idiopathic trigeminal neuralgia, the neurological examination is virtually always normal, including imaging studies; the clinical entity of atypical trigeminal neuralgia differs in that in addition to the episodic lancinating pain, there exists a component of a more persistent aching or burning pain.⁴

Careful attention deserves sensory loss on the face, suggesting an extrinsic compressive lesion such as a tumor or vascular malformation.

HISTORY

Tic douloureux was the standard type of neuralgia for which neurolytic block was tried. Trigeminal neuralgia was treated for the first time by alcohol injection into the nerve by Pitres in 1902.⁴ He was followed by other

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authors who gave this technique a great deal of publicity. By 1905, Schlosser⁵ had reported 68 cases of severe trigeminal neuralgia successfully treated by alcohol nerve block. According to Cushing,⁶ Hartel was the first to block the Gasserian ganglion (GG) itself with alcohol.

Kirschner⁷ used diathermy of GG in the early 1930s for the relief of trigeminal neuralgia, Putnam and Hampton⁸ recommended x-ray control during the procedure, Haakanson in 1983⁹ advocated GG injection with glycerol and Sweet and Wepsic from 1969 through 1986¹⁰ developed percutaneous thermal retrogasserian rhizotomy for the treatment of trigeminal neuralgia.

ANATOMY

The trigeminal nerve is the largest one among the cranial nerves. Sensation of the oral mucosa, anterior and middle cranial fossa, tooth pulp, surrounding gingiva and periodontal membrane is maintained by the trigeminal nerve. It originates from the gasserian ganglion named after a Viennese anatomist, Johann Laurentius Gasser.

The ganglion lies within the cranium in a cave—Meckel's cavity—close to the apex of the petrous part of the temporal bone. Medially, the trigeminal ganglion is bounded by the cavernous venous sinus; superiorly, by the inferior surface of the temporal lobe of the brain; and posteriorly, by the brain stem.

Inferiorly the ganglion gives off 3 branches intracranially; the upper divisions, ophthalmic and maxillary and the lower division is the mandibular branch. The upper branches are sensory while the mandibular branch is partly motor. The trigeminal ganglion is somatotopically located. The ophthalmic branch is located dorsally, the maxillary branch intermediate and the mandibular branch ventrally.

GG contains sensory and motor fibers of the face, nasal and oral mucosae, teeth and anterior two-thirds of the tongue, and motor fibers for the masticatory muscles.

GG links with the autonomic nervous system via the ciliary, sphenopalatine, otic and submaxillary ganglia and communicates with the oculomotor, facial, and glossopharyngeal nerves.

PATIENT SELECTION

Indications

Idiopathic V cranial nerve Neuralgia.

Some secondary V cranial nerve neuralgias (eg, multiple sclerosis).

Alleviation of cancer pain in the head and neck.

Alleviation of acute trigeminal herpes zoster - Pulsed-radiofrequency (PRF).

Cluster headache management (PRF).

Contraindications

Neuropathic pain in the trigeminal area.

Local infection. Sepsis.

Coagulopathies.

Increased intracranial pressure.

Major psychopathology.

TECHNIQUE OF RF LESIONING

(Transforaminal approach)

Material.

SMK[®] cannula – 10 mm. 22 gauge. 0.2 cm active tip.

Patient's position.

Decubitus supine. Frontal-mentonian plane parallel to the table. Head securing to the table with lateral bands.

Anesthesia.

Intravenous (IV) sedation (propofol or Brietal). The patient must be able to collaborate when the stimulation test is performed.

Local anaesthesia of the zone to be punctured is not needed.

Anatomical references.

Entry point: 2-3 cm lateral to the corner of the mouth homolateral to the lesion.

Cannula insertion should be performed following the bisector (45°C) of the sagittal plane, which passes through the homolateral pupil and the frontal-mentonian plane.

X-Ray.

1. Submentovertex projection. Lateral inclination of approximately 30° towards the side of the lesion, with caudal inclination of approx. 30°. The mentonian arch must be seen and, in the upper-internal quadrant to it, the foramen ovale.
2. Lateral projection. Performed when the cannula has already been inserted into the foramen ovale. Its usefulness is to calculate the insertion of the cannula into the bony tunnel of the foramen ovale. The tip of the cannula must not exceed 2 mm in distance from the plane of clivus.

Risks.

Haemorrhage at the insertion site (perform compression).

Perforation of the oral cavity (to avoid this, the cannula should be guided with the index finger placed intra-orally).

Liquorrhage. Frequent. If abundant, dural puncture is produced. In such cases, continuation of the technique must be assessed. If cerebrospinal fluid (c.s.f) is not abundant, the technique may be continued.

Stimulation parameters.

Voltage 0-1 V.

Sensory - 50 Hz. Paraesthesia between 0.05 and 0.3 V must be noted in the painful zone.

Motor - 2 Hz. Little or no motor contraction of the masseter muscle must be present with 0.6-1V. If no motor contraction happens, the tip of the needle is positioned in the I or II branch of the V cranial nerve.

Lesion parameters.

1st lesion: 60 seconds at 65°C. When the lesion is induced, check the bilateral corneal reflex and pain sensitivity in the neuralgic and contralateral zones.

2nd lesion: 60 seconds at 70°C. Proceed in a similar manner.

3rd lesion: 60 seconds at 72°C to 75°C. Proceed in a similar manner.

A 4th lesion may be assessed at 75°C if pain involves 2 branches of the V cranial nerve.

COMPLICATIONS

Percutaneous interventions related with the trigeminal nerve are not free of complications. The complications of percutaneous lesioning are as follows: annoying dysesthesia and anesthesia dolorosa, loss of corneal reflex, neurolytic keratitis, visual loss, retrobulbar hematoma, hematoma in the cheek, significant motor root deficit, carotid puncture, and meningitis. Inadvertent intracranial placement of the electrode can result in intracranial hemorrhage. Penetration through the wrong foramen can cause defects in the other cranial nerves.

Comparison of complications among techniques

In selected series, Taha and Tew compared the results and complications of percutaneous techniques. Total number of patients were 6205 for radiofrequency (RF) rhizotomy, 1217 for glycerol rhizotomy and 759 for balloon compression.¹² Facial numbness occurred in 98% of the patients after RF rhizotomy, in 72% after balloon compression and in 60% after glycerol injection.

Anesthesia dolorosa occurred in 1.5%, 1.8%, and 0.1%, respectively.¹² According to other series anesthesia dolorosa occurred at a rate of 0.3% to 4% in RF lesioning¹³⁻¹⁵ and for glycerol injection from 0% to 2%.¹⁶⁻²⁰ For balloon compression, ipsilateral masticatory weakness, hypoesthesia, dysesthesia, anesthesia dolorosa may occur in 3% to 5% of the cases.²¹⁻²³

The overall incidence of corneal reflex loss and neurolytic keratitis is 0.6% to 1.8% depending upon the technique used. Corneal anesthesia was the highest for RF rhizotomy, 7%, less for glycerol, 3.7%, and 1.5% for balloon compression.¹²

Motor deficit occurs during the lesioning of the third branch, the mandibular branch. The incidence is the highest with balloon compression (66%), for RF rhizotomy (24%), and for glycerol injection (1.7%). The motor deficit improves within time (usually 1 year).¹²

If the needle is misadvanced to the retrobulbar space, retrobulbar hematoma may develop. This is a dramatic complication to the patient, although it is relieved by a conservative method without any sequelae. The eyeball is pushed from the retrobulbar space and exophthalmos develops. Compression over the eye will cease the bleeding. It subsides during the following days.

Hematoma in the cheek may develop, if the needle passes through a vessel while it is introduced. Compression over the cheek by cold pack after the needle is withdrawn may be helpful.

The incidence of infection from the Sweet series was 24 cases of meningitis in 7,000 cases of trigeminal RF lesioning. One of these patients died.²⁴ The possibility of ocular motor paralysis and cavernous sinus fistula²⁵ is a possibility as well as an intracranial hemorrhage²⁶ that has been reported to be fatal. Misplacement of needles into incorrect skull base foramina can lead to vascular damage and secondary hypertension that in turn can lead to bleeding.²⁷

THE OUTCOME OF RADIOFREQUENCY LESIONING; COMPARISON OF TECHNIQUES

The 3 most popular transforaminal techniques for treating trigeminal neuralgia: RF rhizotomy, retrogasserian glycerol injection and percutaneous compression of the GG, have several advantages and disadvantages.

The advantages of RF lesioning, as documented in a review of 25-year experience with 1600 patients seem to be high pain relief rate, low relapse rate, and effectiveness.²⁸ The sensorial deficit, which may lead to anesthesia dolorosa and annoy the patient, is the main drawback for RF lesioning of the trigeminal nerve.

There is slight sensorial deficit for retrogasserian glycerol injection, although shorter duration of pain relief, higher recurrence rate and fibrosis development, enhancing the entrance through the foramen ovale in further attempts are the main disadvantages.

Slight sensorial deficit and moderate rate of recurrence may be the advantages of gasserian ganglion compression. However, it can not be confined to a single branch and the gauge of the needle entering the foramen ovale is larger than the cannulae used in previous methods which may destroy the tissue itself.

The outcome parameters must be measured in terms of technical success, pain relief, recurrence, side effects and complications (facial numbness, dysesthesia, corneal anesthesia, keratitis, trigeminal motor dysfunction, permanent cranial nerve deficit, intracranial hemorrhage or infarction, perioperative morbidity, infection and perioperative mortality).

The technical success rate varies between 97.4% to 100% for RF lesioning at the initial phase. This rate is 94% for glycerol injection and 99% for balloon compression.¹² However in another study technical failure for glycerol was reported as high as 15%.¹⁷

Initial pain relief was highest with RF lesioning (98%), for glycerol rhizotomy it varied between 72% to 96%²⁹ and balloon compression relieved pain in 89.9% to 100%.^{12, 24}

To evaluate pain, recurrence is not easy because of the heterogeneity of the follow-up reported, but the highest rate of recurrence is 54 % for glycerol rhizotomy with a mean follow-up of 4 years.¹² Although in several series this varies between 18.5% to 72% over a follow-up period of 3 to 72 months. RF lesioning had a recurrence rate of 20% in 9 years and 15% in 5 years. For balloon compression, the recurrence rate is 55% to 77.4% at 36 months. The majority of recurrence occurs by 48 months.²⁹

In a recent retrospective study of a series of 108 percutaneous RF thermocoagulation performed in 81 patients, it has been reported an initial success rate of 87% and a pain-free status after 1, 2, and 11 years of 65%, 49%, and 26%, respectively. Patients with typical symptoms had a better long-term efficacy than those with atypical presentations, and patients who had not undergone a previous surgical procedure also had a better outcome.³⁰

New data comparing the long-term outcome after RF thermocoagulation and microvascular decompression (MVD) (Jannetta intervention) for the treatment of idiopathic trigeminal neuralgia, show that there was a 50%

risk for recurrence of pain 2 years after RF thermocoagulation. Conversely, 64% of patients who underwent MVD remained completely pain free 20 years postoperatively.³¹

Detailed analysis of the medical literature regarding the various techniques currently employed for the treatment of idiopathic or classic trigeminal neuralgia suggests that new, well-designed prospective clinical trials must be developed in order to establish the indications and limits of every technique and define specific candidates for each interventional approach.

SPHENOPALATINE NEURALGIA

Sphenopalatine neuralgia (Sluder's syndrome) consists of paroxysmal pains that begin on the medial side of the nose or medial canthus of the eye and radiate to the roof of the mouth, retroorbitally, or rarely to the ipsilateral neck, shoulder, and upper extremity.³²

Paroxysmal pain may be accompanied by unilateral lacrimation and conjunctival injection, being precipitated by sneezing or preceded by a sensation of nasal congestion.

Vidian neuralgia, or Vail's syndrome, is a variant of sphenopalatine neuralgia. It is a paroxysmal unilateral facial pain mediated by the vidian nerve, an afferent branch of the sphenopalatine ganglion. Pain may radiate backward into the ear, nape of the neck, and shoulder, and may be occasionally associated with tinnitus and vertigo.³²

HISTORY

In 1913, Greenfield Sluder published the first description of what he called "upper-half headache," and later sphenopalatine neuralgia, and its treatment by a transnasal sphenopalatine ganglion block (SPGB).³³

Through the following decades, medical literature showed little interest among clinicians in this approach, and during the last decade new interest have grown as a useful therapy for various cranio-facial pain conditions.³⁴⁻³⁷

ANATOMY

The sphenopalatine ganglion (pterygopalatine, nasal, or Meckel's ganglion) is located in the pterygopalatine fossa, posterior to the middle nasal turbinate, and is covered by a 1 to 1.5 mm layer of connective tissue and mucous membrane.

The pterygopalatine fossa is limited anteriorly by the posterior wall of the maxillary sinus, posteriorly by the medial plate of the pterygoid process, medially by the per-

pendicular plate of the palatine bone, superiorly by the sphenoid sinus, and laterally it communicates with the infratemporal fossa.³⁶

This neural structure has branches to the gasserian ganglion, trigeminal nerves, carotid plexus, facial nerve and the superior cervical ganglion. Only the parasympathetic fibers originating in the facial nerve are believed to synapse in the ganglion, sending postganglionic fibers to the lacrimal gland and the nasal and palatine mucosa via the maxillary, lacrimal, and zygomatic nerves, and to the palatine and nasal glands. An afferent sensory root connects the maxillary nerve to the ganglion by way of the branches of the maxillary nerve that extend from the nasopharynx, nasal cavity, palate, and orbit.

INDICATIONS

Sphenopalatine neuralgia
Migraine headache
Cluster headache
Pain in I and II distributions of V cranial nerve neuralgia

TECHNIQUE OF RF LESIONING³⁸

(Infrazygomatic arch approach)

Material.

SMK[®] cannula – 10 cm, 0.5 cm active tip. 20 or 22 gauge or SMK[®] cannula – 10 cm, curved, blunt tipped, with a 5-10 mm active tip.

Position.

Patient in supine decubitus position.
Head securing to the table with lateral bands.

Anaesthesia.

Mild I.V. sedation. Propofol I.V. if needed.
Local anesthesia of the zone to be punctured.
Patient's monitorization: E.K.G, pulsioximeter.

X-Ray.

1st lateral: Define the pterigopalatine fossa (PPF), sella turcica, clivus, petrous bone. The PPF is situated below the anterior portion of the petrous bone, which underlies approximately the clinoid apophysis anterior to the sella turcica. Insert the needle perpendicular to the skin as far as the PPF.
Using lateral x-ray, place a metallic marker above the PPF. Perform the puncture in the upper zone of the mandibular arch and progress perpendicularly until the patient notices paraesthesia in the jawbone.

2nd Antero-Posterior (AP): Vary the radioscope at an AP projection and advance the cannula medially until it is adjacent to the lateral wall of the nasal cavity. Insert the cannula 1-2 mm until it slides within the recess.

The tip of the needle should pass over the vomer bone by approximately 1-2 mm, with care taken not to pierce the partition between the nostrils.

Risks.

Lesion of the second branch of the V cranial nerve in the initial section of the puncture.

Stimulation parameters.

Voltage: 0-1 V.

Sensory: 50 Hz. 0.3-0.4 V. Considered positive when paraesthesia are achieved in the palate, and particularly in the nasal region. If the stimulation is only in the palate, insert the cannula slightly.

Motor: 2 Hz. Verify absence of maxillary contraction.

Lesion parameters.

Prior to performing the lesion, inject 1 mL of lidocaine 2%

1st lesion: 60 seconds 80°C, in the PPF.

2nd lesion: 60 seconds 80°C, somewhat more medial (1-2 mm).

3rd lesion: 60 seconds 80°C, somewhat more medial (3 mm).

Pulsed-RF can be used, a single lesion for 4 minutes or 3 lesions, 1 minute duration each in diverse locations, same as with conventional RF.

Comments.

Correct placement of the electrode is always controlled by impedance (normal, around 250 mΩ). If an air place (eg, nostrils) is reached, impedance increases significantly (between 800 and 1,000 mΩ).

Postoperative discomfort of 2 weeks' duration may ensue.

Some patients present decreased sensitivity in the soft palate.

COMPLICATIONS AND SIDE EFFECTS

Epistaxis is the major complication of this technique due to the vicinity in the PPF of the maxillary artery and its branches.

Hematoma of the maxillary area may develop if the venous plexus over the PPF is punctured.

Patients may experience orthostatic hypotension and should be monitored closely as reflex bradycardia can be developed during RF lesioning.³⁹

Numbness of upper teeth or hard palate, usually transient, can result after RF lesioning.

EFFICACY

Efficacy studies show that this procedure can be used as a second line alternative in patients who have failed pharmacologic or other surgical therapies.

Despite the new drugs introduced for the treatment of cluster headache and migraine, there are patients who fail to control their pain. SPGB has been utilized during the last decade for relieving such conditions with varying success. Sanders and Zuurmond reported the efficacy of SPGB in 66 patients suffering from cluster headaches, refractory to conventional therapies including surgery. They were treated by means of RF lesions at 70° C for 60 seconds, reporting complete pain relief during a mean follow-up of 29 months in 60% of patients with episodic cluster headaches and in 30% of patients with chronic cluster headache.³⁵

In another study, Salar and coworkers used RF lesions for the treatment of sphenopalatine neuralgia in 7 patients, reporting complete pain relief during a follow-up period from 6 to 34 months.³⁶

Controlled studies are needed to establish the indications of this procedure, the parameters of RF lesioning and the subsets of patients that could benefit from it.

GLOSSOPHARYNGEAL NEURALGIA

Glossopharyngeal or vagoglossopharyngeal neuralgia is a rare syndrome that consists of episodic bursts of pain in the sensory distribution of the ninth and tenth cranial nerves.⁴⁰

The pathophysiology of this type of neuralgia is unknown, however, like in trigeminal neuralgia, vascular cross-compression by ectatic vessels in the posterior cranial fossa has been suggested as a possible etiology.

Thus, microvascular decompression of the glossopharyngeal root (Jannetta's procedure) is the neurosurgical procedure of choice for intractable glossopharyngeal neuralgia.⁴¹

Scant literature has been found where RF lesioning had been used for the relief of this pain condition.⁴² This procedure is reserved for cases that have failed all the treatments for intractable glossopharyngeal neuralgia and in patients whose physical status precludes more invasive neurosurgical treatments.

OCCIPITAL NEURALGIA

Occipital neuralgia is much more common than other cranial neuralgias. It is characterized by pain in the distribution of the second (greater occipital nerve) or third (lesser occipital nerve) cervical dorsal root. Pain may occur together with paresthesias in the occipital nerve distributions, that is, unilaterally from the suboccipital area to the vertex (C2), or to the retromastoid and supra-auricular areas (C3).

The pathophysiology of occipital neuralgia is unknown, although it may be secondary to acceleration-deceleration injuries (whiplash) as well as local or systemic diseases. It has been advocated as a mechanism related to increased muscle activity in the cervical region, or entrapment of the C2 root or dorsal root ganglion by paravertebral ligamentous structures.⁴³⁻⁴⁷

CONCLUSION

Chronic pain arising in the head, face, or neck have been traditionally a confusing area with a lack of a universally accepted classification and a standardized treatment.

The outcome of current techniques used for the treatment of idiopathic or classic trigeminal neuralgia, indicates that new prospective clinical trials must be done in order to establish the proper place of RF procedures amongst the interventional techniques, as only in recent years clinical research in this area has grown beyond the stage on retrospective uncontrolled studies.

Recent advances in the knowledge of the physiology and anatomy of the cervical spine as well as the development of pulsed RF procedures have led to broaden the indications of RF technology in the treatment of atypical facial pain and cervicogenic headache. At present time, the results of outcome studies are not yet available.

REFERENCES

1. De Jong RN. *The Neurologic Examination*. New York, NY: P.B. Hoeber; 1958.
2. White JC, Sweet WH. *Pain and the Neurosurgeon*. Springfield, IL: Charles C. Thomas; 1969.
3. Burchiel KJ, Burgess JN. Facial and cranial pain. In: North RB and Levy RM, eds. *Neurosurgical Management of Pain*. New York, NY: Springer-Verlag; 1997:83-99.
4. Cusick JF. Atypical trigeminal neuralgia. *JAMA*. 1981; 245:2328-2329.
5. Swerdlow M. The history of neurolytic block. In: Racz GB, ed. *Techniques of Neurolysis*. Boston, Mass: Kluwer Academic Publishers; 1989: 1-12.
6. Cushing H. The role of deep alcohol injections in the treatments of trigeminal neuralgia. *JAMA*. 1920;75:441-443.

7. Kirschner M. Zur Electrochirurgie. *Arch Klin Chir.* 1931;161: 761–768.
8. Putnam TJ, Hamptom AO. The technique of injection into the Gasserian ganglion under roentgenographic control. *Arch Neurol Psychiat.* 1936;35:92–98.
9. Haakansson S. Retrogasserian glycerol injection as a treatment of tic douloureux. *Ad Pain Res Therapy.* 1983;5: 927.
10. Sweet WH, Wepsic JG. Controlled thermocoagulation of trigeminal ganglion and root for diferential destruction of pain fibers. *J Neurosurg.* 1974;39:143–156.
11. Apfelbaum RI. Trigeminal nerve and ganglion procedures. In: North RB, Levy RM (eds). *Neurosurgical Management of Pain.* New York, NY: Springer-Verlag; 1997:221–242.
12. Taha JM, Tew JM Jr. Comparison of surgical treatments for trigeminal neuralgia; Reevaluation of radiofrequency rhizotomy. *Neurosurgery.* 1996;38:865–871.
13. Broggi G, Franzini A, Lasio G, Giorgi C, Servello D. Long term results of percutaneous retrogasserian thermorhizotomy for “Essential” trigeminal neuralgia. *Neurosurgery.* 1990; 26:5:783–787.
14. Burchiel K, Steege T, Howe J, Loeser J. Comparison of percutaneous radiofrequency gangliolysis and microvascular decompression for the surgical management of tic douloureux. *Neurosurgery.* 1981;9:111–119.
15. Fraoili B, Esposito V, Guidetti B, Crucci G, Mangredi M. Treatment of trigeminal neuralgia by thermocoagulation, glycerolization and percutaneous compression of gasserian ganglion and or retrogasserian rootlets; long term results and therapeutic protocol. *Neurosurgery.* 1989;24:239–245.
16. Fujimaki T, Fukushima T, Miyazaki S. Percutaneous retrogasserian glycerol injection in the management of trigeminal neuralgia, long term follow-up results. *J Neurosurg.* 1990; 73:212–216.
17. Burchiel K. Percutaneous retrogasserian glycerol rhizolysis in the management of trigeminal neuralgia. *J Neurosurg.* 1988;69:361–366.
18. Wilkinson H. Trigeminal nerve peripheral branch phenol/glycerol injections for tic douloureux. *J Neursurg.* 1999; 90:828–832.
19. North RB, Kidd DH, Piantadosi S, Carson BS. Percutaneous retrogasserian glycerol rhizotomy; predictors of success and failure in treatment of trigeminal neuralgia. *J Neurosurg.* 1990;72:851–856.
20. Sweet WH, Poletti CE, Macon JB. Treatment of trigeminal neuralgia and other facial pains by retrogasserian injection of glycerol. *Neurosurgery.* 1981;9:647–654.
21. Lobato RD, Rivas JJ, Rosario S, Lamas E. Percutaneous microcompression of the gasserian ganglion for trigeminal neuralgia. *J Neurosurg.* 1990;72:546–553.
22. Belber CJ, Rak RA. Balloon compression rhizolysis in the surgical management of trigeminal neuralgia. *Neurosurgery.* 1987;20:908–913.
23. Brown JA, McDaniel M, Weaver MT. Percutaneous trigeminal nerve compression for treatment of trigeminal neuralgia: results in 50 patients. *Neurosurgery.* 1993;32:570–573.
24. Sweet WH. Complications of treating trigeminal neuralgia, an analysis of literature and response to questionnaire. In: Rovit RL, Janetta PJ (eds). *Trigeminal Neuralgia.* Baltimore, Md: Williams and Wilkins; 1990:251–279.
25. Sekhar LN, Heros RG, Kerber CW. Carotid cavernous fistula following retrogasserian procedures. *J Neurosurg.* 1979; 51:700–706.
26. Rish BL. Cerebrovascular accident after percutaneous radiofrequency thermocoagulation of the trigeminal ganglion. *J Neurosurg.* 1976;44:376–377.
27. Sweet WH, Poletti CE, Roberts JT. Dangerous risks in blood pressure upon heating of trigeminal rootlets, increased bleeding time in patients with trigeminal neuralgia. *Neurosurgery.* 1985;17:843–844.
28. Kanpolat Y, Savas A, Bekar A, Berk C. Percutaneous controlled radiofrequency trigeminal rhizotomy for the treatment of idiopathic trigeminal neuralgia: 25 years experience with 1600 patients. *Neurosurgery.* 2001;48:524–5534.
29. Burchiel K. Pain in neurology and neurosurgery. Tic douloureux (trigeminal neuralgia). *Pain.* 1996;68:41–60.
30. Yoon KB, Wiles JR, Miles JB, Nurmikko TJ. Long-term outcome of percutaneous thermocoagulation for trigeminal neuralgia. *Anaesthesia.* 1999;54:803–808.
31. Tronnier VM, Rasche D, Hamer J, Kienle AL, Kunze S. Treatment of idiopathic trigeminal neuralgia: comparison of long-term outcome after radiofrequency rhizotomy and microvascular decompression. *Neurosurgery.* 2001;48:1261–1267.
32. Aubry M, Pialoux P. Sluder’s syndrome. In: Vinken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology.* Amsterdam, The Netherlands: Elsevier North Holland; 1968:350–361.
33. Sluder G. Etiology, diagnosis, prognosis and treatment of sphenopalatine neuralgia. *JAMA.* 1913;61:1201–1216.
34. Levovits A, Alfred H, Lefkowitz M. Sphenopalatine ganglion block: clinical use in the pain management clinic. *Clin J Pain.* 1990;6:131–136.
35. Sanders M, Zuurmond W. Efficacy of sphenopalatine ganglion blockade in 66 patients suffering from cluster headaches: a 12 to 70 month follow-up evaluation. *J Neurosurg.* 1997;87:876–880.
36. Salar G, Ori C, Iob I. Percutaneous thermocoagulation for sphenopalatine ganglion neuralgia. *Acta Neurochir (Wien).* 1987;84:24–28.
37. Day M. Sphenopalatine ganglion analgesia. *Curr Rev Pain.* 1999;3:342–347.
38. Raj P, Rauck R, Racz G. Autonomic Nerve Blocks. In: Raj P, ed. *Pain Medicine: A Comprehensive Review.* St. Louis, Mo: Mosby; 1996.
39. Konen A. Unexpected effects due to radiofrequency thermocoagulation of the sphenopalatine ganglion: two case reports. *Pain Digest.* 2000;10:30–33.

40. Laha RK, Jannetta PJ. Glossopharyngeal neuralgia. *J Neurosurg.* 1977;47:316–320.
41. Fraioli B, Esposito V, Ferrante L. Microsurgical treatment of glossopharyngeal neuralgia. *Neurosurgery.* 1989;25:630–633.
42. Arbit E, Krol G. Percutaneous radiofrequency neurolysis guided by computerized tomography for the treatment of glossopharyngeal neuralgia. *Neurosurgery.* 1991;29:580–583.
43. Poletti CE. Proposed operation for occipital neuralgia: C-2 and C-3 root decompression. *Neurosurgery.* 1983;12:221–224.
44. Ehni G, Benner B. Occipital neuralgia and the C1-2 arthrosis syndrome. *J Neurosurg.* 1984;61:961–965.
45. Chambers WR. Posterior rhizotomy of the second and third cervical nerves for occipital pain. *JAMA.* 1954;155:431–432.
46. Koch D, Wakhloo AK. CT-guided chemical rhizotomy of the C-2 root for occipital neuralgia. *Neuroradiology.* 1992;34:451–452.
47. Lord S, Barnsley L, Wallis B, Bogduk N. Third occipital headache: a prevalence study. *J Neurol Neurosurg Psychiatry.* 1994;57:1187–1190.