A 75-year-old woman presented to the pain clinic with severe right-sided facial pain. A diagnosis of TN was made. Carbamazepine, baclofen, phenytoin, and gabapentin had been previously prescribed for the patient, with limited or no improvement in her symptoms. At the time of her visit to the clinic, she was unable to speak due to exacerbating pain in the right side of her face. The pain was mostly of mandibular and maxillary distribution, with some ophthalmic involvement; the eye was spared, however.

Her medical history was significant for multiple sclerosis that was present at remission, hypertension, hyperlipidemia, type 2 diabetes mellitus, and coronary artery disease. She had undergone multiple stenting procedures. Her ejection fraction was noted to be 45%. She had full dentition and a Mallampati class II airway.

CALL FOR CONTRIBUTIONS

If you have an idea for a CME lesson and would like to share it with the readers of Anesthesiology News, please e-mail Dr. Elizabeth A.M. Frost at ElzFrost@aol.com.

TARGET AUDIENCE
Anesthesiologists

NEEDS STATEMENT

Pain clinics, frequently, if not always, incorporate the services of an anesthesiologist. Trigeminal neuralgia (TN) is an extremely painful condition that has been shown to respond to several therapeutic measures. Approximately 30% of patients with TN are refractory to medical management and referred for surgical treatment. Ganglion ablation is a relatively new modality. Glycerol gangliolysis—a successful surgical treatment for TN—requires general anesthesia; there are associated operative risks of morbidity and mortality. Also, the procedural technique varies among institutions. The clinical anesthesiologist should be knowledgeable about the indications, contraindications, and possible complications of this procedure.

CASE HISTORY

A 75-year-old woman presented to the pain clinic with severe right-sided facial pain. A diagnosis of TN was made. Carbamazepine, baclofen, phenytoin, and gabapentin had been previously prescribed for the patient, with limited or no improvement in her symptoms. At the time of her visit to the clinic, she was unable to speak due to exacerbating pain in the right side of her face. The pain was mostly of mandibular and maxillary distribution, with some ophthalmic involvement; the eye was spared, however.

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CALL FOR CONTRIBUTIONS

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**Table 1. Differential Diagnosis in Facial Pain**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Pain Type</th>
<th>Pain Distribution</th>
<th>Pain Triggers</th>
<th>Other Clues</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trigeminal neuralgia</td>
<td>Paroxysmal, lancinating</td>
<td>Trigeminal only; V2 most frequent</td>
<td>None</td>
<td>History of trigeminal neuropathy</td>
</tr>
<tr>
<td>Glossopharyngeal neuralgia</td>
<td>Paroxysmal, lancinating</td>
<td>Ear, throat</td>
<td>Swallowing</td>
<td></td>
</tr>
<tr>
<td>Trigeminal neuropathic pain</td>
<td>Constant, burning, dull throbbing</td>
<td>Trigeminal only</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Postherpetic neuralgia</td>
<td>Constant, crawling; May have paroxysmal component</td>
<td>Trigeminal only; V1 most frequent</td>
<td>Touch</td>
<td>History of herpes zoster ophthalmicus</td>
</tr>
<tr>
<td>Anesthesia dolorosa</td>
<td>Constant, burning, itching in an insensitive region</td>
<td>Trigeminal only</td>
<td>None</td>
<td>History of trigeminal nerve lesion</td>
</tr>
<tr>
<td>Malignancy</td>
<td>Constant, but may have paroxysmal component</td>
<td>In area of neoplasm or referable to nerve compression</td>
<td>Possible if trigeminal nerve involved</td>
<td>Head/neck neoplasm</td>
</tr>
<tr>
<td>Altypical facial pain</td>
<td>Constant</td>
<td>Nonanatomic, often bilateral</td>
<td>None</td>
<td>Prominent psychiatric component</td>
</tr>
</tbody>
</table>


**Figure 1. Algorithm for the medical management of trigeminal neuralgia.**

BCF, baclofen; CBZ, carbamazepine; MVD, microvascular decompression; TN, trigeminal neuralgia

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innervation to the jaw, chin, lower lip, lower teeth, tongue, associated mucous, chewing, talking, etc. from the muscles of mastication. There is very little overlap among the divisions of the trigeminal nerve or between the trigeminal nerve and cervical dermatomes.6

The ophthalmic division enters the intracranial cavity through the superior orbital fissure, the mandibular division through the foramen rotundum, and the maxillary division through the foramen ovale. The divisions then enter the gasserian ganglion, which is located near the apex of the petrous bone in the middle cerebral fossa. The nerve roots then exit the gasserian ganglion and enter the trigeminal nucleus in thepons. The 3 sensory nuclei in the trigeminal complex are the mesencephalic nucleus, the main or principal trigeminal nucleus, and the spinal trigeminal nucleus. The mesencephalic nucleus receives large myelinated fibers from the muscles of mastication. Sensory information from the trigeminal nerve enters the pons, where the nerve divides into several distinct branches, which then further divide and enter specific sections of each nucleus related to proprioception, vibration, pressure, and pain and temperature sensation, respectively. From the pons, sensory information enters the ventral–posterior–medial (VPM) nucleus of the thalamus where it is relayed to the primary and secondary somatosensory cortices.8

**Etiology and Pathogenesis Of Trigeminal Neuralgia**

The etiology of TN has not been fully elucidated because of 2 still unanswered dilemmas. First, the painful paroxysms that characterize TN are not mediated by A delta- and C-fibers that normally transmit painful stimuli, but rather by A beta-fibers that are associated with the transmission of mechanical stimuli. Also, TN responds to some antiepileptic and antispasmodic drugs that do not have analgesic properties, while conventional analgesic drugs are usually ineffective. Second, it is unclear whether the origin of the pain is in the central or peripheral nervous system.9 Most of the clinical data appear to support a peripheral cause, while most of the experimental data support a central etiology.

Trousseau was the first to comment on the clinical similarities between TN and some types of epilepsy. He even suggested that TN be named “epileptiform neuralgia” because of the similarities between the paroxysmal discharges in the trigeminal system and cerebral paroxysmal discharges in epilepsy. Kugelberg and Lindblom demonstrated the presence of a measurable latency period between the onset of stimulus and an attack of TN. They also found that, once it began, an attack was self-sustained, and was often followed by a refractory period. It has been noted that lesions associated with TN usually affect the trigeminal roots near the pons. Tumors that affect the gasserian ganglion more commonly cause chronic pain and/or a sensory deficit. The only disease of the central nervous system with a definite association with TN is multiple sclerosis. Plaques have been found at the trigeminal root entry zone of the pons in patients with both diseases who have been examined at autopsy or during surgery.9

The production of epileptogenic lesions in the spinal trigeminal nucleus of animals by a variety of agents has been shown to cause a marked overreaction by touching the face of the animal, in addition to spontaneously occurring attacks of pain. The same agents applied to the gasserian ganglion did not produce pain. Experimental demyelination of trigeminal roots provoked repetitive action potentials that appeared to arise ectopically from the local areas of demyelination. However, the animals showed increased pawing and grooming of the face rather than contact avoidance. These data suggest a central (trigeminal nucleus) etiology rather than a peripheral (trigeminal roots) etiology for TN.2

Experiments to study the mechanism of action of drugs that clinically alleviate the symptoms of TN have proved useful. Carbamazepine and phenytoin depress the mecanoreceptor response of neurons in the trigeminal nucleus orals of cats with maximal nerve stimulation. In addition, these drugs enhance the afferent or segmental inhibition elicited by delivering a conditioning stimulus to the maxillary nerve prior to delivering the test stimulus. Such effects suggest that the mechanism of action of drugs that relieve the pain of TN involves the ability to facilitate segmental or afferent inhibition and depress excitatory transmission in the trigeminal nucleus. It also indicates that the failure of this inhibitory mechanism is a possible etiology of TN.20

According to Fromm et al., “It is likely that chronic compression or irritation of the trigeminal roots leads to a continued reduction in primary afferent depolarization. The efficiency of the segmental inhibition is diminished, resulting in neuronal hyperactivity and a paroxysmal response of interneurons in the trigeminal nucleus orals to tactile
stimulation of the face. If the paroxysmal discharge of these interneurons is severe enough, it may activate nociceptive interneurons in the nucleus oralis that in turn trigger nociceptive trigeminothalamic neurons in the nucleus caudalis, resulting in conscious perception of a paroxysm of trigeminal neuralgia.1,3

Currently, the most plausible explanation for the etiology of TN involves a combination of central and peripheral factors. Chronic irritation of the trigeminal roots starts a cascade of events resulting in initial failure of segmental inhibition in the trigeminal nucleus and increased activity in the trigeminal nerve as the result of increased ectopic action potentials. The combination of increased activity in primary afferent fibers and impairment of inhibitory mechanisms in the trigeminal nucleus leads to paroxysmal firing of interneurons in the trigeminal nucleus oralis in response to a tactile stimulus.3 Thus, TN appears to have a peripheral etiology and a central pathogenesis.

Medical Treatment of Trigeminal Neuralgia

Medical treatment of TN had been completely unsuccessful until 1940 when Bengoumian used phenytoin in response to Trousseau’s suggestion that the painful paroxysms of TN were similar to the paroxysmal discharges of cerebral origin found in epilepsy patients. The relative effectiveness of phenytoin led to the use of other antiepileptic agents for treatment. Thus far, carbamazepine and baclofen are the only drugs to have been subjected to a double-blind evaluation, and are currently the drugs of choice in the treatment of TN.3-13

Carbamazepine therapy is started at 100 mg to 200 mg bid. The dose is increased by 200 mg every other day until the patient is pain-free or side effects occur. At the usual dose of 600 mg to 1,200 mg per day, carbamazepine can control the paroxysms of TN within 24 to 48 hours. The most common side effects—drowsiness, dizziness, unsteadiness, nausea, and anorexia—occur in about 40% of patients and usually subside within a few weeks. About 10% of patients discontinue the drug because of continued or increased severity of side effects. Increasing the dose slowly can lessen the unwanted actions of carbamazepine, but often the pain of the attacks supersedes the side effects.3

There have been reports of aplastic anemia in patients taking carbamazepine.3 A complete blood count should be obtained from the patient before starting therapy, and repeated every 2 weeks for the first 2 months of therapy, and every 3 months thereafter. Rare side effects from carbamazepine include the syndrome of inappropriate antidiuretic hormone release, and congestive heart failure. Both resolve with discontinuation of the drug. Since TN is characterized by spontaneous remissions, carbamazepine should be tapered gradually after a patient has been symptom-free for 4 to 6 weeks, and resumed only if an exacerbation occurs.3

Baclofen therapy is started at 5 mg to 10 mg tid and increased by 10 mg every other day until the patient is symptom-free or side effects occur. The usual maintenance dose is 50 mg to 60 mg per day. The side effects associated with baclofen are not life threatening, as are some side effects of carbamazepine. The most common side effects of baclofen are drowsiness, dizziness, and gastrointestinal distress. As with carbamazepine, side effects can be minimized by starting at a low dose. About 10% of patients must discontinue baclofen medication because of the severe side effects. Baclofen should also be tapered and then discontinued if the patient remains symptom-free for 4 to 6 weeks. The abrupt cessation of baclofen can lead to hallucinations and seizures.14 Phenytoin was formerly considered the second drug of choice (after carbamazepine) for the treatment of TN; phenytoin has now been replaced by baclofen because only 25% of patients are relieved of symptoms with the use of phenytoin.3 It is now used as an adjunct to baclofen in patients who cannot tolerate carbamazepine. Common side effects of phenytoin include swollen and bleeding gums, which may result in hypertrophy (sufficient to interfere with maintenance of the airway under anesthesia) and difficult intubation. At very high dosages, phenytoin may cause diplopia and dizziness, although 10% to 15% of patients develop these symptoms at subtherapeutic levels.3

Recommendations for the medical treatment of patients with TN generally start with baclofen because of its relatively mild side-effect profile. If baclofen is ineffective or its side effects cannot be tolerated, then the patient is started on carbamazepine. If carbamazepine is ineffective or its side effects are too severe, a combination of baclofen and phenytoin, or other medications such as gabapentin, can be used. In cases of failed medical therapy, surgical cures are attempted. Unfortunately, some patients who undergo surgical therapy experience a recurrence of paroxysms and are then started on another trial of medical therapy. Figure 1 illustrates an algorithm for the medical management of TN.

Surgical Procedures

Approximately 30% of patients are refractory to medical management and referred for surgical treatment.35 Surgical procedures for TN can be divided into minor and major procedures. Minor surgical procedures include alcohol block, alcohol gangliolysis, peripheral neurectomy, glycerol gangliolysis, and radioregulation gangliolysis. Of these, glycerol gangliolysis has been found to be superior for long-lasting pain relief, minimal or no subsequent neurologic deficit, and safety of administration. Major operative procedures include microvascular decompression, rhizotomy, and medullary tractotomy. These techniques require general anesthesia and are associated with operative risks of morbidity and mortality.3 Table 2 outlines an outcomes algorithm for the medical management of TN.

Table 2. Outcomes Analysis for Surgical Resolution of Trigeminal Neuralgia

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Initial pain relief</th>
<th>Recurrence at 2 years</th>
<th>Complications:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glycerol Gangliolysis, %</td>
<td>Initial pain relief</td>
<td>Recurrence at 2 years</td>
<td>Masticator weakness</td>
</tr>
<tr>
<td>Balloon Compression, %</td>
<td>Initial pain relief</td>
<td>Recurrence at 2 years</td>
<td>Dysthesias</td>
</tr>
<tr>
<td>Radiosurgery, %</td>
<td>Initial pain relief</td>
<td>Recurrence at 2 years</td>
<td>Corneal anesthesia</td>
</tr>
<tr>
<td>Microvascular Decompression, %</td>
<td>Initial pain relief</td>
<td>Recurrence at 2 years</td>
<td>Keratitis</td>
</tr>
<tr>
<td>Mortality</td>
<td>Initial pain relief</td>
<td>Recurrence at 2 years</td>
<td>Anesthesia dolorosa</td>
</tr>
<tr>
<td></td>
<td>Initial pain relief</td>
<td>Recurrence at 2 years</td>
<td>Cranial nerve deficit</td>
</tr>
<tr>
<td></td>
<td>Initial pain relief</td>
<td>Recurrence at 2 years</td>
<td>Ipsilateral hearing loss</td>
</tr>
<tr>
<td></td>
<td>Initial pain relief</td>
<td>Recurrence at 2 years</td>
<td>Cerebrospinal fluid leak</td>
</tr>
<tr>
<td></td>
<td>Initial pain relief</td>
<td>Recurrence at 2 years</td>
<td>Mortality</td>
</tr>
</tbody>
</table>

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analysis for some of the surgical procedures for TN. A neurosurgeon, S. Hakanson, was the first to report lasting relief of pain for patients with TN by the injection of glycerol into Meckel’s cistern. The pain relief was associated with a further 19% to 23% with supplemental medication. There is now evidence that glycerol may preferentially exert its effects on the damaged large, myelinated fibers. The procedural technique for glycerol gangliolysis varies among institutions. Essentially, a spinal needle is introduced into Meckel’s cistern under fluoroscopic guidance and general anesthesia. After cerebrospinal fluid is seen flowing into the needle, pure glycerol is injected into the space. Figure 2 illustrates placement of the spinal needle. The patient is then awakened and asked about current pain, relative to previous pain. The presence of eye pain is specifically sought to determine whether the ophthalmic division of the trigeminal nerve has been damaged. Facial pain peaks after the injection and then diminishes within minutes. Overall, 89% to 96% of patients can expect complete relief of pain from this procedure.14 Patients with multiple sclerosis appear to respond well.15 However, 7% to 10% of patients experience an early (within 6 months) recurrence of pain. After 6 months, recurrence rates are low. After 3 injections, both for patients with recur- rent pain yields lasting remission in 68% to 77% of patients.16 The long-term success of glycerol injection ranges from 67% to 80% of patients without medication, and a further 19% to 23% with supplemental medication. Therefore, 90% to 99% of patients get good results from glycerol injection.17,18 Facial and corneal sensation loss is minor in most cases and gradually subsides over time.19 As with most neurodestructive procedures, about 25% of patients may experience an erosion of herpes simplex virus within the affected area—due mainly to the destruction of compo- nents of cell-mediated immunity in the area.20

Preeanesthetic Assessment

A preeanesthetic assessment of patients undergoing surgical procedures for the resolution of TN includes a thorough review of drug regimens and a neurologic assessment. Most patients with TN are on high doses of carbamazepine and/or phenytoin.21 As noted previously, patients on phenytoin therapy may experience swollen, bleeding gums that can complicate airway management. Carbamazepine is a known inducer of cytochrome P450 enzymes, which can alter the metabolism and duration of action of many anesthetic drugs, including volatile agents, narcotics, local anesthetics, and benzodiazepines.22 All patients should have a complete labora-tory workup including blood count, chemistries, and liver function tests to check for evidence of hematopoetic, renal, and hepatic dysfunction.23 In patients on baclofen therapy, there have been reports of severe bradyarrhythmia and hypoten-sion under general anesthesia—perhaps because of the potentiation of gamma-aminobutyric acid (GABA) receptors; these signs can be treated with ephedrine. Emergency drugs, such as ephedrine, phylephrine, and atropine, should be immediately available, similar to all anesthetics. Currently, discontinuation of baclofen therapy (via a gradual withdrawal) is recommended before the administration of anesthetics to avoid the hemodynamic problems mentioned previously.24 Also, most of these patients will be on high-dose opioid and anxiolytic therapy that should be tapered, if tolerable to the patient, before surgery. The patient should understand that the resolution of pain is not immediate in all cases and that he or she may experience pain on awak-en-ing.25 Neurologic assessment includes checking for any loss of protective airway reflexes and the need for possible post-operative intubation, in addition to careful documentation of any pre-existing neuropathies.26

Anesthetic Considerations

The most important aspect of anesthetic management is the requirement for intermittent awakening of the patient between injections in order to assess both the effectiveness of the injections and any corneal or eye damage. An intra-venous anesthetic technique using propofol and remifen-tanil is now used.27 Remifentanil is a short acting opioid that is noncumulative.28 It has a short duration of action, and precise and titratable effects due to rapid onset and offset; it is also noncumulative.29

Case Management

Because of the patient’s multiple medical problems and age, she was started on a low dose of both propofol (25 mcg/kg per minute) and remifentanil (0.02 mcg/kg per minute). A 20-mg bolus of propofol was given to allow the patient to tolerate placement of the needle in Meckel’s cis- tern. The dose of remifentanil was then increased as the injection proceeded, to a maximum of 40 mcg/kg bolus (facilitated by the injection of glycerol). After the third wake-up test, 100 mcg of fentanyl was titrated in 25-mcg boluses as allowed by the respiratory rate for postoperative pain control. During this type of proce- dure, the means to emergently secure the airway (including a laryngeal mask airway) should always be available as apnea may occur at any time; the risk of rapid and life-threatening desaturation can be managed with the place- ment of an airway and positive pressure ventilation.

References
Lesson 238: Post-test

Select the single-letter response that most correctly answers the question or completes the sentence.

1. The least likely pathognomonic diagnostic feature of trigeminal neuralgia (TN) is:
   a. paroxysmal pain with pain-free intervals.
   b. no objective clinical findings.
   c. distinct pathologic findings postmortem.
   d. pain restricted to the area of the trigeminal nerve.

2. Which of the following is false regarding the diagnosis of TN?
   a. Pain is characterized as paroxysmal and lancinating.
   b. Pain occurs in the distribution of the trigeminal nerve only.
   c. Pain is triggered by touching, chewing, and talking.
   d. Nonspecific triggers frequently elicit pain.

3. Trigeminal neuralgia:
   a. has been associated with multiple sclerosis.
   b. has no latency period between the onset of stimulus and onset of attack.
   c. never involves the mandibular branch of the trigeminal nerve.
   d. occurs mostly in men older than 40.

4. The fifth cranial nerve:
   a. has 4 divisions.
   b. carries both motor and sensory fibers.
   c. supplies all sensation to the entire head.
   d. is the primary source of motor innervation to the face.

5. Which of the following is a true statement about the etiology and pathogenesis of TN?
   a. The painful paroxysms that characterize TN are transmitted by A delta- and C-fibers.
   b. The etiology of TN has not been fully elucidated.
   c. TN arises from a central rather than peripheral etiology.
   d. Most clinical evidence suggests that TN arises equally from peripheral and central sites.

6. Which of the following is a true statement about the medical management of TN?
   a. Carbamazepine and phenytoin are no longer the drugs of choice.
   b. The most common side effects of baclofen therapy are drowsiness, dizziness, and gastrointestinal distress.
   c. Phenytoin has frequently been associated with reports of aplastic anemia.
   d. Carbamazepine does not affect the cytochrome P450 system.

7. Which of the following is a false statement?
   a. Glycerol gangliolysis has the least number of complications.
   b. Minor blocks are the least invasive and should be the first line of treatment for TN.
   c. Radiofrequency gangliolysis is, initially, the least effective.
   d. Of the pharmaceutical agents used to treat TN, baclofen is the most likely to be associated with life-threatening side effects.

8. Which of the following is a true statement concerning surgical procedures for TN?
   a. Glycerol gangliolysis is the most likely to be associated with life-threatening side effects.
   b. Carbamazepine controls painful paroxysms usually within 24 to 48 hours.
   c. Carbamazepine controls painful paroxysms usually within 24 to 48 hours.
   d. Carbamazepine does not affect the cytochrome P450 system.

9. Which of the following is a true statement regarding remifentanil?
   a. It is equipotent with fentanyl.
   b. An amide linkage makes it a unique opioid.
   c. Cumulative effects are troublesome after long infusions.
   d. It has a long duration of action.

10. For patients undergoing glycerol gangliolysis, the treating clinician must have:
   a. short-acting angesic agents available.
   b. awareness of all current drug therapies.
   c. complete laboratory profiles of patients including hematopoietic, hepatic, and renal evaluations.
   d. all of the above.

To receive CME credit, you must complete this form, including the time attestation and evaluation, score ≥70% on the quiz, and return the form with a check for $10 made payable to MSSM-Anesthesia before February 28, 2006, to Mount Sinai School of Medicine, Department of Anesthesia, One Gustave L. Levy Place, Box 1010, New York, NY 10029. Credit will be awarded only for lessons completed with a signed time attestation that are postmarked before the expiration date.

1. a  b  c  d
2. a  b  c  d
3. a  b  c  d
4. a  b  c  d
5. a  b  c  d
6. a  b  c  d
7. a  b  c  d
8. a  b  c  d
9. a  b  c  d
10. a  b  c  d

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1. This lesson met the stated objectives.
   Strongly Agree  Agree  Disagree  Strongly Disagree

2. This lesson met my expectations.
   Strongly Agree  Agree  Disagree  Strongly Disagree

3. This lesson enhanced my knowledge.
   Strongly Agree  Agree  Disagree  Strongly Disagree

4. This lesson improved my skills.
   Strongly Agree  Agree  Disagree  Strongly Disagree

5. I am satisfied with the content of this lesson.
   Strongly Agree  Agree  Disagree  Strongly Disagree

6. The information I gained from this lesson will be applicable to my practice.
   Strongly Agree  Agree  Disagree  Strongly Disagree

7. The information I gained from this lesson will assist me in improving the health of my patients.
   Strongly Agree  Agree  Disagree  Strongly Disagree

8. This lesson was free of promotional and commercial bias.
   Strongly Agree  Agree  Disagree  Strongly Disagree

9. The format used for this lesson was appropriate.
   Strongly Agree  Agree  Disagree  Strongly Disagree

10. Which parts of the lesson were most useful?

11. Were any parts of the lesson unsatisfactory or inappropriate? If so, which?

12. What topics would you like to see included in future programs?

   __________________________________________________________

13. Were you able to call the CME office at (212) 241-4441 or e-mail Josephine.Greene@mssm.edu if you required administrative assistance for a question about content?
   __ Yes          __ No          __ Not Applicable

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