

Percutaneous Balloon Compression for Trigeminal Neuralgia

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The purpose of this article is to review the history of the development of balloon compression, the scientific basis for performing it, and details regarding the current technique, results, pitfalls and morbidity associated with the surgery.

Neurosurgeons first conceived of minimal access, percutaneous approaches to treat trigeminal neuralgia in the early 20th century.³⁰ In 1910, Harris injected the gasserian ganglion with alcohol.³⁰ Four years later, in 1914, Haertel described a technique for percutaneous injection through the foramen ovale that is still used today.³⁰ Kirschner built an early stereotactic apparatus that he used to accurately locate the foramen and electrically coagulate the ganglion.⁹ Sweet improved on Kirschner's coagulation technique.³² In 1950, he described a percutaneous technique of injection using radiographic imaging. He then developed an approach that he believed created selective, controlled thermocoagulation lesions using a radiofrequency generator that he designed in association with Radionics, Inc. (Burlington, MA). This technique became most frequently used percutaneous approach to treat trigeminal neuralgia for the next two decades.

In the 1950s, neurosurgeons believed that trigeminal neuralgia occurred because of scar tissue that compressed the nerve root or ganglion in the middle fossa. Taarnhoj³³ thought that the dural canal over the margin of the petrous ridge, the porous trigeminous, could be the source. Sheldon et al.²⁷ reviewed the results of a series of patients on whom they had operated by decompressing either the maxillary or mandibular division at the exiting foramina or at the root level. Love and Svien^{17,31} realized that the surgical outcome was better when the surgery caused facial numbness. Thereafter, neurosurgeons treated trigeminal neuralgia by performing middle fossa craniotomies to directly rub or compress the ganglion.⁴

Early in his career, Mullan described a percutaneous technique for cervical cordotomy using a strontium needle and later a radiofrequency lesion.^{21,22} Then, in 1983, he reported a clinical series using a percutaneous modification of Taarnhoj's operation performed by inserting a transforaminal balloon to squeeze the nerve fibers.²³ Since this first report,

the results of more than 1,000 cases have been evaluated in the world literature.³

Trigeminal neuralgia, *tic douloureux*, or, in the past, *spasme cinique* is a neuropathic pain syndrome. It is unique among neuropathic pain syndromes because it is treatable by an ablative surgical technique and is not associated with easily detectable numbness. The trigger point for trigeminal pain may represent a zone of hyperesthesia generated by focal injury caused by vascular compression of the nerve root. Jannetta argued that careful physical examination elicits a region of hypoesthesia in trigeminal neuralgia patients. Nerve injury at the site of vascular compression is the likely cause. Peripheral ablation is effective in relieving pain because it causes a distal anesthetic block. After the trigeminal neuralgia pain has become neuropathic, that is, when the compression is sufficient to cause demonstrable numbness or significant constant burning, peripheral ablation should no longer be performed. Additional nerve injury, even if peripheral, at this point may add to the degree of dysesthetic pain. This is especially true if the pain no longer has an intermittent, severe electric shock element triggered by a peripheral stimulus.

If trigeminal neuralgia is a progressive disease, then it is better to perform percutaneous ablation during the early phase of the disease. This is the phase before there is permanent clinical nerve injury, experienced as burning dysesthesia. The burning sensation may be quite bothersome, but is not as severe as the sudden, lightning bolts of pain felt during the hyperesthetic early phase.

Biopsy specimens of trigeminal roots at the site of compression obtained during microvascular decompression surgery for clinical trigeminal neuralgia demonstrate axonal loss, demyelination and dysmyelination, myelin debris, excess collagen, and closely opposed axons without intervening glial processes. Such findings are consistent with a hypothesis that trigeminal neuralgia occurs because of changes in the electrical excitability of abnormal afferent trigeminal fibers. These findings also support the idea that trigeminal neuralgia is a neuropathic entity.¹¹

Balloon compression causes anatomic injury different from that caused by thermal or chemical injury. An under-

standing of the injury caused by balloon compression helps one to select the most appropriate surgical option.

Trigeminal balloon compression causes a trigeminal depressor response. When a rabbit's trigeminal ganglion is percutaneously compressed with a no. 2 embolectomy catheter, this depressor response is reproduced. In this model, compression preserves ganglion cells. The perikaryonal distribution of neurofilaments in compressed and control trigeminal ganglia is the same. Three months after compression, there are still focal demyelination and Schwann cell proliferation in the trigeminal sensory root. Such proliferation precedes remyelination. Cell size measurement and immunocytochemical study show that the number of small ganglion cells and fine-caliber primary afferent fibers is unchanged. Transganglionic degeneration is most dense in the deeper brainstem layers of the subnucleus caudalis. These are the termination sites of large myelinated fibers. Injured areas of the nerve had higher numbers of surviving small ($<3\text{-}\mu\text{m}$ diameter) axonal fibers. This differential injury of axons and sparing of trigeminal ganglion cell bodies suggest that axonal regeneration is possible and may contribute to the recovery of motor and sensory function in patients after percutaneous balloon compression.^{5,25}

Selective preservation of unmyelinated, pain-responsive fibers seems to be an unlikely goal for pain treatment. It is easily explained, however. Balloon compression relieves trigeminal pain by injuring the large myelinated axons involved in the sensory trigger. This is consistent with what patients report. Touching the face in a hyperesthetic zone or exposing it to a cool breeze, for example, triggers the trigeminal pain. This is a large fiber-mediated sensation. Unmyelinated pain fibers mediate nociceptive pain transmission, but they are not the source of the sensory trigger. It is not necessary to injure tissue for trigeminal pain to occur. This is the nature of a neuropathic pain syndrome.

Patient Selection

Which patients are candidates for trigeminal balloon compression? Those patients with Burchiel type 1 trigeminal neuralgia are candidates for percutaneous balloon compression.⁷ First, try anticonvulsant therapy. The most commonly used medications are carbamazepine, gabapentin, diphenylhydantoin, and oxcarbazepine. Pain relief with these drugs, at the appropriate dose, helps to confirm the diagnosis of trigeminal neuralgia. There is no identified drug level to assist in deciding whether the dose is adequate. Experience and empirical observation are required. How long medical therapy should continue before one considers having an ablative procedure depends on the patient's tolerance of the medication or of the concept of a lifetime of medical treatment. Trigeminal neuralgia does not resolve with medical treatment. Pain recurs after medical therapy ceases. Because the disease is progressive, dose escalation occurs. Disturbing

additional effects from the anticonvulsant medications emerge. These include signs of cognitive dysfunction such as drowsiness, inattention, diminished reaction time, and speed of information processing.¹²

Patients must be able to tolerate light general anesthesia. Because the median age at diagnosis of trigeminal neuralgia is 65 years, there are many patients taking aspirin, warfarin, or clopidogrel (Plavix). Reverse the effects of blood thinners before surgery. Contralateral jaw weakness is not a contraindication, despite the expectation of temporary masseter and pterygoid muscle weakness.

Brief bradycardia occurs during compression, and there is often reflex hypertension. Preoperative evaluation should include an electrocardiogram to identify those patients with cardiac arrhythmias. Stabilize hypertension. Patients with a history of cold sores may take oral acyclovir to alleviate a postoperative outbreak. There is, however, no clinical evidence to support the effectiveness of doing this.

Operative Technique

The operation is easiest to perform in the operating suite using a portable fluoroscope for imaging (*Fig. 14.1*) Imaging is easier, however, in the radiology suite using multiplanar imaging. Continue oral anticonvulsant agents and infuse prophylactic antibiotics. The likelihood of oral puncture and contamination is low. In my experience, it has only happened on one occasion in 25 years.

Induce light general anesthesia, and then place an external pacemaker. Set the pacemaker to trigger automatically should the heart rate drop to less than 45 beats per minute during compression. The anesthesiologist should confirm capture by the pacemaker before proceeding. The external pacemaker blocks the bradycardia faster than intravenous atropine can be infused. If the bradycardia persists during compression, administer 0.4 mg atropine intravenously. Draw the atropine into a syringe to prepare for this situation. This depressor response consists of both bradycardia and brief hypotension, often with reflex hypertension after triggering

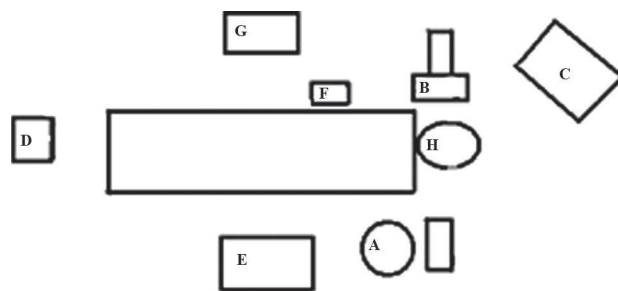


FIGURE 14.1. Operating room setup for a patient with right-sided trigeminal neuralgia. Surgeon (A), image intensifier (B), image intensifier screen (C), external pacemaker (D), equipment table (E), digital pressure monitor (F), anesthesia equipment (G), patient's head (H).

the pacemaker. Preoperative atropine inhibits the depressor response but limits its use as an indication of adequate nerve compression.

Place the patient in the supine position and place a cloth roll under the shoulders to keep the neck in neutral position. Some surgical tables have a steel bar at the head of the table. Remove this table piece. The bar interferes with the modified submental image and obstructs the view of the foramen ovale. Rotate the head 15 to 30 degrees to the opposite side. Because older patients often have degenerative cervical spondylosis, further extension and rotation may not be possible. Prepare the perioral region with a sterile solution. Lubricate and tape the eyes closed, then surround the puncture site with plastic drapes. Mark the entry point on the cheek 2.5 cm lateral to the angle of the lip in a skin crease. For first-division pain treatment, this mark is set slightly more lateral. Position a digital pressure monitor (Merit Medical, South Jordan, UT) on an adjacent intravenous pole located at waist level opposite the surgeon. Drape the fluoroscope imaging units on the side away from the surgeon. Place a half-sized drape over the unit on the side of surgery. That side will not remain sterile because of the need to rotate it to obtain submental and anteroposterior images. Fill the insufflation syringe (Merit Medical) with radiographic dye.

A U.S. Food and Drug Administration–approved kit for this procedure is available. It includes sharp and blunt trocars, an introducing cannula, curved and straight guiding stylets, and a no. 4 balloon (Cook Vascular, Inc., Leechburg, PA). The insufflation syringe connects to the balloon catheter and measures intraluminal pressures in atmospheres. The target intraluminal pressure is 1.3 to 1.5 atm. It is not essential to measure intraluminal pressure. Inflate the no. 4 balloon catheter with 0.75 to 1.0 mL of contrast until a pear shape is observed. Pressure measurement does provide an additional guide to measure the appropriate degree of compression delivered to the trigeminal nerve root. If the patient is allergic to the imaging dye used, premedicate with steroids to reduce any risk from rupture of the balloon.

The first image viewed is the lateral image. Set the entry point of the cannula so that that it approaches the radiographic intersection of the clivus and petrous bone, in line with the angle of the petrous bone. This is the approach for a patient with pure third-division pain. For maxillary-division pain, select a more oblique angle. For first-division pain, the oblique angle is even wider. Hold the 14-gauge cannula with the sharp obturator inserted against the cheek and obtain a lateral fluoroscopic image. Adjust the superior-inferior site of insertion to obtain an insertion path consistent with the targeted division. Then, nick the cheek with a no. 11 blade. The incision should accommodate the cannula easily without requiring a suture at the end of the procedure. Slide the 45-degree sharp obturator into the cannula and use it to puncture the skin of the cheek. Replace the sharp obturator

with the blunt one. Understand that the 14-gauge cannula *does not pass beyond the foramen ovale*. Only the guiding stylets and balloon catheter pass into the cranial cavity.

After the cannula reaches the skull base, obtain a submental view. Identify the foramen ovale on the image intensifier. Advance the cannula using repeated fluoroscopic submental images. Align the approach angle with that of the image intensifier. Direct the imaging unit at an angle of approximately 30 degrees under the chin. Rotate the head 15 degrees to the opposite side. The view shows the foramen ovale. The foramen ovale is medial to the mandible, lateral to the maxilla, and directly above the petrous bone (Fig. 14.2). The depressor response briefly occurs when the cannula engages the foramen ovale. There is some resistance on the cannula while it engages. The cannula should not penetrate any further. No cerebrospinal fluid drains because the cannula has not entered the subarachnoid space surrounding the trigeminal ganglion. This is different from the expectations regarding appropriate positioning that occurs with glycerol and thermal rhizotomy. Once the cannula has engaged the foramen ovale, remove the blunt obturator and insert a straight guiding stylet. Venous bleeding occurs if the cannula is outside the foramen where there is an epidural venous complex. If so, it will stop when the cannula engages the foramen ovale. Insert a straight guiding stylet until it passes beyond the edge of the cannula and the inner aspect of the foramen ovale. There is a “pop,” similar to the tactile feeling when puncturing the dura during a lumbar puncture. If there



FIGURE 14.2. Modified submental image showing a cannula penetrating the right foramen ovale. The foramen is visible superior to the petrous ridge, medial to the mandible, and below the maxillary sinus.

is excessive resistance to advancement of the stylet, then the cannula is not in the correct position.

Obtain an anteroposterior view. Center the petrous bone in the ipsilateral orbit in this image. There is a dip in the petrous bone that is the proximal entrance to Meckel's cave, the porous trigeminus. Direct the stylet to the center of the porous for second-division pain or multidivisional pain. Direct it toward the lateral porous for third-division pain. Direct it toward the medial porous for first-division pain (*Fig. 14.3*). In patients with predominantly first-division pain, aim toward a more lateral to medial position to enable the stylet to pass into the medial segment of the porous. The entrance to the porous is located approximately 17 mm beyond the foramen ovale. When the cannula sits at the foramen ovale, it is not possible to pass the guiding stylet beyond the porous trigeminous into the posterior fossa. The curved stylet allows mobility within the porous to reach the medial or central aspect. Direct the curve caudally during passage through the cannula. Rotate the tip superomedially after it is beyond the cannula. This maneuver decreases the risk of dural perforation. If the dura is perforated, redirect the stylet to remain interdural so that successful balloon compression may occur. The only indication that dural perforation has occurred may be that a pear shape fails to appear during inflation despite the anteroposterior and lateral images suggesting proper balloon position.

Obtain a lateral view. To place the balloon catheter at the best angle for third-, multidivisional, or first-division pain, it is best to set the imaging beam parallel to the skull



FIGURE 14.3. Anteroposterior radiographic intraoperative image showing the position of the straight inner guiding stylet within the 14-gauge cannula when properly positioned for treatment of second-division pain. The tip of the stylet is at the center of the dip in the petrous bone representing the entrance to Meckel's cave. The balloon catheter will be positioned along the same path with its tip approximately 2 mm beyond the petrous bone edge, as seen through the orbit on the image intensifier.

base. Superimpose the planum sphenoidale and posterior clinoids on the fluoroscopic image to give the best view of the sella turcica. This view will accurately show the cannula tip at the floor of the middle fossa. Use the lateral view during the balloon compression. For second- or third-division pain, the stylet is parallel and adjacent to the petrous bone. For first-division pain, the stylet lies more above the petrous bone. Once the proper trajectory is set, remove the stylet. Place the balloon catheter in the same place as the guiding stylet. The catheter has a thin inner wire that identifies its position.

For third-division pain, use the anteroposterior image intensifier view to place the tip of the balloon catheter approximately 2 mm beyond the edge of the petrous bone. For first-division pain, insert the catheter slightly farther, another millimeter deeper. This is because the catheter is positioned superomedially in the nerve to reach the first-division fibers. These fibers are in the superior portion of the trigeminal root. Third-division fibers are in the inferior trigeminal root. The balloon is traversing the root obliquely from the fixed entry site at the foramen ovale.

Once properly positioned, remove the stylet within the catheter and slide the blocking device down to the cannula, firmly securing it on the balloon at the edge of the cannula. This maneuver prevents the balloon from sliding into the posterior fossa when it is inflated. Evacuate air from the balloon with a tuberculin syringe connected to a 3-way stopcock. The balloon is then connected to the insufflation syringe and then to the monitor. Allow it to stabilize at a zeroed pressure.

Slowly inflate the balloon while repeatedly monitoring blood pressure and observing intermittent lateral fluoroscopic images. When the balloon inflates within the porous, the characteristic pear shape appears on the lateral view. If the catheter tip is not within the porous, then the characteristic pear shape does not occur, there is less numbness, and mild pain relief is limited to the third division. Proximal to the porous, the balloon lifts the dura off the ganglion. Within the porous, it compresses the retrogasserian fibers against the firm edge of the dura and the petrous ridge as the dura splits, allowing the nerve to pass into Meckel's cave. There is no danger if the balloon ruptures.

When properly inflated, using the catheter provided in the kit, the intraluminal balloon pressure is 1.3 to 1.6 atm (*Fig. 14.4*). After reaching the target pressure and observing a pear shape, leave the balloon inflated for 1 minute. Leave it inflated longer if you seek more numbness. The depressor response occurs. This briefly triggers the pacemaker. There may be a secondary increase in blood pressure, especially after triggering the pacemaker. Control this hypertensive response with additional anesthetic. Deflate the balloon and remove the catheter and cannula together. This prevents tearing the balloon. Blood-tinged cerebrospinal fluid drips

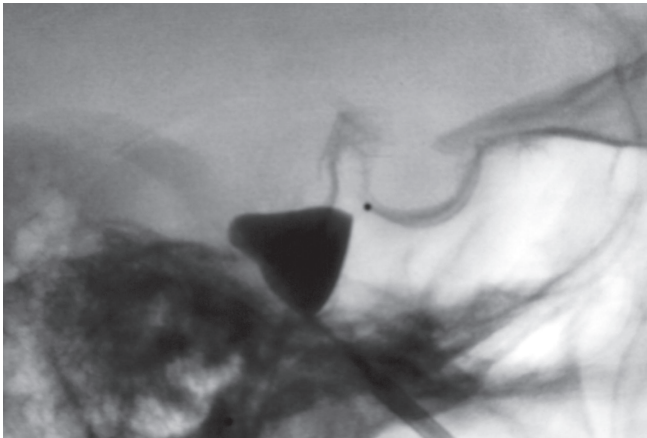


FIGURE 14.4. Lateral intraoperative radiograph showing the pear shape formed by a correctly positioned balloon with an intraluminal pressure of 1.5 atm. Kaplan-Meier analysis of pain recurrence after balloon compression for trigeminal neuralgia in patients who experienced initial pain relief. The mean time until recurrence was 13 months. (From Brown JA, Pilitsis JG: Percutaneous balloon compression for the treatment of trigeminal neuralgia: Results in 56 patients based on balloon compression pressure monitoring. *Neurosurg Focus* 2005;18:E10.³)

when the catheter is taken out before removing the cannula. This is because compression opens the subarachnoid space surrounding the ganglion in Meckel's cave. After removing the cannula, compress the cheek against the maxilla for 5 minutes. Close the entry point with a sterile tape and small bandage.

Postoperative Management

In the recovery room, give mild analgesics for surgical pain. Place an ice pack against the cheek to reduce postoperative swelling. The patient may be discharged home a few hours after the procedure or remain hospitalized until the next day. Evaluate the patient for the adequacy of his or her pain relief. Typically, the patient awakens without pain, except for local discomfort at the needle entry site. If the compression has been mild, trigeminal pain may persist for 1 or 2 days postoperatively and then subside. Assess for the extent of numbness. A decrease in touch and pinprick sensation occurs in two-thirds of patients. Patients may find the associated subjective numbness slightly uncomfortable at first, but usually adjust to it in the first 3 to 4 weeks. The numbness generally decreases substantially by 3 to 6 months. Taper the anticonvulsant medications over several weeks. More rapid taper schedules than this may cause withdrawal symptoms. Pain relief tends to persist even after the numbness resolves. Decreased sensation is most common in the third division. Despite this absence of sensory change, the patient may have complete pain relief. Rarely, patients report dysesthesia, i.e.,

unpleasant hypalgesia (decreased sensitivity to pain) and hypoesthesia (decreased sensitivity to touch).

The corneal reflex is usually not decreased, perhaps because compression selectively preserves A-delta and C fibers.¹⁰ This selectivity makes balloon compression especially useful as a percutaneous treatment of patients with first-division trigeminal neuralgia pain. Thermal rhizotomy injures all fiber types nonselectively at temperatures required to create a clinical lesion. Gamma knife radiosurgery has similar effects. Glycerol rhizotomy is not division selective.

Patients may develop labial eruptions after surgery. Most often, there will be a history of cold sores. The pain quality from these lesions is different from trigeminal neuralgia. Mild ipsilateral temporal and masseter muscle weakness occurs in two-thirds of patients, but usually is not bothersome. Patients generally have avoided chewing strongly that side of their mouths for years. Ipsilateral pterygoid muscle weakness from motor root injury may cause temporomandibular joint pain. This is likely because of muscular imbalance irritating the joint. Treat the joint pain with oral anti-inflammatory medication until resolution. Usually, this will be within a few weeks.

RESULTS

One study of this procedure using Kaplan-Meier curves found a recurrence rate of 26% after balloon compression, with a mean time until recurrence of 18 months.^{3,4,6} The recurrence rate is 30% at 10 years.¹⁵ Recurrence rates are higher in patients with multiple sclerosis and may reach 50%. Manage recurrent pain with anticonvulsant medication. If this approach fails or is not effective or tolerated, repeat the compression. Repeat procedures do not have increased technical difficulty. Lopez et al. summarized morbidity seen in 3 of the largest series of patients reviewed after balloon compression treatment for trigeminal neuralgia. There is a 1.5% incidence of cranial nerve deficits, 2.6% incidence of meningitis, 2% incidence of vascular complications, and 10% incidence of troublesome dysesthesias. The vascular complications occurred early in the history of the procedure, before development of a U.S. Food and Drug Administration–approved kit that included a blunt obturator.

Numerous other series have been published over the past 20 years and have recognized other infrequent complications.^{1,2,8,13,14,16,18–20,24,28,34,35} A single case report described an intraoperative subarachnoid hemorrhage leading to death. The surgeon used a sharp obturator that passed through the foramen ovale.²⁹ A carotid cavernous fistula and an external carotid fistula have also been reported to be associated with the use of sharp obturators in the penetrating cannula.²⁶

SUMMARY

Percutaneous balloon compression is a simple and effective treatment for trigeminal neuralgia. It is especially useful in patients with first-division pain because it does not injure the myelinated fibers that mediate the blink reflex. It is most helpful in patients with pain that has spread across multiple divisions because it does not require multiple lesions. It is also helpful in patients with whom it would be difficult to communicate during selective thermal rhizotomy. It is a relatively easy to perform once you understand the principles of the technique.

Disclosure

Dr. Brown has a financial relationship with Cook Vascular, Inc., Leechburg, PA.

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