Percutaneous balloon compression of the trigeminal nerve for treatment of trigeminal neuralgia

Jeffrey A. Brown, M.D., Christopher J. Chittum, B.S., David Sabol, B.S., and Jan J. Gouda, M.D.

Department of Neurological Surgery, Medical College of Ohio, Toledo, Ohio

This manuscript is accompanied by five movie clips that demonstrate the instruments and technique. Please be advised that the download time for these movies may be substantial, depending on the equipment that you use.

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The technique of percutaneous balloon compression for treatment of trigeminal neuralgia is demonstrated by using embedded audiovisual kernels. A text-based description with linked images is also provided to accommodate varying computer hardware capabilities. A new needle system for guiding the balloon catheter to the entrance of Meckel's cave and a balloon pressure monitoring system for the procedure is described and demonstrated. Results from a series of 141 consecutive patients treated during the period between 1983 and 1995 indicate an initial success rate of 92%. Fifty-seven percent of patients have postoperative numbness, which is described as mild to moderate by 94% of them. Sixteen percent have ipsilateral masseter-terygoid weakness after compression. The overall recurrence rate is 26%. A Kaplan-Meier survival curve indicates that 60% of patients are pain free 8 years after surgery without recurrence requiring reoperation. The recurrence rate does not significantly differ from patients with first division pain to patients without first division involvement. An absent corneal reflex has not occurred,
nor has anesthesia dolorosa. Balloon compression injures the myelinated fibers that mediate the "trigger"
to the lancinating pain of trigeminal neuralgia. Because the corneal reflex is mediated by unmyelinated
fibers, selective, monitored compression of myelinated fibers should preserve the corneal reflex when
first division pain is present.

Key Words * trigeminal nerve * trigeminal neuralgia * pain

Percutaneous trigeminal nerve compression evolved from the work of Shelden, et al.,[21] in the 1950s. They decompressed the fifth nerve at the division level, whereas Taarnhoj[25] later decompressed the
ganglion, each surgeon using a subtemporal approach. Shelden, et al., reviewed their results, which
showed that patients whose trigeminal nerve was injured at the time of decompression fared better.
However, no consistent means of injuring the nerve via the middle fossa approach was available. The
nerve was simply rubbed intraoperatively with a dissecting instrument. Jannetta's[10] use of the operating
microscope beginning in the 1960s and his rediscovery of Dandy's posterior fossa approach toward
decompression of the root entry zone redirected neurosurgeons to the more proximal root. Sweet and
Wepsic's[23] innovative application of radiofrequency current to injure retrogasserian pain fibers
simplified the reproducible neurosurgical treatment of trigeminal pain. Mullan and Lichtor,[18] whose
earlier work had transformed open cordotomy to percutaneous injury, developed the innovative
percutaneous compressive technique by using a Fogarty embolectomy catheter to compress the ganglion
and retrogasserian nerve.

The purpose of this article is to demonstrate the current technique of percutaneous balloon compression
by taking advantage of the capabilities of computerized media, using embedded audiovisual kernels and
linked images. A new needle system for guiding the balloon catheter to the entrance of Meckel's cave and
a balloon pressure monitoring system for the procedure are demonstrated. Results for a series of 141
consecutive patients treated during the period between 1983 and 1995 are presented.

Clinical Material and Methods

Patient Selection

Patients in whom medical therapy for classic trigeminal neuralgia or trigeminal neuralgia in association
with multiple sclerosis has failed are candidates for percutaneous balloon compression. Atypical facial
pain or postherpetic pain is not treatable by balloon compression. Contralateral masseter muscle
weakness is a relative contraindication because balloon compression often causes temporary masseter
weakness. Young patients are candidates for balloon compression if they are willing to accept the
associated mild numbness that usually occurs after surgery. If there is a known ectatic vertebrobasilar
artery seen on magnetic resonance or computerized tomography imaging, then there may be a higher
recurrence rate expected. Elderly patients tolerate the short general anesthetic well, perhaps better than an
intravenously administered anesthetic with a lesser protected airway and erratic intraoperative pain
control.

Operative Technique

Technique, Part 1 (QuickTime Movie, 4.4 MB)
Preoperative magnetic resonance or computerized tomography imaging of the posterior fossa is obtained to rule out an associated arteriovenous malformation, meningioma, or other mass lesion. The incidence of trigeminal neuralgia caused by adjacent mass lesions is low. Plain skull radiographs of the foramen ovale via the submental vertex view can identify those patients in whom the foramen cannot be visualized or in whom it is too small to allow penetration of the No. 14 gauge cannula.

The procedure is most easily accomplished in the angiography suite where multiplane fluoroscopy is available.[4] A C-arm fluoroscope with digital image refinement may also be used. Three views are used to perform this procedure: modified submental (Fig. 1), anteroposterior, and lateral. A general anesthetic is used, usually with isofluorane. After induction of anesthesia, an external pacemaker is placed and set to trigger at 45 beats/minute should bradycardia occur during balloon compression, which it does in two of three patients. Belber and Rak[3] blocked the trigeminal depressor response that occurs with administration of pre- and intraoperative atropine. This depressor response consists of both bradycardia and brief hypotension, often with reflexive hypertension after triggering the pacemaker (Fig. 2). It is consistently different from the pressor response of tachycardia and hypertension seen during radiofrequency lesioning in rhizotomy, probably because different fibers are injured during these
procedures. The pacemaker will respond more quickly than the anesthesiologist can inject atropine. Blocking the depressor response completely, although apparently safer, will eliminate one of the surgeon's cues that the nerve is being injured.

A roll is placed beneath the patient's shoulders with the neck slightly extended and the head is rotated approximately 15° to 30° toward the opposite side. The imaging unit is directed at an approximately 29° angle so that the foramen ovale is seen just medial to the mandible, lateral to the maxilla, and just above the petrous bone. Hartel guidelines are used for the needle insertion. The modified thinwall No. 14 gauge cannula can then be guided parallel to the x-ray beam, directly toward the easily seen foramen. Figure 3 depicts the insoufflation syringe and balloon catheter used in the procedure.

Traditional submental vertex views are more difficult to obtain and require greater neck extension. They are not always well tolerated by elderly patients who often have spondylosis and limited neck mobility. When the needle is inserted according to Hartel guidelines and using the technique of Nugent[19] and van Loveren, et al.[27] for the lateral view, the position of the foramen must be estimated. After the cannula penetrates the skin, a blunt penetrating stylet is inserted to replace the 45° angle sharp stylet (Fig. 4).
Fig. 4. Upper: Photograph of the penetrating cannula with inner penetrating blunt stylet used in the procedure. Lower: Photograph of the penetrating cannula with inner penetrating sharp stylet.

The foramen is engaged while the cannula is directed to the center of the foramen, as visualized in the modified submental view. If atropine is not administered, the patient will experience a brief bradycardia when the foramen is engaged. The surgeon will also feel the needle engage the foramen, but this should not be the sole means of providing feedback regarding the needle position. The lateral view will show the needle to be at the middle fossa base. Figure 5 illustrates the cannula and stylets used in the procedure.
Once the foramen is engaged, an anteroposterior view is obtained with the petrous bone radiographically centered in the orbit. The slender guiding stylet can then be advanced to the entrance of Meckel's cave, usually 17 to 22 mm beyond the foramen. In this radiographic view, a dip is seen medially where the trigeminal nerve enters Meckel's cave, passing over the petrous bone. Nugent[19] has described this position as being 9 mm medial to the lateral border of the internal auditory canal. The guiding stylet creates a path through which the catheter will easily pass. If the stylet is directed lateral to this dip, then a curved stylet is inserted with the curve directed superomedially. Once the track has been made, the stylet is removed and the embolectomy catheter is advanced to the same position at the edge of the petrous bone. The catheter has an thin inner wire that allows its position to be verified on the image intensifier. Often the catheter will wander from this point if the cannula is not properly directed toward it. The catheter is soft and blunt and may limit the risk of hemorrhage if used alone, but if the catheter does not reach the entrance of Meckel's cave, the retrogasserian fibers will not be compressed by the balloon. It is here that the highest pressures are obtained. The balloon lifts the inelastic dura from the ganglion, limiting the pressure at which it is compressed. At the entrance of Meckel's cave, the balloon compresses the retrogasserian fibers against the firm edge of the dura and petrous ridge as the dura splits allowing the nerve to pass into Meckel's cave (Fig. 6). This opening is 9 mm X 2 mm. When the balloon inflates within it, the characteristic pear shape is seen. It is important that the balloon catheter tip be positioned at the entrance of Meckel's cave. If the tip is left short of the entrance, the degree of numbness engendered will be limited, most likely to the third division of the trigeminal nerve. If the tip extends into the subarachnoid space beyond the petrous bone, the brainstem could be compressed, but most certainly the nerve would not be sufficiently injured. No harm has been known to occur from allowing the balloon to slip into the posterior fossa, but the operation will not succeed regardless.

The balloon can be inflated with 0.75 to 1 cc of 180 mg% radiopaque dye either with a tuberculin syringe or with an insoufflator syringe with attached pressure transducer. The transducer can be calibrated to read up to 2000 mm Hg of pressure. When properly inflated, the intraluminal balloon pressure is 1200 to 1500 mm Hg. The resulting tissue compression pressure is 650 to 950 mm Hg, because the in vitro pressure of the inflated balloon is 550 mm Hg. Overinflation can lead to temporary sixth nerve palsy or severe numbness. The balloon is inflated for 1 minute, or up to 1.5 minutes if there have been multiple recurrences. When using a simple tuberculin syringe to inflate the balloon, the surgeon must develop a feel for the pressure by the feedback to the fingers. The insoufflater has the disadvantage of not providing concurrent sensory feedback; however, it is a simpler way to perform the procedure. Gerber[8] recommends removing the air from the balloon before inflating it. This stabilizes the intraluminal pressure and changes the configuration of the balloon, although rupture of the balloon has never led to any morbidity and does not lead to any risk from subarachnoid or subdural air release. If air is to be removed from the balloon, it should be done before connecting it to the pressure monitor.
Fig. 7. Intraoperative radiograph of the inflated balloon forming a characteristic pear shape after inflation with 1 cc of radiopaque dye. The thin neck of the pear represents the portion of the balloon inflated within the porus trigeminus, the entrance of Meckel's cave bordered by the petrous bone and free dural edge.

When properly inflated, the pear shape is seen and the depressor response occurs (Fig. 7). If the pacemaker is triggered, it should be only briefly and is often followed by a hypertensive response that may be treated by an increase in anesthetic or infusion of nitroprusside. After deflation, the balloon and cannula are removed concurrently and the cheek is compressed against the maxilla for 5 minutes. Cerebrospinal fluid (CSF) will leak through the cannula if the catheter is removed separately.

RESULTS

One hundred forty-one consecutive patients with classic symptoms of unilateral trigeminal neuralgia underwent 182 percutaneous compressions between 1983 and 1995. The patients' mean age was 66 years (range 27-97 years). First division pain was present in 59 patients (42%). Forty-six patients (33%) had undergone previous destructive procedures and nine patients (6%) had multiple sclerosis. None of the patients had an associated tumor or malformation. All were treated initially with carbamazepine, some with dilantin and/or baclofen. The indications for surgery were similar to those for other percutaneous procedures for the treatment of trigeminal neuralgia: either elderly patients at higher risk for morbidity from microvascular decompression or young patients who sought a percutaneous operation with an expectation of mild-to-moderate numbness after consultation. Microvascular decompression was recommended to younger patients who had no history of previous peripheral destructive procedures before they considered balloon compression because of their sensitivity to facial numbness.

Magnetic resonance imaging with gadolinium enhancement was routinely obtained preoperatively along with skull radiographs of the foramen ovale. When MR imaging was not possible, 5-mm slice CT was obtained in the region of Meckel's cave. The mean follow-up period for the 182 procedures was 22 months (range 2 months to 10 years). Follow-up study was either by patient examination or telephone interview.

One hundred thirty patients (92%) were initially relieved of their pain. Eighty patients (57%) had numbness at their last evaluation: this was mild in 64 (80%), moderate in 11 (14%), and severe in five (6%). Twenty-three patients (16%) had minor ipsilateral masseter muscle weakness that resolved in 1 year or less. Five percent of patients developed postoperative aseptic meningitis consisting of headache, fever, stiff neck, and altered mental status, all occurring within 6 hours of surgery. All symptoms resolved within 48 hours without residual deficits. Cerebrospinal fluid cultures showed no growth. A CSF red blood cell count suggested that the cause of the symptoms was mild subarachnoid blood. However, CT scanning never showed subarachnoid blood to be present radiographically. Anesthesia dolorosa and loss of the corneal reflex did not occur in any patient.

The overall recurrence rate was 26%. Twenty-eight patients with recurrence had a repeat balloon
compression, which provided pain relief in 19 (68%). Fifty-nine patients (42%) had involvement of the first division either alone or in combination with other sensory divisions. Pain relief without recurrence was achieved in 40 (68%) of 59 patients with first division pain. This rate was similar to the success rate (52 (68%) of 82 patients) without first division pain after their first balloon compression (Fig. 8).

Discussion

Technical Problems

The balloon can slip into the prepontine cistern if it is inflated so that the midpoint is beyond the edge of the petrous bone. Once this happens, reinflation will lead to repeated slips unless the balloon is fixed to the cannula either by holding it or by a fixation system attached to the cannula through which the catheter is threaded.

If a pear shape is not seen (Fig. 9), the balloon may not have been advanced far enough to have entered the porus. The lateral view on fluoroscopy will show whether the balloon has reached the clival line. If advancing the catheter does not lead to a pear shape, or if inflation pressure is near the 550 mm Hg intraluminal pressure (which occurs in vitro), the catheter may have penetrated the temporal lobe dura. The best chance of success will be to remove the needle and catheter and reposition it using a stylet. Otherwise the catheter will tend to slide through the same path, repeatedly penetrating the dura in the same site. If repositioning the needle and catheter does not work, the procedure should be abandoned and repeated later.
Venous bleeding is usually present from epidural veins at the foramen outside the skull base. If venous bleeding is present when the needle is near the foramen, it should be advanced slightly until it is firmly engaged. A lateral view will prevent advancement of the cannula into the middle fossa. Arterial bleeding should not occur.

Pain relief is usually present once the patient has fully awakened from the anesthetic, but has on occasion been delayed for several days. The patient should not be drowsy, but should drowsiness occur, the patient should be further investigated. Minor subarachnoid bleeding can cause aseptic meningitis: headache, fever, and confusion lasting 24 to 48 hours. Such patients will complain of headache immediately upon awakening from anesthesia. Treatment is symptomatic of the headache. A CT scan will not demonstrate subarachnoid blood.

If the patient's pain is not relieved, the procedure may be repeated, but at least a week should pass before performing a repeat procedure because secondary injury to the nerve may cause progressive injury and pain relief in the next several days. It is not more difficult to insert the needle and balloon if the procedure is repeated within days.

If numbness is initially bothersome to the patient, the surgeon should help the patient understand that the numbness will improve considerably during the first several days after surgery. Severe numbness will not resolve, but will improve. Numbness will continue to improve during the 1st year, gradually plateauing. Similarly, jaw weakness will gradually improve over several weeks.

Patients in the early stage of trigeminal neuralgia may be easily treated, but those with longstanding pain may develop a trigeminal neuropathy with a component of constant aching pain. These patients may have associated areas of preoperative numbness. The constant pain may resolve gradually after the lancinating pain has ceased. Some patients will have a combination of trigeminal neuralgia, neuropathy, atypical facial pain, and temporomandibular joint pain. Usually, the trigeminal neuralgia is the worst source of pain and the patient is well pleased to be rid of it, although aware of the residual source of complaints.

**Postsurgical Morbidity**

Numbness is a goal of the procedure, although the majority of the patients will indicate that their numbness is only mild and certainly tolerable. The small percentage of patients who believe their numbness is severe are still grateful to be without pain. Associated with the numbness are several other elements that are a consequence of the motor innervation of the trigeminal nerve. Patients may complain of otalgia related to weakness of the tensor tympani muscle. The jaw may deviate to the side from pterygoid weakness. Occasionally patients have developed hemotypanum from blood entering the eustachian tube. Dysesthesias are rare and seldom bothersome. There may be a sense of intermittent "crawling" sensation. The precise location of the numbness generated by balloon compression is not predictable; however, most often it is located in the perioral area of second and third divisions if the balloon is properly positioned radiographically. More medial placement of the catheter in the porus, by more a more lateral entrance point is desirable if first division numbness is sought.

One patient in this series had a temporary sixth nerve palsy that spontaneously resolved. Presumably this was because of balloon over inflation and cavernous sinus compression; this occurred before pressure monitoring was routinely done. Lasting corneal anesthesia does not occur, although a brief reduction in the reflex has been seen. Keratitis and anesthesia dolorosa have not occurred and are unlikely because of the nature of the compressive injury to large myelinated fibers only. Because the corneal reflex is
mediated by unmyelinated fibers, selective, monitored compression of myelinated fibers should preserve the corneal reflex when first division pain is present.[4]

Literature Review

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of Patients</th>
<th>Recurrence Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Esposito, et al., 1985</td>
<td>50</td>
<td>22%</td>
</tr>
<tr>
<td>Belber &amp; Rak, 1987</td>
<td>33</td>
<td>24%</td>
</tr>
<tr>
<td>Meglio, et al., 1987</td>
<td>47</td>
<td>55%</td>
</tr>
<tr>
<td>Fraioli, et al., 1989</td>
<td>15</td>
<td>9.8%</td>
</tr>
<tr>
<td>Lobato, et al., 1990</td>
<td>144</td>
<td>9.7%</td>
</tr>
<tr>
<td>Mullan, 1990</td>
<td>100</td>
<td>28%</td>
</tr>
<tr>
<td>Brown (present study)</td>
<td>141</td>
<td>26%</td>
</tr>
<tr>
<td>totals</td>
<td>674</td>
<td>25%</td>
</tr>
</tbody>
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The overall initial success rate for balloon compression is 92% [6,14,26] (Table 1). The mean recurrence rate after balloon compression in seven published series totaling 674 patients is 25%, the lowest rate being 9.7% occurring 10 and 35 months after surgery. Mullan[17] reported a 28% recurrence rate after 1 to 10 years of follow-up study. Belber and Rak[3] had a 24% recurrence rate at 6 months to 7 years of follow-up evaluation. Fraioli, et al.,[7] had a 9.8% recurrence rate at 3.5 years of follow up. Meglio, et al.,[16] had the highest recurrence rate at 54%. Masseter muscle weakness occurs in approximately 10% of the cases. Weakness also occurs with radiofrequency thermorhizotomy lesioning. Van Loveren, et al.,[27] report a 7% incidence of weakness from radiofrequency thermorhizotomy lesioning using a curved electrode. Mild-to-moderate hypesthesia occurs in approximately one-half of the patients. The rate of dysesthesia is 6%. Only one case (0.1%) with anesthesia dolorosa has been reported. The risk of corneal anesthesia from balloon compression is 0.1%.[4]

Of 2318 cases of trigeminal neuralgia treated by microvascular decompression the average initial good or excellent result rate was 93% (range 86%-100%).[5,20,24,26,28] The mean time to recurrence was 2 years. Forty-seven percent of recurrences occurred in the 1st year. Thereafter, the likelihood of recurrence was 2% per year for 6 years. Repeat microvascular decompression in patients who have previously undergone microvascular decompression and either did not benefit from initial surgery or had recurrent symptoms produced pain relief in approximately 50% of patients.[1,2]

The mortality rate from microvascular decompression is 0.7% (16 deaths in 2318 cases).[5] The most common serious complications associated with microvascular decompression are aseptic meningitis (1.6%), CSF leak (0.8%), pulmonary complications (0.5%), and cerebellar infarction (0.4%).[11] Aseptic meningitis occurs at a comparable rate to that seen in balloon compression, although the mechanism may not be the same. Eighth nerve injury occurs in 4% of patients with microvascular decompression and fourth nerve palsy and diplopia occur in 1%. Postoperative hearing disturbances are usually conductive losses from fluid or blood accumulation in the middle ear rather than from nerve injury, and they are usually transient. Balloon compression can also cause hemotympanum, most likely from blood within the eustachian canal, and this has been associated with a 2% incidence of sixth nerve palsy from adjacent compression of the cavernous sinus.

Immediate relief was obtained using radiofrequency thermorhizotomy lesioning in 98% of 6235 patients reported in the literature. The average recurrence rate ranges from 2% to 18% at 3 years and 25% to 37%
at 5 years. The risk of death is less than 0.06%. Injury to the temporal lobe, brain abscess, carotid puncture, and carotid-cavernous fistula rarely occur. Corneal anesthesia is the most significant disadvantage, occurring in 7% of cases overall. Less than one-half of these patients develop keratitis. By using the cordotomy-type needle and relying on an awake patient response, the incidence of corneal anesthesia has dropped to 0.5%, as low as with balloon compression. Dysesthesias occur in 5% to 24% of the cases, whereas the anesthesia dolorosa rate varies between 1% to 25% depending on the lesioning philosophy. If the goal is hypesthesia and not anesthesia, then the incidence is reduced.

When using glycerol rhizolysis the initial relief rate is 90%.[13] This pain relief may not be instant and may take up to 21 days. Excellent results in patients with multiple sclerosis have been reported with glycerol rhizotomy as well as with balloon compression.[15] Recurrence may be more likely than with other percutaneous techniques, with rates of 25% at 2 years and 50% at 5 years reported.[7,9,12,13,22] No deaths have been reported. The corneal sensation may be diminished in approximately 3% of patients. Aseptic and/or bacterial meningitis have also been rare with glycerol rhizotomy (1.6% of cases). The average major hypesthesia risk is approximately 1.3%, although minor hypesthesia may occur in up to one-third of cases. Dysesthesias occur in up to 8.8% of cases, possibly from direct injection of glycerol into the trigeminal fibers. When using glycerol rhizotomy anesthesia dolorosa does not occur.

CONCLUSIONS

This report demonstrates the technique of percutaneous balloon compression and summarizes the results of treatment in a consecutive series of 141 patients with classic trigeminal neuralgia or trigeminal neuralgia associated with multiple sclerosis. By using embedded audiovisual kernels and Internet technology, it is possible to communicate accurately subtleties of technique and instrumentation otherwise relegated to time-consuming workshops. A text-based report is provided concurrent with the more sophisticated audiovisual supplements to accommodate the varied hardware available for document review.

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*Address reprint requests to:* Jeffrey A. Brown, M.D., Department of Neurological Surgery, Medical College of Ohio, Toledo, Ohio 43599-0008.

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Quicktime Movies

Instruments used (QuickTime Movie, 3.7 MB)

Technique, Part 1 (QuickTime Movie, 4.4 MB)

Technique, Part 2 (QuickTime Movie, 4.4 MB)
Technique, Part 3 (QuickTime Movie, 5.1 MB)

Technique, Part 4 (QuickTime Movie, 4.6 MB)