Technique of microvascular decompression

Technical note

PETER J. JANNETTA, M.D., MARK R. MCLAUGHLIN, M.D., AND KENNETH F. CASEY, M.D.

Department of Neurosurgery, Drexel University College of Medicine and Allegheny General Hospital, Pittsburgh, Pennsylvania; and Princeton Brain and Spine Care, LLC, Langhorne-Newtown, Pennsylvania

Vascular compression of the trigeminal nerve in the cerebellopontine angle is now generally accepted as the primary source or “trigger” causing trigeminal neuralgia. A clear clinicopathological association exists in the neurovascular relationship. In general, pain in the third division of the trigeminal nerve is caused by rostral compression, pain in the second division is caused by medial or more distant compression, and pain in the first division is caused by caudal compression.

This discussion of the surgical technique includes details on patient position, placement of the incision and craniectomy, microsurgical exposure of the supralateral cerebellopontine angle, visualization of the trigeminal nerve and vascular pathological features, microvascular decompression, and wound closure. Nuances of the technique are best learned in the company of a surgeon who has a longer experience with this procedure.

KEY WORDS • trigeminal neuralgia • tic douloureux • intermediate nerve neuralgia • facial pain • cranial nerve vascular compression • microvascular decompression • operative technique

BACKGROUND

The application of magnification by using the binocular microscope, use of new computerized imaging modalities, neurophysiological monitoring, and safer anesthetic agents has enabled neurosurgeons not only to do older, standard procedures better but to perform new, innovative ones. In many institutions, MVD is now considered the benchmark surgical procedure for intractable TN of any variety, as well as other cranial nerve problems.1–22,25–27 Mclaughlin, et al.,24 described six steps in performing MVD; their paper is most helpful in learning the technique. Most important, we believe it is useful for the neophyte to learn from someone who has a broad experience in the procedure. It seems to us that residents are learning the nuances of microsurgical techniques and of MVD with less difficulty than in the past. We cannot explain this. It may be new instrumentation, better teaching, better preparation; it may be all kinds of things, but whatever the reason, the phenomenon seems to be real.

Abbreviations used in this paper: MVD = microvascular decompression; SCA = superior cerebellar artery; TN = trigeminal neuralgia; V1 = first division of the trigeminal nerve; V2 = second division of the trigeminal nerve; V3 = third division of the trigeminal nerve.

TECHNIQUE OF MVD

After anesthesia is induced and intubation is performed, the patient is placed in the lateral position with the head secured in a three-point head holder. (The head holder can be placed before or after the patient is turned.) Pressure points are padded and an axillary roll is placed. The neck is minimally stretched with mild flexion and rotation approximately 10˚ toward the affected (“up”) side. For trigeminal nerve procedures, the vertex is tilted down approximately 10˚. The point of these maneuvers is to expose the occipital boss. The shoulder is taped down and out of the way. The patient is taped into position so that the table can be rotated laterally or adjusted for the Trendelenburg or reverse Trendelenburg position (Fig. 1).

Next, the incision is defined. The mastoid eminence is traced out with a marker. A line is drawn from the external auditory canal to the inion, bisected, and a point placed a finger breadth caudally to this halfway point. This should be the focus of the first drill hole. The line between the inion and the external auditory canal will define the transverse sinus. The digastric groove line will show the juncture of the sinuses. A Burr hole can be placed a fingerbreadth posterior and caudal to this junction. A short (3–4 cm), somewhat arcuate or straight incision is centered over this area, with the open (concave) side facing the ear (Fig.
2). The incision and craniectomy are placed more posteriorly in a large, muscular, and/or dolichocephalic patient. The osseous landmarks must be well identified before the first opening is made in the bone. (The location of the mastoid emissary vein may not be a good sign of the location of the sigmoid sinus.) A perforator is used to open the bone, and if bleeding occurs, the bone should be sealed with wax. A dural separator should be used before rongeuring the bone. The craniectomy opening for MVD of the trigeminal nerve should generally be in the shape of an isosceles triangle, with the hypotenuse facing away from the sinuses. The mastoid air cell should be thoroughly filled with wax. An incision is made in the dura mater and extended. The dura adjacent to the junction of the sigmoid and lateral sinuses must be clearly identified. It is important that if mastoid air cells are opened, the opening be enlarged enough so that the air cells can be filled with muscle or bone wax or both (Fig. 3).

The next part of the procedure involves entering the supralateral angle of the posterior fossa. This must be done carefully and patiently. After the dura mater is sutured back out of the way, the operating microscope is brought into

Fig. 1. Upper: Drawing showing the patient’s head placed at the foot of the operating table to allow more leg room for the surgeon during the microsurgical part of the procedure. The head is secured with three-point fixation and the patient is turned in the lateral decubitus position. The head is rotated slightly away from the affected side and flexed to approximately two finger breadths from the sternum. Center and Lower: Drawings showing how the angle of the vertex is tailored depending on the cranial nerve approach. For trigeminal or cochlear nerve approaches, the vertex of the head is kept parallel to the floor to keep the seventh–eighth cranial nerve complex at a more inferior position with respect to the trigeminal nerve. For an approach to the seventh or lower cranial nerve, the vertex is dropped 15° toward the floor to rotate and expose the proximal aspect of the seventh nerve and to rotate the vestibulocochlear complex more cephalad. The Roman numerals designate cranial nerves.

Fig. 2. Upper: Drawing depicting how the intersection of the digastric groove and the inomental line defines the junction of the transverse and sigmoid sinuses. With a “mind’s eye” view of optimal burr hole and craniectomy placement, an incision is drawn centered over the planned craniectomy. Lower: Drawings showing different surgical incisions based on the size of the patient’s neck. Short- and thick-necked patients require a more posteriorly (medially) directed incision. This angled incision positions the thicker neck musculature more posteriorly (medially) and out of the operative field. This small adjustment is critical in allowing freer movement of the surgeon’s hands and instruments during the microsurgery.

Fig. 3. Drawing depicting how bone exposure should reveal the junction of the transverse and sigmoid sinuses. Surgical exposure can then be tailored according to the cranial nerve approach desired.
play. A self-retaining, malleable tapered retractor should be placed over a rubber dam and cottonoid in the upper lateral corner and the area carefully visualized. If the dural opening is satisfactory, one can look for the presence of bridging veins. Occasionally such a vessel will come laterally and caudally and open into subarachnoid space below the superior surface of the cerebellum. If this exposure is not easily attained, one should move caudally and open into subarachnoid space in the basal cisterns. No lumbar drain is necessary.

Cerebrospinal fluid should be drained away, and as this is done, the nerves begin to be seen. Usually the eighth cranial nerve is seen first. The retractor is then placed a bit rostrally to look for any problem with bridging veins being stretched on tension. The purpose of the cerebellar retraction (support) is mainly to bring the cerebellum slightly up off the cranial nerves and toward the surgeon and not simply to compress it medially. Hearing the pulse rate monitor is helpful in this regard. We also, of course, maintain monitoring of brainstem auditory evoked responses throughout these operations in an attempt to ensure that we do not hurt the patient’s hearing. The superior petrosal venous complex may or may not have to be partly or totally sacrificed. We coagulate the vein at low power over a distance, partly section it, coagulate again, section it fully, coagulate again, and then perform a Valsalva maneuver. If bleeding occurs from the supratentorial vein, it is usually found to be torn at the brainstem and must be treated carefully. To accomplish this, one can raise the head of the table and one can compress gently, coagulate on the cerebellar side, divide, and then compress the dural side for a time (Fig. 4). The trigeminal nerve is located anterior (deep) and rostral to the eighth and seventh cranial nerves.

The neurovascular relationships in TN generally have a clear clinicopathological correlation. First, it is important that the surgeon understand, except in some cases of multiple sclerosis, that TN is caused by a blood vessel. Second, he or she must understand that not only is there one blood vessel involved but there may be more, and it is up to the surgeon to find all of them. The involvement of these vessels may occasionally be subtle. The compression may be proximal or distal, and it may be located under the ala of the cerebellum. We have seen three patients in whom the vessel was located totally inside the trigeminal nerve.

The vascular compression causing hyperactive symptoms in the trigeminal nerve can occur anywhere from the brainstem to the Meckel cave. There are approximately 100 fascicles in the nerve just inside the Meckel cave, and they each settle into the fibrous cone of the sensory root of the trigeminal nerve (portio major, Fig. 5). Compression anywhere, therefore, from brainstem distally, can cause TN. Rostral compression of the nerve causes pain in the V₁, and this is most commonly due to the SCA looping downward and upward again (Fig. 6). As the artery elongates, it compresses the middle portion of the nerve, causing pain in the V₁ in addition to the V₂.

We have seen a long, looping SCA compress the caudal side of the nerve from below without compressing the rostral side and causing isolated pain in the V₁. If a long SCA loop is mobilized from the proximal to distal direction with the aid of multiple pieces of shredded polytetrafluoroethylene felt, the artery will pop out into a horizontal position (Fig. 7). The relatively rare, isolated pain in the V₁ is caused by a vessel on the caudal side of the nerve. Isolated pain in the V₁ is most common in older men, cigarette smokers, and patients with dolichocephalic features in whom the verte-
brobasilar system arteries compress the nerve from the caudal side. Isolated pain in the V2 is most common in younger women and is caused by a bridging vein that may be quite distal on the nerve.

In a patient with dolichocephalic features, an enostosis may be present over the distal part of the nerve (“Kamal hump”), hiding the compressing vein that is causing the TN. One must take care to look around caudally, rostrally, and underneath the enostosis, using a mirror if necessary to ensure that there is not a vessel in this region. Compression in this area often softens the nerve and one must be careful not to traumatize it.

Duration of compression as well as location and size of the blood vessel cause changes in TN. In time, an artery that is elongating will stretch the nerve, causing constant pain that may be burning in nature and may cause numbness. A vessel compressing the motor–proprioceptor fascicles distally causes constant pain that is usually burning in character and is hard to localize. If the motor–proprioceptor fascicle is stretched by a blood vessel, the patient can experience hyperactive autonomic dysfunction and a cluster headache syndrome. If the individual reports a cluster headache syndrome, one must also look for compression of the intermediate nerve, usually on the caudal side of the seventh and eighth cranial nerve bundle or between the nerves. This is treated with section and decompression of the intermediate nerve.

Surface veins cause a special problem, because they are prone to recollateralize if coagulated and divided. Most early recurrences (<1 year) are the result of these recollateralized veins. Subsequent recurrence (0.5%/year) is due to new blood vessels, especially arteries, pressing on the nerve as a result of the continuation of the aging process.

**WOUND CLOSURE**

The Valsalva maneuver is performed several times before closing. If there is any venous oozing, 1 ml of hydrogen peroxide can be injected carefully into the area, which is then irrigated with saline. Once hemostasis is assured, the retractor, rubber dam, and cottonoid are removed and further irrigation is performed. We like to use a bulb for irrigation rather than a syringe, because we prefer a high-volume, low-pressure fluid stream. We close the dura mater with interrupted and running sutures of 4-0 nylon over Duragen and place Duragen over the suture line. The bone edge is again sealed with wax. A cranioplasty of titanium wire mesh is completed with three or four self-driving screws (available from KLS Martin, L.P., Jacksonville, FL). The wound is then closed in layers in the usual way.

Postoperatively, the patients are kept overnight in a well-staffed nursing unit but not in a continuous care or intensive care unit. If there are any problems, of course, we move them into the intensive care unit. We mobilize the patients the night of the operation or the next day. The mean duration of stay in the hospital is 1.7 days for our patients. If someone has excessive pain, headache, or nausea, we will keep them in the hospital longer. Patients are seen in an office visit on the 5th postoperative day, and those from out of town return to their homes thereafter.

Our patients have had minimal hearing loss problems and infections, and the infections have been superficial in recent
Technique of microvascular decompression

years, but occasional leaks of cerebrospinal fluid continue to plague this procedure. These are treated by closed spinal drainage; using the method of simple oversewing of the wound advocated by McCallum, et al., is not sufficient. Rarely, the wound must be explored, the bone rewaxed, and the dura mater resutured. The mortality rate associated with this procedure is 0.1% in our series.

References


Manuscript received March 15, 2005. Accepted in final form April 15, 2005.

Address reprint requests to: Peter J. Jannetta, M.D., Department of Neurosurgery, 420 East North Avenue, Suite 302, Pittsburgh, Pennsylvania 15212. email: mbirgele@wpahs.org.