MICROVASCULAR DECOMPRESSION FOR GLOSSOPHARYNGEAL NEURALGIA: LONG-TERM EFFECTIVENESS AND COMPLICATION AVOIDANCE

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OBJECTIVE: To establish the long-term safety, efficacy, and durability of microvascular decompression (MVD) for the treatment of glossopharyngeal neuralgia, this study presents the immediate (<6 mo) postoperative and long-term results of a large series of 47 patients with treated with MVD.

METHODS: Operative reports and hospital charts were analyzed to collect demographic information, clinical presentation, and surgical findings. Surgical results and complications were ascertained by direct patient contact or by contact with the patient's family or physician if the patient was dead. Long-term (>10 yr) personal follow-up was available for 29 of 47 patients.

RESULTS: Forty-six (98%) of 47 patients experienced complete relief of pain immediately after MVD. Long-term follow-up was available for 29 of these 47 patients (range, 125–211 mo; median, 152 mo, or 12.7 yr), and 28 of these 29 patients continued to be pain-free. Permanent neurological deficits (>6 mo) attributed to the surgery were observed in 5 (11%) of 47 patients. Of these patients, 4 of 5 had mild hoarseness or dysphagia or both, and one had a Grade II/VI facial nerve paresis.

CONCLUSION: This study demonstrates that MVD is a safe, effective, and durable surgical procedure for producing prolonged pain relief in patients with medically intractable glossopharyngeal neuralgia.

KEY WORDS: Facial pain, Glossopharyngeal nerve diseases, Microvascular decompression, Nerve compression syndromes

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Idiopathic glossopharyngeal neuralgia (GN) is a relatively rare condition characterized by severe, paroxysmal episodes of lancinating pain. The pain is similar to that experienced with trigeminal neuralgia (TN) but is instead localized to the external ear canal, the base of the tongue, the tonsil, or the area beneath the angle of the jaw. In addition, these painful attacks can be associated with hemodynamic instability resulting from reflexive autonomic outflow that can lead to life-threatening syncopal episodes (1, 8, 15, 20, 21, 24–26). Several surgical approaches to medically intractable GN have been described, but most rely on the destruction of the glossopharyngeal or vagus nerves. More recently, microvascular decompression (MVD) has been shown to be a potentially effective treatment for GN (9), but patient series have generally been small or have had relatively short follow-up (7, 12, 16, 22). Recently, a previously reported series of 40 patients (16) with GN treated with MVD at the University of Pittsburgh was extended to a remarkable 217 patients (12), but follow-up for more than 4 years was available for only 20 of the 217 reported patients. The lack of long-term follow-up has not allowed for wide acceptance of MVD as the sole and primary surgical treatment for GN. In this article, we present a new series of 47 patients who all underwent MVD for GN more than 10 years ago. Our results demonstrate the safety, efficacy, and durability of MVD for GN in a large series of patients with long-term follow-up.

PATIENTS AND METHODS

Patient Population

Between 1984 and 1991, 47 consecutive patients were treated for GN with MVD in our

series. A retrospective review of these patients was performed, with particular attention to presenting symptoms, operative findings, and long-term clinical outcomes. Additional data included age and sex of the patient, the localization and characteristics of the presenting symptoms, and the preoperative duration of symptoms. Original operative notes, hospital charts, and direct patient follow-up were used. Patients received the diagnosis of typical idiopathic GN if their symptoms were consistent with the classic description by Dandy (5) or met the guidelines of the International Headache Society (6).

Operative Technique

There are a number of advisable measures that may be used by the anesthesiologist to reduce some of the complications associated with this procedure. For example, giving atropine prophylactically and anesthetizing the oropharynx, particularly on the side of the pain, with topically applied lidocaine before intubation will reduce stimulation in this area, which may lead to significant hemodynamic changes (17). Although prophylactic atropine is also used routinely before manipulation of the offending vessels from around the nerves, it will not guarantee cardiac and hemodynamic stability, and it should be noted that correction of cardiac arrhythmias alone may not be sufficient to correct hemodynamic derangements if they should occur (1, 17, 26). Finally, because of poor oral intake, these patients may be depleted of intravascular volume, making hypotensive episodes even more likely, so such fluid deficits should be corrected before surgery with intravenous fluid administration (17).

Because we believe that intraoperative evoked potential monitoring reduces complications, we routinely use intraoperative monitoring of auditory and somatosensory evoked potentials during surgery in the cerebellopontine angle. The anesthesiologist is informed of the intent to monitor evoked potentials so that the volatile general anesthetic agents, such as isoflurane, that may interfere with such monitoring are kept at a concentration below 1%. If the vagus nerve is to be stimulated intraoperatively, an electrode can be inserted into the ipsilateral false vocal cord at the time of intubation (3, 22, 23). In five patients, we have monitored the glossopharyngeal and vagus nerves with commercially available endotracheal tubes supplemented with reference electrodes. We have not found that monitoring of the lower cranial nerves has allowed us to predict postoperative deficits in these nerves, however, so this supplementary technique is not used routinely. This also allows our anesthesiologists to use muscle relaxants, which improves monitoring of auditory and somatosensory evoked potentials.

The patient is positioned to optimize exposure of the occipital condyle and the surgeons' working angle (*Fig. 1A*). The dependent part of the neck is lifted upward to decompress the contralateral venous drainage system. The skin is incised to the level of the hypodermis, where a layer of fascia, akin to Camper's fascia in the abdominal wall, can be harvested to use

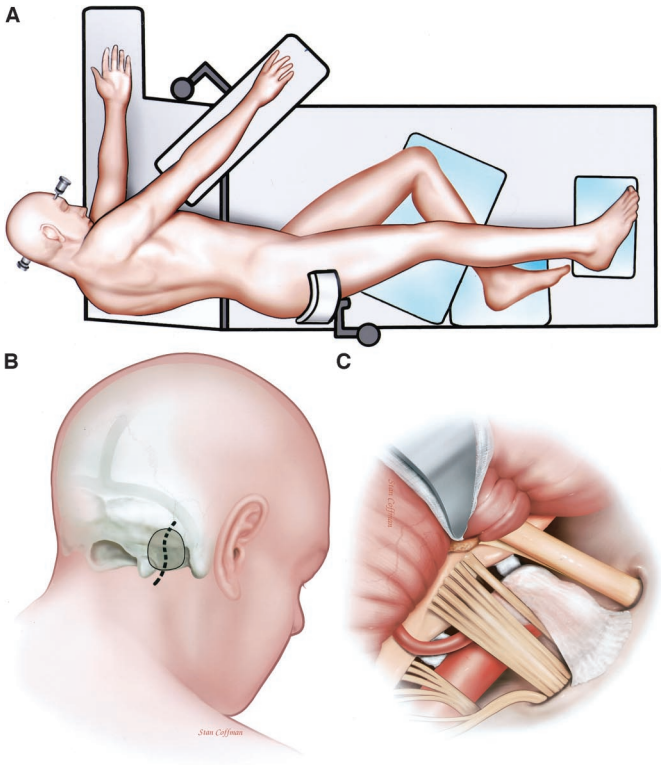

FIGURE 1. A, *drawing showing positioning of the patient. The patient is positioned on his or her side. The head is moderately flexed and the nose rotated upward slightly to bring the body of the mastoid into the highest position on the operative field and to provide a better working angle once the bone removal has been completed. The vertex is angled slightly downward. The head of the bed is flexed 15 degrees upward, and then the entire table is angled in reverse Trendelenburg to lower the head 10 degrees so as to bring the operative area into the horizontal plane. The ipsilateral shoulder is rotated toward the chest and away from the head to maximize exposure.* B, *drawing showing incision and craniectomy. For exposure of the glossopharyngeal and vagus nerves, a 3-cm incision is made just inside the hairline. The pinna will need to be folded anteriorly in some patients to adequately visualize the skin incision. This should be done carefully, however, so as not to interfere with auditory evoked potential signals. The craniotomy extends superior to the inferior nuchal line, skeletonizes the sigmoid sinus laterally, and extends to the horizontal portion of the occipital bone inferolaterally and the occipital condyle inferomedially.* C, *drawing showing mobilization of vascular compression with Teflon sling and spacers. A Teflon sling has been passed over and around the VA and PICA to retract them completely away from the compressed nerves. Teflon spacers (also shown) may also be necessary to mobilize a large, tortuous, and atherosclerotic VA away from the compressed nerves.* D, *drawing showing dural closure using subcutaneous fascia graft. The fascia graft is sutured into place initially with single stitches that force it to stay entirely beneath the dural opening. Thus, intracranial CSF pressure actually serves to push the graft tighter against the dural opening, thus reinforcing the watertight closure.*

for dural closure. The craniectomy is centered below the inferior nuchal line and digastric groove and is extended inferiorly until the horizontal portion of the occipital bone is encountered (*Fig. 1B*). The craniectomy is extended laterally to skeletonize the sigmoid sinus and inferomedially to remove a portion of the occipital condyle. The removal of this additional bone may increase the risk of bleeding from the sigmoid sinus, but with the use of a stable pneumatic drill system, we have not encountered this complication. Furthermore, in our opinion, the small risk of bleeding from the sigmoid sinus is outweighed by the improved anterior and inferior viewing angle, which greatly limits the need for cerebellar retraction. With this improved exposure, the lower cranial nerves are immediately visible on opening of the dura, and only minimal elevation of the base of the cerebellum is necessary to visualize the entire length of the glossopharyngeal and vagus nerves.

The dura mater is initially opened inferomedially to decompress the cerebellomedullary cistern, obviating the need for cerebrospinal fluid (CSF) drainage through a lumbar catheter, which can occasionally cause significant postoperative discomfort. A retractor with a 2-mm-wide tip is introduced at an angle 60 degrees inferior to the angle of the cerebellar folia, and the spinal accessory nerve is followed over the jugular tubercle to the jugular foramen. To protect the lower cranial nerves, the arachnoid is dissected only on the cerebellar side, leaving the arachnoid above the nerves intact. Although this approach may seem counterintuitive, we have found that in this region, the lower cranial nerves are frequently enveloped in an arachnoid envelope that extends under the choroid plexus of the lateral recess. Leptomeningeal vessels and small branches of the anteroinferior cerebellar artery (AICA) and posteroinferior cerebellar artery (PICA) are often adherent to this arachnoid sleeve, and preserving it does not impair access to the nerve root entry zone and permits added protection of the nerves and vessel branches.

At this point, it is critical to assess the anatomy and course of perforating branches off of the vertebral artery (VA) and PICA to prevent injury to them. The offending vessel or vessels, which usually arise from below and ventral to the nerves, are typically repositioned superiorly and laterally away from the nerves with a Teflon sling that is secured to the petrous dural wall with fibrin glue (*Fig. 1C*). Although displacement of the vessels inferiorly can also be effective, we find that superolateral displacement requires less manipulation of the glossopharyngeal and vagus nerves. The technique of using a Teflon sling to suspend the offending vessels away from the nerves has been reported previously (11). In our patients, one sling is usually sufficient for adequate mobilization, but occasionally, large vessels, especially when the VA is involved, may require that two or three slings be placed. We have also found larger Teflon sponges useful to mobilize large and atherosclerotic VA away from the compressed nerves. Although aseptic meningitis and recurrent pain caused by Teflon granulomatous reactions have been reported (2, 4, 14), we believe that use of a Teflon sling to reposition the vessels, leaving nothing in contact with the nerves, reduces the likelihood that scar formation around the nerves may occur and lead to recurrent pain. We will not risk stretching or kinking a perforator vessel to do this; however, when necessary, we will use the more traditional approach of placing a Teflon sponge between the nerve and vessel. This has been necessary in only a few of our patients, however.

The dura mater is then closed in a watertight manner with the fascial graft harvested during the opening (*Fig. 1D*). We believe that in addition to thoroughly waxing closed any mastoid air cells that have been opened, CSF leaks can be virtually eliminated by use of this grafting technique. The graft is sutured into place entirely beneath the dural opening so that intracranial CSF pressure actually serves to push the graft tighter against the dural opening, thus reinforcing the watertight closure. This closure is then reinforced with fibrin glue and covered with a dural substitute. The craniectomy site is then covered with a titanium plate, and the soft tissues are closed tightly in anatomic layers.

RESULTS

Between 1984 and 1991, 47 patients underwent MVD for idiopathic GN. Thirty-two (68%) of the 47 patients presented with paroxysmal attacks of pain throughout the sensory distribution of the glossopharyngeal and vagus nerves, whereas 14 patients (30%) presented with pain only in the throat or oropharynx, and 1 patient (2%) presented with pain only in the external ear canal. In addition, pain in the region of the mandible was present in 4 (9%) of the 47 patients. Other cranial nerve neuralgias were present concurrently with symptoms of GN in 5 patients (11%). Two of these 5 patients (4%) presented with symptoms of GN and TN, and the other 3 patients (6%) presented with GN and superior laryngeal nerve neuralgia.

Patient Demographics

The patient population in this study consisted of 18 men (38%) and 29 women (62%). The age range of the patients was 29 to 82 years (mean, 56.4 yr). The majority of patients presented with left-sided symptoms (72%, 34 of 47 patients). On average, patients were symptomatic for 7.1 years before surgery (range, 0.5–35 yr). Medical therapy was unsuccessful in all patients before surgical intervention. In addition, 17 (36%) of the 47 patients had unsuccessfully undergone one or more previous interventional therapies, including percutaneous alcohol block (14 patients), peripheral neurotomy (2 patients), partial rhizotomy (1 patient), and stylomastoid process resection (1 patient). Moreover, two patients had undergone MVD for TN (which we believe was misdiagnosed) without pain relief.

Surgical Findings

In our series of 47 patients, a single artery alone was found to be compressing the glossopharyngeal or vagus nerve in 33 patients (70%) (*Table 1*). In 32 of these 33 patients, the PICA

TABLE 1. Offending vessel involvement[a]

Vessel	No. of patients
Single arteries	3/47 (70%[b])
PICA	32/47 (68%)
VA	1/47 (2%)
Multiple arteries	8/47 (17%)
PICA + AICA	5/47 (11%)
AICA + VA	1/47 (2%)
PICA + AICA + VA	2/47 (4%)
Vein alone	3/47 (6%)
Vein + artery (PICA)	3/47 (6%)

[a] PICA, posteroinferior cerebellar artery; VA, vertebral artery; AICA, anteroinferior cerebellar artery.
[b] Percentages do not total 100% because of rounding off of percentages.

alone was implicated, and in 1 of them, the VA alone was implicated. In 2 of the 33 patients, superior cerebellar artery compression of the trigeminal nerve was also observed. A combination of multiple arterial loops was found in 8 (17%) of the 47 patients. Of these 8 patients, the PICA and AICA were both involved in 5 patients, the AICA and VA were involved in 1 patient, and all three of these vessels were found to compress the nerve roots in 2 patients. Venous compression was found in 6 of the 47 patients. In 3 (6%) of them, the PICA and a vein were found to be compressing the nerves, whereas in the other 3 patients (6%), a single vein alone was found. None of the patients in this series underwent selective sectioning of the glossopharyngeal or vagus nerves, but if vascular compression is not identified, this approach would not be unreasonable if the surgeon was convinced of the diagnosis.

Surgical Outcomes

All 47 patients were seen in follow-up 6 months or longer after surgery. Complete relief from GN was obtained for 46 (98%) of the 47 patients. The clinical pain syndrome remained unchanged in 1 patient (2%). It is interesting to note that the patient who did not experience any postoperative pain relief also had no obvious arterial compression of the glossopharyngeal or vagus nerves on surgical exposure. In this patient, a suspicious vein encircling the glossopharyngeal nerve was coagulated and cut without relief.

Long-term follow-up of more than 10 years was available by direct patient contact for 29 (62%) of the 47 patients. An unbiased attempt was made to contact all patients for this review; however, given that only patients with surgery performed more than 10 years previously were included in this study, some patients were lost to follow-up during the long period of time since surgery. Overall, postoperative follow-up was between 124 and 211 months (median, 152 mo, or 12.7 yr). A recurrence of GN was seen in only one patient. This patient presented with symptoms consistent with GN and TN of the V3 distribution and had previously undergone unsuccessful MVD for TN. On secondary surgical exposure, no compression of the trigeminal or the glossopharyngeal nerves was found. The PICA seemed to be compressing only the vagus nerve. This was decompressed, and a partial rhizotomy of the Vth nerve was performed, with only partial relief of the TN. This patient had a recurrence of intermittent GN pain 8 months after surgery. However, the pain in this patient was now controllable with carbamazepine, whereas it had been medically intractable before surgery.

There was no surgical mortality or life-threatening complications in this series. No CSF leaks occurred, and there was no hearing loss. Nevertheless, 16 (34%) of 47 patients experienced some postoperative neurological deficits (*Table 2*); however, most of these were mild and reversible. Immediately after surgery, 5 patients (11%) complained of dysphagia only, 3 patients (6%) complained of hoarseness only, and 2 patients (4%) had facial paresis only (House-Brackman Grade II/VI). In addition, 5 patients (11%) complained of hoarseness and dysphagia, and 1 patient (2%) complained of hoarseness and dysphagia and had facial paresis. In all but 5 patients, how-

TABLE 2. Postoperative neurological deficits

Neurological deficit	Immediate postoperative period	Permanent
Dysphagia only	5/47 (11%)	1/47[a] (2%)
Hoarseness only	3/47 (6%)	1/47 (2%)
Facial paresis only	2/47[b] (4%)	1/47[c] (2%)
Hoarseness and dysphagia	5/47 (11%)	2/47 (4%)
Hoarseness, dysphagia, and facial paresis	1/47 (2%)	
Total	16/47 (34%)	5/47 (11%)

[a] May have resolved. Patient lost to follow-up.
[b] House-Brackmann Grade II–III/VI. Suspected to be caused by disruption of perforating vessel from posteroinferior cerebellar artery supplying facial nucleus.
[c] House-Brackmann Grade II/VI.

ever, the symptoms and signs resolved completely. Thus, permanent neurological deficits attributable to surgery were observed in 5 patients (11%). Of these patients, 4 had symptoms of mild hoarseness or dysphagia, and 1 had a persistent Grade II/VI facial nerve paresis.

DISCUSSION

Since the first description of surgical treatment of GN by Sicard and Robineau (19) in 1920, a number of surgical treatments of GN have been proposed. Dandy (5), in his seminal article on the subject, described two cases of GN cured by surgical sectioning of the glossopharyngeal nerve intracranially, proximal to the jugular foramen. As in these early reports, the definitive treatment of GN has traditionally relied on destruction of the nerve by sectioning of the glossopharyngeal nerve and selected rootlets of the vagus nerve or destruction by percutaneous rhizolysis.

Rushton et al. (18), in a large retrospective analysis of the medical and surgical treatment of GN, described 217 cases of GN treated at the Mayo Clinic between 1922 and 1977. Of these patients, 129 were treated surgically. The majority of patients were treated by intracranial section of the glossopharyngeal nerve at the jugular foramen, as endorsed by Dandy. This technique was further refined in 1948 to include the upper rootlets of the vagus when sectioning of only the glossopharyngeal nerve did not achieve clinical relief in several patients. Of the 129 patients who underwent surgery for GN, 110 patients (85%) received good relief of pain. In addition to a 5% operative mortality, the morbidity of the procedure was relatively high, including 25 patients (19%) who experienced permanent or temporary dysphagia after the procedure. In their review, Rushton et al. (18) did not focus on the pathogenesis of GN; however, they did report that in the operative reports of 19 patients, a specific comment was made indicating that there was compression of the IXth or Xth nerve by an arterial loop.

The first association between GN and vascular compression of the glossopharyngeal nerve was reported in 1889 by Pope (13), who described a patient who experienced pain and loss of taste as a result of compression of the glossopharyngeal nerve by a dilated thrombosed VA. In 1934, Dandy (5) first described the relationship between TN and vascular arterial contacts compressing and thereby irritating the dorsal root of the trigeminal nerve. Shortly thereafter, in 1936, Lillie and Craig (10) described an anomalous arterial loop as the cause of GN.

The theory that compression of the glossopharyngeal and vagus nerves by an aberrant vascular structure as the major cause of GN was popularized only relatively recently. In 1977, Laha and Jannetta (9) proposed that GN could be treated by surgically relieving the pressure that offending vascular structures imposed on the glossopharyngeal and vagus nerves. They reported six cases of GN in which aberrant loops of the VA or PICA were found to compress the glossopharyngeal or vagus nerves. Two of these patients underwent section of the glossopharyngeal nerve with division of the upper rootlets of the vagus nerve, three others underwent MVD by mobilization of the offending artery, and the final patient underwent MVD and sectioning. Each of the three patients who underwent division of the rootlets experienced complete resolution of the symptoms. Of the three patients who underwent MVD alone, one experienced complete resolution of symptoms, another continued to experience pain, and the third died after surgery after experiencing an intracerebral hemorrhage. Although these results were by no means ideal, the authors concluded that vascular compression was an important cause of GN and that GN could be relieved by separation of the nerve from the offending vessel without having to destroy portions of the nerves.

Our series is the second large clinical series supporting MVD as an efficacious and safe treatment for GN. However, this is the first large series to include significant long-term follow-up extending over more than 1 decade. Long-term follow-up for a minimum of 10 years (median, 12.7 yr) was ascertained by direct contact for 29 (62%) of the 47 patients in this series. Resnick et al. (16) achieved excellent postoperative surgical results in 79% of patients, and 76% continued to have excellent long-term relief of symptoms. However, in their series, follow-up was evaluated at 1 to 13 years after surgery, with a mean follow-up of only 4 years. More recently, this University of Pittsburgh series has been extended to a remarkable 217 patients (12), but follow-up for more than 4 years was available for only 20 of the 217 reported patients. Kondo (7) reported on a smaller series of 17 patients with a mean follow-up of 11.6 years (range, 5–16 yr) without pain recurrences. In his discussion, he emphasizes the importance of long-term follow-up, noting that earlier reports of MVD for TN found recurrence of symptoms in some patients 5 years after surgery. Taha and Tew (22) also reported four cases of GN treated with a combination of MVD and rhizotomy with excellent results and follow-up of 5 to 12 years, but they also commented that although MVD seems promising as a sole treatment, longer follow-up data in larger series were needed. Of our 29 patients for whom long-term data were available in our series, 28 experienced pain relief after MVD, and 27 continued to be pain-free for more than 10 years after treatment. This is particularly noteworthy given that 17 (36%) of the 47 patients had undergone previous destructive procedures. Although we cannot reliably correlate our surgical responses with these previous procedures, we did note that very few of the patients treated previously with destructive procedures had evidence of neurological deficits at the time of our surgery. This lack of neurological deficit resulting from previous destructive procedures may be important in selecting patients who might benefit from MVD surgery for this condition. The one patient with recurrence did receive temporary relief of symptoms after MVD; her pain recurred 8 months after surgery, but it was controllable with carbamazepine. We concede that a bias could exist in our data, inasmuch as a significant portion of our initial patient group could not be contacted more than 10 years after surgery. However, as patients are

followed up over longer periods of time, this becomes unavoidable, and we may have to accept that better data on long-term follow-up may never be available. Furthermore, even if we make the highly unlikely assumption that all patients lost to follow-up had pain recurrences, we were still able to confirm that 27 (57%) of 47 patients obtained pain relief for more than 10 years. It therefore seems very likely that MVD provides an effective treatment for GN that is at least comparable to selective nerve sectioning and that its benefits are likely to be durable.

Resnick et al. (16) reported a postoperative paresis of the IXth and Xth nerves in four patients (10%), of whom only three (8%) had permanent deficits. In our series, 16 (34%) of 47 patients experienced immediate postoperative symptoms consistent with cranial nerve deficits; however, most of these deficits were minor and temporary. Only 5 (17%) of 29 patients for whom long-term data were available experienced lasting deficits: 1 patient had a persistent facial nerve palsy, and the other 4 patients had symptoms of mild dysphagia or hoarseness. Although one of the purported advantages of MVD over selective sectioning of the glossopharyngeal and vagus nerves is the reduced incidence of injury to the glossopharyngeal and vagus nerves, it is clear from our series that permanent injury to these nerves will not always be avoided by MVD. Although such deficits after MVD tend to be transient and mild, we concede that they may also play a role in pain relief. Resnick et al. (16) reported a surgical mortality of 5% (2 of 40 patients); these deaths were all secondary to intraoperative hemodynamic instability. In our series, there were no deaths as a result of surgery and no indication of serious intraoperative hemodynamic instability in any of the patients.

CONCLUSION

The techniques and results described in this article demonstrate that MVD can be a safe and effective treatment for GN and that the benefit derived from MVD seems to be durable. Although most centers still have limited experience with MVD, this series, along with the experience from other centers frequently performing MVD for GN, suggests that MVD can be a safe and effective primary and sole surgical treatment modality for GN.

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COMMENTS

In this long-term follow-up of patients who underwent microvascular decompression (MVD) for glossopharyngeal neuralgia (GN), Sampson et al. have extended the follow-up period by a median of 1 year with a nice range, and they demonstrate the continuing durability of MVD for this particular problem. Their article represents a clear, succinct description of the work and one or two additional elements to the

exposure introduced for the general readership's evaluation. We do not think that removal of the occipital condyle needs to be a regular part of the procedure, and the exposure to but not over the sigmoid and transverse sinuses represents the cornerstone of lateral exposure while maintaining safety in and around these venous sinuses. Careful attention to intraoperative or potential electrophysiology with direct recording from the IXth and Xth cranial nerves as well as the brainstem evoked potential will continue to keep the long-term complication rate in the subjacent cranial nerves to a minimum.

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Peter J. Jannetta
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This well-written, fluent, synthetic, clear article is perhaps the last thing that is missing to validate, without doubt, the efficacy of MVD for the long-term control of GN and most likely its permanent cure. The results are truly excellent, with all except 1 of 47 patients who underwent surgery more than 10 years previously pain-free early after surgery, and all except 1 of 29 patients who could be contacted 10 years later are still pain-free. Also remarkable is the low surgical morbidity at evaluation 6 months after surgery; also interesting are the technical tricks to which readers have become accustomed from one of the authors: the strategy for dural closure and the suspension of the offending vessel away from the nerves. The authors add an important contribution to the topic of MVD.

Albino Bricolo
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Sampson et al. provide conclusive evidence that MVD is an effective long-term treatment for patients with GN. Initial and long-term pain relief exceeds 90%, and major subgroups (28%) were largely transient. My colleagues and I think that MVD is the treatment of choice for patients with GN when vascular compression is evident on the basis of surgical exploration. In the rare case in which compression is not found, sectioning of Cranial Nerve IX and monitoring of the cortical section of Cranial Nerve X are indicated.

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The computer HAL plays chess with Frank in this still from Stanley Kubrick's film 2001: A Space Odyssey*, which was released in 1968. Kubrick went to great lengths to make sure the machines in his film resembled actual technologies, even going so far as to consult with experts from NASA, IBM, Boeing, and other industry leaders. The character of HAL used computational power to usurp control from his human counterparts, who relied on reason and knowledge.*