CASE REPORT

Fully Endoscopic Vascular Decompression of the Facial Nerve for Hemifacial Spasm

Joseph B. Eby, M.D.,¹ SungTae Cha, M.D.,¹ and Hrayr K. Shahinian, M.D.¹

ABSTRACT

Hemifacial spasm is an uncommon disorder manifesting as a unilateral, involuntary, sporadic contraction of the musculature innervated by the seventh cranial nerve. Although debated, the etiology of hemifacial spasm is generally accepted as compression of the facial nerve by vessels of the posterior circulation. Early surgical techniques were ineffective and fraught with morbidity. Over the past 25 years microvascular decompression surgery has allowed the safe and effective treatment of hemifacial spasm. Recent reports combining microsurgical and endoscopic techniques have documented the advantages of the endoscope in exposing the anatomy of this region. Enhanced visualization allows a less traumatic dissection and increases the surgeon's ability to locate nerve-vessel conflicts often difficult to identify through the limited view of the microscope. This article reviews the history of hemifacial spasm and describes the first three cases of fully endoscopic vascular decompression for hemifacial spasm, emphasizing the advantages of this novel surgical approach.

KEYWORDS: Endoscopy, hemifacial spasm, vascular decompression

Hemifacial spasm (HFS) is commonly described as a unilateral, involuntary, paroxysmal contraction of the musculature innervated by the seventh cranial nerve. The incidence of HFS is approximately 0.8 per 100,000 persons.¹ Fukushima in 1995 published one of the largest series of patients (2890) undergoing decompressive surgery for HFS.² This series demonstrated a preponderance of adult females with a F:M ratio of greater than 2:1 and a mean age of 51.²

Crucial to understanding and treating HFS is obtaining a proper diagnosis. The differential diagnosis of HFS includes but is not limited to essential blepharospasm, facial myokymia, focal seizures, tardive dyskinesia, Meige's syndrome, synkinesis, and tics, a full discussion of which is beyond the scope of this paper.³⁻⁵

Beginning with some of the earliest descriptions of HFS by Shultze in 1875 and Gowers in 1899, the etiology of hemifacial spasm and loca-

Skull Base, volume 11, number 3, 2001. Address for correspondence and reprint requests: Hrayr K. Shahinian, M.D., Division of Skull Base Surgery, Cedars-Sinai Medical Center, 8635 West Third Street, Suite 490W, Los Angeles, CA 90048. E-mail: hrayr.shahinian@cshs.org.

¹Division of Skull Base Surgery, Cedars-Sinai Medical Center, Los Angeles, California. Copyright © 2001 by Thieme Medical Publishers, Inc., 333 Seventh Avenue, New York, NY 10001, USA. Tel: +1(212) 584-4662. 1531-5010,p;2001,11,03,189,198,ftx,en;sbs00230x.

tion of the abnormality have been debated for more than a century.²⁻⁴ Oppenheim in 1911 as well as Ehni and Woltman in 1945 reported on patients with hemifacial spasm who sustained contralateral strokes. These patients were left with a facial droop, which did not relieve the spasm; the researchers therefore concluded that the facial nerve nucleus was not the origin of this disorder.^{3,6} Surgical treatment for HFS in the early 20th century included neurolysis, stretching the facial nerve, and high-pressure irrigation of the nerve with lactate Ringer's solution. Medical regimens of the time involved injection of the nerve with ethanol, electrical stimulation, the application of toxic compounds (nitrate of silver, zinc, arsenic, bromides) as well as medications such as Dilantin or other anticonvulsants.^{2-4,6-8}

Watts, McKillop, and Lelli in the 1930s and Sutherland in the 1940s studied cadaveric anatomy of the posterior circulation at the cerebellopontine angle, its close proximity, and occasional incidental compression of the cranial nerves.^{2,4,9,10} Dandy first hypothesized in 1934 that compression of the trigeminal nerve was the cause of trigeminal neuralgia.¹¹ Then in 1945 Revilla and Dandy documented their experience with 160 patients with tumors of the cerebellopontine angle and described HFS in six of these patients.¹² In 1945, Ehni and Woltman analyzed 106 cases of HFS and concluded that the facial nerve abnormality must lie between the facial nucleus and the stylomastoid foramen.⁶ In 1947, Campbell and Keedy presented two cases of HFS resulting from an aneurysm of the vertebral artery at the cerebellopontine angle, providing additional evidence that vascular compression of the facial nerve may indeed be the cause of HFS.¹³

In 1960 Gardner published a case report of a patient with HFS treated by vascular decompression and demonstrated an excellent result with 5-year follow-up.¹⁴ Then in 1962, reporting on 19 patients operated on for HFS, Gardner noted that aneurysms, AVMs, or ectatic vessels were inti-

mately associated with the facial nerve in the majority of cases.⁸ From this he concluded: "hemifacial spasm is the expression of a reversible pathologic state commonly (in 13 of 19 cases) produced by mild, long continuing compression of the 7th cranial nerve by a vascular structure in the cerebellopontine angle."⁸ At this time treatment of HFS was primarily medical or involved neurolysis.^{2-4,6,7} Gardner's excellent results in his first study, immediate relief in 12 patients, delayed relief in 5, and 2 failures (where the nerve was not compressed by any visible structure), supported his opinion that vascular compression was the likely cause of HFS.^{2,8,15} Gardner's early success, however, was followed by significant operative complications in later reports; as a result, his decompression operation lost much of its early appeal.²

Additional advances in understanding the etiology and improving the treatment of HFS did not occur until the mid-1970s.^{16,17} Jannetta's 1977 article documented 47 cases of HFS all decompressed using the operating microscope and illustrated nerve-vessel conflicts (or cholesteatoma in one case) located at the root exit zone (REZ) of the facial nerve in all cases.¹⁷ The REZ is where the central glial axonal insulation of the nerve ends and the peripheral axonal myelination begins. Biopsies of the REZ reported by Ruby and Jannetta demonstrated degeneration of axons, denuded axis cylinders, and interrupted myelin.¹⁸ Jannetta's results strengthened the theory that vascular compression was the primary cause of HFS, and he proposed that the longstanding effects of compression of a specific region of the facial nerve (the REZ) result in nerve dysfunction. Moreover, this paper demonstrated that by using the operating microscope to guide decompression of the nerve, a safer and more complete operation could be performed.^{16,17}

Three theories have been proposed to explain the facial nerve dysfunction found in HFS. Gardner first proposed ephaptic transmission in 1962.^{19,20} Work by Digre and Corbett as well as

by Nielsen and Moller support this theory, which states that normal electrical activity can cross from one demyelinated neuron to another resulting in a false synapse. The second theory involves abnormal axonal activity at the REZ secondary to compressive damage/demyelination. The third theory, the "kindling theory," involves increased excitability of the facial nerve nucleus due to feedback from a damaged facial nerve.^{3-5,21-23} The mainstay of medical therapy for HFS in the 21st century involves injection of the muscle with botulinum toxin. Additional treatments include the use of anticonvulsants such as Carbamazepine or Dilantin.^{3,4}

In the past 25 years since the introduction of the operating microscope, ample evidence from several large series' reports has reinforced the position that compression of the facial nerve by ectatic vessels of the posterior fossa is the principle cause of HFS.^{1-5,8,15-18,22,24-41} Overall, microvascular decompression yields an "excellent result" (complete resolution of symptoms) with minimal morbidity in 70 to 95% of patients undergoing this procedure.^{1-5,17,24-32,35-38,40-42} The dramatic improvement in symptoms and minimal complications have led some authors to claim that HFS is primarily a surgical disease, and as such, they recommend early operative decompression.^{2,20} However, several reports have documented the failures, recurrences, and complications related to microscopically decompression.⁴²⁻⁴⁷

In 1994, Magnan introduced a combined microscopic and endoscopic approach to vascular decompression of the facial nerve for HFS.³³ Several papers and an anatomic atlas also have described the endoscopic anatomy of the posterior fossa.^{33,34,48,49} However, no reports have documented a completely endoscopic approach to vascular decompression of the facial nerve for hemifacial spasm. We therefore present three patients suffering from hemifacial spasm who underwent a fully endoscopic vascular decompression of the facial nerve and experienced full resolution of their symptoms.

CASE REPORTS

Before the procedures were formed, the protocol was reviewed and approved by the hospital's Institutional Review Board Committee, and informed consent to perform fully endoscopic vascular decompression was obtained from each patient.

Case 1

A 72-year-old female had a 6-year history of left hemifacial spasm treated with repeated botulinum toxin injection. The patient presented for definitive therapy, as she was unhappy with the pain of the injections, the temporary period of relief (3 to 4 months), and the slight facial weakness she experienced after these injections. On examination, frequent involuntary contractions of muscles of the left face primarily involved the orbicularis oculi and orbicularis oris muscles. Sagittal and coronal T1-weighted images, thin-section axial pre- and postgadolinium images with fat saturation, and T2-weighted axial proton density images demonstrated a tortuous left anterior inferior cerebellar artery close to the cisternal portion of the left facial nerve.

A completely endoscopic surgical procedure was performed via a retrosigmoid keyhole approach as outlined in the surgical technique section. At surgery, a loop of the anterior inferior cerebellar artery (AICA) and the main left vertebral artery were both compressing the REZ of the left facial nerve (Fig. 1). These structures were separated, and Teflon pledgets were placed between the nerve and arteries to provide complete decompression (Fig. 2).

The patient was taken to the surgical intensive care unit for overnight neurologic monitoring. The next day she was transferred to the floor and subsequently discharged home on postoperative Day 3. Her facial spasm resolved completely with no facial weakness. At a 7-month follow-up examination, her hemifacial spasm had not recurred.

Figure 1 Case 1. An intraoperative photograph taken from the endoscope reveals a loop of the anterior inferior cerebellar artery (white arrow) and the main left vertebral artery (outlined arrow) both compressing the facial nerve (black arrowhead).

Case 2

A 70-year-old male with a 15-year history of right-sided hemifacial spasm had undergone more than five botulinum toxin injections over a 4-year period with only a temporary improvement in his symptoms. The patient exhibited active involuntary facial twitching, primarily in the orbicularis oculi and right upper cheek. He also had mild facial asymmetry, with a mild right lower facial droop thought

Figure 2 An intraoperative photograph of the same patient (Case 1) after endoscopic vascular decompression and placement of Teflon pledgets to prevent recurrent nerve compression.

to be secondary to multiple botulinum injections. Facial nerve electromyography revealed some evidence of deinnervated facial musculature. Preoperatively, T1- and T2-weighted, proton-density, parasagittal, coronal, and axial slices demonstrated ectatic vertebral and basilar arteries located in the right cerebellopontine angle.

A completely endoscopic surgical procedure was performed via a retrosigmoid keyhole approach as outlined in the surgical technique section. At surgery, a tortuous loop of the AICA and the main right vertebral artery were found to be compressing the REZ of the right facial nerve (Fig. 3). These structures were separated and Teflon pledgets were placed between the nerve and arteries to provide complete decompression (Fig. 4).

The patient was transferred to the surgical intensive care unit and extubated within 12 hours of the operation. He was neurologically intact but lethargic. CT demonstrated tension pneumocephalus, which was treated with bed rest and oxygen. Repeat CT within 24 hours showed >50% reduction in the amount of intracranial air. The patient's hemifacial spasm resolved completely. Immediately after surgery he exhibited mild gait instability and mild nystagmus (L>R) gaze. Before discharge both findings improved significantly.

Figure 3 Case 2. An intraoperative photograph taken from the endoscope reveals a tortuous loop of the anterior inferior cerebellar artery (white arrow) and the right vertebral artery (outlined arrow) compressing the facial nerve (black arrowhead).

Figure 4 An intraoperative photograph of the same patient (Case 2) after endoscopic vascular decompression and placement of Teflon pledget (black arrow) to prevent recurrent nerve compression.

The patient remained in the hospital for acute rehabilitation and was discharged home on postoperative Day 7. Follow-up at one year revealed no hemifacial spasm, complete resolution of the gait disturbance, and no remaining nystagmus or facial weakness.

Case 3

A 51-year-old female with a 6-year history of right-sided hemifacial spasm had undergone multiple botulinum toxin injections over the course of her illness, most of which provided relief for about 3 months. Multiple paroxysms of right facial spasm involved the entire facial nerve distribution. The patient also demonstrated mild right facial weakness.

A completely endoscopic surgical procedure was performed via a retrosigmoid keyhole approach as outlined in the surgical technique section. At surgery, a complex compression of the facial nerve by the AICA, which pierced the acousticofacial bundle between the facial nerve and the vestibulocochlear nerve, was noted. The AICA was dissected free from the facial nerve, and two 3-mm Teflon pledgets were placed between them.

The patient was taken to the surgical intensive care unit overnight; her hemifacial spasm resolved completely. She was transferred to the floor on postoperative Day 1 and discharged on postoperative Day 3. Immediately after surgery the patient demonstrated grade III-IV facial paresis, which had completely resolved by her 4-week follow-up visit. At her 6-month follow-up examination, she reported no recurrence of her hemifacial spasm.

SURGICAL TECHNIQUE

Under general anesthesia without paralysis, the patient is placed in a park-bench position. The head is secured in mild flexion to expose the affected mastoid region. Intraoperatively, the facial nerve is monitored in all cases. A 3-cm retroauricular scalp incision is made, and the dissection continues to the cranium where a 1.5-cm retrosigmoid craniotomy is performed. The dura is incised and cerebrospinal fluid (CSF) is drained, allowing the structures of the posterior fossa to fall away without retraction. A 4-mm 0-degree rigid endoscope (Karl Storz of America, Culver City, CA) is introduced into the posterior fossa between the posterior aspect of the petrous bone and the cerebellum and is then advanced slowly to visualize the cerebellopontine angle. The acousticofacial bundle is located, and the facial nerve is stimulated and identified positively. Once identified the surrounding vascular structures are surveyed to find any nerve-vessel conflicts or other compression of the facial nerve. (In all cases one or more offending vessels were identified.) Using microdissection and gentle manipulation the adhesions and compressions from the vessel(s) on the facial nerve are lysed, and the nerve and vessel(s) are freed from one another. After this dissection, a Teflon pledget is placed between the two structures and secured with fibrin glue. The dura is reapproximated and sutured to provide a watertight closure. The craniotomy is re-

constructed using the bone plug, while the soft tissues and skin are reapproximated using sutures. The patient is awakened from general anesthesia, extubated, and transported to the surgical intensive care unit for observation.

RESULTS

Each of these three cases demonstrated one or more nerve-vessel conflicts. Relieving the facial nerve compression completely resolved their hemifacial spasm. The endoscope was used to perform the vascular decompression in each case. The anatomy of the cerebellopontine angle and the area of nerve-vessel conflicts were well visualized with the endoscope alone. The improved visualization enhanced the surgeon's ability to perform the surgical dissection and to assess the adequacy of the decompression, both of which are often difficult to appreciate using the operating microscope.

Complications were limited to mild temporary facial weakness, and one patient experienced postoperative tension pneumocephalus that resolved with medical management and time. There were no cases of hearing loss, permanent facial paresis, or other neurological deficits. All patients were discharged within 1 week of surgery. Follow-up examinations showed complete resolution of their hemifacial spasms.

DISCUSSION

Although tumors, bony abnormalities, aneurysms, AVMs, and plaques from multiple sclerosis have all been described as causing hemifacial spasm, these abnormalities account for less than 10% of cases. Most cases of HFS are due to abnormal compression of the facial nerve by ectatic vessels of the posterior cerebral and/or cerebellar circulation.^{1-5,8,15-18,22,24-41}

The key aspects of a safe and effective vascular decompression operation for HFS include optimal visualization of the cerebellopontine angle to identify normal and abnormal anatomic relationships, identification of significant nerve-vessel conflicts, atraumatic dissection and separation of these structures, and placement of a permanent barrier to ensure that the compression does not recur.

Access to the posterior cranial fossa via a retrosigmoid approach was first described by Magnan and Bremond in 1974.⁵⁰ In the late 1970s, Jannetta introduced the current standard treatment for this disorder involving microscope-assisted vascular decompression.^{16,17} The complex anatomy of the posterior fossa and the limited size of the craniotomy make adequate visualization of the facial nerve using an operating microscope difficult.^{15,33,34,48,49} Jannetta has pointed out some of the microscope's limitations: "The facial nerve may not be clearly visualized medially at this time because the offending arterial loop, flocculonodular lobe of the cerebellum, or choroid plexus of the lateral recess may be in the way."¹⁷

The earliest recognized use of endoscopy for surgery of the posterior fossa was in 1917 when Doyen used endoscopes to perform trigeminal root neurectomy.⁵¹ After this early report little was written about an endoscopic approach to this region. Then in a cadaveric study in 1993, O'Donoghue and O'Flynn documented the anatomy of the cerebellopontine angle and highlighted the advantages of the endoscope over the operating microscope,⁴⁸ commenting: "The microscope has distinct limitations: the operator can only visualize objects directly ahead and is unable to see around objects or down narrow, tortuous pathways; therefore, access to the side of disease may need wide exposure and retraction of adjacent structures."⁴⁸ Retraction injury of the cerebellum or brainstem during decompressive surgery for HFS can be a considerable source of morbidity.⁴⁴ During our procedures we found that the unimpeded endoscopic view provided excellent panoramic views of

the posterior fossa, cerebellopontine angle, and REZ of the facial nerve, and allowed identification of the nerve-vessel conflicts in all cases without the need for brain retraction.

In 1994 Magnan also demonstrated the utility of the endoscope in exposure of the cerebellopontine angle stating that the endoscope yields "magnified high resolution images and provides an unobstructed view of all nervous and vascular structures in the area."³⁴ In 1997 he used the endoscope to assist microscopic vascular decompression of the facial nerve for HFS, and demonstrated an additional 72% accuracy rate in identifying nerve-vessel conflicts.¹⁵ We found that the endoscope improved visualization of the cerebellopontine angle and avoided the need for retraction; both factors should reduce the number of complications resulting from dissection in this region.

Microscopically assisted vascular decompression for HFS is reported to have a mortality rate of 0.2% and an overall complication rate of approximately 5 to 25% for temporary dysfunction and 2 to 10% for permanent neurologic impairment.^{1-5,15,17,24-32,35-38,40-42} Most complications involve auditory or facial nerve dysfunction. The reported rate of auditory nerve impairment is 3 to 5% for temporary disturbance and 2 to 3% for permanent hearing loss. Facial nerve dysfunction occurs temporarily in about 4% of patients, while 1 to 2% experience permanent facial nerve deficit.^{1,2,4,17,26,27,31,32,35-38,41} Monitoring the facial nerve and brainstem auditory evoked potentials helps reduce these major complications.^{41,46,47} Facial nerve monitoring was performed in all three patients, while brainstem auditory evoked potentials were monitored in two. One patient experienced mild temporary facial nerve weakness. There were no cases of hearing loss or permanent facial nerve weakness.

Hemifacial spasm treated by microscopic decompression has an initial failure rate of 3 to 5% and a recurrence rate of 10 to 20%.^{1-5,15,17,24-32,35-38,40-42} In a 1998 review of cases of recurrent HFS, Kureshi and Wilkins found that 37.5% of patients re-

explored had an artery or vein compressing the facial nerve.⁴² This study also noted an extremely high (75%) complication rate for patients undergoing reoperation for HFS.⁴² As discussed, endoscopy may identify a significant number of nerve-vessel conflicts that are not appreciated using the operating microscope alone.^{15,33,49} Endoscopic identification of the often-obscure areas of nerve compression during the primary operation should improve initial success rates and avoid the morbidity of re-exploration for missed neurovascular conflicts.

CONCLUSION

Overall, these cases demonstrate that the endoscope can be used exclusively to visualize the neurovascular conflicts leading to hemifacial spasm and to decompress the facial nerve safely. Other reports have documented the use of endoscopy at the cerebellopontine angle and have used endoscopy as an adjunct to microscopic dissection. These three patients, however, represent the first documented cases of fully endoscopic decompression of the facial nerve for hemifacial spasm. Additional studies involving a larger series of patients will likely reveal endoscopic vascular decompression of the facial nerve for hemifacial spasm to be a safer, more complete operation, with a lower incidence of complications and recurrence.

REFERENCES

1. Auger RG, Whisnant JP. Hemifacial spasm in Rochester and Olmsted County, Minnesota, 1960 to 1984. *Arch Neurol* 1990;47:1233-1234
2. Fukushima T. Microvascular decompression for hemifacial spasm: Result in 2890 cases. In: Spetzler RF ed. *Neurovascular Surgery*. New York: McGraw Hill Inc; 1995: 1133-1145
3. Hansen C, Montgomery JEB. Hemifacial spasm: Medical treatment. In: Spetzler RF ed. *Neurovascular Surgery*. New York: McGraw Hill Inc; 1995:1119-1131

4. Wilkins RH. Hemifacial spasm: a review. *Surg Neurol* 1991;36:251-277
5. Digre K, Corbett JJ. Hemifacial spasm: differential diagnosis, mechanism, and treatment. *Adv Neurol* 1988;49:151-176
6. Ehni G, Woltman HW. Hemifacial spasm. Review of 106 cases. *Arch Neurol Psychiatry* 1945;53:205
7. Coleman CC. Surgical treatment of facial spasm. *Ann Surg* 1937;105:647-657
8. Gardner JW. Hemifacial Spasm-A reversible pathophysiologic state. *J Neurosurg* 1962;27:240-247
9. Sutherland S. Neurovascular relations and anomalies at the base of the brain. *J Neurol Neurosurg Psychiatry* 1948;11:243-258
10. Sunderland S. The arterial relations of the internal auditory meatus. *Brain* 1945;68:23-27
11. Dandy W. Concerning the cause of trigeminal neuralgia. *Am J Surg* 1934;24:447-455
12. Revilla AG, Dandy W. Neurinomas of the cerebellopontile recess. *Johns Hopkins Hosp Bull* 1947;80:254-296
13. Campbell E, Keedy C. Hemifacial spasm: a note on the etiology in two cases. *J Neurosurg* 1947;4:342-347
14. Gardner JW. Five-year cure of hemifacial spasm. *Cleve Clin Q* 1960;27:219-221
15. Magnan J, Caces F, Locatelli P, Chays A. Hemifacial spasm: endoscopic vascular decompression. *Otolaryngol Head Neck Surg* 1997;117:308-314
16. Jannetta PJ. Trigeminal neuralgia and hemifacial spasm: etiology and definitive treatment. *Trans Am Neurol Assoc* 1975;100:89-91
17. Jannetta PJ, Abbasy M, Maroon JC, Ramos FM, Albin MS. Etiology and definitive microsurgical treatment of hemifacial spasm. Operative techniques and results in 47 patients. *J Neurosurg* 1977;47:321-328
18. Ruby JR, Jannetta PJ. Hemifacial spasm: ultrastructural changes in the facial nerve induced by neurovascular compression. *Surg Neurol* 1975;4:369-370
19. Gardner JW. Concerning the mechanism of trigeminal neuralgia and hemifacial spasm. *J Neurosurg* 1962;19:947-958
20. Gardner JW. Cross talk-the paradoxical transmission of a nerve impulse. *Arch Neurol* 1966;14:149-156
21. Nielsen VK. Electrophysiology of the facial nerve in hemifacial spasm: ectopic/ephaptic excitation. *Muscle Nerve* 1985;8:545-555
22. Nielsen VK, Jannetta PJ. Pathophysiology of hemifacial spasm: III. Effects of facial nerve decompression. *Neurology* 1984;34:891-897
23. Moller AR. Hemifacial spasm: ephaptic transmission or hyperexcitability of the facial motor nucleus? *Exp Neurol* 1987;98:110-119
24. Auger RG, Piepgras DG, Laws ER, Jr., Miller RH. Microvascular decompression of the facial nerve for hemifacial spasm: clinical and electrophysiologic observations. *Neurology* 1981;31:346-350
25. Wilkins RH. Hemifacial spasm: treatment by microvascular decompression of the facial nerve at the pons. *South Med J* 1981;74:1471-1474
26. Iwakuma T, Matsumoto A, Nakamura N. Hemifacial spasm. Comparison of three different operative procedures in 110 patients. *J Neurosurg* 1982;57:753-756
27. Loeser JD, Chen J. Hemifacial spasm: treatment by microsurgical facial nerve decompression. *Neurosurgery* 1983;13:141-146
28. Eidelman BH, Nielsen VK, Moller M, Jannetta PJ. Vascular compression, hemifacial spasm, and multiple cranial neuropathy. *Neurology* 1985;35:712-716
29. Wilkins RH. Neurovascular compression syndromes. *Neurol Clin* 1985;3:359-372
30. Auger RG, Piepgras DG, Laws ER, Jr. Hemifacial spasm: results of microvascular decompression of the facial nerve in 54 patients. *Mayo Clin Proc* 1986;61:640-644
31. Niemeyer Filho P, Bezerra M, Mufarrej G. Hemifacial spasm: results of microvascular decompression in 53 patients. *Arq Neuropsiquiatr* 1990;48:210-216
32. Huang CI, Chen IH, Lee LS. Microvascular decompression for hemifacial spasm: analyses of operative findings and results in 310 patients. *Neurosurgery* 1992;30:53-56
33. Magnan J, Chays A, Caces F, et al. Role of endoscopy and vascular decompression in the treatment of hemifacial spasm. *Ann Otolaryngol Chir Cervicofac* 1994;111:153-160
34. Magnan J, Chays A, Lepetre C, Pencroffi E, Locatelli P. Surgical perspectives of endoscopy of the cerebellopontine angle. *Am J Otol* 1994;15:366-370
35. Barker FG II, Jannetta PJ, Bissonette DJ, Shields PT, Larkins MV, Jho HD. Microvascular decompression for hemifacial spasm. *J Neurosurg* 1995;82:201-210
36. Zhang KW, Shun ZT. Microvascular decompression by the retrosigmoid approach for idiopathic hemifacial spasm: experience with 300 cases. *Ann Otol Rhinol Laryngol* 1995;104:610-612
37. Caces F, Chays A, Locatelli P, et al. Neuro-vascular decompression in hemifacial spasm: anatomical, electrophysiological and therapeutic results apropos of 100 cases. *Rev Laryngol Otol Rhinol* 1996;117:347-351
38. Illingworth RD, Porter DG, Jakubowski J. Hemifacial spasm: a prospective long term follow up of 83 cases treated by microvascular decompression at two neurosurgical centers in the United Kingdom. *Neurol Neurosurg Psychiatry* 1996;60:72-77
39. Kondo A. Follow-up results of microvascular decompression in trigeminal neuralgia and hemifacial spasm. *Neurosurgery* 1997;40:46-51
40. Moller AR. Vascular compression of cranial nerves. I. History of the microvascular decompression operation. *Neurol Res* 1998;20:727-731
41. McLaughlin MR, Jannetta PJ, Clyde BL, Subach BR, Comey CH, Resnick DK. Microvascular decompression of cranial nerves: lessons learned after 4400 operations. *J Neurosurg* 1999;90:1-8

42. Kureshi SA, Wilkins RH. Posterior fossa reexploration for persistent or recurrent trigeminal neuralgia or hemifacial spasm: surgical findings and therapeutic implications. *Neurosurgery* 1998;43:1111-1117
43. Haines SJ, Torres F. Intraoperative monitoring of the facial nerve during decompressive surgery for hemifacial spasm. *J Neurosurg* 1991;74:254-257
44. Hanakita J, Kondo A. Serious complications of microvascular decompression operations for trigeminal neuralgia and hemifacial spasm. *Neurosurgery* 1988;22:348-352
45. Nishihara K, Hanakita J, Kinuta Y, Kondo A, Yamamoto Y, Nakatani H. Importance of intraoperative monitoring of ABR and compound action potential of the eighth cranial nerve during microvascular decompression surgery. *No Shinkei Geka* 1986;14:509-518
46. Radtke RA, Erwin CW, Wilkins RH. Intraoperative brainstem auditory evoked potentials: significant decrease in postoperative morbidity. *Neurology* 1989;39:187-191
47. Moller AR, Moller MB. Does intraoperative monitoring of auditory evoked potentials reduce incidence of hearing loss as a complication of microvascular decompression of cranial nerves? *Neurosurgery* 1989;24:257-263
48. O'Donoghue GM, O'Flynn P. Endoscopic anatomy of the cerebellopontine angle. *Am J Otol* 1993;14:122-125
49. Jennings CR, O'Donoghue GM. Posterior fossa endoscopy. *J Laryngol Otol* 1998;112:227-229
50. Bremond G, Garcin M, Magnan J, Bonnaud G. L'abord à minima de l'espace pon to-cerebelleux. *Cah ORL* 1974;9:443-460
51. Doyen E. Surgical therapeutics and operative techniques. In: Balliere, Tindall, Cox, eds. vol 1. London, Inc; 1917: 599-602