Failed microvascular decompression surgery for hemifacial spasm due to persistent neurovascular compression: an analysis of reoperations

Mark Gregory Bigder, MD, and Anthony M. Kaufmann, MD, MSc, FRCSC

Centre for Cranial Nerve Disorders, Section of Neurosurgery, Department of Surgery, University of Manitoba, Winnipeg, Manitoba, Canada

OBJECTIVE Microvascular decompression (MVD) surgery for hemifacial spasm (HFS) is potentially curative. The findings at repeat MVD in patients with persistent or recurrent HFS were analyzed with the aim to identify factors that may improve surgical outcomes.

METHODS Intraoperative findings were determined from review of dictated operative reports and operative diagrams for patients who underwent repeat MVD after prior surgery elsewhere. Clinical follow-up was obtained from the hospital and clinic records, as well as telephone questionnaires.

RESULTS Among 845 patients who underwent MVD performed by the senior author, 12 had been referred after prior MVD for HFS performed elsewhere. Following repeat MVD, all patients improved and complete spasm resolution was described by 11 of 12 patients after a mean follow-up of 91 ± 55 months (range 28–193). Complications were limited to 1 patient with aggravation of preexisting hearing loss and mild facial weakness and 1 patient with aseptic meningitis without sequelae. Significant factors that may have contributed to the failure of the first surgery included retromastoid craniectomies that did not extend laterally to the sigmoid sinus or inferiorly to the posterior fossa floor in 11 of 12 patients and a prior surgical approach that focused on the cisternal portion of the facial nerve in 9 of 12 patients. In all cases, significant persistent neurovascular compression (NVC) was evident and alleviated more proximally on the facial root exit zone (fREZ).

CONCLUSIONS Most HFS patients will achieve spasm relief with thorough alleviation of NVC of the fREZ, which extends from the pontomedullary sulcus root exit point to the Obersteiner-Redlich transition zone.

<http://thejns.org/doi/abs/10.3171/2015.1.JNS142714>

KEY WORDS hemifacial spasm; microvascular decompression; facial root entry zone; neurovascular compression; surgical volume; reoperation; functional neurosurgery

HEMIFACIAL spasm (HFS) is relatively rare, with an estimated incidence of 0.8 per 100,000 persons/year and an estimated prevalence of 7.4 per 100,000 men and 14.5 per 100,000 women.² The disease typically begins as infrequent twitches of an eyelid that progress over time in intensity, frequency, and extent of involvement of the ipsilateral facial muscles. HFS may cause significant disability due to annoying and distracting twitches and forceful spasms, and also by the loss of binocular vision associated with involuntary eye closure. The greatest impact on the quality of life for many patients is the psychological and social impact of the facial disfigurement caused by the disorder.^{15,17}

Surgical cure of HFS can be achieved with microvascular decompression (MVD) surgery, as pioneered by Jannetta in 1967,¹² and several series have demonstrated high rates of success and safety.^{3,4,6–8,10,11,13,20,21} The aim of this surgery is to alleviate culprit neurovascular compression (NVC) upon the facial nerve, most commonly at the facial root exit zone (fREZ). Only in rare cases is HFS caused by severe NVC of the cisternal portion of the facial nerve.^{4,5} The extent of the fREZ, however, has been variably defined and is often considered as only the Obersteiner-Redlich zone of transition between central oligodendrocytes and peripheral Schwann cell–derived myelin, just distal to the facial nerve detachment from the lateral pons.¹⁹ We have previously em-

ABBREVIATIONS AICA = anterior inferior cerebellar artery; AS = attached segment; FIESTA = fast imaging employing steady-state acquisition; fREZ = facial root exit zone; HFS = hemifacial spasm; MVD = microvascular decompression; NVC = neurovascular compression; PICA = posterior inferior cerebellar artery; RDP = root detachment point; RExP = root exit point; STF = shredded Teflon felt; TZ = transition zone.

SUBMITTED December 1, 2014. **ACCEPTED** January 29, 2015.

INCLUDE WHEN CITING Published online August 21, 2015; DOI: 10.3171/2015.1.JNS142714.

phasized, however, that culprit NVC may occur anywhere along the fREZ, which is defined as the entire length of the exposed centrally myelinated facial nerve fibers.^{4,15} These facial nerve fibers emerge from the pontomedullary sulcus, then remain adherent to the pontine surface for approximately 8 mm before separating from the brainstem, and then 2 mm further distal they transition from central to peripheral myelin before continuing as the cisternal portion that courses through the subarachnoid space.^{4,15,22} These distinct segments of the fREZ may be identified as the root exit point (RExP), attached segment (AS), root detachment point (RDP), and transition zone (TZ), respectively. We have also observed that the failures of MVD surgery to alleviate HFS are commonly due to persistent culprit NVC somewhere upon this fREZ. In this study, we analyzed the findings at repeat MVD in patients with persistent or recurrent HFS, with the aim to identify factors that may improve surgical outcomes.

Methods

All MVD surgeries performed by the senior author (A.M.K.) had been recorded in a prospective database, including 91 at the University of Pittsburgh Medical Center (1996–1997), 139 at the University of Calgary Foothills Hospital (1998–2000), and 615 at the University of Manitoba Health Sciences Centre (2001–2014). Among these, 307 patients were referred for HFS, including the 12 patients who were referred after undergoing prior MVD surgery elsewhere and who are the subjects of this report. All these repeat MVD surgeries were performed between 1998 and 2012. Prior surgeries had been performed by 10 different surgeons in 5 Canadian provinces. Selection for repeat MVD surgery was based on an established HFS diagnosis and significant persistence or recurrence of spasms. Reoperation was offered if at least 1 year had passed since a prior unsuccessful MVD, or sooner if persistent NVC was clearly seen on MRI (Fig. 1). No patients seeking repeat MVD surgery who met either of these criteria were denied surgery. Preoperative MRI or other diagnostic imaging was not otherwise used to select or exclude candidates for repeat MVD.

The intraoperative findings were determined from review of the dictated operative reports and operative diagrams completed by the senior author immediately following each of the reoperations. Clinical details and outcomes were determined from review of the hospital and clinic records. The most recent follow-up was performed by telephone interview with a standardized questionnaire administered by a research assistant. Contacted patients were also asked to provide a rating of their satisfaction with the repeat MVD surgery results, where a score of 0 was considered not at all satisfied and 10 was considered completely satisfied. The medical ethics committee at the University of Manitoba approved this study.

Repeat MVD Technique

The details of our standard MVD surgical technique for HFS have been previously published.^{1,4,15} The lateral position was used, and our standard 5- to 8-cm linear retroauricular incision was opened utilizing either a por-

FIG. 1. Case 5. Magnetic resonance images. FIESTA sequence images prior to first MVD showing incidental vessel contact at the cisternal portion of the facial nerve (A) and the culprit PICA impinging deeply at fREZ (B). After the initial surgery, there remained persistent culprit NVC on the coronal T2-weighted image showing the severity of impingement at the facial RExP at the pontomedullary sulcus, as well as on the attached segment of the fREZ (C). The axial image shows persistent compression at the fREZ (asterisk), implant material positioned along the cisternal portion of the facial nerve (oval), and the craniectomy positioned too medially.

tion of the preexisting incision line or a new one. Prior cranioplasty material was removed, in whole or in part, and additional bone was removed as required to clearly visualize the edge of the sigmoid sinus and the posterior fossa floor. The total required craniectomy used measured less than 3×2 cm. The dura was typically adherent to the underlying cerebellar convexity and opened with the aid of magnification. If no subdural CSF was encountered, CSF diversion was achieved with intraoperative lumbar puncture using a 25-gauge needle, performed by the anesthesiologist. The microsurgical exposure proceeded over the anterior-inferior aspect of the cerebellum, utilizing retraction applied with 5-F suction and microinstruments over a 1-cm-wide cottonoid overtop a rubber “slider.” The cerebellum was elevated from the rootlets of lower cranial nerves IX–XI, and the arachnoid was opened sharply. The fREZ was exposed with gentle elevation of the cerebellar flocculus and choroid plexus, often utilizing a 3-mm tapered self-retaining retractor blade. The location of prior implant material was noted, and adhesions between the implant material and vessels, nerves, or brain were lysed as required. The culprit NVC on the fREZ was then identified, and offending vessels were mobilized away from the nerve and brainstem and then maintained in the new position with shredded Teflon felt (STF) implants (Bard Peripheral Vascular, Inc.). In all cases, intraoperative monitoring was used, including continuous brainstem auditory evoked responses and facial and endotracheal lead electromyography. More recently, we have also included routine monitoring of facial motor evoked potentials.^{2,18,23}

Results

The 12 patients who underwent repeat MVD surgery for persistent or recurrent HFS included 8 females and 4 males who were 37–75 years of age (mean 55 ± 11 years [$\pm$ SD]) at the time of repeat surgery. The total duration of HFS since onset had been 76–324 months (mean $124 \pm$

87 months), and the interval between the 2 surgeries was 6–312 months (mean 70 ± 89 months) (Table 1). Operative findings included prior craniectomies that did not extend to the sigmoid sinus and/or posterior fossa floor in 11 of 12 cases, such that additional bone removal was required. Intraoperative CSF diversion with lumbar puncture was used in 3 cases. Thorough exposure of the fREZ was achieved, and persistent NVC was alleviated in all cases.

Prior implant material from the first surgery was found in 11 of 12 cases and consisted of an Ivalon sponge ($n = 2$), unshredded Teflon felt ($n = 1$), or STF ($n = 8$). In 8 of 12 cases, the prior implant material was situated along the cisternal portion of the facial nerve in association with the anterior inferior cerebellar arteries (AICA) rather than at the fREZ (Fig. 2). In these cases, the arachnoid over the fREZ had not been previously opened such that the dissection there was straightforward and not impeded by any adhesions related to prior surgery. Significant NVC was uncovered at the fREZ, alleviated by mobilization of the culprit vessels, and then the vessel was maintained in the new position with placement of the STF implants (Figs. 3 and 4). In Case 6, a 4-mm incidental aneurysm arising from the posterior inferior cerebellar artery (PICA) origin was contributing to compression on the attached segment of the fREZ, as discovered at repeat MVD surgery. The aneurysm was clipped and the vessels were mobilized such that culprit compression was alleviated. In 3 other cases (Cases 1, 2, and 9) where prior implant material was found in the region of the fREZ, persistent NVC was also discovered more proximally on the fREZ along the attached segment and RExP and was also alleviated at repeat MVD surgery.

No patient suffered severe complications such as death, stroke, facial paralysis, infection, or CSF leak. One patient (Case 9) suffered a transient worsening of preexisting mild postoperative facial paresis, which improved again to House-Brackmann Grade II, and the worsening of preexisting hearing deficit. These complications were associated with severe adherence of the prior unshredded Teflon felt implant to the cisternal portion of the facial and vestibulocochlear nerves found at repeat MVD. Another patient developed aseptic meningitis 10 days after repeat MVD that resolved without sequelae (Case 12).

Postoperative spasm relief was assessed by telephone interview for the 10 patients who could be reached at 91 ± 55 months (range 28–193), or from the most recent clinic notes for Cases 9 and 10 at 7 and 3 months, respectively. Complete spasm resolution was described by 11 of 12 patients, including 2 patients who noted only an occasional quiver of the eyelid; another patient (Case 2) reported over 75% reduction of spasms. No patients underwent additional surgery after repeat MVD. One patient (Case 7) received a single botulinum toxin injection at 5 months after surgery but prior to complete spasm resolution. Among those patients reached for the telephone questionnaire, the patient satisfaction ratings for the repeat MVD surgeries were all 9 or 10 out of 10.

Discussion

MVD has been proven as an effective treatment for

TABLE 1. Operative findings and outcomes of repeat MVD for HFS

Case No.	Age (yrs), Sex	HFS Duration (yrs)	First MVD Performed Elsewhere		Interval Btwn MVD Procedures (mos)		Redo MVD		
			Complications	Outcome	MVD Procedures (mos)	Prior Implant	Persisting NVC	Complications	Outcome (mos)
1	30, F	2	None	2 yrs of spasm relief	68	STF @ RDP	AICA @ AS	None	Resolved (24)
2	33, F	7	None	No improvement	17	STF @ RDP, AS	AICA, LV @ AS	None	>75% reduction (106)
3	64, F	2	None	No improvement	15	IS @ CP	AICA, PICA, VA @ AS	None	Resolved (28)
4	42, M	7	None	No improvement	73	STF @ CPI/AC	AICA @ AS	None	Resolved (89)
5	37, M	6	Mild facial paresis	No improvement	10	STF @ CP	PICA, AICA @ AS, RExP	None	Resolved, OQ (58)
6	37, F	5	None	No improvement	44	STF @ CPI/AM	PICA @ AS	None	Resolved (88)
7	49, F	1	None	10 yrs of spasm relief	312	STF @ CP	AICA @ RExP	None	Resolved (35)
8	47, F	3	None	No improvement	6	IS @ CP	AICA @ AS	None	Resolved, OQ (137)
9	45, M	1	Mild facial paresis, severe hearing loss	No improvement	166	usTF @ CP, RDP	Vein, AICA @ AS	Mild facial paresis, severe hearing loss	Resolved (7)
10	46, F	4	None	3 mos of spasm relief	28	STF @ CPI/AM	AICA @ AS	None	Resolved (3)
11	48, F	2	Mild facial paresis	5 yrs of spasm relief	91	None	VA, AICA @ AS, RExP	None	Resolved (193)
12	33, M	10	Severe hearing loss, transient facial palsy	No improvement	12	STF @ CP	VA, AICA @ AS	Aseptic meningitis	Resolved (145)

CP = cisternal portion; IAM = internal auditory meatus; IS = Ivalon sponge; OQ = occasional quiver; usTF = unshredded Teflon felt; VA = vertebral artery.

FIG. 2. Diagram of surgical exposure. **Left:** The cisternal portion of the vestibulocochlear and facial nerves as seen from a lateral approach over the cerebellar hemisphere. The locations of prior implant materials from the first MVD surgery are depicted (X). **Right:** Elevation of the cerebellar flocculus with careful retraction provides exposure of the proximal facial nerve and its root exit zone. The locations of persistent NVC seen at repeat MVD are depicted (+). Copyright Jon Stepaniuk. Published with permission.

HFS, with spasm relief achieved in 86% to 97% of patients.^{3,4,6–8,10,11,15,20,24} Culprit NVC in HFS is almost always found at the fREZ. We and others have emphasized that this area comprises exposed centrally myelinated facial nerve fibers as they emerge at the pontomedullary sulcus (i.e., RExP), then ascend approximately 8 mm attached along the pontine surface (AS) before detaching from the brainstem (RDP) and finally transitioning to peripheral myelin a few millimeters more distal along the nerve (TZ) to the cisternal portion.^{4,15,22} The majority of culprit NVC is found on the AS-fREZ. In 10% of patients, however, culprit NVC is exclusively at the RExP, an area immediately above the supraolivary fossa that may be difficult to visualize medial to the overlying glossopharyngeal nerve rootlets.⁷ The best way to visualize the entire extent of the fREZ is by a subfollicular approach, which requires thorough dissection of the arachnoid between the cerebellum and lower cranial nerves IX–XI, then gentle upward re-

traction or elevation of the flocculus and often some of the associated choroid plexus that emerges from the foramen of Luschka.^{9,15} It is also important to account for all of the potential culprit vessels, which are often multiple and almost always arteries. It also bears emphasizing that the common association between the AICA and the cisternal portion of the facial nerve is not a usual cause of HFS. Although contact may be obvious and vessels may even course between tightly apposed cranial nerves VII and VIII, truly culprit NVC at the cisternal portion is seen in only 1% to 4% of cases, and in these cases only when the cisternal portion of the facial nerve is physically distorted in its course by this distal compression.^{6,9}

MVD is no guarantee of HFS cure. In our own experience performing over 300 MVD procedures for HFS, nearly 10% of patients have undergone repeat MVD for persistent or recurrent HFS. Among these, persistent or recurrent NVC was usually discovered. This often was related to an elongated vertebral artery that had again shifted toward the fREZ despite previous mobilization and placement of the STF implants. For this reason, we have more recently adopted some form of sling technique whenever feasible for such large arteries.¹ The AICA has also been problematic in some cases when tethered by the pontine perforating vessels, limiting a wide mobilization away from the fREZ. At repeat surgery, the vessel may sometimes be better mobilized, although not at the expense of any perforators. As in prior reports, success rates have generally been lower than reported for initial surgery,^{3,16,25} although we found that two-thirds of patients became spasm-free after our own repeat MVD. Similar to the results of our study, Barker and Jannetta did differentiate the results of surgery between patients treated initially by their own group versus other centers and found repeat MVD cure rates to be significantly higher in the latter.³ Based on these observations, we undertook the current study with the aim of identifying potential factors that may help improve the results of MVD for HFS.

FIG. 3. Case 3. Magnetic resonance image, FIESTA sequence prior to repeat MVD. The PICA arising from the ipsilateral vertebral artery impinges on the fREZ (up arrow). In the prepontine cistern, the AICA is seen in association with the faintly visualized Ivalon sponge implant material and the cisternal portion of the facial nerve (down arrow).

FIG. 4. Case 3. Intraoperative photographs and corresponding diagrams. **A and B:** Previously placed Ivalon sponge in association with the AICA and the cisternal portion of cranial nerves VII–VIII. **C and D:** Sharp dissection of the arachnoid overlying the fREZ. **E and F:** Dissection over the rootlets of cranial nerves IX and X, and the dissector elevating the PICA arising from the elongated vertebral artery. The fREZ remains indented from the neurovascular compression. **G and H:** Newly placed STF between the brainstem and culprit arteries with alleviation of NVC on the fREZ.

It is noteworthy that the utilization of MVD for HFS is low in North America, with less than 10% of patients undergoing this surgery. This impacts the surgical caseload, such that few operations are done by many surgeons. In the United States, the median annual MVD caseload was 3 cases per year, and the majority of these operations were for trigeminal neuralgia rather than HFS, as reported by Kalkanis et al.¹⁴ These authors also noted the average number of MVD procedures for HFS performed at non-federal hospitals between 1996–2000 was 237 cases per year. Similarly, in Canada 40% of the 50 annual MVD procedures for HFS were performed at 12 neurosurgical centers, each with caseloads of less than 5 per year (Canadian Institutes of Health Information data 2004–2014). While gaining a large volume of experience and expertise in MVD for HFS is difficult given the rarity of this disease, attention to established techniques will enable favorable outcomes and potentially a raise in the surgical utilization rates.

Conclusions

The keys to surgical success in performing MVD for HFS have been well illustrated and widely published. Exposure to the culprit NVC relies on a surgical corridor gained by bony removal to the limits of the sigmoid sinus laterally and posterior fossa floor inferiorly. A subfollicular approach to the fREZ generally provides adequate exposure and minimizes lateral retraction that may traumatize the cerebellum and vestibulocochlear nerve. It bears emphasis that the fREZ extends from its emergence at the pontomedullary sulcus and extends a couple of millimeters beyond the more superior detachment from the pons. All NVC along this 10 mm length should be alleviated, while vascular contact along the cisternal portion of the facial nerve root is generally incidental. MVD for HFS is effective when all NVC upon the fREZ is thoroughly alleviated. Optimization of surgical outcomes may lead to a greater utilization of this potentially curative treatment for HFS.

Acknowledgments

We thank the clinical staff at our Centre for Cranial Nerve Disorders, Mss. Olive Cotroneo, Andrea Kinnear Michelle Terrio, and Eleanor Topacio. Special thanks to Mr. Norman Delos Reyes for tireless administrative assistance and Mr. Jon Stepaniuk for artwork.

References

1. Attabib N, Kaufmann AM: Use of fenestrated aneurysm clips in microvascular decompression surgery. Technical note and case series. *J Neurosurg* **106**:929–931, 2007
2. Auger RG, Whisnant JP: Hemifacial spasm in Rochester and Olmsted County, Minnesota, 1960 to 1984. *Arch Neurol* **47**:1233–1234, 1990
3. Barker FG II, Jannetta PJ, Bissonette DJ, Shields PT, Larkins MV, Jho HD: Microvascular decompression for hemifacial spasm. *J Neurosurg* **82**:201–210, 1995
4. Campos-Benitez M, Kaufmann AM: Neurovascular compression findings in hemifacial spasm. *J Neurosurg* **109**:416–420, 2008
5. Chang WS, Kim HY, Chung SS, Chang JW: Microneurovascular decompression in patients with hemifacial spasm caused by vascular compression of facial nerve at cisternal portion. *Acta Neurochir (Wien)* **152**:2105–2111, 2010
6. Chung SS, Chang JH, Choi JY, Chang JW, Park YG: Microvascular decompression for hemifacial spasm: a long-term follow-up of 1,169 consecutive cases. *Stereotact Funct Neurosurg* **77**:190–193, 2001
7. Fukushima T: Microvascular decompression for hemifacial spasm: results in 2890 cases, in Carter LP, Spetzler RF, Hamilton MG (eds): *Neurovascular Surgery*. New York: McGraw Hill, 1995, pp 1133–1145
8. Goto Y, Matsushima T, Natori Y, Inamura T, Tobimatsu S: Delayed effects of the microvascular decompression on hemifacial spasm: a retrospective study of 131 consecutive operated cases. *Neurol Res* **24**:296–300, 2002
9. Hitotsumatsu T, Matsushima T, Inoue T: Microvascular decompression for treatment of trigeminal neuralgia, hemifacial spasm, and glossopharyngeal neuralgia: three surgical approach variations: technical note. *Neurosurgery* **53**:1436–1443, 2003
10. Hyun SJ, Kong DS, Park K: Microvascular decompression for treating hemifacial spasm: lessons learned from a pro-

- spective study of 1,174 operations. **Neurosurg Rev** **33**:325–334, 2010
11. Illingworth RD, Porter DG, Jakubowski J: Hemifacial spasm: a prospective long-term follow up of 83 cases treated by microvascular decompression at two neurosurgical centres in the United Kingdom. **J Neurol Neurosurg Psychiatry** **60**:72–77, 1996
 12. Jannetta PJ: Microsurgical exploration and decompression of the facial nerve in hemifacial spasm. **Curr Top Surg Res** **2**:217–220, 1970
 13. Jeon CJ, Kong DS, Lee JA, Park K: The efficacy and safety of microvascular decompression for hemifacial spasm in elderly patients. **J Korean Neurosurg Soc** **47**:442–445, 2010
 14. Kalkanis SN, Eskandar EN, Carter BS, Barker FG II: Microvascular decompression surgery in the United States, 1996 to 2000: mortality rates, morbidity rates, and the effects of hospital and surgeon volumes. **Neurosurgery** **52**:1251–1262, 2003
 15. Kaufmann AM, Wilkinson MF: Microvascular decompression surgery for hemifacial spasm, in Schmidek HH, Roberts DW (eds): **Schmidek & Sweet Operative Neurosurgical Techniques: Indications, Methods, and Results**, ed 5. Philadelphia: Elsevier, 2005, pp 1473–1490
 16. Kureshi SA, Wilkins RH: Posterior fossa reexploration for persistent or recurrent trigeminal neuralgia or hemifacial spasm: surgical findings and therapeutic implications. **Neurosurgery** **43**:1111–1117, 1998
 17. Mattick RP, Clark JC: Development and validation of measures of social phobia scrutiny fear and social interaction anxiety. **Behav Res Ther** **36**:455–470, 1998
 18. Miller LE, Miller VM: Safety and effectiveness of microvascular decompression for treatment of hemifacial spasm: a systematic review. **Br J Neurosurg** **26**:438–444, 2012
 19. Obersteiner H, Redlich E: Über Wesen und Pathogenese der tabischen Hinterstrangsdegeneration. **Arch Neurol Inst Univ Wien** **1-3**:158–172, 1894
 20. Samii M, Günther T, Iaconetta G, Muehling M, Vorkapic P, Samii A: Microvascular decompression to treat hemifacial spasm: long-term results for a consecutive series of 143 patients. **Neurosurgery** **50**:712–719, 2002
 21. Shin JC, Chung UH, Kim YC, Park CI: Prospective study of microvascular decompression in hemifacial spasm. **Neurosurgery** **40**:730–735, 1997
 22. Tomii M, Onoue H, Yasue M, Tokudome S, Abe T: Microscopic measurement of the facial nerve root exit zone from central glial myelin to peripheral Schwann cell myelin. **J Neurosurg** **99**:121–124, 2003
 23. Wilkinson MF, Kaufmann AM: Monitoring of facial muscle motor evoked potentials during microvascular decompression for hemifacial spasm: evidence of changes in motor neuron excitability. **J Neurosurg** **103**:64–69, 2005
 24. Zhong J, Li ST, Zhu J, Guan HX, Zhou QM, Jiao W, et al: A clinical analysis on microvascular decompression surgery in a series of 3000 cases. **Clin Neurol Neurosurg** **114**:846–851, 2012
 25. Zhong J, Zhu J, Li ST, Li XY, Wang XH, Yang M, et al: An analysis of failed microvascular decompression in patients with hemifacial spasm: focused on the early reoperative findings. **Acta Neurochir (Wien)** **152**:2119–2123, 2010

Disclosure

Parts of this material are based on data and information provided by the Canadian Institute for Health Information. The analyses, conclusions, opinions, and statements expressed herein are those of the authors and not necessarily those of the Canadian Institute for Health Information.

Author Contributions

Conception and design: both authors. Acquisition of data: both authors. Analysis and interpretation of data: both authors. Drafting the article: both authors. Critically revising the article: both authors. Reviewed submitted version of manuscript: both authors. Approved the final version of the manuscript on behalf of both authors: Kaufmann. Statistical analysis: both authors. Administrative/technical/material support: both authors. Study supervision: Kaufmann.

Correspondence

Anthony M. Kaufmann, Centre for Cranial Nerve Disorders, Section of Neurosurgery, Department of Surgery, University of Manitoba, GB 139 GH, 820 Sherbrook, Winnipeg, MB R3A1R9, Canada. email: akaufmann@hsc.mb.ca.