

Hemifacial Spasm Caused by Veins Confirmed by Intraoperative Monitoring of Abnormal Muscle Response

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■ **BACKGROUND:** Hemifacial spasm (HFS) is a benign disease caused by the hyper excitement of facial nerves owing to vessel compression. The offending vessels are usually arteries, such as anterior and posterior inferior cerebellar or vertebral arteries, but there are few reports of vein involvement cases.

■ **OBJECTIVE:** The aim of this study was to investigate veins as offending vessels in patients with HFS confirmed by abnormal muscle response (AMR).

■ **METHODS:** We analyzed 5 patients with HFS caused by veins among 78 patients with HFS over the past 10 years. All patients underwent microvascular decompression (MVD) with AMR monitoring, whereas 3 of them underwent a second MVD. The mean follow-up time was 97 months.

■ **RESULTS:** Arteries were thoroughly decompressed in 3 patients with a failed first MVD surgery who received a second surgery, during which veins at the root exit point (RExP) were decompressed with the disappearance or a significant decrease in the amplitude of AMR. Two patients showed spasm resolution after the first surgery when veins were decompressed together with the disappearance of AMR. The location of veins was RExP and the cisternal portion. All patients had excellent outcomes within 3 months, and no complications were observed.

■ **CONCLUSIONS:** Veins can be offending vessels in HFS patients. AMR is useful to determine the endpoint in these cases. Once arteries are decompressed thoroughly with

residual AMR, surrounding veins at unusual sites, such as the RExP or the cisternal portion, must be checked to prevent persistent HFS. Complete decompression of veins leads to a good clinical outcome.

INTRODUCTION

Hemifacial spasm (HFS) is a hyperactive symptom mainly caused by vessel compression at the root exit zone of facial nerves. The offending vessels are usually arteries; such as anterior (AICA) or posterior (PICA) inferior cerebellar artery, or vertebral artery, but veins are rarely reported, and estimated at fewer than 5%.¹ Microvascular decompression (MVD) is widely accepted as a first choice for HFS, which leads to spasm resolution in over 90% of patients.² An abnormal muscle response (AMR) is a typical abnormal muscle reaction in patients with HFS, which is particularly valuable when AMR disappears just after the decompression of facial nerves³ that is important to help surgeons confirm whether adequate decompression has been achieved. We performed this study applying AMR monitoring during MVD to confirm vein compression in HFS patients.

MATERIAL AND METHODS

Patient Data

We reviewed 78 patients who underwent MVD for typical HFS in the Department of Neurosurgery, Tokyo Women's Medical University Medical Center East from 2007–2018. Among them, 5

Key words

- Abnormal muscle response
- Hemifacial spasm
- Vein

Abbreviations and Acronyms

- AICA:** Anterior inferior cerebellar artery
- AMR:** Abnormal muscle response
- AS:** Attached segment
- CP:** Cisternal portion
- HFS:** Hemifacial spasm
- MVD:** Microvascular decompression
- PICA:** Posterior inferior cerebellar artery
- RDP:** Root detachment point
- RExP:** Root exit point

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Table 1. Summary of 5 Patients with Facial Spasm Caused by Veins

Patient Number	Sex	Age (years)	Side	Interval Between MVD	Offending Vessels at First MVD	AMR Findings	Outcome	Offending Vessels at Second MVD	AMR Findings	Outcome
1	F	57	Rt	15 Month	PICA	No change	Persist	Vein (REXP), Teflon prosthesis	Disappeared after decompression	Immediate relief
2	F	69	Rt	35 Month	AICA	No change	Persist	Vein (REXP), coagulate	Decreased to one third	Relief within 3 months
3	F	47	Lt	4 Month	PICA	Disappeared during procedure	Persist	Vein (REXP), coagulate	Disappeared after decompression	Relief within 3 months
4	F	60	Rt	N/A	PICA/vein (CP), Teflon prosthesis	Disappeared after decompression of vein	Immediate relief	N/A	N/A	N/A
5	M	48	Lt	N/A	AICA/vein (REXP), coagulate	Disappeared after decompression of vein	Relief within 3 months	N/A	N/A	N/A

MVD, microvascular decompression; AMR, abnormal muscle response; F, female; Rt, right; PICA, posterior inferior cerebellar artery; REXP, root exit point; AICA, anterior inferior cerebellar artery; Lt, Left; N/A, not available; CP, cisternal portion; M, male.

patients were confirmed to have veins as offending vessels according to intraoperative findings and AMR monitoring. Patients consisted of 4 women and 1 man, whose mean age was 55.6 years old (ranging from 43–72 years old). Three patients had a failed first MVD surgery and received a second surgery: the mean interval between the 2 operations was 16.3 months (4–36 months). The mean follow-up period was 97 months (74–140 months). The patients' clinical characteristics are presented in **Table 1**. The study was approved by the institutional review board of Tokyo Women's Medical University (IRB #4879).

Surgical Procedure

One dose of short-acting muscle blockage agent was administered before intubation; total intravenous anesthesia is maintained during the whole operation. The patients were operated on in the lateral decubitus position, using a 3-skullpin head holder to fix the head. Craniotomy was performed using a routine retro-sigmoid approach.³ After the edge between the transverse and sigmoid sinuses was explored, we opened the dura and arachnoid tissue, gradually exposed the lower cranial nerves, looked upward through the infra-flocculus, and exposed the root exit point (REXP), attached segment (AS), root detachment point (RDP) transition zone, or cisternal portion (CP)⁴ of the facial nerve. When the offending vessels were identified, we used a transposition method⁵ for decompression for arteries; for veins, we performed dissection and coagulation, or inserted a Teflon prosthesis to achieve decompression.

Intraoperative Monitoring

The auditory brain stem response was monitored in all cases during MVD to preserve hearing. The AMR was used to identify the recognition of offending vessels and to evaluate the effectiveness of decompression. We stimulated the zygomatic branches and recorded the AMR wave from the mentalis muscle.⁶

Follow-Ups

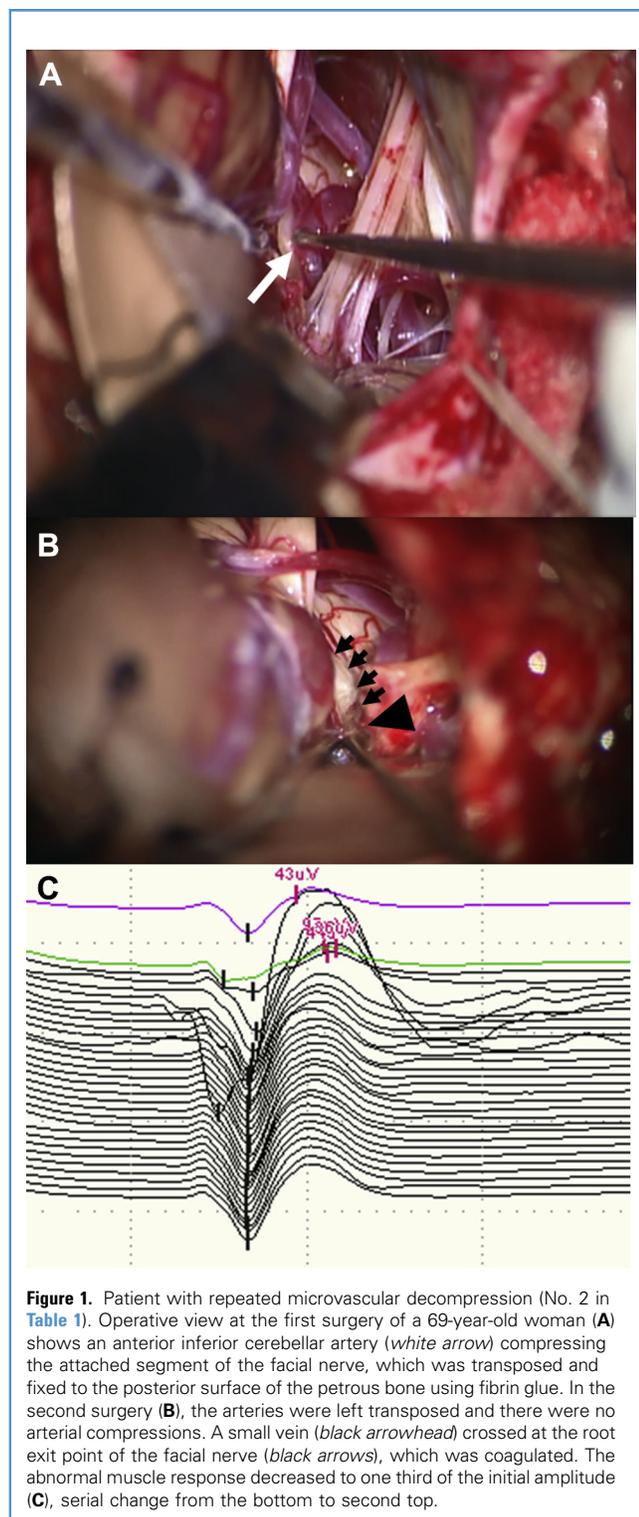
We checked the symptoms on the first postoperative day, at discharge, and in the outpatient clinic later with a face-to-face inquiry.

RESULTS

All 5 patients diagnosed with HFS completed preoperative exams in the outpatient clinic and received scheduled MVD by 1 doctor. None had received Botox treatment previously, and none had been clearly diagnosed with vein compression preoperatively (**Table 1**). Three patients had a failed first MVD and received a second surgery. Among their first procedures, PICA was revealed as the offending vessel in 2 patients, and AICA in 1 patient. AMRs persisted in 2 patients, although they disappeared during the procedure in 1 patient. In their second surgeries, the original decompressed arteries remained transposed from the facial nerve, but veins were found to have adhered or formed girdles around the nerve root. Finally, we coagulated veins or inserted a Teflon prosthesis to complete the decompression. All veins were located at REXP. AMRs disappeared in 2 of the patients, although the peak wave decreased to one third in 1 patient (**Figure 1**). In the fourth case, PICA was found to compress AS. The AMR did not change when PICA was transposed, which disappeared immediately when the vein under the facial nerve at CP was freed using a Teflon prosthesis (**Figure 2**). In the fifth case, we first alleviated AICA compression but AMR showed no change. We finally found a small branch of the transversospine vein crossing at REXP, which was carefully coagulated, and AMR fully resolved.

Outcomes

HFS of the 5 patients disappeared within 3 months. Up to the present, HFS has not recurred in these patients. None of the patients experienced any complications, such as hearing loss, facial palsy, or cerebrospinal fluid leakage. The intraoperative findings and outcomes are detailed in **Table 1**.



DISCUSSION

HFS is caused by neurovascular conflicts involving facial nerves, especially the root exit zone. MVD, which was standardized by Gardner⁷ and Jannetta et al.,⁸ is widely accepted as the first

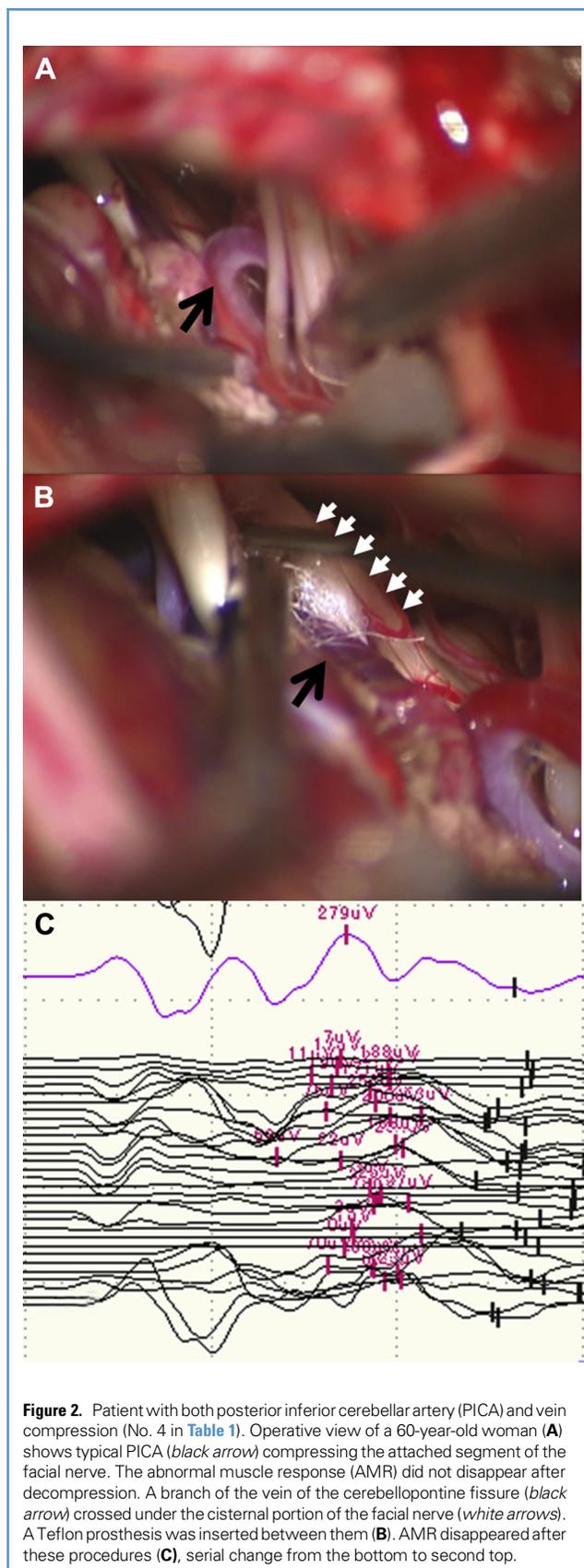


Table 2. Distribution of Conflicting Veins in Patients with Hemifacial Spasm

Author (year)	Total	Veins with Arteries (%)	Veins Alone (%)	Location of Compression	AMR Monitoring
Huang et al., 1992 ²²	310		2 (0.6)	Not mentioned	Not mentioned*
Barker et al., 1995 ^{23,§}	648	132 (20.4)	19 (2.4)	Not mentioned	Not mentioned
Samii et al., 2002 ^{24,§}	145	2 (1.4)	6 (4.1)	Not mentioned	Not mentioned
Park et al., 2006 ^{25,†}	13		1 (7.7)	1: RExP†	Not mentioned
Park et al., 2008 ²⁶	236		1 (0.4)	Not mentioned	Not mentioned
Campos-Benitez and Kaufmann 2008 ⁴	115		4 (3.4)	3: AS; 1:RDP	Not mentioned
Sindou and Keravel 2009 ²⁷	147	(13)	(0.7)	Not mentioned	Not detailed
Zhong et al., 2012 ^{9,§}	1327	(3)	0	Not mentioned	Not mentioned
Wang et al., 2013 ^{18,†}	33		19 (57.58)	Not mentioned	Detailed
Present study (2018)	78		5(6.4)	4: RExP; 1: CP	Detailed

AMR, abnormal muscle response; RExP, root exit point; AS, attached segment; RDP, root detached point; CP, cisternal portion.
 *Not mentioned: no data or analysis of AMR; not detailed: only brief data for AMR monitoring.
 †Judging from figure.
 ‡Focus on second surgery.
 §Including second surgery.

therapeutic choice for HFS, with a satisfactory outcome. A variety of methods are employed to improve the effectiveness of procedures, including intraoperative neurophysiological monitoring. An AMR—also called a lateral spread response—is widely used in MVD. Detection of the disappearance of AMR has been reported to be helpful in identifying offending vessels and confirming adequate decompression.^{3,6-9} A meta-analysis conducted by Sekula et al.¹⁰ indicated that the chance of cure if AMR was resolved during surgery was 4.2 times greater than if AMR persisted. This suggests the value of confirming AMR. However, there is still some debate on the value of AMR.¹¹⁻¹³ Some authors suggested that AMR monitoring does not provide significant benefit with respect to the outcome of MVD for HFS.² It is true that AMR disappears just after dural incision in some patients, and 1 patient in our series showed disappearance during the first surgery.

Kim et al.¹⁴ studied 299 HFS patients and compared outcomes between groups with the disappearance of AMR before and after MVD, and pointed out that patients with disappearance of AMR after decompression had better outcomes during either short or long-term follow-up. Mooij et al.¹⁵ detailed its role according to diverse categories in guiding or confirming, which resulted in a positive contribution of approximately 87% for intraoperative facial monitoring in MVD for HFS. Wang et al.¹⁶ also reported that intraoperative monitoring with AMR is an effective tool to evaluate adequate decompression involving veins. In patients with AMR persisting at the end of the procedure, facial nerve compression caused by a vein should be examined. We demonstrated that AMR is useful to determine the endpoint in HFS caused by veins.

MVD for HFS leads to satisfactory cure rate, which may reach 90%.² Some reasons for failure included misidentification of the actual offending vessels, displacement of an inserted prosthesis,

or recompression owing to vessel displacement.^{9,17} Some authors reviewed the second MVD, and offending arteries were found rostral to the VII nerve, between VII and VIII nerves, or in the cisternal segment of the VII root.⁹ The inability to identify these arteries or small arterioles led to the inefficiency of MVD, but vein compression was not mentioned as a cause of reoperation. Others found veins as offending vessels in significantly more patients who underwent a second MVD compared to the first MVD.¹⁸ Similar to our study of 3 patients with a second MVD, the arterial vessels were fully decompressed and the main cause of failure was the overlooking of vein compression.

Li et al.¹⁹ reported 13 early repeat MVD (several days after the first surgery) based on the following inclusion criteria: (1) The first MVD failed, the spasm was not resolved at all or became more severe, and (2) AMR persisted during the first MVD or disappeared once but re-emerged after the closure of the dural incision with the same or even at a higher amplitude. All 13 patients had good or excellent spasm resolutions immediately after the reoperation, which involved whole-range exploration and intraoperative AMR monitoring. One patient received a second MVD within a 4-month interval (No. 3 in Table 1) who completely failed the first MVD. However, once patients show partial relief, the outcome is different. We question the necessity of early repeat surgeries. According to our experience, many patients show delayed relief—symptoms disappearing after 3 months or longer is quite common. Jo et al.²⁰ found that among 70 patients with residual or recurrent spasms >1 year after surgery, 11 showed gradual improvement over 3 years, and 1 showed delayed improvement >3 years after surgery. Intraoperative resolution of AMR after decompression and severe indentation were significant predictors of a good long-term outcome after MVD for HFS. Chang et al.²¹ reported a probability of delayed

recurrence of 1.0%, 1.7%, and 2.9% at 1 year, 2 years, and 5 years after surgery. Our policy is observation, especially in patients whose symptoms show partial relief. We were concerned about missing responsible vessels, and discussed the possibility of a second MVD.

We list 9 reports including our series that focused on veins (Table 2). Among them 0%–6.4% of facial spasms were caused by veins.^{4,9,18,22–27} In the second surgery, the number of veins increased. Campos-Benitez and Kaufmann⁴ determined the nature of neurovascular compression in 115 consecutive patients undergoing their first MVD for HFS. The primary culprit location was at RExP in 10%, AS in 64%, RDP in 22%, and CP in 3%. They found 3 cases of venous compression in AS, and 1 case in RDP. In our series, 4 cases of venous compression were

found in RExP, and 1 in CP, which is not a common location. We would like to stress that we should carefully look for vessels not only in common locations but also at RExP and CP at the second surgery or when AMR persists.

CONCLUSIONS

Veins can be offending vessels in HFS patients, although this is not very common (6.4% in our database). AMR is useful to determine the endpoint in these cases. Once arteries are decompressed thoroughly with residual AMR, surrounding veins at unusual sites, such as RExP or CP, must be checked to prevent persistent HFS. Complete decompression of veins leads to a good clinical outcome.

REFERENCES

- Mercier P, Sindou M. The conflicting vessels in hemifacial spasm: literature review and anatomical-surgical implications. *Neurochirurgie*. 2018;64:94-100.
- Wei Y, Yang W, Zhao W, et al. Microvascular decompression for hemifacial spasm: can intraoperative lateral spread response monitoring improve surgical efficacy? *J Neurosurg*. 2018;128:885-890.
- Møller AR, Jannetta PJ. Microvascular decompression in hemifacial spasm: intraoperative electrophysiological observations. *Neurosurgery*. 1985;16:612-618.
- Campos-Benitez M, Kaufmann AM. Neurovascular compression findings in hemifacial spasm. *J Neurosurg*. 2008;109:416-420.
- Ryu H, Yamamoto S. A simple technique for neurovascular decompression of the cranial nerves. *Br J Neurosurg*. 2000;14:132-134.
- Møller AR, Jannetta PJ. Monitoring facial EMG responses during microvascular decompression operations for hemifacial spasm. *J Neurosurg*. 1987;66:681-685.
- Gardner WJ. Concerning the mechanism of trigeminal neuralgia and hemifacial spasm. *J Neurosurg*. 1962;19:947-958.
- 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.
- Zhong J, Li ST, Zhu J, et al. A clinical analysis on microvascular decompression surgery in a series of 3000 cases. *Clin Neurol Neurosurg*. 2012;114:846-851.
- Sekula RF Jr, Bhatia S, Frederickson AM, et al. Utility of intraoperative electromyography in microvascular decompression for hemifacial spasm: a meta-analysis. *Neurosurgical Focus*. 2009;27:E10.
- Von Eckardstein K, Harper C, Castner M, Link M. The significance of intraoperative electromyographic "lateral spread" in predicting outcome of microvascular decompression for hemifacial spasm. *J Neurol Surg B Skull Base*. 2014;75:198-203.
- Joo WI, Lee KJ, Park HK, Chough CK, Rha HK. Prognostic value of intra-operative lateral spread response monitoring during microvascular decompression in patients with hemifacial spasm. *J Clin Neurosci*. 2008;15:1335-1339.
- Hatem J, Sindou M, Vial C. Intraoperative monitoring of facial EMG responses during microvascular decompression for hemifacial spasm. Prognostic value for long-term outcome: a study in a 33-patient series. *Br J Neurosurg*. 2001;15:496-499.
- Kim CH, Kong DS, Lee JA, Park K. The potential value of the disappearance of the lateral spread response during microvascular decompression for predicting the clinical outcome of hemifacial spasms: a prospective study. *Neurosurgery*. 2010;67:1581-1588.
- Mooij JJA, Mustafa MK, van Weerden TW. Hemifacial spasm: intraoperative electromyographic monitoring as a guide for microvascular decompression. *Neurosurgery*. 2001;49:1365-1371.
- Wang X, Thirumala PD, Shah A, et al. The role of vein in microvascular decompression for hemifacial spasm: a clinical analysis of 15 cases. *Neurol Res*. 2013;35:389-394.
- Engh JA, Horowitz M, Burkhart L, Chang YF, Kassam A. Repeat microvascular decompression for hemifacial spasm. *J Neurol Neurosurg Psychiatry*. 2005;76:1574-1580.
- Wang X, Thirumala PD, Shah A, et al. Microvascular decompression for hemifacial spasm: focus on late reoperation. *Neurosurg Rev*. 2013;36:637-644.
- Li S, Hong W, Tang Y, et al. Re-operation for persistent hemifacial spasm after microvascular decompression with the aid of intraoperative monitoring of abnormal muscle response. *Acta Neurochir (Wien)*. 2010;152:2113-2118.
- Jo KW, Kong DS, Park K. Microvascular decompression for hemifacial spasm: long-term outcome and prognostic factors, with emphasis on delayed cure. *Neurosurg Rev*. 2013;36:297-302.
- Chang WS, Chung JC, Kim JP, Chung SS, Chang JW. Delayed recurrence of hemifacial spasm after successful microvascular decompression: follow-up results at least 5 years after surgery. *Acta Neurochir (Wien)*. 2012;154:1613-1619.
- Huang CI, Chen IH, Lee LS. Microvascular decompression for hemifacial spasm: analyses of operative findings and results in 310 patients. *Neurosurgery*. 1992;30:53-57.
- Barker FG, Jannetta PJ, Bisonette DJ, Shields PT, Larkins MV, Jho DJ. Microvascular decompression for hemifacial spasm. *J Neurosurg*. 1995;82:201-210.
- 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*. 2002;50:712-718.
- Park YS, Chang JH, Cho J, Park YG, Chung SS, Chang JW. Reoperation for persistent or recurrent hemifacial spasm after microvascular decompression. *Neurosurgery*. 2006;58:1162-1167.
- Park JS, Kong DS, Lee JA, Park K. Hemifacial spasm: neurovascular compressive patterns and surgical significance. *Acta Neurochir (Wien)*. 2008;150:235-241.
- Sindou M, Keravel Y. Neurosurgical treatment of primary hemifacial spasm with microvascular decompression. *Neurochirurgie*. 2009;55:236-247.

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