Prognostic ability of intraoperative electromyographic monitoring during microvascular decompression for hemifacial spasm to predict lateral spread response outcome

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OBJECTIVE Abnormal lateral spread response (LSR) is a typical finding in facial electromyography (EMG) in patients with hemifacial spasm (HFS). Although intraoperative monitoring of LSR has been widely used during microvascular decompression (MVD), the prognostic value of this monitoring is still debated. The purpose of this study was to determine whether such monitoring exhibits prognostic value for the alleviation of LSR after treatment of HFS.

METHODS Between January 2009 and December 2013, a total of 582 patients underwent MVD for HFS with intraoperative EMG monitoring at Kyung Hee University Hospital. The patients were categorized into 1 of 2 groups according to the presence of LSR at the conclusion of surgery (Group A, LSR free; Group B, LSR persisting). Patients were assessed for the presence of HFS 1 day, 6 months, and 1 year after surgery. Various parameters, including age, sex, symptom duration, offending vertebral artery, and offending perforating artery, were evaluated for their influence on surgical and electrophysiological results.

RESULTS Overall, HFS was alleviated in 455 (78.2%) patients 1 day after MVD, in 509 (87.5%) patients 6 months after MVD, and in 546 (93.8%) patients 1 year after MVD. Patients in Group B were significantly younger than those in Group A ($p = 0.022$). Patients with a symptom duration of less than 1 year were significantly more likely to be classified in Group A than were patients whose symptoms had persisted for longer than 10 years ($p = 0.023$); however, analysis of the entire range of symptom durations did not reveal a significant effect ($p = 0.132$). A comparison of Groups A and B according to follow-up period revealed that HFS recovery correlated with LSR alleviation over a shorter period, but the same was not true of longer periods; the proportions of spasm-free patients were 80.6% and 71.1% ($p = 0.021$), 89.4% and 81.9% ($p = 0.022$), and 93.5% and 94.6% ($p = 0.699$) 1 day, 6 months, and 1 year after surgery in Groups A and B, respectively.

CONCLUSIONS Although intraoperative EMG monitoring during MVD was beneficial for identifying the offending vessel and suggesting the most appropriate surgical end point, loss of LSR did not always correlate with long-term HFS treatment outcome. Because the HFS cure rate improved over time, revision might be considered for persistent LSR when follow-up has been performed for more than 1 year and the spasm remains despite adequate decompression.

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KEY WORDS outcome prediction; hemifacial spasm; lateral spread response; electromyography; microvascular decompression; functional neurosurgery

HEMIFACIAL spasm (HFS) is a movement disorder characterized by involuntary contraction of facial muscles that often results in severe psychosocial stress.¹¹ Although the mechanism of this disorder has not yet been clearly defined, its primary cause is considered to be vascular compression of the facial nerve root entry

zone (REZ).^{1,18,19,25,27} Hyperexcitability of the facial nucleus and ephaptic transmission of the facial nerve are generally accepted as its main pathological mechanisms.¹⁷ In a small number of cases, tumor and vascular diseases such as aneurysm and arteriovenous malformation have also been shown to cause HFS.^{2,3,6} Microvascular decompression

ABBREVIATIONS EMG = electromyography; HFS = hemifacial spasm; LSR = lateral spread response; MVD = microvascular decompression; REZ = root entry zone; VA = vertebral artery.

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sion (MVD) is the gold-standard treatment based on the etiology of HFS. This treatment yields high cure rates and has an excellent safety profile.^{4,16}

The lateral spread response (LSR) is an abnormal electrophysiological finding on electromyography (EMG) in patients with HFS. This response is elicited by the stimulation of 1 branch of the facial nerve; the responses of the muscles innervated by the other facial nerve branches are then recorded.^{9,10,15,21} Intraoperative monitoring of the LSR is now widely performed, because it can help identify the offending vessels and confirm facial nerve decompression from neurovascular contact.^{20,24} However, the prognostic value of intraoperative monitoring, especially over long-term follow-up periods, is still under debate. We performed a large-scale long-term study to investigate the relationships between EMG monitoring, LSR alleviation during MVD, and recovery from HFS.

Methods

A total of 885 patients with HFS underwent MVD accompanied by EMG monitoring in Kyung Hee University Hospital between January 2009 and December 2013. The LSR had been identified on preoperative EMG in each patient. The exclusion criteria were as follows: less than 1 year of follow-up, secondary HFS (such as tumorous or vascular lesion-related HFS^{3,23}), no LSR detected on preoperative EMG, and intraoperative EMG monitoring failure as a result of technical problems or the effects of muscle relaxants. In all, 582 (65.8%) patients were included in this retrospective study. The mean patient age was 54 years (range 16–79 years), and more females than males were present in the group (male/female ratio 1:2.59). The mean symptom duration was 4.8 years (range 0.5–40.0 years), and the spasms appeared on the left side of the face in 312 (53.6%) patients. A demographic comparison of the included and excluded patients was performed. We retrospectively reviewed patient clinical data to determine the prognostic value of intraoperative EMG monitoring during MVD for HFS.

Each patient underwent a preoperative MRI evaluation that included 3D time-of-flight angiography and CT examinations. A 3D angiogram was also generated in each case to evaluate the extent of vertebral artery (VA) anatomical variation.

EMG Monitoring

Each patient underwent continuous intraoperative monitoring with facial EMG and brainstem auditory evoked potentials. Electromyography monitoring was performed on a Cascade system; responses were recorded from needle electrodes that were inserted intradermally in the frontalis, orbicularis oculi, orbicularis oris, and mentalis muscles. Stimulating waves (300- μ sec pulse wave, 5–25 mA) were administered to the zygomatic and buccal branches of the ipsilateral facial nerve. The LSRs were recorded simultaneously at the orbicularis oris and mentalis muscles after stimulation of the zygomatic branch of the facial nerve and at the frontalis and orbicularis oculi muscles after stimulation of the buccal branch of the facial nerve. Muscle-relaxing agents were administered with the induction of anesthesia and followed immediately by the tempo-

rary alleviation of EMG signals in most cases. The muscle relaxants were maintained at a minimal dose to preserve signals from at least 3 of the following 4 nerves: both ulnar nerves, the median nerves, and the posterior tibial nerves. The patients were monitored with EMG throughout their surgery. Measurements were obtained at initiation of the surgery, the dural incision followed by CSF drainage, Teflon insertion, and surgery completion. Each patient was categorized into 1 of 2 groups according to the persistence of LSR at the conclusion of surgery. Patients for whom LSR was alleviated at the end of surgery were assigned to Group A, whereas patients for whom LSR remained at the end of surgery were assigned to Group B. Patients for whom LSR transiently disappeared but recurred at the conclusion of surgery were also assigned to Group B.

Surgical Procedure

Craniotomy was performed via the retrosigmoid suboccipital approach. The incisions were made adjacent to the sigmoid and transverse sinuses, and the section of the skull removed measured 2.5 $\times$ 1.5 cm. After dural incision, the CSF was drained by mild aspiration for gentle cerebellar retraction. The arachnoid trabeculae, including the lateral pontomedullary membrane, were dissected as meticulously as possible to enable further CSF drainage. This procedure resulted in a secure, large working space and helped prevent lower cranial nerve injury. After retraction of the cerebellar hemisphere and the choroid plexus, accompanied by further arachnoid dissection, the REZ of the seventh nerve was identified, and its relationship with the offending vessels was determined. One or more pieces of Teflon felt were inserted between the offending vessels and the compressed REZ or proximal facial nerve as necessary. If the LSR was not alleviated after these decompressive procedures, even after additional refinement efforts were made (such as rechecking the REZ and the offending vessels), no additional measures were taken, and the procedure was completed.

Follow-Up and Statistical Analysis

Patients were classified according to the presence of spasm and required drugs. “HFS free” was defined as a spasm-free state in which no antispasmodic agents (diazepam and gabapentin in this series) were required. “HFS remaining” was defined as the state in which facial spasm continued to occur with or without the need for antispasmodic drug administration. During the follow-up period, each patient visited the outpatient clinic at various intervals according to the presence of remaining symptoms and need for drug administration. When a patient became HFS free without recurrence, the time until the next follow-up visit was increased.

The presence of remaining HFS was estimated at 1 day, 6 months, and 1 year after surgery (short-term, mid-term, and long-term, respectively). To assess the prognostic value of intraoperative LSR monitoring, the numbers of HFS-free patients in Groups A and B were compared with the numbers of HFS-remaining patients (both HFS unchanged and HFS improved) in each group over each follow-up period. Moreover, several potential contributing factors in both groups, including age, sex, duration

of symptoms, and particular offending vessel, were also assessed. The data were analyzed using the independent-sample t-test, the chi-square test, logistic regression analysis, and repeated-measures ANOVA using SPSS 21.0 software (IBM Corp.). A p value of < 0.05 indicates statistical significance.

The institutional review board of Kyung Hee University Hospital approved this study.

Results

Demographic Characteristics

In the group of included patients, the mean age was 54 years (range 16–79 years), and there were more females than males (male/female ratio 1:2.59). The mean symptom duration was 4.8 years (range 0.5–40.0 years), and spasms appeared on the left side of the face in 312 (53.6%) patients.

When excluded and included patients were compared, neither age, sex, symptom duration (analysis over the entire range of symptom durations in all the patients), nor involved side were significantly different (Table 1). However, the proportion of patients with a symptom duration of > 5 years was significantly larger in the excluded group ($p = 0.001$).

Alleviation of LSR

The LSR was alleviated at the end of surgery in 433 (Group A, 74.4%) of the 582 patients (Table 1). Of the demographic factors compared between Groups A and B, age, sex, and lateralization showed no significant differences. The persistence of LSR was not significantly associated with symptom duration when the full range of durations was taken into account. However, more patients with a symptom duration of less than 1 year were present in Group A than in Group B ($p = 0.023$). These find-

ings were also significant in the multivariate analysis ($p = 0.014$) (Table 2).

The anterior inferior cerebellar artery was the most common offender and was involved in 62.71% of all cases. The next most common arteries were the posterior inferior cerebellar artery and the VA. Although the VA acted as an offending vessel in 84 (14.43%) cases, it acted as a single offender in only 2 (0.34%) cases. The presence of the VA as an offending artery (either alone or in combination) was not significantly associated with patient classification in either group.

Correlation Between MVD Results and LSR Alleviation

Of the 582 patients who underwent MVD, 455 (78.2%) were HFS free 1 day after surgery. These patients included 349 (80.6%) in Group A and 106 (71.1%) in Group B (Fig. 1). After 6 months of follow-up, 69 patients who had been classified as HFS remaining became HFS free. In addition, 46 patients became HFS free after 1 year of follow-up. In contrast, 15 and 9 HFS-free patients exhibited HFS recurrence after 6 months and 1 year of follow-up, respectively. Overall, 546 (93.8%) patients became HFS free by 1 year, including 405 (93.5%) in Group A and 141 (94.6%) in Group B. Of the 18 patients for whom LSR reappeared during surgery, the spasm-free rates were 50.0%, 77.8%, and 100% 1 day, 6 months, and 1 year after surgery, respectively. Intraoperative change in LSR and HFS alleviation were significantly correlated 1 day and 6 months after surgery ($p = 0.048$ and 0.020 , respectively) but not 1 year after surgery ($p = 0.608$) (Table 3).

Discussion

MVD is a causative treatment method with a dramatic cure rate. In this study, 93.8% of all patients with HFS were cured by 1 year after surgery. The LSR is a typical

TABLE 1. Demographic and LSR-related characteristics of patients with HFS who underwent MVD with intraoperative EMG monitoring

Demographic/Characteristic	Overall (n = 885)			p Value	Included (n = 582)		p Value
	Overall (n = 885)	Included (n = 582)	Excluded (n = 303)		Group A (n = 433)	Group B (n = 149)	
Age in yrs (mean $\pm$ SD)*	53.68 $\pm$ 10.70	53.82 $\pm$ 10.66	52.86 $\pm$ 10.31	0.619	55.20 $\pm$ 10.76	52.87 $\pm$ 10.32	0.207
Sex (no. male/no. female)†	283:602	162:420	121:182	0.807	121:312	41:108	0.920
Duration of symptoms							
Mean $\pm$ SD in yrs*	4.66 $\pm$ 4.98	4.82 $\pm$ 4.81	5.37 $\pm$ 5.64	0.17	4.63 $\pm$ 5.01	5.36 $\pm$ 5.64	0.132
<1 yr† (no.)	69	39	30	0.092	35	4	0.023‡
≥ 1 yr, <5 yrs† (no.)	505	331	174	0.875	248	83	0.739
≥ 5 yrs, <10 yrs† (no.)	238	178	60	0.001‡	129	49	0.480
>10 yrs† (no.)	73	34	39	0.000‡	21	13	0.082
Lateralization (no. rt/no. lt)†	409/476	270/312	139/164	0.884	199/234	71/78	0.721
Surgical findings (%)							
VA§ offender†	125	84	41	0.715	63	21	0.891
Perforating artery§ offender†	61	42	19	0.598	30	12	0.647
Multiple vessels†	258	178	80	0.194	128	50	0.361

Group A = patients for whom LSR was absent at the end of surgery; Group B = patients for whom LSR persisted at the end of surgery.

* Independent-sample t-test was used.

† Chi-square test was used.

‡ $p < 0.05$.

§ Either alone or in combination.

TABLE 2. Multivariate analysis results of demographic and LSR-related characteristics of patients in Groups A and B

Demographic/Characteristic	p Value	OR	95% CI
Age	0.094	0.985	0.967–1.003
Sex	0.714	1.083	0.708–1.655
Duration of symptoms	0.641	0.985	0.926–1.049
<1 yr	0.014*	0.168	0.040–0.695
≥1 yr, <5 yrs	0.151	0.513	0.206–1.275
≥5 yrs, <10 yrs	0.185	0.588	0.268–1.289
>10 yrs	0.170	1.732	0.790–3.797
Lateralization	0.936	0.984	0.667–1.453
Surgical findings			
VA† offender	0.629	0.839	0.412–1.709
Perforating artery† offender	0.890	0.945	0.422–2.115
Multiple vessels	0.275	1.368	0.779–2.402

* p < 0.05.

† Either alone or in combination.

finding in patients with HFS on EMG and thus has diagnostic value. Intraoperative EMG monitoring has become widely used, because the alleviation of LSR during surgery indicates sufficient decompression and, thus, a suit-

able surgical end point. Intraoperative monitoring is useful for identifying the offending vessel, especially when multiple vessels are involved. However, the prognostic value of such monitoring has been debated. The results of studies that investigated the correlation between LSR loss and HFS alleviation are summarized in Table 4.^{7,8,12,14,15,22,24,26,28} These studies have yielded discrepant results. In our study, LSR alleviation was correlated significantly with spasm alleviation 1 day and 6 months after surgery but not after 1 year of follow-up. Joo et al.¹² reported results similar to those of our study, and they suggested that the reason was that the LSR results were from not only pulsatile irritation but also REZ demyelination, which can require several months for recovery. In addition, Hatem et al.⁷ hypothesized that the late alleviation of HFS is a result of delayed recovery of motor nucleus hyperexcitability. The results of our study also support this hypothesis. In this study, 80.6% in Group A and 71.1% in Group B were HFS free on the first postoperative day. Although this difference was statistically significant, it should be noted that more than 70% of patients with “persistent LSR” became spasm free immediately after surgery, and only 6.6% of the patients had experienced recurrence 6 months after surgery.

We hypothesized that chronic vascular compression is related to longer symptom duration. We found that short symptom durations (less than 1 year) were significantly

FIG. 1. Chronological changes in HFS after MVD in Groups A and B.

TABLE 3. MVD results in Groups A and B according to follow-up period

Follow-Up Period	No. of Patients (%)			p Value*
	Overall (n = 582)	Group A (n = 433)	Group B (n = 149)	
1 day after surgery				0.048†
Spasm remaining	127 (21.8)	84 (19.4)	43 (28.9)	
No spasm	455 (78.2)	349 (80.6)	106 (71.1)	
6 mos after surgery				0.020†
Spasm remaining	73 (12.5)	46 (10.6)	27 (18.1)	
No spasm	509 (87.5)	387 (89.4)	122 (81.9)	
1 yr after surgery				0.608
Spasm remaining	36 (6.2)	28 (6.5)	8 (5.4)	
No spasm	546 (93.8)	405 (93.5)	141 (94.6)	

* Repeated-measures ANOVA.

† $p < 0.05$.

related to immediate loss of LSR after decompression, whereas the opposite was observed for long symptom durations (more than 10 years). However, symptom duration as a whole was not correlated significantly with a change in LSR during decompression. This finding suggests that early revision surgery can be considered for patients with remaining or persistent HFS with a < 1-year symptom duration. However, these findings cannot be generalized yet, and more evidence is needed to confirm this hypothesis. Moreover, many patients with a longer symptom duration were excluded from this patient population.

In 18 patients, the LSR was lost but then reappeared before Teflon insertion. This temporary disappearance of the LSR occurred in almost all cases immediately after the dural incision. Haines and Torres³ proposed that this condition results from a temporary shift in the neurovascular relationship and, thus, is not a reliable sign of decompression. Kim et al.¹³ reported that patients who experienced this premature LSR loss exhibited similar anatomical conditions, including thick arachnoid trabeculae and poor MVD results. We did not evaluate the effects of thick arachnoid trabeculae, because arachnoid thickness could not be classified objectively. A careful evaluation of whether the REZ was decompressed correctly was performed intraoperatively in cases of premature LSR loss. In our study, all the patients with premature LSR loss experienced delayed complete HFS recovery.

The published rates of LSR loss have varied widely, from 55.6% to 94.4%. In contrast, the recovery rates for patients with HFS have all been close to 90%. We suggest that this discrepancy results from the following factors. First, subjective surgeon-related factors play a role. These factors include the method of decompression (i.e., craniotomy size, amount of CSF drainage, extent of arachnoid dissection, and/or Teflon insertion) and the response to changes in the EMG monitoring results. Anesthesia-related factors, predominantly related to the administration of a muscle relaxant, may also be involved. Finally, mechanical factors might also play a role. These factors include the specific EMG system used for monitoring and the system settings. Future meta-analyses that investigate the roles of these factors are expected to be highly informative.

TABLE 4. Previous reports regarding the prognostic value of intraoperative EMG monitoring during MVD

Authors & Year	No.	LSR Alleviation (%)	Follow-Up Period		
			Short Term (≤1 wk)	Midterm (1–6 mos)	Long Term (≥9 mos)
Kiya et al., 2001	38	NA	X	—	—
Hatem et al., 2001	33	NA	—	O	X
Kong et al., 2007	263	87.4	X	O	O
Joo et al., 2008	72	55.6	O	X	—
Huang et al., 2009	36	94.4	O	—	—
Sekula et al., 2009	69	76.8	—	O	—
Neves et al., 2009	24	62.5	X	O	O
Thirumala et al., 2011	259	79.9	O	—	X
Ying et al., 2011	241	93.9	O	O	—
Present study	582	74.4	O	O	X

NA = not available; O = significant correlation between LSR loss and HFS alleviation; X = no correlation between LSR loss and HFS disappearance; — = not determined.

This study did have some limitations. First, this was a retrospective study and thus likely exhibits some selection bias. Among all the patients who underwent MVD at Kyung Hee University Hospital during the same period, only 65.8% were included in the study. Moreover, the excluded patients exhibited significantly longer symptom duration. The major reason for exclusion was a follow-up period shorter than 1 year. This exclusion might have affected our results, because patients who became completely HFS free after surgery were likely to refuse longer follow-up. Second, the end of each operation was decided subjectively on the basis of EMG changes. In addition, objective information on symptom duration was obtained on the basis of subjective patient statements. Third, we did not include every possible contributing factor in our analysis and omitted facial palsy, history of botulinum toxin injection, and other medical conditions. Last, follow-up ended after 1 year in this study; studies with longer follow-up are needed to evaluate the prognostic value of EMG during MVD. We are planning studies to address these limitations. We also intend to investigate the contribution of various surgical findings and aspects of LSR that influence the outcomes of patients after MVD.

Conclusions

To the best of our knowledge, this is the largest single-center study to have investigated the prognostic ability of intraoperative EMG monitoring during MVD for HFS. Patients who had less than 1 year of symptoms showed a significantly higher rate of LSR loss after decompression compared with patients who had more than 10 years of symptoms. EMG monitoring was helpful for identifying the offending vessel(s), especially when multiple offending vessels were involved, and was also useful for confirming facial nerve decompression after Teflon sheet insertion from neurovascular contact. Intraoperative monitoring of

the LSR during MVD for HFS had some prognostic value over a short-term follow-up period; however, this was not true over long-term follow-up periods. When the LSR was not alleviated even after correct decompression, our data indicate that extending follow-up for longer than 1 year is a useful solution.

References

- Campos-Benitez M, Kaufmann AM: Neurovascular compression findings in hemifacial spasm. **J Neurosurg** **109**:416–420, 2008
- Choi SK, Rhee BA, Lim YJ: Hemifacial spasm caused by epidermoid tumor at cerebello pontine angle. **J Korean Neurosurg Soc** **45**:196–198, 2009
- Choi SK, Rhee BA, Park BJ, Lim YJ: Hemifacial spasm caused by fusiform aneurysm at vertebral artery-posterior inferior cerebellar artery junction. **J Korean Neurosurg Soc** **44**:399–400, 2008
- Costa J, Espírito-Santo C, Borges A, Ferreira JJ, Coelho M, Moore P, et al: Botulinum toxin type A therapy for hemifacial spasm. **Cochrane Database Syst Rev** (1):CD004899, 2005
- Haines SJ, Torres F: Intraoperative monitoring of the facial nerve during decompressive surgery for hemifacial spasm. **J Neurosurg** **74**:254–257, 1991
- Han IB, Chang JH, Chang JW, Huh R, Chung SS: Unusual causes and presentations of hemifacial spasm. **Neurosurgery** **65**:130–137, 2009
- 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** **15**:496–499, 2001
- Huang BR, Chang CN, Hsu JC: Intraoperative electrophysiological monitoring in microvascular decompression for hemifacial spasm. **J Clin Neurosci** **16**:209–213, 2009
- Ishikawa M, Ohira T, Namiki J, Ajimi Y, Takase M, Taya S: Abnormal muscle response (lateral spread) and F-wave in patients with hemifacial spasm. **J Neurol Sci** **137**:109–116, 1996
- Ishikawa M, Ohira T, Namiki J, Kobayashi M, Takase M, Kawase T, et al: Electrophysiological investigation of hemifacial spasm after microvascular decompression: F waves of the facial muscles, blink reflexes, and abnormal muscle responses. **J Neurosurg** **86**:654–661, 1997
- Jannetta PJ: The cause of hemifacial spasm: definitive microsurgical treatment at the brainstem in 31 patients. **Trans Sect Otolaryngol Am Acad Ophthalmol Otolaryngol** **80**:319–322, 1975
- 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** **15**:1335–1339, 2008
- 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** **67**:1581–1588, 2010
- Kiya N, Bannur U, Yamauchi A, Yoshida K, Kato Y, Kanno T: Monitoring of facial evoked EMG for hemifacial spasm: a critical analysis of its prognostic value. **Acta Neurochir (Wien)** **143**:365–368, 2001
- Kong DS, Park K, Shin BG, Lee JA, Eum DO: Prognostic value of the lateral spread response for intraoperative electromyography monitoring of the facial musculature during microvascular decompression for hemifacial spasm. **J Neurosurg** **106**:384–387, 2007
- McLaughlin MR, Jannetta PJ, Clyde BL, Subach BR, Comey CH, Resnick DK: Microvascular decompression of cranial nerves: lessons learned after 4400 operations. **J Neurosurg** **90**:1–8, 1999
- Møller AR: The cranial nerve vascular compression syndrome: II. A review of pathophysiology. **Acta Neurochir (Wien)** **113**:24–30, 1991
- Møller AR: Hemifacial spasm: ephaptic transmission or hyperexcitability of the facial motor nucleus? **Exp Neurol** **98**:110–119, 1987
- Møller AR: Vascular compression of cranial nerves: II: pathophysiology. **Neurol Res** **21**:439–443, 1999
- Møller AR, Jannetta PJ: Hemifacial spasm: results of electrophysiologic recording during microvascular decompression operations. **Neurology** **35**:969–974, 1985
- Møller AR, Jannetta PJ: Physiological abnormalities in hemifacial spasm studied during microvascular decompression operations. **Exp Neurol** **93**:584–600, 1986
- Neves DO, Lefaucheur JP, de Andrade DC, Hattou M, Ahdab R, Ayache SS, et al: A reappraisal of the value of lateral spread response monitoring in the treatment of hemifacial spasm by microvascular decompression. **J Neurol Neurosurg Psychiatry** **80**:1375–1380, 2009
- Rhee BA, Kim TS, Kim GK, Leem WL: Hemifacial spasm caused by contralateral cerebellopontine angle meningioma: case report. **Neurosurgery** **36**:393–395, 1995
- Sekula RF Jr, Bhatia S, Frederickson AM, Jannetta PJ, Quigley MR, Small GA, et al: Utility of intraoperative electromyography in microvascular decompression for hemifacial spasm: a meta-analysis. **Neurosurg Focus** **27**(4):E10, 2009
- Sindou MP, Polo G, Fischer C, Vial C: Neurovascular conflict and hemifacial spasm. **Suppl Clin Neurophysiol** **58**:274–281, 2006
- Thirumala PD, Shah AC, Nikonow TN, Habeych ME, Balzer JR, Crammond DJ, et al: Microvascular decompression for hemifacial spasm: evaluating outcome prognosticators including the value of intraoperative lateral spread response monitoring and clinical characteristics in 293 patients. **J Clin Neurophysiol** **28**:56–66, 2011
- Valls-Solé J: Facial palsy, postparalytic facial syndrome, and hemifacial spasm. **Mov Disord** **17** (Suppl 2):S49–S52, 2002
- Ying TT, Li ST, Zhong J, Li XY, Wang XH, Zhu J: The value of abnormal muscle response monitoring during microvascular decompression surgery for hemifacial spasm. **Int J Surg** **9**:347–351, 2011

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: BJ Park, Lee. Acquisition of data: BJ Park, Lee, CK Park, Rhee. Analysis and interpretation of data: Lee, Shin. Drafting the article: Lee. Critically revising the article: BJ Park. Reviewed submitted version of manuscript: BJ Park, Lim. Statistical analysis: Shin. Administrative/technical/material support: CK Park, Rhee. Study supervision: Rhee, Lim.

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