# ORIGINAL ARTICLE

# Microvascular decompression for hemifacial spasm: focus on late reoperation

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Abstract The objective of this study is to investigate late repeat microvascular decompression (MVD) with persistent or recurrent hemifacial spasm (HFS) and to compare the clinical characteristics, intraoperative findings, complications, and outcomes with first MVD. We analyzed MVDs performed at the University of Pittsburgh Medical Center between January 1, 2000 and December 31, 2007. Thirty-three patients who underwent late redo MVDs were classified as group I and 243 patients who underwent their first MVD as group II. Clinical data were collected to analyze the difference between the two groups. The mean follow-up period was 54.48 months (range, 9-102 months). There is no significant difference in preoperative clinical characteristics (gender, age, side of MVD, botox usage, facial weakness) between the two groups. In present study, we found a vein as the offending vessel in significantly more number of patients who underwent repeat MVD as compared to first MVD (P=0.02). The

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lateral spread response disappeared in 66 % of patients during repeat MVDs, which is not different from those undergoing their first MVD. No difference in the relief rate was found during the immediate postoperative, discharge, or follow-up stages between repeat and first MVD. Moreover, no difference was found in the incidence of complications between repeat MVD and first MVD. Late repeat MVD for HFS is an effective and safe procedure. No specific preoperative clinical characteristics were identified in patients with repeat MVD. Intraoperative monitoring with lateral spread response (LSR) is an effective tool to evaluate adequate decompression. In patients with persistent LSR at the end of the procedure, facial nerve compression from a vein should be examined. We believe that it is important to undergo a repeat MVD for failed HFS relief irrespective of the timing of the operation.

**Keywords** Hemifacial spasm · Microvascular decompression · Reoperation · Lateral spread response · Botox

# Introduction

Hemifacial spasm (HFS) is a condition involving involuntary, repetitive, unilateral contraction of the muscles innervated by the facial nerve (cranial nerve CN VII) [1]. Typical HFS is caused by facial nerve irritation secondary to vascular compression at the root exit zone (REZ) [2, 3], leading to involuntary, intermittent spasms beginning at the orbicularis oculi muscle and progressing to the mentalis muscle. Retromastoid craniotomy and facial nerve microvascular decompression (MVD) have been proven to be effective cure for patients [4, 5]. However, wide variations in the cure rate for HFS have been reported ranging from 86 to 92 % [2, 6]. There is no agreement concerning what is the suitable time for the treatment of persistent or recurrent patients with HFS after the original MVD [7, 8]. So far, little has been published regarding repeat MVD for HFS in those patients who failed their initial operation [9-11], and less has been reported about patients who had late repeat MVD [9]. In present study, the main objective was to investigate characteristics, outcomes, and complications of the late repeat MVD (beyond 1 month after the original MVD) for persistent or recurrent HFS after initial failure of MVD.

# Methods

# Study design

A retrospective study was conducted with institutional review board approval from the University of Pittsburgh (IRB #: PR008120394). Of 276 in 293 patients with typical HFS, 33 patients with HFS underwent repeat MVD due to persistent or recurrent spasm (group I), and 243 patients underwent their first MVD (group II). In group I, 28 patients underwent reoperation for clinical failure or recurrence after original MVD, and five patients had two prior MVDs. The interval to repeat MVD ranged from 1.6 months to 16 years (Fig. 1). Clinical outcome data were obtained after the immediate operation, at discharge (mean, 4.00±2.00 days), and at a follow-up phone call during June 2008. Follow-up data were collected from 195 patients who had a minimum follow-up period of 9 months (mean, 54.48±27.84 months).

#### Microvascular decompression

Between January 2000 and December 2007, we reviewed patients who had HFS and underwent MVD procedures at the University of Pittsburgh Medical Center (UPMC). The operation was performed using a routine retrosigmoid approach [12, 13]. Short-acting neuromuscular junctionblocking medications were used for intubation. No additional paralytic agent was administered during electromyography

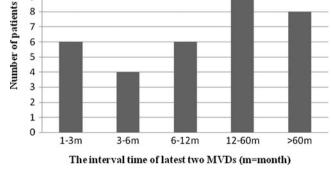


Fig. 1 Number of patients and interval time of latest two MVDs

10

9

8 7

6

(EMG) monitoring. The dissection was started from the caudal cranial nerves, using withdrawal of CSF and gentle exposure of pertinent anatomy with handheld suction. Following careful exposure of the CN VII-CN VIII complex, any suspect arteries or veins compressing CN VII anywhere from the brainstem to beyond the REZ were treated. For redo MVDs, the previous conflict site was reexamined and checked to confirm whether a satisfactory decompression was completed. The operation was complete when the nerve no longer demonstrated any visible evidence of vascular compression. After confirming that there were no further offending vessels, the surgeon terminated the procedure and closed the craniotomy in a routine fashion.

Intraoperative neurophysiological monitoring and alarm criteria

During surgery, free run EMG (f-EMG) monitoring of the facial, glossopharyngeal, and vagus nerves was performed, in addition to lateral spread response (LSR). Lateral spread response is a delayed abnormal muscle response recorded in the mentalis muscle following the stimulation of zygomatic branch of the facial nerve [5]. All instances of f-EMG activity, regardless of type (spikes, bursts, neurotonic discharges), were made audible to and also immediately reported to the surgeon and recorded in the patient's record. Auditory nerve function was monitored using brainstem auditory evoked potentials performed with our institution's alarm criteria [14]. Physician oversight and interpretation were performed using a combined on-site and remote model utilized by UPMC [15].

# Data analysis

Statistical analyses were performed using SAS version 9.1.3 (SAS Institute, Cary, NC). Continuous variables were presented as mean  $\pm$  standard deviation and categorical variables as frequency (in percent). Group differences in demographic, clinical characteristics, and outcomes were assessed using  $\chi^2$  tests, and the Fisher exact test correction was used when needed. P <0.05 was considered as statistically significant.

# **Results**

# Demographics

Two hundred seventy-six patients with HFS had a mean age of 52.17±12.13 years (range, 17-82 years), with a femaleto-male ratio being 1.9:1 and left-to-right ratio being 1.2:1. Medical and surgical histories were obtained from each patient undergoing MVD. Two hundred two patients (73.19 %) received prior botox treatment. Of the 276

Table 1 Comparison of pre erative clinical characteristic between group I and group

Table 1         Comparison of preop- erative clinical characteristics between group I and group II	Variable	Group I, <i>n</i> (%)	Group II, $n$ (%)	P value	
	Number of patients	33 (100.0)	243 (100.0)	_	
	Times of undergoing MVDs				
	One	0	1 (100.0)	_	
	Two	28 (84.85)		_	
	Three	5 (15.15)		_	
	Gender				
	Female	23 (69.70)	156 (64.20)	0.53	
	Male	10 (30.30)	87 (35.80)		
	Side				
	Left	20 (60.61)	131 (53.91)	0.47	
	Right	13 (39.39)	112 (46.09)		
	Botox usage	28 (84.85)	173 (71.19)	0.10	
	H-B classification of facial weakness				
	Grade II	12 (36.36)	72 (29.63)	0.43	
	Grade III	7 (21.12)	46 (18.93)	0.75	
	Grade IV	0 (0.00)	11 (4.53)	0.37	
	Preoperative symptom				
Group I, 33 patients underwent repeat MVDs; group II, the remaining 243 patients with first MVD	Decreased corneal reflex	5 (15.15)	53 (21.81)	0.38	
	Tinnitus	1 (3.03)	15 (6.17)	0.70	
	Decreased hearing	4 (12.12)	27 (11.11)	0.77	
	Tonus	18 (54.55)	155 (63.79)	0.30	
<i>H-B</i> House–Brackmann score, 1985	Platysmal involvement	16 (48.48)	109 (44.86)	0.81	

patients, 33 underwent repeat MVDs, among which, 29 patients were referred to UPMC after prior operations elsewhere, and four patients had their first operation at UPMC. These patients underwent reoperation at UPMC beyond 1 month of their original decompression. No patients exhibited bilateral HFS.

# Preoperative characteristics

There is no significant statistical difference in preoperative clinical characteristics (gender, age, side of MVD, botox usage, facial weakness, etc.) between the two groups (Table 1).

# Intraoperative findings

# Compressing vasculature seen near facial nerve REZ

The vessels compressing the REZ, as identified by the surgeon, are summarized in Table 2. A majority (70.7 %) of patients had multiple compressing vessels. The compression was commonly caused by the anterior inferior cerebellar artery (AICA), posterior inferior cerebellar artery, vertebral artery, and some veins in both groups.

Significantly higher number of patients had a vein as offending vessel intraoperatively in group I as compared to group II (P=0.02; Table 2).

Intraoperative lateral spread monitoring

Data regarding intraoperative monitoring of the LSR during MVD were available for 255 (92.39 %) of the 276 patients. LSR disappeared in 66.67 % (20/30) of the patients in group I as compared to 65.33 % (147/225) in group II (P=0.89; Table 2).

Table 2 Comparison of intraoperative findings including LSR, offending vessels, and outcomes between group I and group II

Variable	Group I, <i>n</i> (%)	Group II, $n$ (%)	P value
LSR disappeared	20/30 (66.67)	147/225 (65.33)	0.89
Offending vessels			
AICA	13 (39.39)	129 (53.09)	0.14
PICA	15 (45.45)	111 (45.68)	0.98
VA	11 (33.33)	67 (27.57)	0.49
Vein	19 (57.58)	90 (37.04)	0.02*
Perforator	7 (21.21)	43 (17.70)	0.62
HFS resolution			
Postoperative	28 (84.85)	217 (89.30)	0.45
Discharge	30 (90.91)	223 (91.77)	0.74
Follow-Up	17/20 (85.00)	162/175 (92.57)	0.22

AICA anterior inferior cerebellar artery, PICA posterior inferior cerebellar artery, VA vertebral artery, LSR lateral spread response \*P<0.05

# Operative outcomes

In 33 patients, 84.85 % experienced immediate postoperative relief of spasm, 90.91 % had relief at discharge, and 85.00 % had relief at follow-up period. No significant difference in spasm relief between two groups was observed at the immediate postoperative, discharge, or follow-up stages.

# Postoperative complications

Postoperative complications were observed in both patient groups (Table 3). Complications included hearing loss, balance problem, CSF leakage, diplopia, headache, and dizziness/vertigo. There was no significant difference in complications between the two groups. Complication rates in repeat MVD were as follows: hearing loss 15.15 %, facial weakness 12.12 % (two cases of grade II, one grade III, and one grade IV, respectively), balance disorder 3.03 %, cerebrospinal fluid leak 3.03 %, diplopia 3.03 %, headache 6.06 %, and wound infection 6.06 %.

# Discussion

HFS is caused by vascular compression of the REZ of the facial nerve [16, 17]. Microvascular decompression is a highly accepted and effective method for treatment of patients with HFS [1, 2, 18]. Much has been published regarding the high efficacy of MVD for HFS, with cure rates ranging anywhere from approximately 85 to 92 %, and excellent long-term results 5 years after MVD in 85 % of patients [7, 19]. Some authors have suggested the possibility of symptom relief within 1 year; the treatment of persistent or recurrent patients with HFS should be more than 1 year after the original MVD [6, 16, 20]. Sindou et al. [7, 8] recommend a waiting period of about 1 year after initial surgery before reoperation. Hyun [21] believed that any

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judgment and decision regarding re-treatments should be done after 12 months after the original surgery, no matter how much the residual spasm existed after the first MVD. In contrast, Kassam [9] and Zhong et al. [22] reported that patients undergoing early reoperation were significantly more likely to be cured or improved than patients undergoing late reoperation. In the present study, we analyzed patients who underwent late repeat MVD. As a matter of fact, the relief of HFS did happen after the late redo MVD in 28 of the 33 patients postoperatively. Therefore, it was noted that late repeat MVD for HFS is an effective and safe procedure.

In previous reports, MVD had immediate spasm relief rates varying from 76.5 to 88.3 % [22-24]. The rest exhibited delayed gradual resolution of spasm after MVD during follow-up observations. After follow-up of 1-3 years after surgery, the successful spasm relief rate increased to 79-94.6 % [10, 22, 24], which is consistent with the pathophysiological mechanisms of primary HFS. Some think the delayed spasm resolution may be attributed to the time required for remyelination of the damaged area, as well as the return of normal excitability of the facial motonucleus [4, 25, 26]. However, because it cannot explain the immediate relief after a successful surgery, it is still unclear about the delayed relief. According to Moller [27], primary HFS may be due to hyperactivity of the facial nucleus, progressively induced by the chronic compression pulsation of the neurovascular conflict, especially at REZ. Thus, it would be physiologically logical that the effect of surgical decompression takes time to decrease and normalize the clinical spasm. However, as a matter of fact, most of the patients rather than a minority of the patient relieved immediately after a successful MVD. Zheng et al. [28] developed a hypothesis about the mechanism of HFS, which is the cross-transmission is bridged by sympathetic nerve fibers between the facial nerve fibers and offending vessels. Hence, this might explain most of patients who were relieved immediately after facial nerve and offending vessels

Group I, <i>n</i> (%)	Group II, <i>n</i> (%)	P value
4 (12.12)	28 (11.52)	1.00
4 (12.12)	11 (4.53)	0.09
2 (6.06)	6 (2.47)	0.24
1 (3.03)	2 (0.82)	0.32
1 (3.03)	3 (1.23)	0.40
1 (3.03)	6 (2.47)	0.59
1 (3.03)	5 (2.06)	0.54
1 (3.03)	12 (4.94)	1.00
2 (6.06)	18 (7.41)	1.00
2 (6.06)	4 (1.65)	0.15
0 (0.00)	8 (3.29)	1.00
	$\begin{array}{c} 4 \ (12.12) \\ 4 \ (12.12) \\ 2 \ (6.06) \\ 1 \ (3.03) \\ 1 \ (3.03) \\ 1 \ (3.03) \\ 1 \ (3.03) \\ 1 \ (3.03) \\ 1 \ (3.03) \\ 2 \ (6.06) \\ 2 \ (6.06) \end{array}$	4 (12.12) $28 (11.52)$ $4 (12.12)$ $11 (4.53)$ $2 (6.06)$ $6 (2.47)$ $1 (3.03)$ $2 (0.82)$ $1 (3.03)$ $3 (1.23)$ $1 (3.03)$ $6 (2.47)$ $1 (3.03)$ $5 (2.06)$ $1 (3.03)$ $12 (4.94)$ $2 (6.06)$ $18 (7.41)$ $2 (6.06)$ $4 (1.65)$

Table 3Comparison of post-<br/>operative complications betweenGroup I and Group II

wall were separated by Teflon. Some of the reasons for failure of first or repeat MVD may include not identifying the real offending vessels, Teflon pledget movement after closure, or incomplete decompression [9, 10]. Therefore, the primary reason to perform a repeat MVD would be to reexplore the entire root and explore missed compressive vessel [22]. The second reason would be to reexamine the relationship between REZ of CN VII and pledget to make ensure complete decompression.

Based on the spasm relief during the immediate postoperative period, the results indicate that intraoperative monitoring is an effective tool in helping identify offending vessels during repeat MVD [5, 29]. Our LSR disappearance rate in both groups was comparable; in addition, it was similar to previously published data [4, 30]. Sekula et al. [31] reported that the chance of resolved HFS if the LSR was abolished during surgery was 4.2 times greater than if the LSR persisted. Given the significant negative predictive value of LSR monitoring, the surgeon can be reassured that an adequate decompression has been achieved, thus avoiding unnecessary operative time and resultant complications especially when multiple vessels are involved [6, 32-34]. Absence of LSR to stimulation during MVD was similar in both groups. In our previous study, we found that patients with residual LSR had higher chance of persistent spasm [5]. We found a significantly higher number of patients in group I to have a vein as the offending vessel at the REZ in comparison to group II. These results suggest that patients with residual LSR should undergo exploration for a vein to prevent persistent HFS. Based on our previous published results [5] and the current study, the use of LSR in redo MVDs may be very valuable.

The common operative complications of MVD for HFS include new or worsening facial weakness, hearing loss, diplopia, headache, balance problem, CSF leakage, and dizziness/vertigo [29, 35, 36]. In our study, there was no significant difference in postoperative hearing decrease and/or loss, diplopia, headache, balance problem and CSF leak, and dizziness and vertigo between two groups and those previously reported. Our hearing outcomes after MVD were similar to our previously reported study [14]. It is possible that experience with reoperations and monitoring the facial and auditory nerve might have contributed to the insignificant difference in the outcomes between the two groups. However, facial weakness was noticed in 12.12 % of the patients after surgery in group I. The following points are important for higher percentage of facial weakness: (1) Repeat MVDs require more time for a careful exposure due to the presence of adhesions which increase the chance for facial weakness. (2) The number of patients who underwent redo MVD is limited, and it could represent the low sample size. (3) The higher incidence of facial palsy in group I could be related to higher botox usage. Although wound infection difference in our study did not reach statistical significance, we found the rate of 6.06 % is higher than that have been reported previously for HFS patients [37]. Given this, what the surgeon needs to do is the dura and muscle should be closed carefully. In our study, an insignificant number of patients had recovery after discharge in both groups I and II. In addition, majority of the patients had very good results. Engh et al. [9] showed that early reoperation was also associated with good results. Based on these data, we think it is important to undergo repeat MVD for failed HFS relief irrespective of the timing of the operation. One limitation of the study is that most of patients in whom a MVD was redone had their first surgery at other medical centers. So, no descriptions of the surgical findings at the first operation were available to better understand the putative cause of the failure from the study.

## Conclusion

Repeat MVD for HFS is an effective and safe procedure. No specific preoperative clinical characteristics were identified in patients with repeat MVD. Intraoperative monitoring with LSR is an effective tool to evaluate adequate decompression. In patients with persistent LSR at the end of the procedure, facial nerve compression from a vein should be examined. We believe that it is important to undergo a repeat MVD for failed HFS relief irrespective of the timing of the operation.

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# Comments

Marc Sindou, Lyon, France

The authors should be acknowledged for their encouraging advice to redo MVD in patients with persistent HFS after a first MVD was considered as failed, irrespective of the timing of the operation.

Our personal policy is to propose reoperation in "resisting cases," but only under very restrictive conditions:

-A delay of at least 1 year: As a matter of fact, in approximately one third of our patients, HFS necessitated several months up to 1 year to be completely relieved, including disappearance of EMG signs. Delayed relief was particularly observed in the patients in whom MVD was little atraumatic for the nerve and with the interposed prosthesis (Teflon) not touching the facial nerve, i.e., not being neo-compressive.

-On MRI, a possible still-compressive loop at brainstem or a deliberately left loop at the porus of internal auditory meatus: In the later, eventuality, hearing function could be at risk due to manipulation of the eighth nerve and/or the labyrinthine artery.

-Acceptance from the patient of occurrence of facial weakness or hearing disturbances, as reoperations entail higher risk of such side effects

As regard to usefulness of intraoperative monitoring of the lateral spread responses, although we think it interesting, we observed some deficiency in reliability [1].

Whatever these reserves, we agree that a patient without satisfactory effect after a first MVD may benefit from reoperation, especially when botulinum toxin injections have consumed their therapeutic effects.

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Dr. Wang et al. did a retrospective investigation concerning redo MVD operations on those with persistent or recurrent HFS in the University of Pittsburgh and concluded that late repeat MVD is an effective and safe procedure. Their works once again confirmed Jannetta and his team's contribution that MVD is a reasonable as well as an effective treatment for HFS.

In the paper, they recognized that veins could be the offending vessel. We also noticed that arterioles could be the offending vessel [12], and the REZ is not the only area where the neurovascular confliction occurs [7]. Accordingly, I believe that, for a HFS patient, his or her ipsilateral facial nerve root should be compressed (though a compression of VII nerve is not always developing to a HFS). The culprit could be any vessel(s) anywhere along the nerve root [8]. The reason for a so-called negative finding of the offending vessel was nothing but (1) the culprit had been transposed while retracting the cerebellum, dissecting the arachnoids, or even suctioning the CSF, and (2) the culprit was not discovered, especially when it is located very caudomedially. Theoretically, for a properly diagnosed HFS, with an appropriate manipulation by a sophisticated neurosurgeon, MVD should lead to a total relief of the symptom immediately after the operation. Nevertheless, a failure of MVD may arise in case of difficult approach to the neurovascular conflict site due to individual anatomical feature [6, 13].

However, there are some reports regarding delayed relief in the literature [1, 2]. I think this may happen when the facial nerve root was not sufficiently decompressed. Our primary study on the mechanism of HFS implied that the emersion of ectopic action potentials in the VII nerve fibers might be triggered by neurotransmitters released from sympathetic endings in the offending artery wall, and the attrition of neurovascular interface was the essence of the etiology [11]. This new hypothesis gave a good explanation for the fact that the episode of spasm is often associated with moods (sympathetic excitement) [5]. It could also explain the partial or delayed relief. It may happen when multiple vessels are involved. Once the larger one is moved away, the symptom may marginally improve as the main problem has been solved. For the smaller vessel, a little movement may allow the lesions at the interfaces to repair over time. With restoring of both the epineurium and adventitia, the nerve may finally be isolated from the vessel [10]

Therefore, we agree on a redo MVD, even an early reoperation if the patient does hope for an immediate cure instead of anxiously expecting of a possible relief. To ensure the curative effect, we had suggested separating all the vessels from the VII nerve including the AICA between the VII and VIII nerves near the internal acoustic meatus [3], but it may raise the complication for a young neurosurgeon. To balance cure with safety [9], I recommend terminating the operation once an apparent offending artery was found in the axil of the VII nerve (especially when a dent was also visualized in the nerve) and the LSR or AMR vanished as the culprit was moved away [4]. Postoperatively, if the symptom does not improve at all, an immediate reoperation with exploration of the entire intracranial segment of the facial nerve is recommended; if the symptom improves a bit, then an alternative is to observe.

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