

Original Article

Reduction in intraoperative lateral spread response as a prognostic neurophysiological parameter in microvascular decompression for hemifacial spasm: A retrospective cohort study

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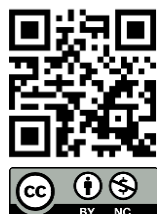
Abstract

Hemifacial spasm (HFS) is caused by vascular compression of the facial nerve at its root exit zone, and microvascular decompression (MVD) remains the gold standard treatment, although intraoperative confirmation of adequate decompression is challenging. The lateral spread response (LSR), an abnormal facial muscle response elicited by nerve stimulation, has been used to guide intraoperative decision-making, but its predictive accuracy remains debated. The aim of this study was to identify an optimal LSR diminution threshold as a dependable prognostic indicator for surgical success. A retrospective observational study was conducted on 34 patients who underwent MVD for primary HFS between January 2023 and December 2025. Intraoperative delta LSR, defined as the change in LSR activation threshold from baseline to post-decompression, was analyzed using receiver operating characteristic (ROC) analysis. Firth penalized-likelihood logistic regression was performed as an exploratory sensitivity analysis. Complete spasm resolution at six months was achieved in 29 patients (85.3%). A delta LSR cut-off of ≥ 15.0 mA discriminated complete resolution from partial improvement with moderate to good discrimination (area under the curve: 0.793; 95% confidence interval (95%CI): 0.614–0.936), corresponding to a 100.0% success rate above this threshold versus 70.6% below it (Fisher's exact $p=0.044$). This association persisted in direction and magnitude after adjustment for age, sex, and symptom onset duration using Firth's method (adjusted odds ratio: 1.18 per 1 mA increase; 95%CI: 0.96–1.45). These findings support intraoperative delta LSR, particularly a threshold of ≥ 15.0 mA, as an adjunct for guiding MVD in HFS. Nevertheless, validation in larger, prospectively followed, multicenter cohorts is warranted before delta LSR is adopted for intraoperative decision-making.

Keywords: Hemifacial spasm, intraoperative neurophysiological monitoring, lateral spread response, microvascular decompression, delta LSR

Introduction

Hemifacial spasm (HFS) is a neurovascular contact disorder characterized by involuntary, paroxysmal, and repetitive contractions of the ipsilateral facial musculature, typically secondary to vascular compression of the seventh cranial nerve at its root exit zone. Microvascular decompression (MVD) remains the gold standard treatment for definitive spasm resolution.



However, intraoperative confirmation of adequate decompression remains challenging. Beyond its motor manifestations, HFS is associated with a significant non-motor burden, as involuntary unilateral facial muscle contractions may contribute to headache, comorbid insomnia, depression, and reduced health-related quality of life [1]. Intraoperative neurophysiological monitoring is therefore commonly used to assess the adequacy and safety of MVD [2]. Among the available monitoring modalities, the lateral spread response (LSR) is an abnormal electromyographic response elicited by stimulation of facial nerve branches. Its intraoperative resolution has been associated with successful nerve decompression and symptom relief [3].

The study by Møller and Jannetta demonstrated a high correlation between persistent intraoperative LSR and unfavorable spasm outcomes. Since then, LSR has been widely investigated as a critical, independent prognostic predictor for evaluating the efficacy of MVD [4,5]. However, its clinical utility and predictive accuracy remain inconsistent across studies, particularly regarding the relationship between intraoperative LSR resolution and postoperative relief of HFS. Recent studies have also reported inconsistent findings regarding the optimal degree of LSR diminution associated with successful spasm resolution [6,7]. Therefore, the aim of this study was to evaluate the intraoperative LSR changes during MVD for HFS and identify an optimal LSR diminution threshold as a prognostic indicator of spasm resolution.

Methods

Study design and setting

This retrospective observational cohort study was conducted at the Department of Neurosurgery, Dr. Cipto Mangunkusumo National General Hospital, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia. The study evaluated patients who underwent MVD for primary HFS between January 2023 and December 2025. Medical records, surgical-registry data, operative records, intraoperative neurophysiological monitoring records, and postoperative follow-up data were reviewed. Record review was performed from March to June 2026, ensuring a minimum postoperative follow-up of six months for all included patients.

The study was reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) recommendations for cohort studies. Ethical approval was obtained from the Health Research Ethics Committee of the Faculty of Medicine, Universitas Indonesia–Dr. Cipto Mangunkusumo Hospital (KEPK FKUI–RSCM; KET-7/2/UN2.F1/ETIK/PPM.00.02/2026). As this retrospective study used existing medical records, the requirement for individual informed consent was waived by the ethics committee. All patient data were anonymized before analysis.

Patient selection

Patients who underwent MVD for HFS during the study period were identified from the departmental surgical registry and screened for eligibility. The inclusion criteria were age ≥ 18 years; a confirmed diagnosis of primary HFS; MVD performed during the study period with intraoperative neurophysiological monitoring that included LSR recording; and availability of postoperative clinical outcome data for at least 6 months. Primary HFS was defined clinically as involuntary, intermittent contractions of facial muscles without an identifiable secondary neurological or structural cause. The diagnostic work-up included preoperative magnetic resonance imaging and electromyography.

Patients were excluded if they had secondary HFS caused by an intracranial tumor, demyelinating disease, or another identifiable structural or neurological disorder; incomplete medical or intraoperative monitoring records; or unavailable six-month clinical outcome data. Previous botulinum toxin treatment was permitted provided that the final injection was administered at least six months before surgery.

Microvascular decompression procedure

MVD was performed for primary HFS under intraoperative neurophysiological monitoring. The facial nerve root exit zone was explored, the offending vessel was identified, and neurovascular decompression was performed. The offending vessel was documented in the operative records

and categorized as the anterior inferior cerebellar artery, vertebral artery, or multiple-vessel compression. The offending vessel was mobilized away from the root exit zone and maintained in the decompressed position with interposed shredded Teflon felt. Adequacy of decompression was confirmed by direct microscopic inspection before watertight dural closure. All procedures were performed by the same operating surgeon, who was aware of LSR findings in real time. Persistent LSR after decompression prompted further exploration for residual or additional neurovascular conflict.

Intraoperative neurophysiological monitoring

Intraoperative monitoring included LSR assessment and brainstem auditory evoked potential (BAEP) monitoring. Monitoring was performed under total intravenous anesthesia, with neuromuscular blockade limited to intubation. LSR was elicited by percutaneous stimulation of the zygomatic branch of the facial nerve using subdermal needle electrodes and recorded from the ipsilateral mentalis muscle.

Square-wave pulses of 0.2 ms duration were delivered at 1 Hz, starting at 2 mA and increased until a reproducible response was obtained. LSR was assessed at baseline, after dural opening, immediately after decompression, and before closure. Final LSR thresholds were transcribed from the intraoperative monitoring records. The institutional protocol specified a maximum stimulation intensity of 20 mA.

Delta lateral spread response measurement

Delta LSR was defined as the increase in the LSR threshold from the baseline pre-decompression measurement to the final post-decompression measurement, calculated as the final post-decompression threshold minus the baseline pre-decompression threshold and expressed in milliamperes (mA). A positive delta LSR indicated that a higher stimulation intensity was required to elicit the abnormal response after decompression. An exact numeric delta LSR was available for 33 of 34 patients. One patient had a final LSR threshold recorded as >20 mA, resulting in unquantifiable delta LSR; this patient was excluded only from analyses requiring a numeric delta LSR. Because the final threshold was capped at 20 mA in the institutional monitoring protocol and several observations clustered at this value, delta LSR was treated as an operational database-derived measure.

Clinical outcome assessment

Postoperative outcomes were assessed at approximately 1 week, 1 month, 3 months, and 6 months after MVD. The primary outcome time point for the prognostic analysis was six months. Clinical outcome was classified as complete resolution, partial improvement, or no improvement. Complete resolution was defined as the absence of clinically evident HFS at six months. Partial improvement was defined as persistent spasm with clinical improvement after surgery, whereas no improvement was defined as persistent symptoms without meaningful clinical improvement. For the primary analysis, clinical success was defined as complete HFS resolution at six months; partial improvement and no improvement were classified as clinical non-success. Overall clinical improvement, comprising complete resolution and partial improvement, was reported descriptively.

Recurrence was defined as the reappearance of HFS during postoperative follow-up after an initial period of clinical improvement or complete resolution. Time-to-event follow-up was measured from the date of surgery to the first documented recurrence. Patients without recurrence were censored at the date of their last documented follow-up.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for distribution and summarized as mean $\pm$ standard deviation (SD) for normally distributed data or median with interquartile range (IQR) for non-normally distributed data. Categorical variables were summarized as frequencies and percentages. Available-case denominators were reported for variables with missing data, and no imputation was performed. All statistical tests were two-sided, with $p < 0.05$ considered statistically significant. Given the exploratory nature of the study, p -values were interpreted

alongside effect estimates and their 95% confidence intervals (CIs) rather than as confirmatory evidence. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Continuous variables were compared using parametric or nonparametric tests according to data distribution.

Delta LSR was initially analyzed as a continuous predictor of complete HFS resolution at six months. Receiver operating characteristic (ROC) analysis was performed to assess its discriminative ability, with the area under the curve (AUC) and corresponding 95%CI estimated from 20,000 stratified bootstrap resamples. An exploratory cutoff was selected using the Youden index. The resulting 15.0-mA threshold was subsequently used only for secondary descriptive analyses. Sensitivity, specificity, positive predictive value, negative predictive value, and overall accuracy were calculated with exact binomial 95%CIs. Because the threshold was derived and evaluated in the same cohort, these threshold-specific estimates were considered post hoc and potentially optimistic. Fisher's exact test was used to compare the proportion of patients achieving complete resolution between the two threshold groups.

Given the sparse number of clinical non-success outcomes, conventional maximum-likelihood logistic regression was not performed. Instead, an exploratory Firth penalized-likelihood logistic regression model was fitted as a sensitivity analysis to estimate the association between continuous delta LSR and complete HFS resolution after adjustment for age, sex, and symptom duration. The model was interpreted as exploratory and was not used to establish independent prognostic value. No post hoc power analysis was performed. Statistical uncertainty was characterized using effect estimates and corresponding 95%CIs.

Results

Patient characteristics

A total of 34 patients who underwent MVD for primary HFS during the study period were included, with no exclusions (**Figure 1**). All 34 patients had available six-month clinical outcome data. The mean age was 49.82 ± 13.83 years, and 21 patients (61.8%) were female. The median symptom duration was 35.5 months (IQR, 12.0–85.8), and left-sided involvement was most common (19/34, 55.9%), followed by right-sided (13/34, 38.2%) and bilateral involvement (2/34, 5.9%). Complete spasm resolution at six months was achieved in 29 patients (85.3%), while five patients (14.7%) had partial improvement; no patient had persistent HFS without clinical improvement.

Baseline LSR was available for all 34 patients, with a mean threshold of 5.87 ± 3.12 mA. Final LSR and exact numeric delta LSR were available for 33 patients, with mean values of 19.61 ± 3.67 mA and 13.59 ± 4.24 mA, respectively. One patient was excluded from analyses requiring a numeric delta LSR because the final LSR threshold was recorded as >20 mA and therefore could not be quantified. The anterior inferior cerebellar artery was the most common offending vessel (27/34, 79.4%), followed by the vertebral artery (6/34, 17.6%) and multiple-vessel compression (1/34, 2.9%). At six months, the median House-Brackmann grade was 1 (IQR, 1–1). Hearing status was normal or unchanged in 28 patients (82.4%), improved in two (5.9%), and remained the same as the preoperative disturbance in four (11.8%) (**Table 1**).

Table 1. Baseline characteristics and clinical outcomes of patients undergoing microvascular decompression for hemifacial spasm (n=34)

Variable	Value
Demographics	
Age (years), mean $\pm$ SD	49.82 $\pm$ 13.83
Sex, n (%)	
Male	13 (38.2)
Female	21 (61.8)
Clinical characteristics	
Symptom onset duration (months), median (IQR)	35.5 (12.0–85.8)
Affected side, n (%)	
Right	13 (38.2)
Left	19 (55.9)
Bilateral	2 (5.9)

Variable	Value
Neurophysiological parameters	
Baseline LSR (mA), mean±SD	5.87±3.12
Final LSR (mA), mean±SD	19.61±3.67
Delta LSR (mA), mean±SD	13.59±4.24
BAEP prolongation (%), median (IQR)	9 (0.0–10.8)
Vascular findings	
Offending vessel type, n (%)	
AICA	27 (79.4)
VA	6 (17.6)
Multiple vessels (AICA and VA)	1 (2.9)
Postoperative complications	
Facial nerve paresis (HB grade), median (IQR)	1 (1–1)
Hearing disturbance, n (%)	
None	28 (82.4)
Improved	2 (5.9)
Same as preoperative	4 (11.8)
Clinical outcomes	
Spasm resolution, n (%)	
Complete resolution	29 (85.3)
Partial improvement	5 (14.7)
No improvement	0 (0.0)

AICA: anterior inferior cerebellar artery; BAEP: brainstem auditory evoked potential; HB: House–Brackmann; IQR: interquartile range; LSR: lateral spread response; SD: standard deviation; VA: vertebral artery.

Data are presented as mean±SD for normally distributed continuous variables, median (IQR) for non-normally distributed continuous variables, and frequency (%) for categorical variables.

Prognostic performance of delta LSR

Among the 33 patients with an exact numeric delta LSR, continuous delta LSR yielded an AUC of 0.793 (bootstrap 95%CI: 0.614–0.936) for discriminating complete HFS resolution from partial improvement at six months. This analytic subset comprised 28 patients with complete resolution and five with partial improvement (**Figure 2**). Performance measures across candidate delta LSR thresholds are presented in **Table 2**. The Youden index identified an exploratory threshold of 15.0 mA. At this threshold, sensitivity was 57.1% (16/28; 95%CI: 37.2–75.5) and specificity was 100.0% (5/5; 95%CI: 47.8–100.0). Because the threshold was derived and evaluated in the same cohort, these performance estimates are likely to be optimistic.

At the 15.0-mA threshold, the positive predictive value was 100.0% (16/16; 95%CI: 79.4–100.0), the negative predictive value was 29.4% (5/17; 95%CI: 10.3–56.0), and overall accuracy was 63.6% (21/33; 95%CI: 45.1–79.6%). The 100% specificity and positive predictive value reflected the absence of false-positive observations in this small cohort and should not be interpreted as evidence of perfect performance in other populations. The positive likelihood ratio was not estimable as a finite value because no false-positive observations occurred, while the negative likelihood ratio was 0.43. Both likelihood-ratio estimates should be interpreted cautiously because only five patients had clinical non-success.

Table 2. Diagnostic performance of delta LSR at selected thresholds

Cut-off value (mA)	Sensitivity (%)	Specificity (%)	Youden index
13.0	67.9	80.0	0.479
14.0	60.7	80.0	0.407
15.0	57.1	100.0	0.571
16.0	42.9	100.0	0.429

The 15.0-mA threshold maximized the Youden index in the same cohort used to estimate its performance. It represents an exploratory candidate threshold and has not been externally validated.

Association between delta LSR and clinical outcome

Complete resolution occurred in all 16 patients with delta LSR ≥15.0 mA and in 12 of 17 patients with delta LSR <15.0 mA; Fisher's exact test yielded $p=0.044$ (**Table 3**). Because the threshold was selected from the same outcome data, this p -value should be interpreted as descriptive rather than confirmatory.

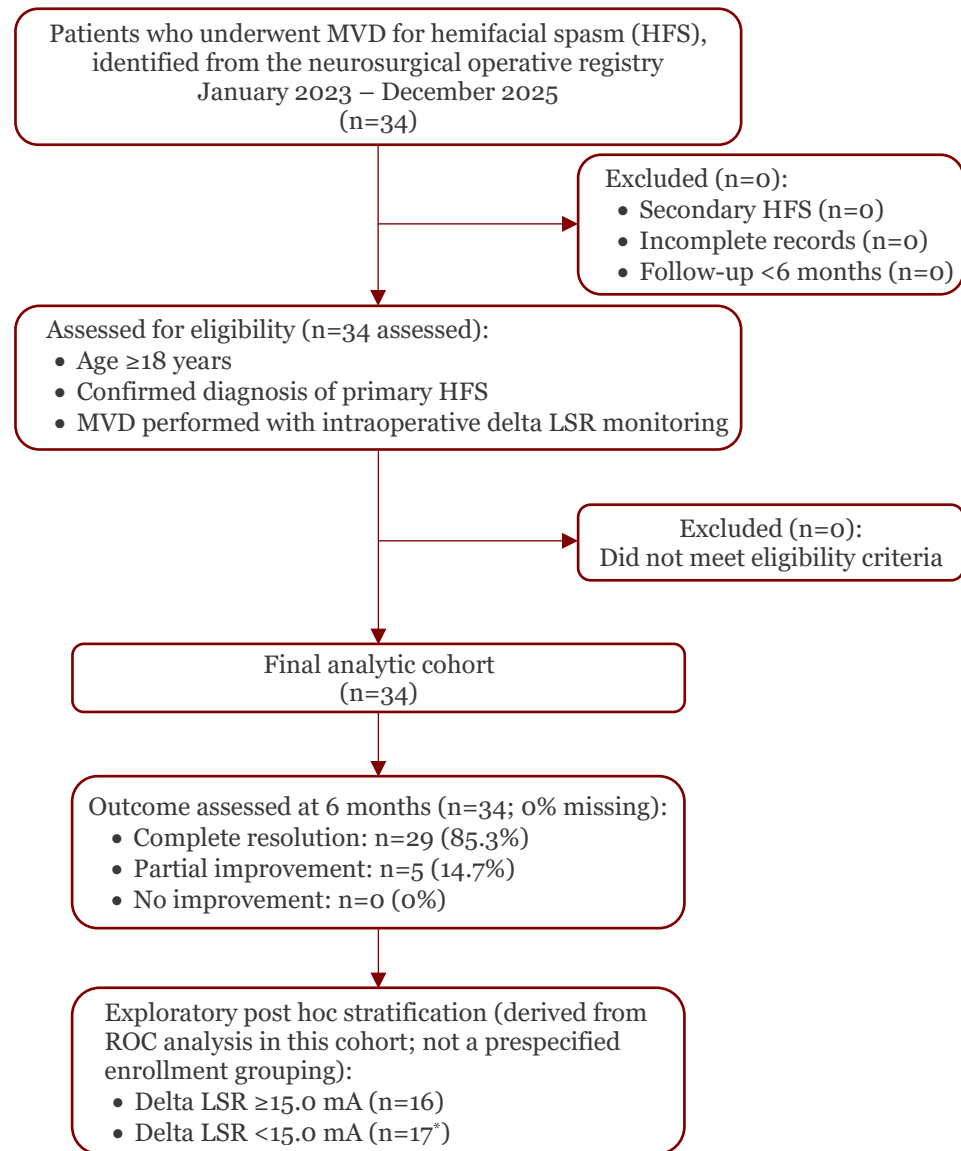


Figure 1. Flow diagram of patient selection and the final study cohort undergoing MVD for HFS. The delta LSR subgroups represent a post hoc exploratory stratification derived from ROC analysis within the same cohort and were not used for patient selection. *One patient had a non-quantifiable post-decompression LSR value (>20 mA); therefore, delta LSR could not be calculated, and the patient was excluded from continuous and threshold-based delta LSR analyses.

Table 3. Association between delta LSR and clinical outcome following MVD surgery

Delta LSR	Complete resolution (clinical success)	Partial/no improvement (clinical non-success)	Total, n (%)
	n (%)	n (%)	
LSR ≥15.0 mA	16 (100.0)	0 (0.0)	16 (48.5)
LSR <15.0 mA	12 (70.6)	5 (29.4)	17 (51.5)
Total	28 (84.8)	5 (15.2)	33 (100.0)

LSR: lateral spread response; MVD: microvascular decompression.

Fisher's exact test: $p=0.044$. Because the cut-off was selected from the same outcome data, this comparison is post hoc, and the nominal p -value should not be interpreted as confirmatory. The full study cohort comprised 34 patients. This table includes only the 33 patients with an exact numeric delta LSR; the excluded patient achieved complete resolution but had a non-quantifiable delta LSR.

Exploratory Firth logistic regression analysis

After adjustment, each 1-mA increase in delta LSR was associated with an estimated 18% increase in the odds of complete resolution (**Table 4**). However, this association was not statistically significant (adjusted OR: 1.18; 95%CI: 0.96–1.45; $p=0.107$). Age, sex, and symptom duration

were also not statistically significant. Because only five non-success outcomes occurred, the model was considered an exploratory sensitivity analysis and was not used to establish independent prognostic value (**Table 4**).

Table 4. Multivariable logistic regression using Firth's penalized likelihood for clinical success

Variable	Adjusted OR	95%CI	p-value
Age (per 1-year increase)	0.98	0.92–1.05	0.632
Sex (female vs male)	2.26	0.30–17.12	0.430
Symptom onset duration (per 1-month increase)	1.01	0.99–1.02	0.581
Delta LSR (per 1 mA increase)	1.18	0.96–1.45	0.107

OR: odds ratio; CI: confidence interval; LSR: lateral spread response.

Firth penalized likelihood was used because of sparse outcome data. With only five non-success outcomes and four predictors, the estimates are unstable and should be interpreted as exploratory.

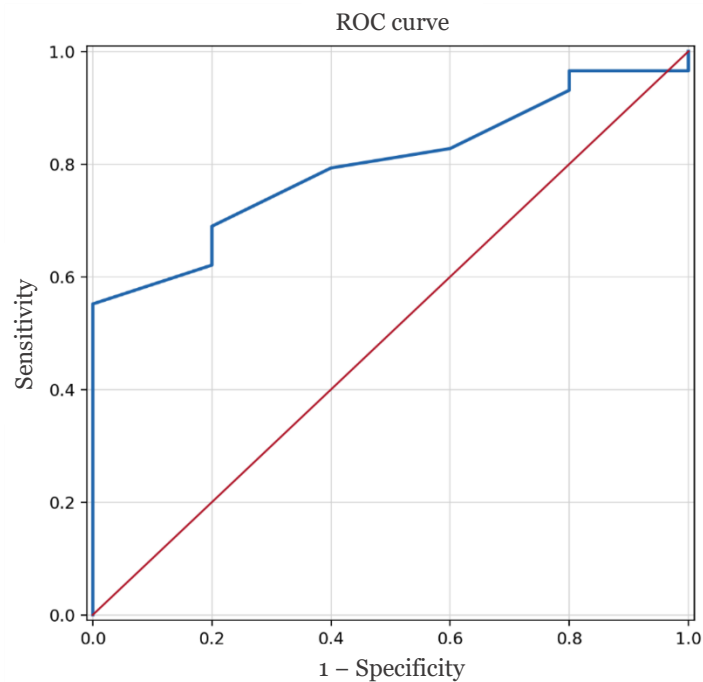


Figure 2. Receiver operating characteristic (ROC) curve of intraoperative delta lateral spread response (LSR) for predicting complete HFS resolution at six months after MVD.

Discussion

Disappearance of the LSR is widely regarded as electrophysiological evidence of successful decompression; however, whether this intraoperative finding reliably predicts postoperative symptom resolution and long-term recurrence remains controversial [4,6-9]. Previous studies have yielded inconsistent results. Some have demonstrated a significant association between intraoperative LSR disappearance and favorable clinical recovery [6,7], whereas others have suggested that its prognostic value is limited once adequate decompression has been achieved microscopically [10,11]. Consequently, LSR has traditionally been regarded as an adjunctive intraoperative monitoring tool rather than a validated prognostic biomarker, and no universally accepted quantitative threshold has been established to guide intraoperative decision-making. Against this background, the present study found a preliminary association between the recorded intraoperative change in LSR activation threshold and complete spasm resolution at six months. The AUC of 0.793 suggests potentially useful discrimination; however, the confidence interval was wide, and only five patients experienced clinical non-success.

Complete postoperative symptom resolution was achieved in 85.3% (29/34) of patients, consistent with durable outcomes reported by high-volume MVD centers worldwide. A previous study involving more than 6,000 patients reported one-year cure rates exceeding 90%, confirming MVD as the most effective definitive treatment for HFS [12]. Unlike botulinum toxin injections, which provide temporary symptomatic relief by inhibiting neuromuscular

transmission, MVD directly addresses the underlying neurovascular conflict by eliminating chronic arterial compression at the facial nerve root exit zone (REZ). Relief of this mechanical irritation interrupts the pathophysiological cascade responsible for ectopic facial nerve excitation, allowing progressive restoration of normal neural function. Moreover, the low incidence of postoperative facial weakness and hearing deterioration observed in the present study further supports the safety of contemporary MVD when meticulous microsurgical techniques are combined with continuous intraoperative neurophysiological monitoring. These findings align with previously reported permanent facial weakness and hearing impairment rates of approximately 1–3% in large surgical series [13,14].

The biological plausibility of these findings is supported by the current understanding of HFS pathophysiology. Chronic pulsatile compression at the facial nerve REZ produces focal demyelination, facilitating ephaptic transmission between adjacent motor fibers and generating abnormal muscle activation. In addition to these peripheral changes, prolonged neurovascular compression appears to induce secondary hyperexcitability of the facial motor nucleus through persistent afferent stimulation. These peripheral and central mechanisms are not mutually exclusive but may interact throughout the course of HFS, contributing collectively to the development and persistence of facial spasms. Following successful MVD, the peripheral source of ectopic excitation is removed immediately; however, normalization of central motor nucleus excitability may occur more gradually. This temporal dissociation provides a plausible explanation for why intraoperative electrophysiological improvement does not always correspond to immediate postoperative symptom resolution. Consistent with this concept, several studies have reported delayed clinical recovery despite technically adequate decompression, with facial spasms continuing to improve over several months following surgery [15-17].

At the candidate 15.0-mA cut-off, all patients above the threshold achieved complete resolution, but sensitivity was only 57.1%. This pattern suggests that a large increase in the recorded threshold may be associated with favorable outcome, whereas a smaller increase does not reliably identify clinical non-success. The apparent perfect specificity was based on only five non-success cases and may reflect threshold overfitting. Persistent LSR should therefore not be interpreted as evidence of inadequate decompression solely on the basis of these findings.

These findings also have important implications for intraoperative surgical decision-making. Historically, persistent LSR has occasionally prompted surgeons to continue exploring the REZ despite satisfactory microscopic decompression, with the goal of achieving complete abolition of the electrophysiological response. Although additional exploration may occasionally reveal an overlooked offending vessel, unnecessary manipulation around the facial and vestibulocochlear nerves inevitably increases the risk of traction-related neurological injury. These results suggest that persistent LSR should not automatically be interpreted as evidence of inadequate decompression when the offending vessel has already been completely mobilized, and appropriate decompression has been confirmed under direct microscopic visualization. Instead, intraoperative LSR should be interpreted within the broader operative context, integrating direct inspection of the REZ, identification and transposition of all offending vessels, preservation of perforating arteries, and complementary neurophysiological monitoring such as BAEPs and facial motor evoked potentials (FMEPs). This multimodal approach is supported by recent evidence demonstrating that combined LSR and BAEP monitoring reduces postoperative hearing impairment without compromising the diagnostic value of LSR for predicting surgical success [18]. Accordingly, LSR should be regarded as one component of a comprehensive intraoperative assessment rather than the sole determinant of operative adequacy.

The variability in previously published evidence is therefore not unexpected. Considerable heterogeneity exists among previous studies with respect to stimulation techniques, anesthetic protocols, definitions of LSR disappearance, electrophysiological equipment, follow-up duration, and surgeon experience, all of which may substantially influence intraoperative recordings and postoperative outcomes [12]. Furthermore, reliance on binary classification inevitably sacrifices valuable information regarding the extent of electrophysiological recovery. A patient demonstrating a 90% reduction in LSR amplitude and another exhibiting no measurable improvement would both be categorized as having “persistent LSR,” despite potentially

representing different physiological states. This limitation may partially explain why qualitative studies have produced inconsistent prognostic estimates despite evaluating the same electrophysiological phenomenon. By preserving information regarding the magnitude of recovery, quantitative assessment provides a more nuanced evaluation of facial nerve function during decompression and may better reflect the biological continuum of neural recovery.

The findings in this study support that intraoperative delta LSR can be used as an adjunct in guiding surgery for HFS cases, specifically with a threshold of 15.0 mA. Several limitations materially restrict interpretation and full adoption of the threshold. First, this was a retrospective, single-center cohort of only 34 patients, with five clinical non-success outcomes. This event count was insufficient for reliable multivariable modeling or stable threshold development. To date, LSR recording and intraoperative monitoring have not been adopted as routine practice in Indonesia, and the capability to perform them remains limited to certain centers. Second, the 15.0-mA cut-off was selected and assessed in the same dataset, introducing potential optimism into the threshold-specific estimates. Third, important clinical and technical confounders could not be addressed adequately because of the small number of outcome events. In addition, LSR recording and intraoperative monitoring are not routinely available across all centers in Indonesia, limiting the generalizability and broader implementation of this approach. Prospective multicentre studies should use standardized monitoring protocols, prespecify outcome definitions and analysis plans, and validate candidate thresholds in independent cohorts before clinical implementation.

Conclusions

In this Indonesian cohort, a greater increase in the recorded LSR activation threshold was associated with complete spasm resolution at six months after MVD for HFS. A post hoc delta-LSR threshold of 15.0 mA showed high specificity but limited sensitivity in the development cohort. Because the threshold was selected and evaluated in the same small sample and final-threshold recording was not fully uniform, these findings are exploratory and should not yet guide intraoperative decision-making. A larger prospective cohort with standardized LSR measurement is required.

Ethics approval

This study was approved by the Health Research Ethics Committee of the Faculty of Medicine, Universitas Indonesia, Dr. Cipto Mangunkusumo Hospital, KET-7/2/UN2.F1/ETIK/PPM.00.02/2026. As this was a retrospective study based on existing medical records, the requirement for individual informed consent was waived by the ethics committee, and patient data were anonymized prior to analysis.

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Competing interests

The authors declare that there is no conflict of interest.

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Underlying data

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Declaration of artificial intelligence use

During manuscript preparation, the authors used AI-assisted tools solely for language editing and formatting checks. All scientific content, data interpretation, and conclusions are the sole responsibility of the authors, who reviewed the manuscript and take full responsibility for its final content.

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References

1. Lee S, Joo KM, Park K. Challenging microvascular decompression surgery for hemifacial spasm. World Neurosurg 2021;151:e94-e99.
2. Cho KR, Lee HS, Kim M, *et al.* Optimal method for reliable lateral spread response monitoring during microvascular decompression surgery for hemifacial spasm. Sci Rep. 2023;13(1):21672.
3. Nugroho SW, Perkasa SAH, Gunawan K, *et al.* Predicting outcome of hemifacial spasm after microvascular decompression with intraoperative monitoring: A systematic review. Heliyon 2021;7(2):e06115.
4. Møller AR, Jannetta PJ. Hemifacial spasm: Results of electrophysiologic recording during microvascular decompression operations. Neurology 1985;35(7):969-974.
5. Sprengers L, Lemmens R, van Loon J. Usefulness of intraoperative monitoring in microvascular decompression for hemifacial spasm: A systematic review and meta-analysis. Br J Neurosurg 2022;36(3):346-357.
6. 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(6):1581-1588.
7. Kang MC, Choi YS, Choi HK, *et al.* Efficacy of the disappearance of lateral spread response before and after microvascular decompression for predicting the long-term results of hemifacial spasm over two years. J Korean Neurosurg Soc 2012;52(4):372-376.
8. Kim M, Cho KR, Park SK, *et al.* Prognostic value of lateral spread response recorded 1 month after microvascular decompression for the treatment of hemifacial spasm. Neurosurgery 2022;91(1):159-166.
9. 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. Acta Neurochir 1999;141(7):737-743.
10. El Damaty A, Rosenstengel C, Matthes M, *et al.* The value of lateral spread response monitoring in predicting the clinical outcome after microvascular decompression in hemifacial spasm: A prospective study on 100 patients. Neurosurg Rev 2016;39(3):455-466.
11. Thirumala PD, Wang X, Shah A, *et al.* Clinical impact of residual lateral spread response after adequate microvascular decompression for hemifacial spasm: A retrospective analysis. Br J Neurosurg 2015;29(6):818-822.
12. Holste K, Sahyouni R, Teton Z, *et al.* Spasm freedom following microvascular decompression for hemifacial spasm: Systematic review and meta-analysis. World Neurosurg 2020;139:e383-e390.
13. Chung SS, Chang JH, Choi JY, *et al.* Microvascular decompression for hemifacial spasm: A long-term follow-up of 1,169 consecutive cases. Stereotact Funct Neurosurg 2001;77(1-4):190-193.
14. Sindou M, Mercier P. Microvascular decompression for hemifacial spasm: Outcome on spasm and complications. A review. Neurochirurgie 2018;64(2):106-116.
15. Al Menabbawy A, Eisold M, El Refaee E, *et al.* Timing matters: evaluating lateral spread response disappearance as a prognostic marker in microvascular decompression for hemifacial spasm: A phenomenological study. Acta Neurochir 2025;167(1):112.
16. Al Menabbawy A, El Refaee E, Elwy R, *et al.* A multivariable prediction model for recovery patterns and time course of symptoms improvement in hemifacial spasm following microvascular decompression. Acta Neurochir 2022;164(3):833-844.
17. Chan RW, Chiang YH, Chen YY, *et al.* Predicting early loss of lateral spread response before decompression in hemifacial spasm surgery. Life 2021;12(1):40.
18. Cheng D, Liu C, Qiu Y, Ji C. Monitoring of the lateral spread response combined with brainstem auditory evoked potentials in microvascular decompression for hemifacial spasm. Front Neurol 2025;16:1516606.