Prognostic value of lateral spread response during microvascular decompression for hemifacial spasm

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Abstract

Objective: This study aimed to investigate the prognostic value of the lateral spread response (LSR) for predicting surgical outcomes following microvascular decompression (MVD) in patients with hemifacial spasm.

Methods: Seventy-three patients with hemifacial spasm underwent MVD with intraoperative LSR monitoring. Surgical outcomes were evaluated I week and I year after MVD and correlations between LSR characteristics and surgical outcomes were analyzed.

Results: The LSR disappeared completely in 61 patients during surgery (Group A; prior to insertion of Teflon felt pledgets in 11, after insertion of pledgets in 50), disappeared partially in nine patients (Group B), and remained unchanged in three patients (Group C). Fifty-five patients showed short-term and 61 patients showed long-term clinical cures during the follow-up period. The short-term and long-term cure rates were significantly higher in Group A than in Group C. There was no correlation between the time of complete LSR disappearance and surgical outcomes.

Conclusions: Disappearance of the LSR during MVD is correlated with the surgical outcomes. Intraoperative LSR monitoring is a reliable approach for predicting the prognosis of hemifacial spasm following MVD, but the time at which LSR disappears is not a prognostic indicator.

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Keywords

Hemifacial spasm, lateral spread response, microvascular decompression, prognosis, surgery, intraoperative monitoring

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Introduction

Hemifacial spasm (HFS) is a rare neuromuscular disorder characterized by paroxysmal involuntary tonic clonic contractions of unilateral facial muscles, commonly beginning in the orbicularis oculi and spreading to the other facial muscles.¹ HFS has been reported to show a female predominance and to have a higher incidence in Asian compared with other populations.² HFS is considered to be caused by vascular compression of the facial nerve at the root exit zone (REZ), and microvascular decompression (MVD) is thus the current gold-standard treatment.³ However, identification of the offending vessel and adequate decompression are sometimes challenging, potentially resulting in unsatisfactory surgical outcomes.

The lateral spread response (LSR), also known as the abnormal muscle response, is an abnormal electrophysiological characteristic of primary HFS: when a certain branch of the facial nerve is stimulated. the responses of the muscles innervated by the other facial nerve branches can be monitored. This phenomenon was originally reported by Møller and Jannetta in 1985.⁴ The LSR can be recorded preoperatively or intraoperatively in most patients with HFS. The LSR disappears after adequate decompression, and according to the literature, intraoperative monitoring of the LSR can help to identify the offending vessels and confirm adequate decompression of the facial nerve from neurovascular contact.⁵ value However, the prognostic of intraoperative monitoring of the LSR remains controversial.^{6,7} In the current study, we retrospectively investigated the value of the LSR for predicting the short-term and long-term surgical outcomes following MVD in patients with HFS.

Materials and methods

Patients

A total of 73 consecutive patients were enrolled the Department at of Neurosurgery, the First Hospital of Jilin University, between April 2013 and December 2016. The patients were diagnosed with primary HFS and underwent MVD. The exclusion criteria were preoperative administration of botulinum toxin, secondary HFS due to intracranial spaceoccupying lesions, or recurrent HFS. This study was approved by the local ethics committee and written informed consent was obtained from each participant.

Intraoperative neurophysiological monitoring and MVD

Anesthesia was induced using remifentanil, propofol, and a single-dose, short-term muscle relaxant (cisatracurium; 0.15 mg/kg), and general anesthesia was performed using continuous intravenous infusion of remifentanil and propofol. Neurophysiological monitoring was prepared following the induction of anesthesia. Paired needle electrodes for stimulation were placed 5 mm apart along the marginal mandibular branches of the facial nerve to evoke LSR. Two pairs of recording needle electrodes were inserted into the ipsilateral orbicularis oculi muscle (to record the abnormal electromyographic responses) and the ipsilateral mentalis muscle (to record the normal electromyographic responses), respectively. The ground electrode was inserted into the orbicularis oris muscle. The electrode stimulation parameters were a single, rectangular wave, for 0.2 ms at 0.5-20 mA. Brainstem auditoryevoked potential monitoring was also performed. Electromyography monitoring of the trigeminal and facial nerves was carried out using muscles innervated by these nerves (e.g. masseter, orbicularis oculi, orbicularis oris). The bandpass filter was set at 30-3000 Hz (Xltek 32, Natus, USA). The train-offour ratio was used to assess the degree of neuromuscular blockade. The LSR waves and amplitudes were recorded at the following time points: 1) after induction of anesthesia: 2) before skin incision: 3) immediately after skin incision; 4) immediately after bone flap opening; 5) during arachnoid incision and cerebrospinal fluid release; 6) immediately after dura mater complete opening; 7) before MVD; 8) after MVD; 9) after dura mater closure; 10) after bone flap closure; and 11) after skin suture. The patients were classified into three groups according to the LSR monitoring results: Group A (complete disappearance of LSR intraoperatively; Figures 1 and 2); Group B (partial disappearance of LSR intraoperatively, amplitude reduced by >50%; Figure 3), and Group C (no change in LSR intraoperatively, amplitude reduced by <50%; Figure 4).

MVD was performed via the retrosigmoid approach in all patients. If the LSR persisted after decompression, we looked again for vessels compressing the distal facial nerve until confirming that there were no additional offending vessels.

Surgical outcome evaluation

Surgical outcomes were evaluated at 1 week and 1 year after MVD. Cure was defined as an HFS-free state, and no cure as partial or no relief of HFS.

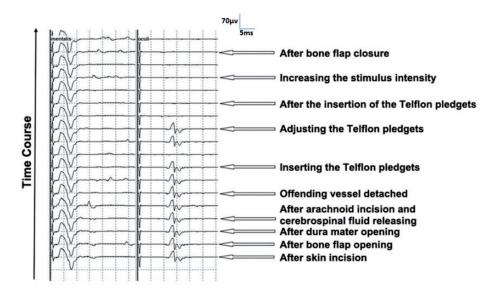


Figure I. Complete disappearance of the LSR after insertion of the Teflon pledgets.

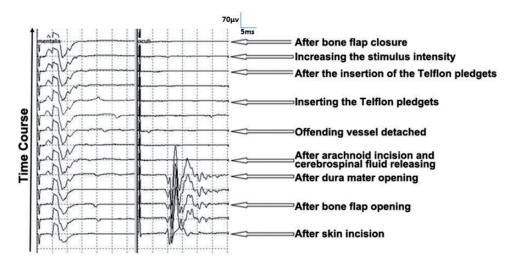


Figure 2. Complete disappearance of the LSR prior to insertion of the Teflon pledgets.

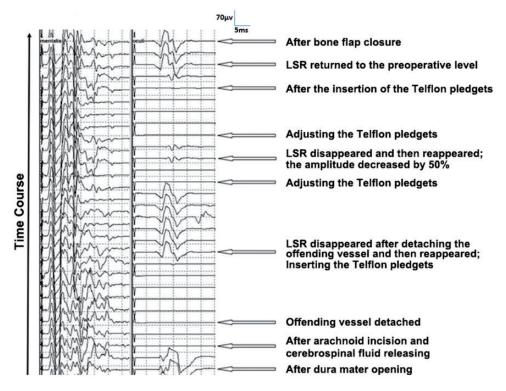


Figure 3. Partial disappearance of the LSR after insertion of the Teflon pledgets (wavelength decreased by 50% and amplitude decreased by 20%).

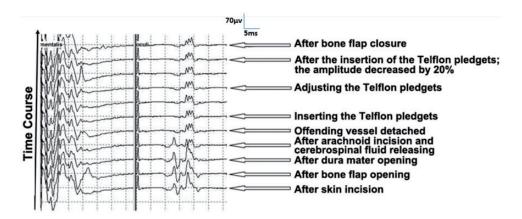


Figure 4. Persistence of the LSR after insertion of the Teflon pledgets.

Statistical analysis

Statistical analyses were performed using SPSS 24.0 software (IBM Corp., Armonk, NY, USA). Numerical data were compared among multiple groups using the Kruskal–Wallis H test, and between two groups using Wilcoxon's test. Values of $P \le 0.05$ were considered significant.

Results

Clinical characteristics of study participants

The study population included 21 men and 52 women, with a mean age (\pm standard deviation) of 50.0 ± 10.5 years (range, 22–68 years). The mean symptom duration was 5.3 ± 4.1 years (range, 0.5–20 years). The HFS was left-sided in 34 patients and right-sided in 39 patients.

LSR monitoring and surgical outcomes

The LSR was observed during MVD in all patients. The LSR disappeared completely during surgery in 61 patients (Group A), disappeared partially in nine patients (Group B), and remained unchanged in three patients (Group C; complete decompression confirmed by surgeons).

Fifty-five patients exhibited short-term clinical cures, of whom 49 showed complete disappearance and six showed partial disappearance of the LSR. Sixty-one patients exhibited long-term clinical cures, of whom 53 showed complete disappearance and seven showed partial disappearance of the LSR, and one showed no change in the LSR. In Group A, the LSR disappeared prior to insertion of Teflon pledgets in 11 patients (7 showed short-term and 9 showed long-term clinical cures) and after insertion of Teflon pledgets in the remaining 50 patients (42 showed short-term and 44 showed long-term clinical cures).

No patients experienced neurophysiological monitoring-related complications. Eight patients had facial paralysis and three patients had hearing loss at 1 week postoperatively, and three patients had facial paralysis and one patient had hearing loss at 1 year postoperatively.

Correlations between LSR characteristics and surgical outcomes

The short-term and long-term cure rates in Group A were significantly higher than in Group C (P < 0.05), but there were no significant differences in short-term or long-term cure rates between Groups A and B

or between Groups B and C (Table 1). There was no correlation between the time of complete LSR disappearance and surgical outcomes (Table 2).

Discussion

The pathogenetic mechanism of HFS remains to be fully elucidated, though regular contractions of the facial muscles in HFS are currently considered to be caused by hyperexcitability of the facial nerve. There are two widely-accepted theories to explain this phenomenon: the central origination theory proposes that the hyperexcitability of the facial nerve is a result of hyperexcitability of the motor nucleus of the facial nerve,⁸ while the peripheral origination theory considers that vascular compression of the facial nerve at the REZ leads to ephaptic transmission and myelin injuries.9 MVD was developed as a treatment for HFS based on the peripheral origination theory. Theoretically, decompression of the facial nerve can reduce its excitability, which may manifest as disappearance of the LSR and lead to clinical improvement of HFS. Intraoperative neurophysiological monitoring, including the F wave, blink reflex, and LSR, is vital for studying the pathophysiology of HFS and for optimizing the efficacy of MVD.^{4,10} The LSR is the characteristic neurophysiological feature of HFS and cannot be detected under normal conditions. The existence of the LSR indicates the existence of abnormal cross connections between facial nerve branches or fibers, though the definitive abnormal connections have yet to be identified.

Previous studies showed that intraoperative LSR monitoring was helpful for identifying the offending vessels and confirming adequate decompression of the facial nerve intraoperatively.^{11–13} Intraoperative LSR monitoring can provide objective evidence to confirm the identity of the offending vessels; if the LSR disappears after a suspected vessel is detached from the facial nerve, that vessel can be confirmed as the offending vessel, but if the LSR persists, there may be other offending vessel(s).

The prognostic value of LSR monitoring during MVD for predicting the surgical outcomes of HFS remains controversial.

 Table 1. Surgical outcomes following MVD in patients classified according to intraoperative change in LSR amplitude.

LSR amplitude	Total (n)	Short-term cure (n)	Long-term cure (n)
Complete disappearance	61	52	59
Partial disappearance	9	6	7
No change	3	0	I
Hc value	_	13.567	17.563
P value	_	<0.01	<0.01

Hc: Kruskal-Wallis H test correction.

Table 2. Surgical outcomes following MVD in patients classified according to the time of complete LSR disappearance.

Time of complete LSR disappearance	Total (n)	Short-term cure (n)	Long-term cure (n)
Prior to insertion of pledgets After insertion of pledgets	 50	7 45	10 49
P value	_	>0.05	>0.05

A previous meta-analysis showed that complete intraoperative disappearance of the LSR was associated with a 4.2-fold increase in the chance of a clinical cure compared with cases in which the LSR persisted.¹⁴ Furthermore, intraoperative disappearance of the LSR was significantly correlated with the surgical outcomes of HFS, and could predict clinical remission.¹⁴ However, other studies concluded that intraoperative LSR monitoring did not predict the surgical outcomes of MVD.¹⁵ El Damaty et al.⁶ prospectively analyzed 100 patients with HFS and found that the LSR could guide adequate decompression of the facial nerve during MVD but did not predict postoperative efficacy. Hatem et al.⁷ also noted that all 10 patients in their study achieved clinical cures, despite persistence of the LSR during MVD, thus shedding doubt on the application value of the LSR in MVD.

The LSR may disappear at varying time points intraoperatively. The current study found no correlation between the time at which LSR disappeared completely and surgical outcomes. We speculate that complete disappearance of the LSR may represent adequate decompression of the facial nerve, and the prognosis may thus be favorable irrespective of the time at which the LSR disappears. However, Kim et al.¹⁶ found that patients in whom the LSR disappeared prior to adequate decompression had poorer outcomes than those in whom LSR disappeared after adequate decompression. These inconsistent findings may be due to the use of different criteria to evaluate the efficacies and to the variable follow-up periods.

The LSR persisted in some patients (n = 3 in the current study) despite adequate decompression of the facial nerve throughout the REZ to the internal auditory canal. The remaining LSR may be caused by hyperexcitability of the motor nucleus of the facial nerve. Additionally, the short-term and long-term cure rates in Group A

(LSR completely disappeared) were significantly higher than those in Group C (LSR persisted), indicating that the LSR can help to predict surgical outcomes and suggesting that peripheral neurogenic HFS may indicate a better prognosis than central neurogenic HFS. Moreover, we speculate that reduced amplitude of the LSR may be caused by both central and peripheral mechanisms.

During MVD, the LSR usually disappears after insertion of the Teflon pledgets. Notably however, the LSR may occasionally disappear after the opening of the arachnoid membrane, because the cerebrospinal fluid drainage may change the anatomical relationship between the facial nerve and vessel.^{17,18} the offending The most common offending vessels in HFS are the anterior inferior cerebellar artery and the posterior inferior cerebellar artery. However, the widespread application of intraoperative LSR monitoring has identified other offending vessels, including the vertebral artery and veins. Facial nerve compression can sometimes occur outside the REZ. Considering that relative anatomical shifting is reversible, intraoperative LSR monitoring should be maintained throughout the surgical procedures.

Further studies are planned to investigate the prognostic values of LSR arising from diverse targets. We are currently carrying out a randomized controlled trial to compare the prognostic values of LSRs generated by stimulation of the zygomatic and marginal mandibular branches.

In the current study, we chose the mentalis muscle for monitoring the LSR for several reasons. First, the marginal mandibular branch of the facial nerve runs along the mandibular angle, and the location of the stimulation point is thus more practicable and stable and the LSR detection rate is relatively high. Second, the ocular muscles are more commonly involved in patients with hemifacial spasm, and stimulation of the marginal mandibular branch and LSR recording on the orbicularis oculi are thus reasonable. Finally, when stimulating the marginal mandibular branch, the distance between the stimulation point and the recording point is longer than in the case of zygomatic stimulation and there are fewer artifacts, thus increasing the stability of the LSR.

Although intraoperative LSR monitoring can help guide MVD and predict the surgical outcomes, the surgical outcomes of HFS are multifactorial and LSR therefore cannot be used as the sole criterion for prognostic evaluation.

In conclusion, intraoperative LSR monitoring can help to identify the offending vessels in HFS and can confirm adequate decompression of the facial nerve. Disappearance of the LSR during MVD is closely associated with the surgical outcomes of MVD. Intraoperative LSR monitoring may thus be a reliable approach for predicting the prognosis of HFS following MVD, though the time of complete LSR disappearance is not a reliable prognostic indicator.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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