Current Science
CS 5 (2), 1820-1826 (2025)
Received 20 **Jan** 2025 | **Revised 21 Feb** 2025 | **Accepted 16 March**. 2025 | **Online Available 15 April** 2025
https://doi.org/ 10.52845/CS/2025-5-2-44

Current Science

Current Science

OPEN ACCESS

ISSN(O)2795-8639

ORIGINAL ARTICLE

Analysis of Facial Nerve Motor Evoked Potentials for Assessing a Central Mechanism in Hemifacial Spasm

Xie Xiangtong[1], Meng Xuan[2], Wang Anqi[1], Guo Wei[1], Zhou Shijun[1], Wangzhimin[3], Yang Debao[1]

[1]**Department of Neurosurgery, Suzhou Kowloon Hospital, Shanghai Jiaotong University School of Medicine, Suzhou 215028**

[2]**Department of Neurosurgery, Suzhou Boe Hospital Suzhou 215217**

[3]**Department of Neurosurgery, Suzhou Dushu Lake Hopital Suzhou 215000**

[*]**Corresponding Author: Wang Zhimin, Yang Debao**

Abstract

Objective: To investigate whether the excitability of the facial nucleus group is enhanced in patients with hemifacial spasm, thereby further exploring the mechanism of hemifacial spasm.

Methods: Microvascular decompression was employed to treat hemifacial spasm in 30 patients, and motor evoked potentials of the affected and healthy sides were monitored. The amplitude and threshold voltage of bilateral facial nerve motor evoked potential were compared using monopulse and multi-pulse stimulations. The inhibitory effect of sevoflurane on facial nerve motor evoked potential was monitored and analyzed statistically.

Results: Stable facial motor evoked potential could be elicited by monopulse in 26 patients on the affected side, while only 5 patients on the healthy side, demonstrating a statistically significant difference between the two groups. The average threshold voltage of neuromotor evoked potential on the affected and healthy sides was 140.3 ± 26.8 V and 177.0 ± 23.2 V, respectively, with significant differences between the two groups. There was no statistical difference in the amplitude of facial motor evoked potential between the two groups. Sevoflurane exhibited a significant inhibitory effect on facial nerve motor evoked potential.

Conclusion: The excitability of the facial nucleus mass was increased in patients with hemifacial spasm.

[Key Words] Hemifacial Spasm; Microvascular Decompression; Facial Nerve Motor Evoked Potentials; Sevoflurane; Mechanism

Introduction

The pathogenesis of hemifacial spasm (HFS) remains highly controversial. In almost all HFS patients, responsible vascular compression has been found at the root entry/exit zone (REZ) of the facial nerve. The long-term pulsatile compression of the facial nerve REZ by blood vessels leads to demyelination of nerve fibers, and the contact between demyelinated nerve fibers forms false synaptic transmission. Therefore, the false synaptic transmission occurring at the site of vascular-nerve contact is considered the pathogenesis of hemifacial spasm (peripheral theory). However, many scholars believe that vascular nerve compression is the initiating factor of hemifacial spasm. The long-term pulsatile compression of the nerve by blood vessels stimulates an increase in excitability of the facial nerve motor nucleus, leading to the establishment of abnormal new synapses between motor neurons, resulting in a short circuit and the occurrence of hemifacial spasm (central theory). This study provides a new method for further research on the pathogenesis of hemifacial spasm by monitoring facial nerve motor evoked potentials (FNMEP) during microvascular decompression surgery for HFS.

1. Materials and Methods

1.1 Clinical Data

A total of 30 cases of hemifacial spasm treated with microvascular decompression (MVD) at the Department of Neurosurgery, Suzhou Kowloon Hospital affiliated with Shanghai Jiao Tong University School of Medicine, were selected from April 2018 to March 2019. Preoperative routine magnetic resonance tomography angiography (MRTA) was performed, utilizing three-dimensional time-of-flight (3D-TOF) and three-dimensional constructive interference in steady state (3D-CISS) imaging techniques to clarify the relationship between the responsible vessel and the facial nerve, assess the anatomical structure of the cerebellopontine angle, and exclude secondary lesions causing hemifacial spasm.

1.2 Anesthesia Method

After entering the room, the patient was placed supine on the operating table. Continuous monitoring of electrocardiogram, non-invasive blood pressure, peripheral oxygen saturation, and respiratory rate was conducted; a peripheral venous line was established, and oxygen was administered via a mask. Lidocaine was used for local anesthesia, and radial artery catheterization was performed to monitor invasive blood pressure and arterial blood gas. While providing oxygen via a mask, intravenous administration of sufentanil 0.3 μg/kg, propofol 1.5-2.5 mg/kg, and cisatracurium 2 mg/kg was carried out, followed by endotracheal intubation with volume-controlled mechanical ventilation selected. After intubation, muscle relaxants were discontinued, and during the procedure, remifentanil, propofol, and dexmedetomidine were infused intravenously for maintenance. The medications were gradually discontinued before the end of the surgery. Communication with the intraoperative and electrophysiological monitoring physician was established regarding the use of inhalational anesthetics, with sevoflurane chosen when inhalational anesthetics were used. The minimum alveolar concentration (MAC) value was recorded when inhalational anesthetics were administered.

1.3 Electrophysiological Monitoring Methods

The electrophysiological monitoring equipment used is the Cascade 32-channel system manufactured by CADWELL. The electrophysiological monitoring device is placed after satisfactory anesthesia. The spiral stimulation electrode anode is positioned at C3/C4 (according to the international 10-20 EEG system). Recording electrodes are needle electrodes placed on the orbicularis oculi, orbicularis oris, and mentalis muscles. The stimulation intensity and stimulation threshold are recorded; the stimulation threshold is defined as the minimum stimulation intensity that causes the amplitude of FNEMP to reach 50μV in 5 out of 10 consecutive stimulations [1,2]. The pulse width is 50μsec, and the filtering range is 30-3000Hz. Initially, multiple pulse stimulation is performed, delivering 4 pulses each time with an interval of 2ms; after determining the threshold voltage and FNMEP amplitude, single pulse stimulation is conducted. Waveforms with a latency of less than 10ms are considered to be induced by stimulation of the extracranial segment of the facial nerve; therefore, in this study, FNMEP waveforms with a latency greater than 10ms are selected as multiphasic waves. After communication between the electrophysiological monitoring physician, anesthesiologist, and the lead surgeon, sevoflurane is administered while recording the amplitude changes of FNMEP on the affected and unaffected sides at different MAC values; all monitoring is completed before the dura mater is opened during craniotomy. Postoperative FNMEP amplitudes are recorded after microvascular decompression is completed.

1.4 Statistical Analysis

Data are statistically analyzed using SPSS 22.0 software. Normal distribution measurement data are expressed as mean ± standard deviation ($\bar{x} \pm s$), and paired t-tests are used for normally distributed data; for pairwise comparisons, non-parametric data are analyzed using the Mann-Whitney U test. To compare the effects of sevoflurane on FNMEP amplitude and mean arterial pressure, one-way ANOVA is used for multiple pairwise comparisons. Rates or composition ratios are used to represent count data, and comparisons are made using the $\chi 2$ test or Fisher's exact probability method. A P value of <0.05 is considered statistically significant.

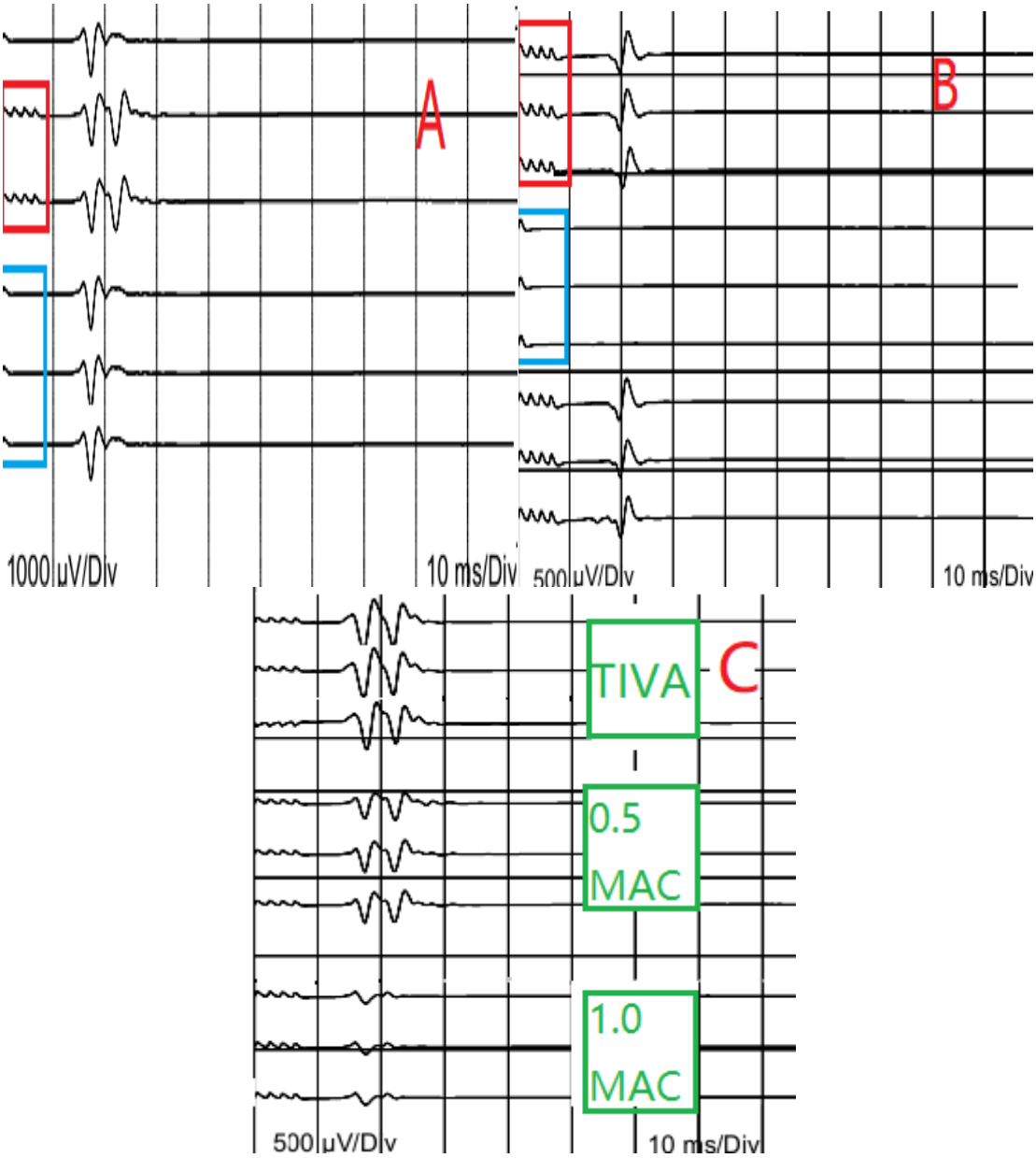


Figure 1: A. In patients with HFS, both multipulse (red) and single pulse (blue) can evoke FNMEP waveforms on the affected side; B. In HFS patients, multipulse (red) can evoke FNMEP waveforms on the unaffected side, while single pulse (blue) cannot evoke FNMEP waveforms; C. In HFS patients, after inhalation of the anesthetic sevoflurane, the amplitude of FNMEP decreases, and the decrease becomes more significant with higher inhalation concentrations.

2. Results

In the monitoring of FNMEP, we found that the waveforms recorded from the mentalis muscle were relatively stable. Therefore, data recorded from the mentalis muscle were selected as the basis for this study in all cases [1,3-4]. Stable FNMEP waveforms were recorded in 30 cases on the affected side of HFS, and stable waveforms were recorded in 29 cases on the unaffected side. A total of 29 patients who had FNMEP waveforms recorded on both sides were included, divided into the HFS affected side group (Group A) and the HFS unaffected side group (Group B). Among the 29 enrolled patients, ages ranged from 36 to 67 years; there were 14 males and 15 females; 17 had left-sided HFS and 12 had right-sided HFS; the duration of the disease ranged from 7 months to 9 years.

The average amplitude of FNMEP recorded on the affected side (Group A) was 207.2±62.1μV, with an average stimulation threshold voltage of 140.3±26.8V for the corresponding amplitude; the average amplitude of FNMEP recorded on the unaffected side (Group B) was 180.2±55.0μV,

with an average stimulation threshold voltage of 177.0±23.2V for the corresponding amplitude. When comparing the stimulation voltages between the affected side (Group A) and the unaffected side (Group B), there was a statistically significant difference, P<0.001 (Figure 2A); however, when comparing the FNMEP amplitudes recorded on the affected side (Group A) and the unaffected side (Group B), the difference was not significant, p=0.885 (Figure 2B).

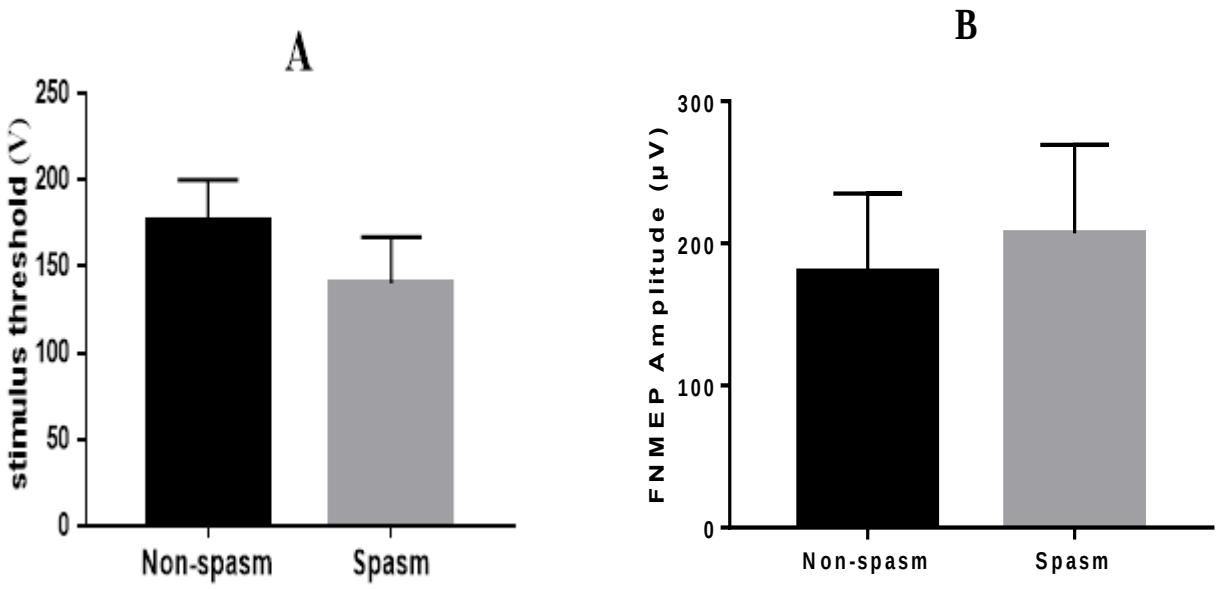


Figure 2: A. Stimulus voltage for inducing FNMEP in the affected side (Group A) and the healthy side (Group B); B. FNMEP amplitude recorded in the affected side (Group A) and the healthy side (Group B). Non-spasm: healthy side; Spasm: affected side; stimulus threshold: stimulus threshold voltage; FNMEP amplitude: FNMEP amplitude.

In the affected side (Group A), 26 cases were able to induce FNMEP waveforms with a single pulse; whereas in the healthy side (Group B), only 5 cases induced FNMEP waveforms with a single pulse. Statistical analysis of the two groups showed P < 0.001, indicating a significant difference between the two groups (Figure 3).

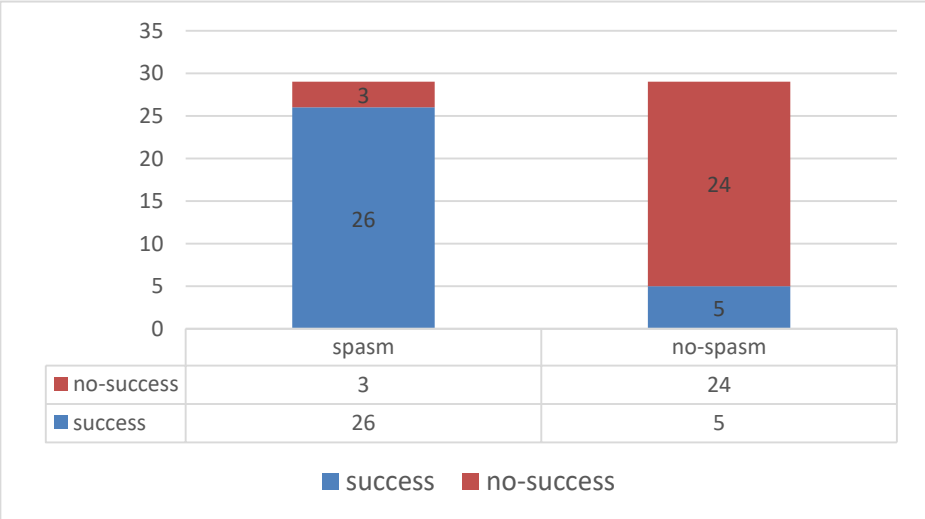
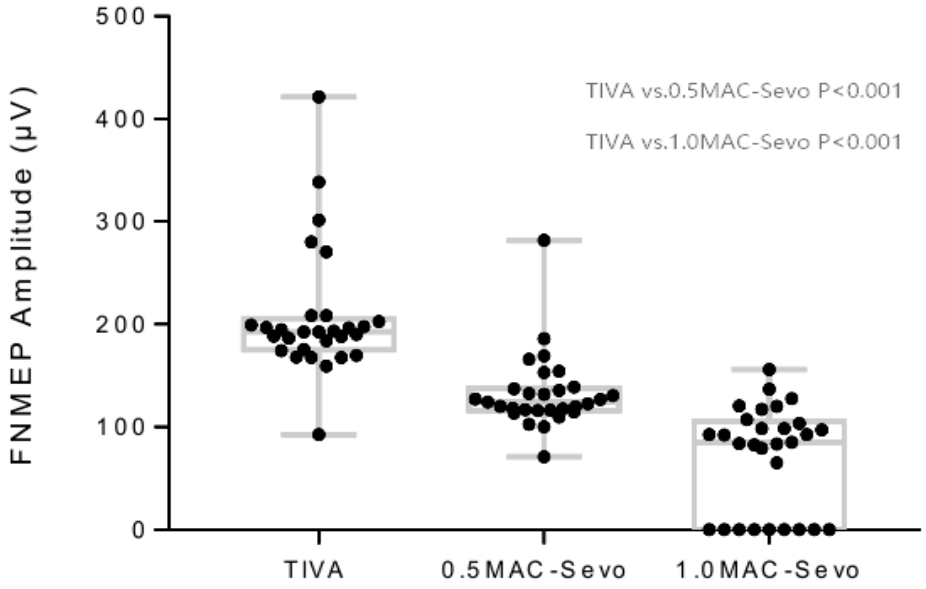


Figure 3: Number of cases obtaining FNMEP amplitude using single pulse stimulation in the affected side and the healthy side groups. Spasm: affected side; no-spasm: healthy side; success: stable FNMEP amplitude obtained; no-success: stable FNMEP amplitude not obtained.

After determining the baseline of FNMEP, patients were administered inhaled sevoflurane at concentrations of 0.5 MAC and 1.0 MAC, with corresponding mean arterial pressures of 75.6±3.5 mmHg, 74.2±3.2 mmHg, and 75.5±2.3 mmHg; there were no significant changes in mean arterial pressure before and after the administration of sevoflurane, with statistical analysis P values of 0.178 (TIVA vs. 0.5 MAC) and 0.987 (TIVA vs. 1.0 MAC).

At an inhaled concentration of 0.5 MAC of sevoflurane, the amplitude of FNMEP measured on the affected side (Group A) was 133.0±36.5 μV; when inhaling 1.0 MAC of sevoflurane, 9 patients were unable to obtain stable FNMEP amplitudes, with the amplitude on the affected side measured at 70.4±40.2 μV; the statistical analysis P values were 0.001 (TIVA vs. 0.5 MAC) and 0.001 (TIVA vs. 1.0 MAC), indicating a significant decrease in FNMEP amplitude after the administration of inhaled sevoflurane, with statistical significance (Figure 4).



(Figure 4: In patients with HFS, the amplitude of FNMEP on the affected side decreased after inhalation of the anesthetic sevoflurane, and the decrease in amplitude became more significant with higher inhalation concentrations. FNMEP amplitude: The amplitude of FNMEP.)

3. Discussion

Motor evoked potentials (MEP) are a method for monitoring the function and integrity of the motor conduction system by recording potential changes. In the 1980s, MEP was first used for monitoring during spinal surgery[5]. MEP can be divided into two modes: transcranial magnetic stimulation and transcranial electrical stimulation. Transcranial electrical stimulation motor evoked potential (TES-MEP) refers to the activation of cortical motor neurons by transcranial electrical stimulation of the cerebral cortex, which excites descending motor neurons to the medulla or spinal cord, allowing for the recording of action potentials in the muscles innervated by the corresponding motor neurons. It can also be recorded in the spinal cord or nerve roots, serving as a neurophysiological monitoring method for motor conduction function. MEP is sensitive to spinal cord compression, ischemia, blunt trauma, and spinal cord traction, and it has high sensitivity in monitoring cortical and subcortical ischemia as well as brain function impairment. TES-MEP can also be applied in skull base tumor surgeries, providing real-time and continuous monitoring of facial nerve motor evoked potentials (FNMEP), thereby monitoring facial nerve function and preventing postoperative facial nerve dysfunction.

FNMEP originates from the facial nerve motor nucleus, which has been recognized by scholars[1,6-10]. Inhalational anesthetics mainly include sevoflurane, desflurane, and isoflurane, which can inhibit the motor cortex of the brain, suppress anterior horn cells and interneurons in

the spinal cord, without affecting the peripheral neuromuscular junction[11-13]. To verify whether inhalational anesthetics have an inhibitory effect on FNMEP, this study administered different concentrations of sevoflurane and compared the amplitude of FNMEP before and after inhalation. After administering sevoflurane at a concentration of 0.5 MAC, a significant decrease in FNMEP amplitude was observed, with statistical analysis showing $P < 0.001$, indicating a significant difference. When the concentration of sevoflurane reached 1.0 MAC, FNMEP amplitude further decreased significantly, with 9 patients unable to obtain stable FNMEP amplitudes. Therefore, the study confirmed that sevoflurane has a significant inhibitory effect on FNMEP, and this effect is significantly enhanced with increasing concentrations of sevoflurane. Since sevoflurane has no significant effect on the peripheral neuromuscular junction, it further confirms that FNMEP originates from the facial nerve motor nucleus. During the process of electrophysiological monitoring, significant changes in mean arterial pressure can affect the obtained amplitude of FNMEP; therefore, in this study, mean arterial pressure was maintained stable before and after inhalation of sevoflurane, with no significant changes, to reduce the impact of arterial blood pressure fluctuations on electrophysiological monitoring.

During intraoperative monitoring of FNMEP in skull base surgery, stable FNMEP waveforms are not always detectable, especially when the patient presents with severe facial numbness or other symptoms of facial nerve dysfunction preoperatively. In the anesthetized state during surgery, it is challenging to obtain stable FNMEP waveforms in patients with skull base tumors using single-pulse stimulation; continuous multi-pulse stimulation is required[7,8,14]. In contrast, for patients with hemifacial spasm, the majority can achieve stable FNMEP waveforms through single-pulse stimulation even under general anesthesia, as confirmed by many researchers[1,9]. In this study, it was also demonstrated that out of 29 patients, 26 achieved stable FNMEP waveforms using single-pulse stimulation, accounting for 89.7%. However, the proportion of HFS patients obtaining FNMEP waveforms on the healthy side using single-pulse stimulation was significantly lower, at only 17.2%. This is believed to be related to the increased excitability of the facial nerve nucleus in patients with hemifacial spasm. If the excitability of the facial nerve nucleus is heightened in these patients, FNMEP may require a lower stimulation threshold to be activated. Therefore, this study further investigated the stimulation thresholds for FNMEP on the affected and healthy sides in HFS patients. The findings revealed that compared to the healthy side, relatively smaller stimulation on the affected side could still yield stable FNMEP amplitudes, with a statistically significant difference between the two groups. Additionally, although the stimulation threshold voltage on the affected side was lower than that on the healthy side, the FNMEP amplitudes obtained at the stimulation threshold were not lower than those on the healthy side, consistent with the results of many other studies. This further illustrates that the neural excitability of the facial nerve nucleus in HFS patients is significantly higher than that on the healthy side, supporting the central theory of hemifacial spasm.

4. Conclusion

The persistent compression of the facial nerve REZ area by the responsible vessel may be the initiating factor for hemifacial spasm. The continuous pulsatile compression of the facial nerve by the responsible vessel ultimately leads to increased excitability of the facial nerve nucleus, resulting in the occurrence of hemifacial spasm, which supports the central theory of hemifacial spasm.

Fund Program: Suzhou Industrial Park Health Talent Project(YQWS202202) and Suzhou Science and Technology Education Revival Health Youth Science and Technology Project (KJXW2020083), Suzhou Science and Technology Education Revival Health Youth Science and Technology Project (KJXW2023090), Suzhou Kowloon Hospital National nature Pre-research Project Fund(SZJL202401) .Suzhou key clinical disease diagnosis and treatment technology special(LCZX2023047).Suzhou science and technology development plan(SYSD2020078).

References

1. Wilkinson MF,Chowdhury T,Mutch WA,et al.Analysis of facial motor evoked potentials for assessing a central mechanism in hemifacial spasm [J].J Neurosurg, 2017,126

(2):379-385.
2. Ridding MC,Taylor JL.Mechanisms of motor-evoked potential facilitation following prolonged dual peripheral and central stimulation in humans[J].J Physiol,2001,537 (2:623-631.
3. Alvaro PL, Josep AS, et al.Responses to rapid- rate transcranial magnetic stimulation of the human motor cortex [J]. Brain, 1994, 117(4):847-858.
4. Verst SM,Chung TM,Sucena AC,et al.Comparison between the C5 or C6-Cz electrode assembly and C3 or C4-Cz assembly for transcranial electric motor activation of muscular response of the contralateral facial nerve [J].Acta Neurochir (Wien),2012, 154 (12):2229-2235.
5. Mertan PA,Morton HB.Stimulation of the cerebral cortex in the intact human subject [J].Nature,1980,285(5762):227.
6. MacDonald DB,Skinner S,Shils J,et al.Intraoperative motor evoked potential monitoring-a position statement by the American Society of Neurophysiological monitoring[J].Clin Neurophysiol,2013,124 (12): 2291-2316.
7. Pechstein U,Nadstawek J,Zentner J,et al.Isoflurane plus nitrous oxide versus propofol for recording of motor evoked potentials after high frequency repetitive electrical stimulation [J].Electroencephalogr Clin Neurophysiol,1998,108(2):175-181.
8. Dong CC,Macdonald DB,Akagami R,et al.Intraoperative facial motor evoked potential monitoring with transcranial electrical stimulation during skull base surgery [J].Clin Neurophysiol,2005,116(3):588-596.
9. Wilkinson MF,Kaufmann AM.Monitoring of facial muscle motor evoked potentials during microvascular decompression for hemifacial spasm:evidence of changes in motor neuron excitability [J].J Neurosurg,2005,103(1):64-69.
10. Matthies C,Raslan F,Scheweizer T,et al.Facial motor evoked potentials in cerebellopontine angle surgery:technique pitfalls and predictive value [J].Clin Neurol Neurosurg,2011, 113 (10):872-879.
11. Acioly MA,Liebsch M,de Aguiar PH,et al.Facial nerve monitoring during cerebellopontine angle and skull base tumor surgery:a systematic review from description to current success on function prediction [J].World Neurosurg,2013,80(6):e271-300.
12. Kawaguchi M,Inoue S,Kakimoto M,et al.The effect of sevoflurane on myogenic motor-evoked potentials induced by single and paired transcranial electrical stimulation of the motor cortex during nitrous oxide/ketamine/fentanyl anesthesia. J Neurosurg Anesthesiol, 1998,10 (3):131-136.
13. Chen Z.The effect of isoflurane and propofol on intraoperative neurophysiological monitoring during spinal surgery.J Clin Monit Comput,2004,18(4):303-308.
14. Legatt AD,Emerson RG.Motor evoked potential monitoring-it's about time.J Clin Neurophysiol,2002,19(5):383-386.