

# Surgical treatment strategies for hemimasticatory spasms

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*To the Editor:* Hemimasticatory spasm (HMS) is a relatively rare type of unilateral trigeminal nerve (TN) motor branch dysfunction, whose pathogenesis is currently unknown. The clinical manifestations of HMS are involuntary paroxysmal convulsions of the muscles innervated by the associated unilateral trigeminal motor branch. Recently, the academic community has proposed two etiologies for HMS: TN compression and central lesion theory.<sup>[1]</sup> Clinically, HMS must be distinguished from oromandibular dystonia, facial muscle spasm, multiple sclerosis pain spasm, and focal epilepsy. Generally, electromyography (EMG) can confirm the diagnosis. The current outcomes of multidrug and botulinum toxin injection treatments remain unsatisfactory. Only patients with cranial neurovascular conflicts have an excellent response to microvascular decompression (MVD) surgery.

We treated six patients with HMS (from May 2010 to February 2019) who were subjected to MVD and/or highly selective trigeminal rhizotomy (HSTR). All patients were administered carbamazepine, clonazepam, and baclofen; the total duration of pharmacological therapy lasted > 5 years. Early initiation of medications was found to be effective. As the disease progressed and the dosage of drugs was gradually increased within the safe dose range, the side effects became more obvious. After discontinuation of pharmacological treatment, symptoms remained unresolved or even deteriorated. Botulinum toxin type A was then injected, but facial nerve paralysis occasionally occurred. Finally, surgical interventions were selected. None of the patients presented with speech and swallowing changes or a history of trauma. Prior to the operation, the spasms had been occurring as frequently as a dozen of times a day, each lasting a few seconds to a few minutes and increasing with fatigue and emotional changes. Contractions and pain of the facial and temporal muscles had been reported. Patient two specified that exacerbations occurred after weather changes; when the pain was severe, it involved the forehead and the top of the same side of the head. Patient five had a contralateral nasal sinus

cyst, which was ruled out as the cause of the illness by otolaryngologist consultation. Patient six had right breast cancer and had underwent radical mastectomy, with four courses of chemotherapy and one course of radiotherapy. Drug-related effects were evaluated, with no obvious correlation between therapy drugs and dystonia. Moreover, no changes were observed in the masseter spasms during cancer treatment. The right masseter and temporal muscles were hypertrophic.

Preoperatively, there were no significant changes in the patients' physical examination, and their facial sensations and limb movements did not decrease. Additionally, there were no specific changes in antinuclear antibodies, rheumatoid factor, and so on, when they were admitted to the hospital. High discharges, a lack of normal rhythm, and plenty of motor unit potentials could be seen in the resting period on EMG. Electroencephalography results were negative. Time-of-flight magnetic resonance angiography (TOF-MRA) suggested the presence of neurovascular conflict or the possible source of blood vessel compression. Psychiatrist consultations excluded anxiety and depression.

Definite vascular compression on TOF-MRA and a strong willingness to undergo surgery were indicated for MVD. HSTR was recommended for patients without definite vascular compression. If neurovascular conflict was observed, a Teflon sponge was used to push the involved blood vessels away from the TN motor roots. If no vascular compression was detected, except for trigeminal neurolysis, neurophysiological monitoring was applied to identify the nerve motor branches corresponding to each muscle. To localize the target nerve fiber, contraction of the masseter muscle was induced by electrical stimulation (0.5 mA). The unaffected nerve fibers were preserved, and the affected fibers were cut off according to the amplitude of the compound muscle action potential (CMAP) from high to low, until CMAP decreased significantly or disappeared. It is suggested that the single nerve bundle that innervated the affected muscle alone should be cut off first. The nerve branches were then dissected in sequence until the

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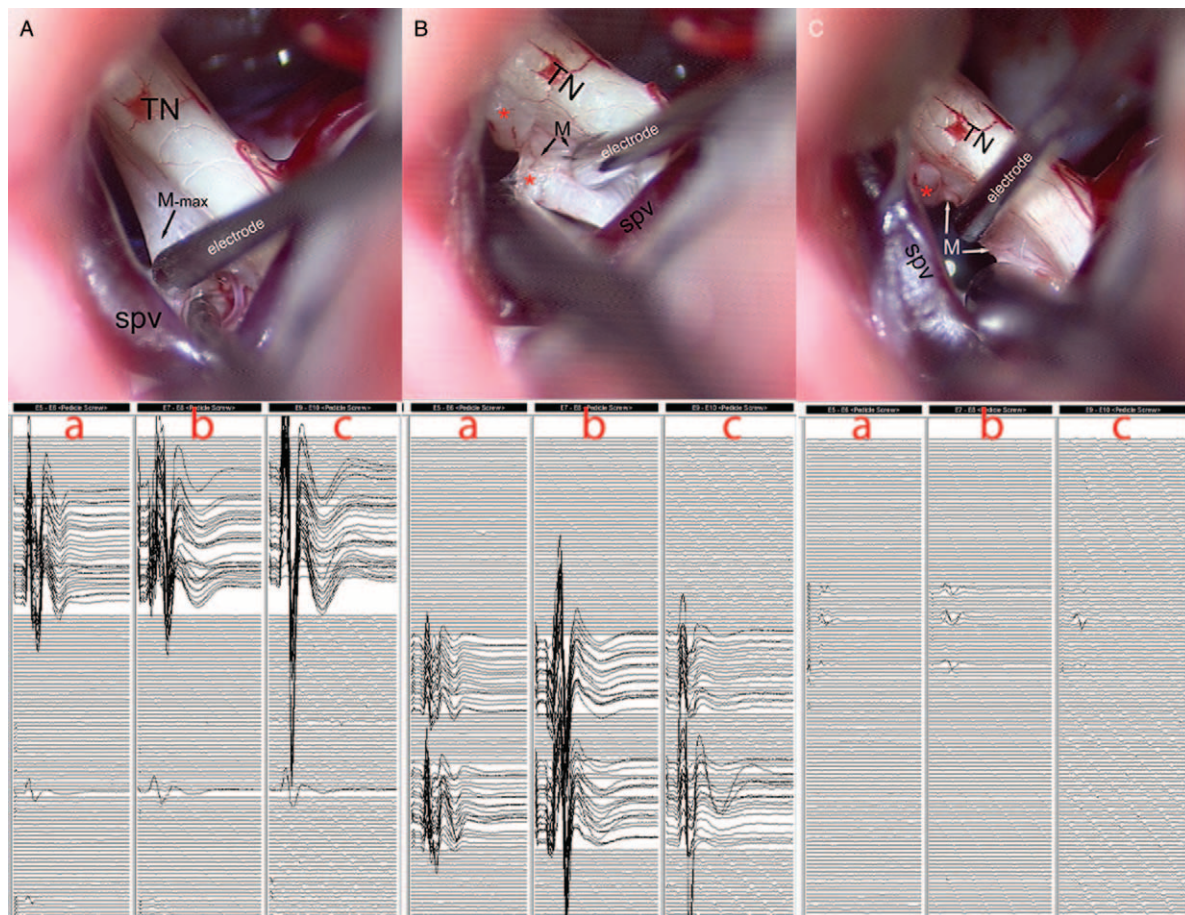
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**Figure 1:** (A) Intraoperative EMG indicated the largest branch of the TN motor root mainly innervating the masseteric muscle. (B) Following removing the largest branch, EMG showed high activity of the anterior temporal muscle. (C) EMG indicated no masseter or temporalis response after amputating the largest and secondary branches. \*Trigeminal motor root stump, (a) electromyographic response of posterior temporal muscle, (b) anterior temporal myoelectric response, (c) masseter myoelectric response. EMG: Electromyography; M: The trigeminal motor root; M-max: The maximum of the trigeminal motor root; SCA: Superior cerebellar artery; SPV: Superior petrosal vein; TN: Trigeminal nerve; Teflon: Teflon Padding.

myoelectric response of the muscles disappeared [Figure 1]. It should be emphasized that neurophysiological monitoring is required after complete cleaning of skeletal muscular relaxants. Bursts of motor unit potentials that correlated with involuntary spasms were detected in the rest stage. In three patients with HMS, TOF-MRA revealed definite vascular compression, and obvious indentations in the TN root were found intraoperatively. In two of the three, this occurred due to compression of the superior cerebellar artery and branches. Following MVD surgery, the spastic symptoms disappeared immediately. One patient had compression of the superior cerebellar and petrosal veins; symptoms were also significantly relieved after MVD. Although another patient's preoperative TOF-MRA suggested the possibility of vascular compression, it was intraoperatively found that the blood vessels were in contact with, but not compressing, the TN. HSTR, assisted by intraoperative EMG, and MVD was performed, and the patient's symptoms were partially relieved. For two patients, preoperative TOF-MRA showed avascular compression, thus HSTR with neurophysiological monitoring was conducted. One patient's spastic symptoms disappeared postoperatively, and the other's symptoms were significantly relieved [Supplementary Table 1, <http://links.lww.com/CM9/B14>]. The motor root usually has several

branches, ranging from three to eight branches. The involved nerve branch was often larger and showed CMAP of higher amplitude, which decreased after neurotomy. Postoperatively, the nerve branches showed a lower amplitude, and no CMAP was preserved.

Yoshida *et al*<sup>[2]</sup> published the cases of 18 patients with HMS who underwent the masseter stripping procedure, with an 80.2% overall average symptom improvement rate. However, 30% of patients still required additional botulinum injections. In recent years, some scholars have attempted endoscope-assisted avulsion of the masseter nerve to treat HMS<sup>[3]</sup>; these attempts resulted in good short-term outcomes with the advantages of minimal trauma and speedy recovery. However, preoperative TOF-MRA revealed that neuro-vascular conflict was absent at the root of the TN in some patients. For those with unclear or no neurovascular conflict, percutaneous procedures and stereotactic radiosurgery are common alternatives that are preferred due to their minimally invasive techniques. However, one of our patients refused repeated puncture treatment due to fear of complications, such as severe facial numbness, corneal keratitis, and dysesthesia. Neurolysis of the TN is typically performed in patients with avascular conflict, but one of our patients underwent HSTR assisted

by intraoperative EMG plus MVD. We conducted trigeminal neurolysis simultaneously, but the spasm did not disappear completely; thus, it is not reliable in the therapy of HMS. The HSTR was then considered due to the preservation of facial sensation. Postoperative neuromuscular electrical stimulation physiotherapy was finally used to reduce facial muscle atrophy. Upon follow-up, no obvious muscle shape atrophy was demonstrated in patients with a lower resection rate. Although those with a higher resection rate had mild masticatory muscle atrophy, the patients' spastic symptoms had disappeared, and there was increased satisfaction with therapy. Moreover, the masticatory power of the patients was not significantly weakened. However, this article has certain potential limitations, especially in the small number of recruited patients and the lack of quantitative standards for postoperative evaluation. Long-term follow-up is needed to evaluate the safety and efficacy of these methods.

#### **Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal.

The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

#### **Conflicts of interest**

None.

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