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Review article

The progress in epidemiological, diagnosis and treatment of primary hemifacial spasm

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ABSTRACT

Hemifacial Spasm is a neurological disorder characterized by persistent and rhythmic spasms of the facial muscles, significantly affecting the patient's quality of life. This condition can be classified into primary and secondary types; this article focuses on the characteristics of primary hemifacial spasm. Epidemiological studies indicate that the condition is more common in women, older adults, and individuals with posterior fossa stenosis or uneven blood flow dynamics, and is associated with gene expression related to demyelinating lesions.

In terms of diagnosis, magnetic resonance imaging can show the location of arterial or venous compression on the facial nerve on a macroscopic level and reveal white matter lesions on a microscopic level. Additionally, optimized electrophysiological techniques can determine the type of neural excitation disorder from both central and peripheral perspectives, thereby improving detection rates.

There are numerous treatment options available. Although early oral medications may have limited effectiveness, botulinum toxin injections can provide temporary relief. Future considerations include balancing injection costs with long-term efficacy. Microvascular decompression remains the preferred treatment approach and can be further optimized with endoscopic techniques. For refractory cases, alternative therapies such as facial nerve massage, radiofrequency techniques, rhizotomy, or acupuncture may be considered.

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ABBRATIONS

HFS	hemifacial spasm
pHFS	primary hemifacial spasm
MRI	magnetic resonance imaging
MVD	microvascular decompression
BoNT	botulinum toxin
PCF	posterior cranial fossa
CSFV	cerebrospinal fluid volume
VA	vertebral artery
3D-T2W MRI	three-dimensional T2-weighted magnetic resonance imaging
3D-TOF MRA	three-dimensional time-of-flight magnetic resonance angiography
DTI	Diffusion tensor imaging
FA	fractional anisotropy
MD	mean diffusivity
AD	axial diffusivity
RD	radial diffusivity
ILF	inferior longitudinal fasciculus
IFOF	inferior fronto-occipital fasciculus
RD	radial diffusivity
AMR	abnormal muscle response
LSR	lateral spread response
ZLR	Z-L response
EMG	electromyography
FCoMEP	Facial corticobulbar motor evoked potential
MEPs	Motor evoked potentials
sEMG	Surface electromyography
HD-sEMG	High-density surface electromyography
FNM	facial nerve massage
RFA	radiofrequency ablation
PRF	pulsed radiofrequency

1. Introduction

Hemifacial Spasm (HFS) is a common neurological disorder characterized by gradual progression among craniofacial movement disorders. It is characterized by painless, intermittent, and involuntary contractions of the facial muscles. Studies have shown that patients with HFS experience increased levels of anxiety, depression, and functional impairment, highlighting the significant impact of HFS on quality of life [1]. HFS can be classified into three types: typical, idiopathic, and secondary. Typical HFS is commonly caused by benign vascular compression of the facial motor nerve, occurring at or near the root exit zone of the brainstem [2]. This condition may occasionally be associated with increased intracranial pressure, posterior fossa flattening, and arachnoid adhesions [3,4]. Although idiopathic HFS presents with similar symptoms, its exact cause remains unclear. Our focus is on idiopathic and typical HFS, collectively referred to as primary HFS (pHFS). Secondary HFS is less common and is caused by identifiable underlying conditions or diseases, such as tumor masses or multiple sclerosis [2].

Given that pHFS resembles other facial disorders (e.g., Meige syndrome), preliminary diagnosis in clinical practice often relies on clinical features and patient history [5]. More precise diagnosis depends on magnetic resonance imaging (MRI) to rule out secondary causes, such as tumor, and to identify the location of neurovascular conflict [6]. Electrophysiological techniques are then used to determine the type of excitatory disturbance [7]. Although these methods have been validated through extensive clinical experience, differences in physician expertise and technical limitations may impact diagnostic accuracy, leading to variations in diagnostic approaches.

In terms of treatment, the currently recognized most effective method for treating pHFS is microvascular decompression (MVD); however, it is limited to patients with clear vascular compression, and reported side effects include facial paralysis, hearing loss, and cerebrospinal fluid leaks [8]. Botulinum toxin (BoNT), as a first-line conservative treatment, heavily relies on the clinical experience of the physician, including timing of injection, selection of target muscles, dosage, and formulation [9,10]. Importantly, BoNT injections can only alleviate symptoms and do not offer a definitive cure. Due to the need for frequent injections and high costs, many patients gradually lose confidence in the treatment [11,12].

The inadequacies in diagnosis and treatment are fundamentally due to a lack of comprehensive understanding of pHFS. This review explores the epidemiological characteristics of pHFS from the perspectives of gender differences, anatomical features, hemodynamics, and genetics, and summarizes the strengths and limitations of MRI and electromyography in clinical practice. Finally, we present methods for optimizing MVD and botulinum toxin injection, as well as potential alternative therapies. We hope this review will provide

insights for future research to deepen the understanding of pHFS, expand the diagnostic and therapeutic fields, and utilize multi-disciplinary approaches to improve patient quality of life.

2. Clinical manifestations

The characteristic feature of pHFS is the involuntary, progressive, irregular, spasmodic, or tonic contractions of the muscles innervated by the facial nerve [1]. Typically, these symptoms are entirely unilateral. Initially, many cases present with involuntary spasms starting in the eyelid region, which then progressively spread to other branches of the facial nerve, including the zygomatic branch (upper orbicularis oculi, zygomaticus major, zygomaticus minor), the temporal branch (lower orbicularis oculi, frontalis), the buccal branch (buccinator and orbicularis oris), the mandibular branch (mentalis), and the cervical branch (platysma) (Fig. 1A and B). This primarily manifests as difficulty in opening the eye during daily activities. In a few cases, patients may experience a typewriter-like tinnitus on the affected side, resulting in significant functional and lifestyle impairments (Fig. 1C and D) [13,14]. Apart from that, facial spasms may persist during sleep, albeit to a lesser degree. This condition can still result in a reduction in sleep quality and an increase in the frequency of awakenings [15]. Research has indicated that patients with pHFS are more prone to anxiety or depression compared to the general population. Moreover, emotional tension and anxiety can exacerbate facial spasms [16].

The differential diagnosis for facial spasms is extensive. Besides pHFS, other types include dystonia [5,17], synkinesias after facial nerve paralysis [18–20], psychogenic facial spasm [21], tardive dyskinesia [22,23], facial myoclonus or myorhythmia [24,25], hemimasticatory spasm [26–28], facial motor tic [29,30], and facial myokymia [31]. Therefore, a comprehensive evaluation that integrates the patient's clinical features, medical history, and diagnostic tests is essential for accurate differentiation (Table 1).

3. Epidemiological characteristics

3.1. Sexual characteristics

According to epidemiological studies in the United States, HFS occur in approximately 14.5 out of every 100,000 women and 7.4 out of every 100,000 men [32]. Although there is a lack of similar epidemiological data in the Asian region, relatively large sample

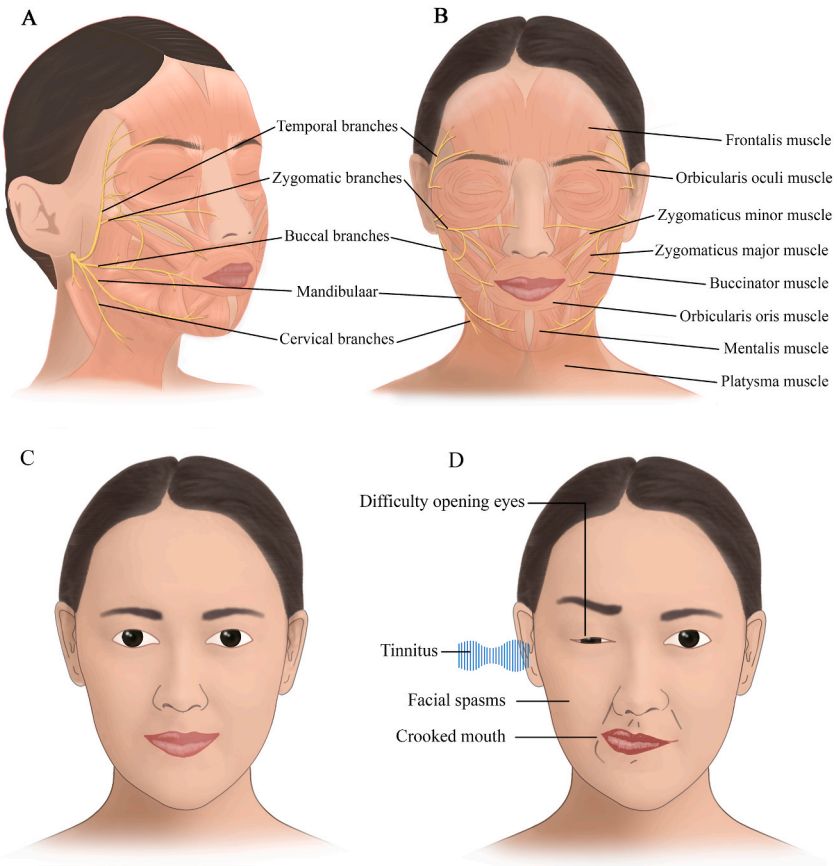

Fig. 1. A, Healthy individuals' facial features; B, Clinical features of patients with hemifacial spasm; C, Branches of the facial nerve, viewed from the lateral aspect; D, Muscles innervated by the branches of the facial nerve, viewed from the front.

Table 1
Differential diagnosis of primary hemifacial spasm.

Diseases	Primary etiology	Differentiating features	Primary treatment
Dystonia [5,17]	Abnormalities in the basal ganglia and motor cortex function (blepharospasm, oromandibular dystonia, Meige syndrome)	• Blepharospasm Clinical features: Increased blinking frequency gradually developing into bilateral, synchronous eyelid contractions. • Oromandibular dystonia Clinical features: Involuntary contraction of masticatory and/or tongue muscles. • Meige syndrome Clinical features: Manifesting as a combination of eyelid spasm and lower jaw muscle tension disorders; this condition may initially be limited to either eyelid spasm or lower jaw muscle tension and then spread to other muscles.	1. Oral medications (e.g., anticholinergic drugs, benzodiazepines) 2. Botulinum toxin injection 3. Deep brain stimulation
Synkinesias after facial nerve paralysis [18–20]	Abnormal regeneration of the facial nerve leading to atypical muscle innervation	1. Facial paralysis history 2. Clinical feature: Persistent facial weakness 3. Needle electromyography: Polyphasic reinnervation potentials 4–10 months after the onset of the lesion.	1. Physical therapy (e.g., neuromuscular retraining) 2. Botulinum toxin injection 3. Surgical treatment (e.g., selective neurotomy, selective myectomy)
Psychogenic facial spasm [21]	Psychological disorders	1. Clinical features: Early onset, asymmetric bilateral movements, downward pull of the mouth corner on the same side, and isolated involvement of the lower face. 2. Lack of the "other Babinski sign'':elevation of the eyebrow caused by contraction of the frontalis muscle ipsilateral to the facial spasm.	1. Psychological intervention 2. Oral medications (e.g., benzodiazepines and selective serotonin reuptake inhibitors)
Tardive dyskinesia [22,23]	Long-term use of antipsychotic medications	1. Psychiatric history 2. Medication history 3. Clinical features: Involves areas beyond the face, with bilateral irregular movements.	1. Reduce/stop/change antipsychotic medications 2. Oral medications (e.g., VMAT2 inhibitors)
Facial myoclonus/ myorhythmia [24,25]	Autoimmune response caused by anti-NMDA receptor antibodies, usually associated with ovarian teratoma (NMDA Encephalitis)、gram-positive intracellular bacillus Tropheryma whipplei (Whipple's Disease)	• NMDA Encephalitis 1.Clinical features: Associated with seizures, psychiatric symptoms, and altered consciousness. 2.Immunohistochemistry: Utilized to detect NMDA receptor antibodies. 3.Electroencephalogram: Epileptiform activity. •Whipple's Disease 1.Clinical features: Characterized by multisystem symptoms including arthralgia, arthritis, diarrhea, and cognitive impairment. 2.Duodenal Biopsy: PAS staining can be employed to identify the pathogenic bacteria.	• NMDA Encephalitis: Immunotherapy • Whipple's Disease: Antibiotic therapy
Hemimasticatory spasm [26–28]	Trigeminal nerve lesions	Clinical features: Primarily involves the masticatory muscles, and includes additional features such as facial hemiatrophy, connective tissue disease symptoms like scleroderma, and skin changes such as morphea.	1. Oral medications (e.g., antiepileptic drugs) 2. Botulinum toxin injection
Facial motor tics [29,30]	The brain regulates the impulses generated by negative emotions	Clinical features: Sudden and brief facial movements that intensify with the escalation of negative emotions, often accompanied by a prodromal sensation.	Manage emotions
Facial myokymia [31]	Fatigue, stress, caffeine overuse, lack of sleep, or eye irritation	1. Clinical features: Sudden, brief facial movements without preceding sensory sensations. 2. Needle electromyography: Single motor unit potentials with bursts ranging from 5 to 150 Hz.	Adjust lifestyle habits

sizes from Asian countries such as China, India, Japan, and the Philippines indirectly indicate a higher incidence rate of HFS in Asia. The gender distribution of pHFS appears to be more inclined towards females, with a male-to-female ratio of approximately 1:1.8. The average onset age is around 40–50 years old, and the risk of developing the condition significantly increases with age [33,34]. Given the onset age of around 50 years and the coinciding menopausal period, factors associated with sex hormones have been demonstrated to influence myelin formation and neuroinflammation. Perhaps further experiments could be designed to explore the pathological mechanisms related to gender and age [35].

3.2. Anatomical characteristics

In the Asian population, the compact configuration of the posterior cranial fossa results in a higher concentration of cranial nerves and vascular structures, increasing the likelihood of neurovascular compression. This is considered a potential factor contributing to the elevated incidence of pHFS in Asians compared to regions such as Europe and America. Advanced three-dimensional magnetic resonance imaging studies have shown that individuals with pHFS have notably increased crowding in the posterior cranial fossa (PCF) compared to those without, accompanied by smaller volumes of cerebrospinal fluid volume (CSFV) in the posterior cranial fossa. Moreover, the PCF crowding index in females is significantly higher than in males, providing an explanation for the prevailing gender distribution among patients [36]. The flattened configuration of the PCF signifies a more restricted space for nerves and blood vessels. This is considered a significant prognostic factor for poor treatment outcomes and diminished long-term efficacy following MVD. Additionally, it increases the risk of pHFS (Fig. 2A and B) [4].

3.3. Hemodynamic characteristics

Based on studies evaluating the development and symmetry of bilateral vertebral arteries (VAs), asymmetry of VAs is considered a normal phenomenon. Generally, the average diameter and velocity of the left VA are higher than those of the right [37]. However, recent research suggests that this asymmetry may pose a risk factor for posterior circulation stroke, as the side with lower flow is more prone to vascular pathology [38]. This dominant vertebral artery is particularly observed in cases of pHFS. Studies indicate that hemodynamic differences in bilateral VA blood flow may cause the basilar artery to shift or twist towards the side with lower flow [39,40]. Computational fluid dynamics analyses reveal that the curvature of vertebral arteries creates pressure differentials between the walls of opposing sides, and as the curvature angle and degree increase, so do the differences in wall shear stress [41]. Progressive displacement and curvature of VAs may ultimately affect the anatomical positions of adjacent arteries such as the posterior inferior cerebellar artery and anterior inferior cerebellar artery, thereby directly or indirectly compressing the facial nerve and potentially triggering pHFS [40]. These also provide an explanation, to some extent, for the phenomenon that the incidence of facial nerve spasms is higher on the left side than on the right. Last but not the least, it is crucial to note that factors such as hypertension, high cholesterol levels, and smoking are believed to be significant contributors to the formation of differences in vascular wall shear stress. These factors accelerate vascular deformation by damaging the vascular wall [42]. Coincidentally, the most commonly observed comorbidities of pHFS are hypertension and hyperlipidemia [43]. Future research could explore how these comorbid conditions interact with the pathophysiology of pHFS to potentially develop targeted therapeutic strategies.

3.4. Genetic characteristics

Family studies on pHFS suggest that in several families, idiopathic pHFS appears to follow an autosomal dominant inheritance pattern, with symptoms consistent with pHFS. This indicates a hereditary etiology in certain cases [44–46]. Interestingly, a high prevalence of pHFS (29.6 %) has been reported in a large family affected by Charcot-Marie-Tooth disease type 1B [45]. Mutations in the MPZ gene in CMT1B patients can lead to demyelination or axonal changes in nerve conduction [47]. Overall, familial genetic studies of pHFS remain limited. As a supplementary clue for exploring its genetic patterns, some studies have identified the expression of potential pathogenic genes as risk factors for pHFS. Clinical observations suggest that pHFS with APOE ε4 expression tend to experience more rapid disease progression, possibly due to the detrimental effects of APOE ε4 on neuronal and myelin regeneration, which may accelerate disease progression and increase the risk of abnormal signal transmission [48,49]. Furthermore, experimental research using pHFS SD rats has found that overexpression of Nav1.8, encoded by the SCN10A gene, may be associated with abnormal muscle responses [50,51]. Upregulation of Nav1.8 expression, particularly when axons or myelin are damaged, leads to persistent

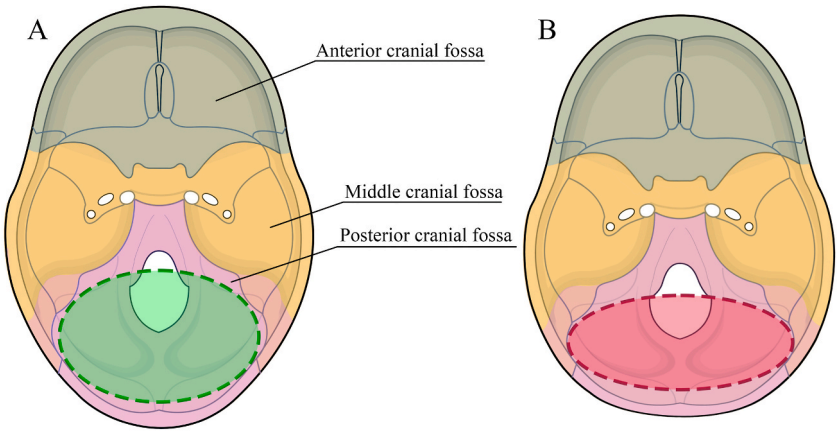

Fig. 2. A, Convex-shaped posterior cranial fossa with favorable prognosis; B, Flat-shaped posterior cranial fossa is associated with poor outcomes.

depolarization of neuronal membranes and increased neuronal excitability, contributing to facial spasms [52–55]. However, existing research is constrained by limited sample sizes and flaws in study design (e.g., the applicability of results from animal model studies), resulting in insufficient evidence to definitively ascertain the genetic mechanisms of pHFS. Consequently, further research is necessary to deepen understanding in this field.

4. Diagnostic studies

The diagnosis and assessment of pHFS usually include two main types of tests: (1) Neuroimaging to exclude secondary causes such as space-occupying lesions from tumors and to identify the location of neurovascular conflict (Table 2). (2) Electrophysiological techniques are used to determine the nature of the neural excitability disorder (Table 3). These methods are widely recognized for their efficacy in diagnosing pHFS.

4.1. Neuroimaging

High-resolution three-dimensional T2-weighted MRI (3D-T2W MRI) is a widely utilized imaging technique in both clinical practice and research fields. This technology offers high-quality images, enabling doctors and researchers to closely observe and analyze the anatomical structures of the nervous and vascular systems [56]. However, the main limitation of these imaging techniques lies in their inability to differentiate between arterial and venous signals, as well as the signal differences between vessels and nerves.

With the development of three-dimensional time-of-flight magnetic resonance angiography (3D-TOF MRA), preoperative vascular visualization has become more convenient. This technique allows arterial blood to appear as high signal, while cerebrospinal fluid appears as low signal. Additionally, signals from the facial nerve and brainstem fall between the two, aiding in tracking the origin of responsible vessels [57]. However, this imaging method suppresses the signals of stationary tissues, making it less favorable for clear visualization of neural structures. Furthermore, it is influenced by blood flow velocity and direction, resulting in poor visualization of smaller diameter and tortuous vessels [58]. Additionally, after craniotomy, the displacement of brain tissue, nerves, and vessels, as well as the release of cerebrospinal fluid, may alter the relationships of surrounding structures, potentially leading to diagnostic errors [8]. Therefore, utilizing three-dimensional fused contrast-enhanced magnetic resonance angiography and 3D-T2W MRI can provide complementary advantages in identifying anatomical structures and responsible vessels, offering higher accuracy and sensitivity in diagnosing neurovascular compression syndromes, especially the combination of 3D-T2-weighted CISS and 3D-TOF has significant application value in the diagnosis of atherosclerosis [59,60].

Since the aforementioned imaging sequences cannot clearly visualize venous vessels, consideration should be given to performing three-dimensional thin-slice T1-weighted scans after injection of gadolinium-based contrast agents [61]. However, due to the fewer occurrences of venous compression of the facial nerve and the smaller veins around the facial nerve, imaging is challenging, and this imaging method is not widely used in the examination of patients with pHFS.

Diffusion tensor imaging (DTI) is a neuroimaging technique that measures the diffusion direction and velocity of water molecules within tissue, revealing the directional orientation and connectivity patterns of neural fiber bundles. Coupled with TBSS technology, which obviates the need for smoothing and nonlinear registration, DTI is adept at elucidating changes in the microstructure of white matter [62]. It has found extensive application in research on Parkinson's disease, Alzheimer's disease, psychiatric disorders, and trigeminal neuralgia [63–65]. Key parameters include fractional anisotropy (FA), mean diffusivity (MD), axial diffusivity (AD), and radial diffusivity (RD). Typically, FA reflects the integrity of white matter [63], MD correlates with cellular degeneration, while AD and RD are associated respectively with axonal and myelin sheath degeneration or damage [64,65]. Studies have shown that in patients with pHFS on the affected side, FA is significantly lower than on the healthy side, while the MD is significantly higher [66]. This indicates that long-term vascular compression in pHFS leads to degeneration of nerve structures and changes in cells, consistent with the pathological mechanism of focal demyelination involved in pHFS [67,68].

Furthermore, chronic pHFS can result in widespread damage to white matter integrity, particularly in the inferior fronto-occipital fasciculus (IFOF) and inferior longitudinal fasciculus (ILF), manifested by reduced FA and increased RD and MD on DTI. Additionally, the higher the RD value of IFOF and ILF, the more severe the facial muscle spasms [69]. These white matter connections are involved in multidimensional experiences of facial movement, facial sensation, and emotional regulation within gray matter regions, providing a

Table 2
Neuroimaging for predicting neurovascular compression[a].

Neuroimaging	Advantages	Limitations
3D-T2W MRI [56,59,60]	Provide detailed anatomical information on the relative positions of nerves and blood vessels.	1. Unable to distinguish arterial and venous signals. 2. Unable to differentiate between signals of blood vessels and nerves.
3D-TOF MRA [57–60]	Trace the source of the responsible blood vessel	Anatomical information is poorly displayed.
Color-coded T1-weighted MRI [61]	Venous compression	The veins around the facial nerve are small, making imaging difficult.
DTI [62–69]	Reflect the degeneration of neural structures and changes at the cellular level.	Reflect only the structural changes at the microscopic level.

[a] 3D-T2W MRI,three-dimensional T2-weighted imaging; 3D-TOF MRA,three-dimensional time-of-flight magnetic resonance angiography; DTI, Diffusion tensor imaging.

Table 3
EMG for recordding neuroelectrophysiological activity[a].

EMG	Definition	Advantages	Limitations
AMR[7, 71–73]	High-frequency electrical stimulation of a branch of the facial nerve records pathological electromyographic responses in other branches of the facial nerve, with a latency of approximately 10 ms.	High specificity in pre- and post-MVD testing	1. Difficulty in obtaining a stable positive response during surgery 2. The disappearance or persistence of LSR delay
ZLR [3, 76–79]	The current passing through the arterial wall in contact with the facial nerve transmits to the facial nerve, with a latency of approximately 7 ms.	High specificity in arterial compression diagnosis	1. Limited diagnostic capability for compressing the arachnoid and veins 2. Possibility of false positives
FCoMEP [80–85]	Assess the integrity of the brainstem motor pathways	Assessment of facial nerve nucleus group excitability	More prospective studies are needed to validate its effectiveness as a predictive indicator.
HD-sEMG [86–88]	Record neuromuscular electrical activity by using a large number of closely spaced electrodes on the skin surface.	1. Provide high-resolution spatiotemporal information 2. Diagnose the pathological states of neuromuscular conditions	More prospective studies are needed to validate its effectiveness as a predictive indicator.

[a] EMG,electromyography; AMR,abnormal muscle response; LSR, lateral spread rresponse; MVD,microvascular decompression; ZLR,Z-L response; FCoMEP, Facial corticobulbar motor evoked potential; HD-sEMG, High-density surface electromyography.

central explanation for the influence of negative emotions on pHFS. However, evidence from DTI studies regarding microstructural changes associated with pHFS remains very limited and is currently in the exploratory stages [70]. In the future, DTI may serve as an assessment tool to investigate the impact of factors such as age of onset, disease duration, and treatment outcomes on pHFS.

Although various sequences of MRI technology have been validated in preoperative diagnosis of pHFS, data on neurovascular compression in asymptomatic individuals are still scarce. Deep et al.'s study demonstrated that neurovascular contact of the facial nerve could be identified on MRI in approximately 37 %–53 % of asymptomatic patients [6]. However, due to limitations in physician experience and technique, such subtle or difficult-to-detect vascular contacts are almost always overlooked. Future research aimed at optimizing MRI imaging sequences, improving imaging evaluation methods for neurovascular relationships, and collecting more postoperative follow-up data from patients to further enhance diagnostic accuracy.

4.2. *Electrophysiological techniques*

Abnormal muscle response (AMR) or lateral spread response (LSR) is a distinct electrophysiological reaction observed in patients with pHFS, characterized by an abnormal muscle response in facial nerve branches other than the stimulated one [71]. Intraoperative monitoring of AMR can assist surgeons in pinpointing the causative vessel, evaluating surgical decompression efficacy, and minimizing procedure-related risks [72]. However, some pHFS with typical symptoms may not display AMR in preoperative facial electromyography, potentially due to variations in surgical techniques or stimulation points impacting AMR detection rates [7,73]. To address this challenge, innovative methods like centrifugal stimulation and multi-branch monitoring have been introduced, significantly enhancing the detection of abnormal muscle responses. These advancements have led to improved accuracy in forecasting the outcomes of MVD and in fine-tuning the decompression effects [74,75]. Although these methods have potential, their effectiveness still relies solely on the analysis of AMR waveform characteristics.

Z-L response (ZLR) serves as a novel intraoperative electromyography (EMG) technique, transmitting current through stimulation of the arterial wall in contact with the facial nerve, playing a unique role in MVD [76]. The stability of the AMR is sometimes compromised, and AMR may disappear during surgery when the facial nerve is not fully decompressed. Research indicates that combined monitoring of AMR and ZLR provides more valuable information compared to AMR alone, aiding surgeons in identifying compressive vessels, particularly the causes of arterial compression [77]. Moreover, when AMR is unavailable or unstable, ZLR may be the sole useful intraoperative EMG indicator in MVD [78]. However, even without vascular compression, stimulation of the arterial wall can still elicit ZLR waves [79]. ZLR wave monitoring has certain limitations, especially in cases where the etiology involves compression by arachnoid or venous structures [3], highlighting the need for further refinement of related ZLR techniques in the future.

Facial corticobulbar motor evoked potential (FCoMEP) is a novel method developed based on recording Motor Evoked Potentials (MEPs) in limb muscles to assess the integrity of motor pathways through the brainstem. This method is applied in various types of neurosurgical procedures involving facial nerve or the facial nuclei [80]. Given that the pathophysiological mechanism of pHFS primarily involves abnormalities in the facial nerve and the facial nerve nucleus, monitoring the electrophysiological changes in related pathways using FCoMEP during MVD surgery may be highly useful. Wilkinson et al. conducted a study comparing the effects of desflurane on MEPs in the spastic and non-spastic sides of patients undergoing MVD for pHFS. The results showed that the success rate of facial stimulation was higher on the spastic side (84 %) compared to the non-spastic side (74 %), and the inhibitory effect of inhaled anesthesia on facial motor neuron excitability was significantly less pronounced on the spastic side [81]. This finding validates that facial motor neurons in pHFS are in an excited state, requiring less depolarizing stimulation to reach the threshold. Similar research results were obtained in studies involving active testing with high-osmolarity glycerol injections into the root entry zone and

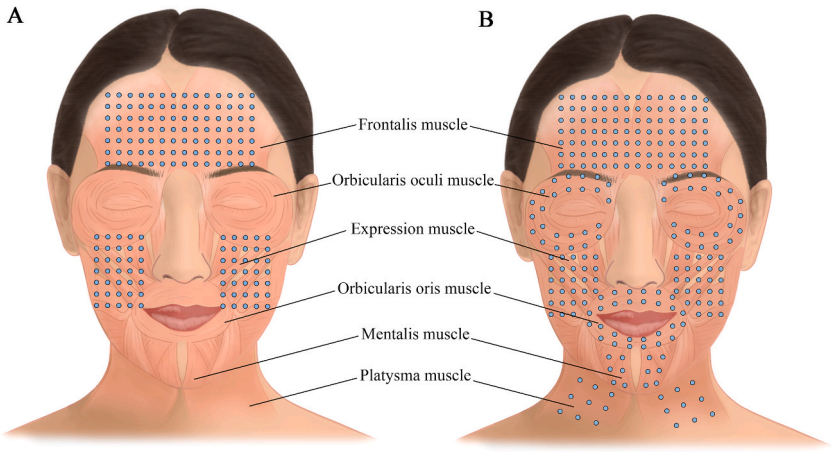

Fig. 3. A, Pattern and characteristics of facial nerve activity analyzed by high-density surface electromyography; B, High-density electromyography distributed based on the areas affected by primary hemifacial spasm.

transcranial magnetic stimulation in awake patients with pHFS [82,83]. The Blink Reflex, as one of the brainstem pathway monitoring modalities, has demonstrated to be a more reliable clinical indicator compared to the LSR during or after MVD [84,85]. These studies not only provide evidence for increased excitability in central facial structures but also offer new predictive indicators for the decompression effects of surgical interventions, warranting further investigation.

Surface electromyography (sEMG) measures the patterns, tension, and coordination of muscle electrical activity by placing electrodes on the skin surface. As a non-invasive form of electromyography, sEMG not only captures muscle discharges in patients with pHFS but also simultaneously detects the AMR [86]. High-density surface electromyography (HD-sEMG), due to its increased electrode density and spatial resolution, can provide pathological information related to neuromuscular conditions similar to needle electromyography [87]. This includes conditions such as muscle fatigue, motor neuron disease, neuropathies, myopathies, spontaneous muscle activity, and Motor Unit discharge rates. In recent years, researchers like Cui et al. have utilized HD-sEMG and facial topography to quantify spatial activation characteristics of facial muscles under different movement patterns in patients with facial paralysis, analyzing the correlation between facial paralysis and electromyography after facial nerve injury (Fig. 3 A) [88]. However, this experimental method does not align with anatomical considerations and cannot fully utilize the advantages of HD-sEMG in disease diagnosis. Therefore, we are considering adjusting the configuration of HD-sEMG based on the distribution of muscles affected by pHFS (Fig. 3 B). This type of objective, non-invasive technology could potentially replace invasive electromyography and be used in daily diagnostics to discover more readily identifiable “digital biomarkers".

5. Treatment

5.1. Oral medications

Based on previous experiences, several cases of pHFS have been reported to be treated with oral medications in clinical practice, including antiepileptic drugs such as carbamazepine, gabapentin, clonazepam, levetiracetam, and baclofen, a muscle relaxant. However, a recent double-blind randomized controlled trial observed that patients treated with clonazepam at doses of 0.25 mg, 0.5 mg, 0.75 mg, and 1 mg twice daily for four weeks did not show significant improvement in symptoms [89]. Considering the potential side effects and treatment efficacy of these medications, they are generally used only in early or mild cases. In the future, large-scale randomized clinical trials are still needed to develop more effective treatment medications and provide more comprehensive evidence (Fig. 6 A).

5.2. Botulinum toxin

Compared to oral medications Botulinum toxin (BoNT) comes in seven serotypes, encompassing a variety of different products. In the treatment of facial spasms, BoNT-A is more common. Compared to oral medications, BoNT has seven serotypes. In the treatment of facial spasm, BoNT-A is more commonly used, with Onabotulinum toxin-A (ONA) and abobotulinum toxin-A (ABO) being the most widely applied types [90]. Research indicates that the concentration and dosage of the product have little significant correlation with the effectiveness of the treatment, but are more related to the duration. Studies indicate that while different products have certain dose conversion ratios, fundamentally they are independent. The concentration and dosage of products are more relevant to the duration of effectiveness rather than the significance of treatment outcomes [9,91]. Therefore, in devising treatment plans, it is advised to avoid dose conversion between different types of BoNTs and instead tailor adjustments based on individual patient factors.

BoNT injection primarily targets facial muscles, with the orbicularis oculi muscle being one of the most affected muscles, divided into orbital and eyelid portions, with the latter further divided into preseptal and pretarsal subparts. Research suggests that compared to traditional preseptal injection of the orbicularis oculi muscle, Pretarsal injection exhibits longer-lasting treatment effects and higher patient satisfaction (Fig. 4A and B). This difference may be attributed to Pretarsal subpart having more skeletal muscle fibers, higher nerve density, and being more susceptible to the effects of BoNT-A [10,92]. However, the rich capillary network in the preseptal subpart may increase the risk of adverse events such as bleeding and congestion. Reliable evidence supporting the most suitable

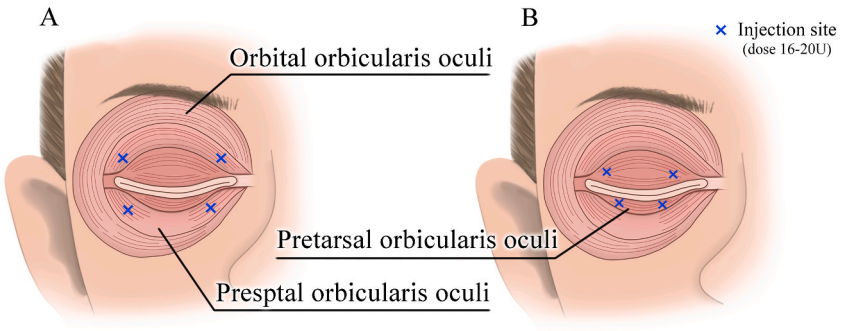

Fig. 4. A, Pretarsal injection increase the risk of adverse events such as bleeding and congestion; B, Pretarsal injection exhibits longer-lasting treatment effects and higher patient satisfaction.

injection method for other areas is currently lacking.

Overall, most patients experience significant improvement after BoNT injection, with effects lasting for 3–4 months. Over time, patients' conditions may gradually worsen, often necessitating a gradual increase in dosage to maintain treatment effects [11]. Known side effects include temporary facial weakness, eyelid ptosis, and drooling, which typically resolve completely within a few weeks. Long-term side effects may include facial asymmetry but can be addressed by injecting unaffected areas [93]. However, this may require higher doses and treatment costs, with the impact on patients' facial perception and quality of life remaining uncertain (Fig. 6 B).

In general, the effectiveness of BoNT in the treatment of HFS is relatively limited. Due to the lengthy treatment cycles and high costs involved, many patients gradually lose confidence in treatment [12]. Future research should focus on achieving BoNT detoxification and enhancement, considering factors such as muscle type, characteristics, as well as onset and duration of treatment. By integrating large-scale data and real-world clinical practice, strengthening individualized treatment strategies, and balancing the relationship between treatment effectiveness and costs, it is hopeful to provide more satisfactory treatment options for patients.

5.3. MVD

Compared to oral medications and BoNT injections, MVD is the most effective treatment for pHFS. The actual decompression process typically involves continuous monitoring of facial electromyography and auditory evoked potential nerve monitoring. It is performed by making an incision behind the occipital bone, exposing the cerebellopontine angle region, inspecting the compressed vascular area, and placing a piece of Teflon sponge between the vessel and brainstem (Fig. 5) [94]. More complex surgeries may require techniques such as Teflon transposition or biomedical adhesive suspension to completely relieve compression [95,96]. The objective of the surgery is to alleviate compression of the neurovascular structures, with an average effectiveness ranging from 84 % to 92 % [8,34,97,98].

In terms of complications, studies have reported that common short-term complications after surgery include brainstem ischemia, meningitis, and cerebrospinal fluid leakage. By contrast, only a few patients experience long-term persistent complications, such as hearing loss and facial paralysis (Fig. 6C) [8]. However, it is reassuring that fully endoscopic MVD has been validated as safe and feasible for treating pHFS. This approach offers clearer visualization of neurovascular conflicts, although it initially presented some challenges, such as susceptibility to blood contamination, lack of three-dimensional information, and requiring a longer learning curve [99].

In recent years, the latest research has reported that endoscopic assistance significantly improves surgical success rates (97 % vs. 89 %), lowers recurrence rates (0.3 % vs.5.7 %), and reduces the probability of complications (12 % vs. 27 %) [100]. These findings indicate the potential of this technique in pHFS treatment, and with technological advancements, its advantages may become more prominent.

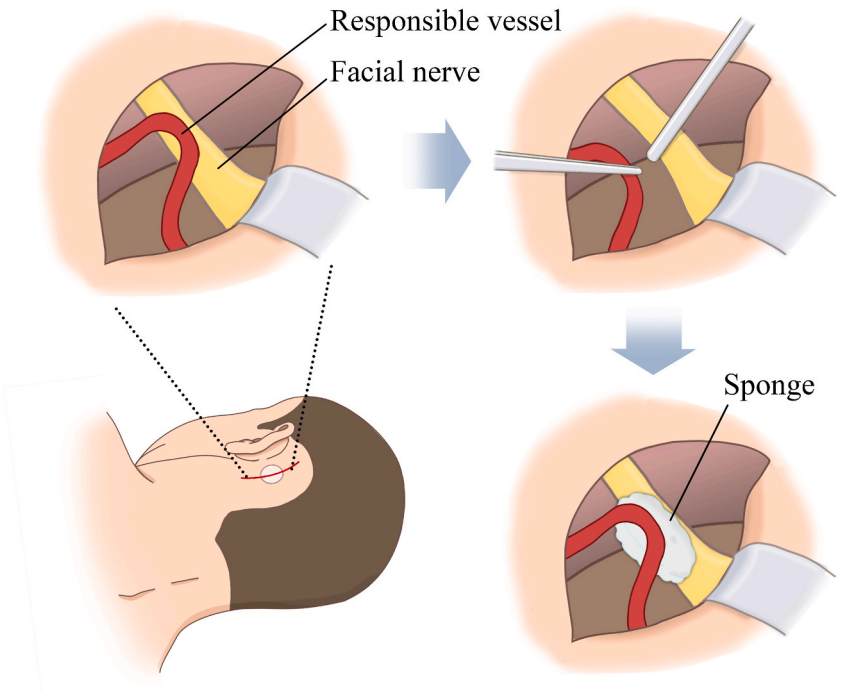

Fig. 5. The various stages of microvascular decompression.

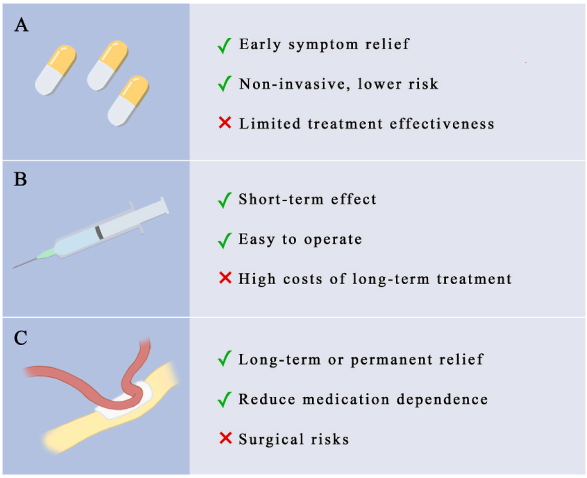

Fig. 6. A, Oral medication; B, Botulinum toxin injection; C, Microvascular decompression.

6. Alternative treatments

6.1. *Facial nerve massage*

When patients fail to achieve sufficient relief with medication and MVD is deemed inappropriate for repeat procedures, facial nerve massage (FNM) is undertaken as an emergency intervention. Under general anesthesia, a minimally invasive procedure is performed to access the cerebellopontine angle. After confirming the absence of vascular compression on the facial nerve, specific locations on the facial nerve are gently rubbed or massaged using miniature dissectors. Studies indicate that, in most patients with pHFS who lack evidence of intracranial vascular compression and have undergone prior MVD, FNM results in persistent and at least partial relief of spasms, with a relatively low complication rate [101]. However, the degree of facial nerve damage caused by the massaging process cannot be quantified, along with potential long-term adverse effects. Disease-specific assessments performed before and after the procedure may enhance understanding of the mechanisms underlying FNM in these patients, gradually promoting its wider application.

6.2. *Radiofrequency techniques*

Radiofrequency techniques for the treatment of pHFS mainly include radiofrequency ablation (RFA) and pulsed radiofrequency (PRF) (Fig. 7) [102,103]. Percutaneous RFA is a minimally invasive therapy that utilizes high-frequency electrical currents to heat specific nerves or tissues, effectively alleviating or eliminating pain sensations. It is a minimally invasive and successful treatment method for short-term and long-term outcomes in trigeminal neuralgia [101]. Studies report that, following CT-guided RFA, 91 % of pHFS (48/53) completely eliminate symptoms. The main postoperative complication is mild to moderate facial paralysis, but the majority recover within one month [103]. CT-guided awake RFA holds promise as an effective and cost-effective alternative treatment for pHFS. In contrast, pulsed PRF operates through short bursts of high-frequency energy directed at nerve tissues, providing effective pain relief without causing significant damage. In a case where PRF was used to treat recurrent pHFS following MVD, the patient experienced significant therapeutic benefits without adverse effects such as sensory numbness or facial muscle weakness [102]. It is important to note that PRF typically offers only temporary relief, whereas RFA may provide a more lasting solution. For patients with pHFS who are resistant to pharmacological treatment, different radiofrequency techniques can be selected based on specific clinical needs [104]. However, the clinical application of radiofrequency technologies remains limited, and their efficacy and safety require further investigation.

6.3. *Rhizotomy*

Rhizotomy is a surgical procedure that involves cutting or removing part or all of a nerve root to reduce nerve conduction and alleviate related symptoms such as spasms or pain [105,106]. Due to the higher risk of facial weakness or paralysis, rhizotomy is generally less preferred than MVD for treating pHFS [105]. However, drawing from the treatment experience of trigeminal neuralgia, which also involves vascular compression, when MVD alone is unsuitable—such as when the offending vessel is a vein or no clear vascular compression is identified—consideration may be given to combining MVD with rhizotomy or using rhizotomy alone [106, 107]. Currently, there is insufficient data on the short-term and long-term clinical efficacy of rhizotomy, either alone or in combination, for treating pHFS.

	RFA	PRF
Temperature	60°C - 100°C	42°C
Output	Continuity	Intermittent
Mechanism	Destroy tissue	Alter nerve function

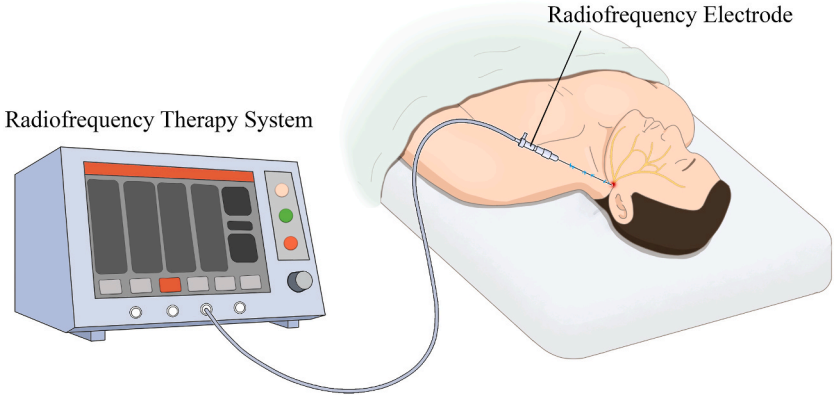

Fig. 7. Radiofrequency techniques for treating primary hemifacial spasm.

6.4. Acupuncture

Acupuncture, a traditional healing method in China, is gaining increasing attention. In Traditional Chinese Medicine, HFS fall under the category of "Spasm Syndrome", involving factors such as Qi and blood imbalance, liver wind disturbance, and kidney deficiency. Some studies have reported the effectiveness of acupuncture in treating pHFS, including methods like gold thread acupuncture [108]. Therefore, we recommend considering acupuncture as an alternative treatment option for patients who prefer conservative management and are dissatisfied with the effects of BoNT. However, the evidence supporting the efficacy of acupuncture treatment is not comprehensive, and the underlying mechanisms.

7. Conclusions

The pathophysiological mechanisms of pHFS are diverse and complex. Current research indicates that factors such as age, gender, anatomical structures, blood components, and genetic variations can influence the presentation of pHFS, leading to variability in clinical manifestations among individuals. However, making a definitive diagnosis remains challenging with current scientific knowledge and technological means. Future diagnostics for pHFS may rely on more objective and precise methods, including the use of advanced imaging techniques and improved electrophysiological technologies.

In terms of treatment, injections of BoNT remain the preferred conservative approach due to their high tolerability and low risk profile. Additionally, alternative therapies such as facial nerve massage, radiofrequency techniques, rhizotomy, and acupuncture have shown to be effective. Nonetheless, MVD stands as the only definitive treatment for pHFS. Physicians are now able to utilize new technologies like endoscopy to optimize the surgical process.

Moving forward, there is a need for more animal experiments and randomized controlled clinical trials to further explore the pathogenesis of pHFS and to develop more effective, better tolerated, and more cost-efficient treatment options.

Data availability

No data was used for the research described in the article.

CRediT authorship contribution statement

Guangfa Xiang: Writing – original draft, Conceptualization. **Minghong Sui:** Writing – original draft. **Naifu Jiang:** Writing – original draft. **Rui Luo:** Visualization. **Jianwei Xia:** Visualization. **Xinling Wei:** Visualization. **Yifeng Lin:** Visualization. **Xingyu Li:** Visualization. **Zixiang Cai:** Visualization. **Junxia Lin:** Visualization. **Shipei Li:** Visualization. **Wanyi Chen:** Conceptualization. **Yang Zhao:** Writing – review & editing, Supervision. **Lin Yang:** Writing – review & editing, Conceptualization. Guangfa Xiang, Minghong Sui and Naifu Jiang contributed equally to this work and share first authorship.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to

influence the work reported in this paper.

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