Special Article

Hemifacial Spasm and Microvascular Decompression: The Treatment of Neurovascular Conflict

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Background: *Hemifacial spasm (HFS) has an impact on physical, social, mental, and occupational perspectives of patients. Botulinum toxin injection is an effective, non-invasive therapeutic procedure; however, the injection cannot get rid of cause of the disease. Microvascular decompression (MVD) is the only treatment which can eliminate the etiology of HFS.*
Objective: *MVD surgery aims to relieve symptoms of HFS by decompression of the facial nerve.*
Material and Method: *The authors reviewed medical literatures regarding pathophysiology, clinical presentation, diagnosis, and treatment of HFS which emphasizes in MVD surgery.*
Results: *The main pathophysiology is focal demyelination of the compressed facial nerve at the root exit zone. The other Babinski sign (the Babinski-2 sign) is commonly found in HFS with high specificity and reliability for the diagnosis. Neurovascular conflict can be obviously visualized in high resolution T2-weighted MRI of the brain and constructive interference in steady stage (CISS) sequence. Conventionally, MVD is performed by microscopic decompression of the facial nerve from offending vessel. Teflon felt pledget is interposed between the nerve and vessel. Neuroendoscopy is useful in visualization of the structures which cannot be seen by microscopic MVD.*
Conclusion: *MVD is an effective surgery which can treat the cause of HFS. Even though operative complications are uncommon, the complications, particularly hearing impairment and facial paresis should be minimized.*

Keywords: *Hemifacial spasm, Microvascular decompression, Neurovascular conflict, Facial nerve, Offending vessel*

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Hemifacial spasm (HFS) is a unique movement disorder characterized by intermittent clonic or tonic contractions of unilateral facial muscles innervated by the ipsilateral facial nerve[1,2]. The clonic movement occurs involuntarily and can be seen on one side of the face. The symptom is often insidious, chronic, and paroxysmal. It is usually aggravated when patients are talking, chewing or feeling excited. The etiology of facial spasm is chronic irritation of the facial nerve caused by pulsation of small arteries located in proximity to the nerve or compression of the nerve root exit zone by the adjacent vessels[3]. This irritation results in hyperactivity of the facial muscles eliciting HFS. Other disorders which have unilateral facial movements should be considered in the differential diagnosis of HFS, such as hemimasticatory spasm, myoclonus, oromandibular dystonia, facial tics, craniofacial tremor, and psychogenic facial spasms[4-6].

HFS has impact to patients in several dimensions, including physical, social, mental, and occupational aspects. A number of patients harboring HFS have visual impairment, social embarrassment, depression, and interference with work performance[7]. Patients with frequently disturbing facial spasms should be treated to improve quality of life. Regarding management of HFS, efficacy of oral medications, such as clonazepam, baclofen, carbamazepine, and anticholinergics, is usually temporary[5]. Botulinum toxin injection is an effective therapeutic option for HFS[1]; however, effect of the toxin is transient, and repeated injection is required in most cases. Neuromuscular complications following injection of the toxin, including ptosis, facial paresis, diplopia, and lagopthalmos, can be found in a number of patients[8]. Neurosurgical treatment by decompression of the facial

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nerve in the cerebellopontine angle known as microvascular decompression (MVD) has become popular, because good postoperative outcomes can be achieved in 80 to 90% of patients with HFS who undergo the surgery[9,10].

This article is a review of HFS with regard to pathophysiology, clinical manifestation, diagnosis and treatment which the authors focus on MVD as a surgical treatment of the disease.

Pathophysiology

The most accepted pathophysiology in the occurrence of HFS is composed of demyelination of the facial nerve root exit zone and ephaptic transmission. This area is a junction between the central and peripheral nervous system of the facial nerve, which is vulnerable to compression forces because the nerve is wrapped by an arachnoid membrane without the epineurium[11,12]. Several hypotheses of the pathophysiology of HFS were reported. The central hypothesis is proposed about hyperexcitability of the facial motor nucleus in the brainstem caused by regressive medullary change following nerve injury[11]. On the other hand, the peripheral mechanism assumes that ephaptic transmission and ectopic excitation in the root exit zone play a major role in pathophysiology of the disease. Ephaptic cross-talk is a pathological impulse transmission between adjacent nerve fibers, and ectopic conduction is instinctive occurrence of aberrant nerve impulses in the compression area[11,13-15]. The most recent study suggested that abnormal facial muscle response in HFS occurs as a result of ephaptic transmission at the site of compression rather than at the facial nucleus[16]. Another possible theory is the sympathetic hypothesis. The outer layer of the offending artery wall contains sympathetic endings which can induce ectopic action potentials from the demyelinated facial nerve fibers causing involuntary facial muscle spasms[17].

The etiology of HFS can be stratified into primary and secondary HFS[18]. The primary disease is characterized by compression of the facial nerve root exit zone by the offending vessel in the cerebellopontine angle. Chronic compression of the root exit zone brings about focal demyelination of the nerve. In most HFS patients, the facial nerve is compressed by the posterior inferior cerebellar artery (PICA) or anterior inferior cerebellar artery (AICA). Compression of the nerve by the vertebral artery or posterior fossa veins is not common[12]. Multiple compressions are encountered in 38% of patients[19]. The incidence of primary HFS increases with age because of progressive ectasia, elongation of the artery in the posterior cranial fossa and vertebrobasilar dolichoectasia[12,20], particularly in HFS patients with underlying arterial hypertension[21-24]. Small volume of the posterior cranial fossa is also reported to be a major risk factor of primary HFS in young patients[24-26]. Nevertheless, contrary to popular belief, the most recent study revealed that arterial hypertension was not associated with HFS, whereas it was significantly correlated with vascular compression of vasomotor center located in the ventrolateral medulla oblongata[27]. The first objective study of tortuosity of the verterbrobasilar arteries in the posterior fossa conducted by Edmond et al showed that hypertension, posterior fossa volume and vascular tortuosity alone is not an important factor in the etiology of HFS[28].

Secondary HFS is defined by HFS caused by lesions along the course of the facial nerve from the posterior cranial fossa to the stylomastoid foramen. These lesions, such as schwannoma, meningioma, brainstem tumor, cavernoma and various types of intracranial cyst, are rare causes of facial muscle spasms[12,29-40]. Furthermore, HFS can be a presenting symptom of Chiari I malformation which has restricted posterior cranial fossa volume[41-45].

Clinical manifestation

Typically, involuntary periodic spasms of the facial muscles are located in one side of the face. At the onset of HFS, the spasms present in the area of the orbicularis oculi muscle, bringing about eyelid closure and eyebrow elevation[1,46-48]. As the disease progresses, the abnormal movement involves a lower half of the facial muscles. In severe cases, the entire unilateral facial muscles, including the platysma muscle, can be involved[12]. The symptoms are aggravated by stress, fatigue, and changing of head position, and relieved by sleep[49,50]. Bilateral distribution of HFS is uncommonly found[51,52].

The other Babinski sign (the Babinski-2 sign or brown-lift sign) is a hallmark phenomenon in HFS. It is paradoxical elevation of the eyebrow during closure of the eye (Fig. 1). When the orbicularis oculi muscle contracts and the eye closes, simultaneous eyebrow elevation occurs owing to contraction of medial portion of the ipsilateral frontalis muscle[53-56]. This distinctive sign is useful for differentiating HFS from other craniofacial movement disorders. Pawlowski et al showed high prevalence of the Babinski-2 sign (86%) in HFS, high specificity (100%) and interrater reliability

(92%) for diagnosis of the disease[56].

Diagnostic evaluation

Electromyography (EMG) is useful for confirming diagnosis and differentiating HFS from other movement disorders of the face[57]. Magnetic resonance image (MRI) is an essential investigation for excluding secondary HFS. High resolution T2-weighted MRI and constructive interference in steady stage (CISS) sequence are excellent diagnostic tool for identifying neurovascular conflict and useful in preoperative planning for MVD[5,12,58] (Fig. 2). Advance virtual MRI techniques, such as image fusion and virtual cisternography can be utilized to demonstrate relationship between the cranial nerves and offending vessels within the cistern[59].

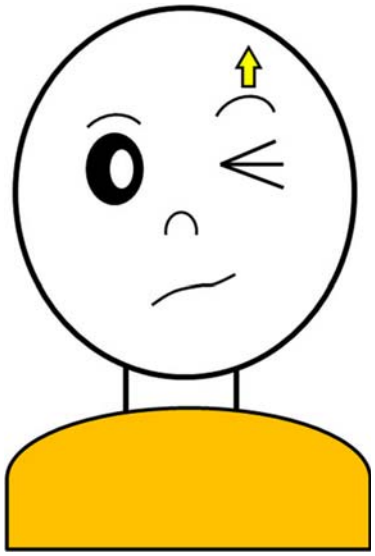

Fig. 1 The other Babinski sign (the Babinski-2 sign) in HFS; eyebrow elevation (arrow) and eye closure occurs concurrently.

Treatment

In management of HFS, treatment should be initiated in HFS patients who have the symptoms disturbing the daily living, such as visual field impairment, sleep disturbance, and social embarrassment[12]. Results of medical treatment, for example, clonazepam, carbamazepine, baclofen, are poor and not consistent[1]. Oral medication should be considered only in HFS patients with mild symptoms[12]. Even though botulinum toxin injection is a common therapy for HFS, this treatment cannot treat the underlying cause of the disease. As a result of this reason, MVD surgery plays a major role in elimination of the symptoms and improvement of quality of life in patients suffering from facial muscle spasms.

Botulinum toxin injection

Botulinum toxin has been introduced since the early 1980s. Nowadays it remains a standard medical treatment of HFS. The toxin can relieve the symptoms and carries low risk of complication. A recent study revealed 72.7 to 75% improvement after the injection[60]. Excellent outcome (>80% improvement)

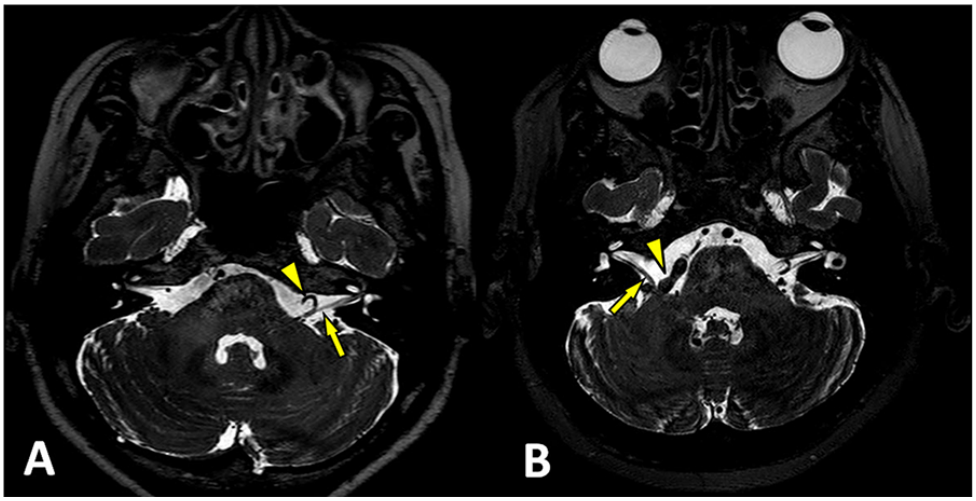

Fig. 2 Constructive interference in steady stage (CISS) sequence of MRI showing: (A) a loop of offending artery (arrowhead) compressing the facial nerve (arrow) near the root exit zone; (B) an ectatic offending artery (arrowhead) compressing the facial nerve (arrow) at the root exit zone.

was found in 96.7% of injection in the other study[61]. Adverse effects are uncommon, transient and usually mild. Common side effects include transient facial paresis, ptosis, diplopia, lagophthalmos, dry eye, eyelid swelling, and ecchymosis[60-64]. A relatively low risk of adverse effects makes this treatment suitable for patients with high anesthetic risk, patients who reject MVD surgery and patients whose symptoms are not caused by neurovascular conflict[12,48]. Major drawback of botulinum toxin injection is limited efficacy of the toxin. Repeated injections are required every 3 to 4 months and the effectiveness of botulinum toxin may decline in some cases after several years of the injection[12].

Microvascular decompression (MVD)

MVD for the treatment of HFS is a neurosurgical procedure which endeavors to relieve neurovascular conflict by separation of the offending vessels from the facial nerve in the cerebellopontine angle. This surgery is curative option to treat the etiology of the disease with long-term relief of the symptoms[12,48]. Many studies reported high success rate of the operation. Eighty-six percentages of patients were symptom free at 10 years after MVD[65]. A large series of 1,174 patients who underwent MVD for treating HFS, 1,105 (94.1%) were classified as cured state at one year postoperatively[66]. Seventy-five percent of patients achieved an excellent result (no residual spasm) in a French series of MVD[67]. Several studies showed excellent outcome (cured HFS) in more than 90% of patients[68-70].

The operation is performed under general anesthesia. Various positions, including the lateral park-bench, supine, and sitting positions can be utilized[71-73]. The most popular position is the lateral park-bench position with the operated side placed up. All pressure areas of the body are padded to prevent positional skin and nerve injury. MVD is seldom performed in sitting posture because of an increased risk of venous air embolism[74,75]. Intraoperative electrophysiologic monitoring (facial nerve EMG and brainstem auditory evoked potentials) is used to detect abnormal EMG responses and avoid intraoperative cranial nerve injury[12,48]. Suboccipital craniotomy with retrosigmoid approach using operative microscope is performed to directly access the cerebellopontine angle. The arachnoid membrane was incised for releasing the cerebrospinal fluid (CSF) from the subarachnoid space to reduce the posterior cranial fossa volume and enlarge the operative corridor. The facial nerve is inspected from the root exit zone to the location where it enters the internal auditory meatus. The offending vessels, usually being the PICA or AICA loop, is meticulously dissected and moved away from the compressed nerve. Care should be taken during the dissection, iatrogenic nerve or vascular injury and excessive retraction of the cerebellar hemisphere must be cautious. After complete isolation of the conflicting vascular loop from the facial nerve by cutting the arachnoid membrane, a single piece or sequential arrangement of Teflon felt pledget is interposed between the vessel and nerve (Fig. 3 and 4). In some complex cases, particularly in case with large dolichoectatic artery compressing the facial nerve, mobilization of the arterial loop and utility of sling retraction technique is helpful for prevention of recurrent disease[12,76]. The utilization of intraoperative EMG for monitoring abnormal muscle response or known as lateral spread response is useful for ensuring adequate decompression of the facial nerve. However, correlation between disappearance of lateral spread response and outcome following MVD is still controversial. Several studies revealed that disappearance of abnormal muscle response was not correlated with operative outcome[77-79]. Sun et al proposed the use of artery-nerve abnormal muscle response or Z-L response, which was more specific than abnormal muscle response. It was electrical stimulation of the offending artery wall and recording of abnormal waveform from the facial muscles. Persistence of artery-nerve abnormal muscle response indicates that decompression of the facial nerve is not adequate and further decompression is required[69]. After complete decompression is carried out, visualization of the vessels should be done to detect kinking of vascular loop. Once kinking of vascular loop is encountered, surgical reposition of the loop is mandatory. Postoperatively, some patients experience minimal or no improvement of the symptoms; this failure is mostly caused by anatomical rationale, including huge dolicoectatic vertebral arteries in limited subarachnoid space, large arteries or veins intervening between the facial nerve and the vestibulocochlear nerve, and migration of Teflon felt[10,46,65].

MVD is considered to be a safe intracranial procedure. Operative complications of MVD are rare. Major complications include temporary or permanent hearing deficit and facial nerve dysfunction. Other uncommon complications are lower cranial nerve dysfunction, intracranial infection and CSF leakage[9,10,46,65-67,70]. Delayed facial palsy is not unusual complication following MVD for HFS. The exact

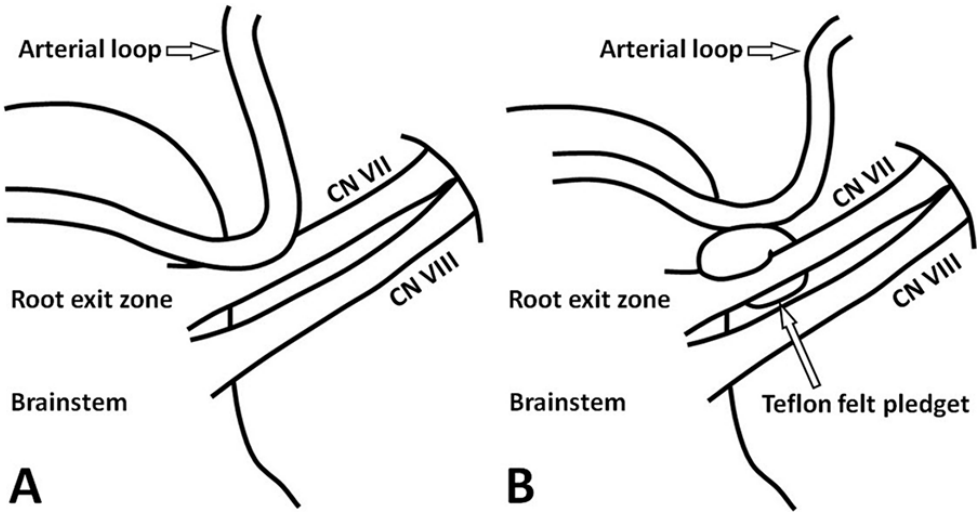

Fig. 3 Schematic illustration of MVD: (A) a loop of offending artery (arterial loop) compressing the facial nerve (CN VII) at the root exit zone; (B) a Teflon felt pledget is interposed between the arterial loop and the facial nerve (CN VII) in MVD surgery.

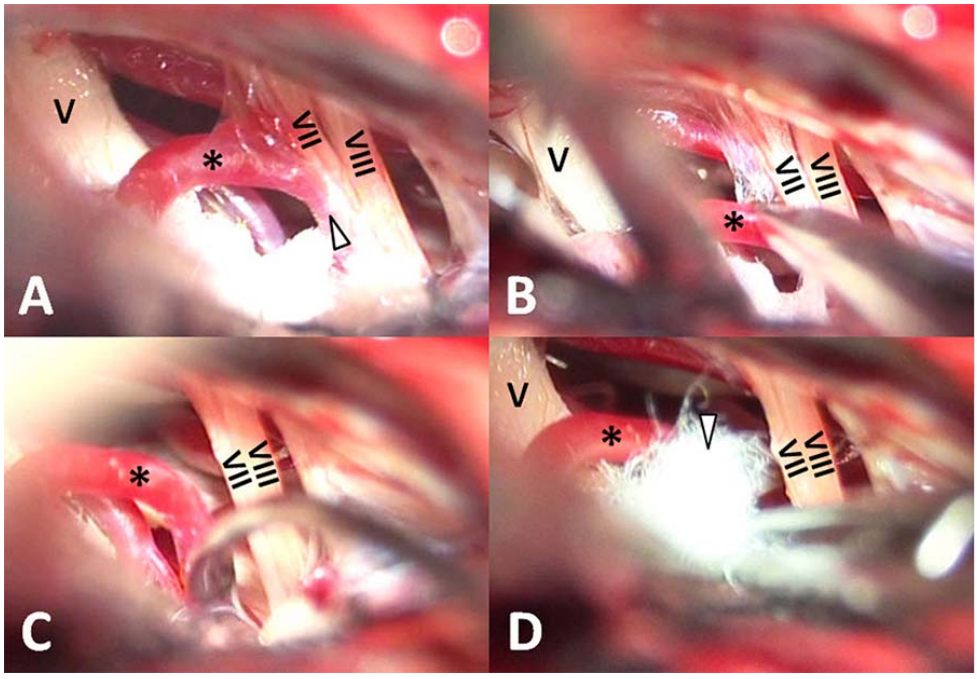

Fig. 4 Operative microscopic view in MVD: (A) neurovascular conflict (arrowhead) between the loop of offending artery (asterisk) and facial nerve (VII); (B) dissection of the arachnoid membrane between the arterial loop and facial nerve; (C) Push away of the arterial loop from the facial nerve; (D) placement of Teflon felt pledget (arrowhead) between the artery and nerve; V = trigeminal nerve; VII = vestibulocochlear nerve.

etiology has been not known yet; one possible hypothesis is that surgical manipulation of the facial nerve may reactivate herpes simplex and varicella zoster viruses. The facial weakness can recover spontaneously with a good or excellent outcome[80-83]. A systematic review of 19 retrospective and 3

prospective studies in terms of safety and efficacy of MVD for HFS showed that 91.1% of patients had complete resolution of symptoms following MVD. The most common transient complication was facial palsy found in 9.5% of patients, followed by hearing deficit (3.2%) and CSF leak (1.4%). The common permanent complications were hearing deficit (2.3%) and facial palsy (0.9%). The risk of stroke was 1 in 1,800 and risk of death was 1 in 5,500[84].

Intraoperative neuroendoscopy is utilized in neuroendoscopy-assisted MVD or fully neuroendoscopic MVD to minimize the risks of brain retraction and postoperative hearing impairment. The endoscope enhances visualization of the ventral surface of the brainstem and the offending vessels which can be missed by microscopic MVD. Furthermore, it is helpful in confirmation of the position of Teflon felt before closure[85-91].

Conclusion

HFS is a distinctive neurological disorder mostly caused by neurovascular conflict at the root exit zone of the facial nerve. Diverse movement disorders masquerading as HFS must be excluded before initiation of the treatment. Cranial MRI and CISS sequence are beneficial for inspection of neurovascular conflict and exclusion of secondary causes of facial muscle spasms. Botulinum toxin injection is a commonly used symptomatic treatment, but it cannot treat the etiology. MVD surgery is an effective procedure to ameliorate cause of the disease and relieve symptoms in a long-term; however, operative risk, particularly cranial neuropathies, must be minimized. Neuroendoscopy has been increasingly utilized for assisting microscopic MVD or used as endoscopic MVD alone.

What is already known from this topic?

HFS is involuntary contraction of the unilateral facial muscles. Medical treatment and injection therapy aim to relieve the symptoms; however, they cannot be used to treat cause of the disease.

What this study adds?

MVD of the facial nerve is the outstanding option for treating neurovascular conflict which is the etiology of HFS. This operation renders high success cure rate with low operative complications. The major drawbacks of MVD includes postoperative sensorineural hearing impairment, and transient facial nerve dysfunction. Role of neuroendoscopy has been increased in the treatment of HFS.

Potential conflicts of interest

None.

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โรคใบหน้ากระตุกครึ่งซีกและการผ่าตัดทางจุลศัลยกรรมในการรักษาภาวะหลอดเลือดกดทับเส้นประสาท

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ภูมิหลัง: *โรคใบหน้ากระตุกครึ่งซีกมีผลกระทบทางด้านร่างกาย สังคม จิตใจ และอาชีพการงานของผู้ป่วย การฉีดโบทูลินัมท็อกซินเป็นการรักษาที่มีประสิทธิภาพวิธีหนึ่ง อย่างไรก็ตามการฉีดโบทูลินัมท็อกซินไม่สามารถรักษาสาเหตุของโรคได้ การผ่าตัดทางจุลศัลยกรรมเพื่อแก้ไขภาวะหลอดเลือดกดทับเส้นประสาทเป็นการรักษาเพียงวิธีเดียวที่สามารถรักษาที่สาเหตุของโรคได้*

วัตถุประสงค์: *การผ่าตัดทางจุลศัลยกรรมเพื่อแก้ไขภาวะหลอดเลือดกดทับเส้นประสาท มีวัตถุประสงค์เพื่อรักษาอาการใบหน้ากระตุกครึ่งซีก โดยแก้ไขภาวะเส้นประสาทควบคุมกล้ามเนื้อใบหน้าถูกกดทับ*

วัสดุและวิธีการ: *คณะผู้นิพนธ์ทบทวนวรรณกรรมทางการแพทย์ในแง่ของพยาธิสรีรวิทยา อาการทางคลินิก การวินิจฉัยและการรักษาโรคใบหน้ากระตุกครึ่งซีกโดยเน้นในส่วนของการผ่าตัดทางจุลศัลยกรรมเพื่อแก้ไขภาวะหลอดเลือดกดทับเส้นประสาท*

ผลการศึกษา: *พยาธิสรีรวิทยาหลักของโรคเกิดจากการเสื่อมเฉพาะที่ของปลอกไมอิลินที่หุ้มเส้นประสาทควบคุมกล้ามเนื้อใบหน้า ซึ่งถูกกดทับในตำแหน่งที่เส้นประสาทออกจากก้านสมอง อาการแสดงบาบินสกีที่ใบหน้า (อาการแสดงบาบินสกี 2) พบบ่อยในโรคกล้ามเนื้อใบหน้ากระตุกครึ่งซีก รวมทั้งมีความจำเพาะและความน่าเชื่อถือสูง ในการวินิจฉัยโรคนี้สามารถเห็นภาวะหลอดเลือดกดทับเส้นประสาทได้ชัดเจนในการตรวจภาพแม่เหล็กไฟฟ้าของสมองในภาพ T2 ที่มีความละเอียดสูงและในภาพ constructive interference in steady stage (CISS) โดยทั่วไปการผ่าตัดจะทำโดยอาศัยกล้องจุลศัลยกรรมสำหรับแก้ไขภาวะเส้นประสาทควบคุมกล้ามเนื้อใบหน้าถูกกดทับจากหลอดเลือดที่อยู่ใกล้เคียง จากนั้นจึงใช้ชิ้นของ Teflon felt วางคั่น ระหว่างเส้นประสาทกับหลอดเลือดดังกล่าว การผ่าตัดโดยวิธีการส่องกล้องเป็นอีกวิธีหนึ่งที่มีประโยชน์สำหรับการมองเห็นส่วนของระบบประสาทซึ่งไม่สามารถ มองเห็นได้จากการผ่าตัดโดยอาศัยกล้องจุลศัลยกรรม*

สรุป: *การผ่าตัดทางจุลศัลยกรรมเพื่อแก้ไขภาวะหลอดเลือดกดทับเส้นประสาทเป็นการผ่าตัดที่มีประสิทธิภาพ ซึ่งสามารถรักษาสาเหตุของโรคใบหน้ากระตุกครึ่งซีกได้ แม้ว่าจะพบภาวะแทรกซ้อนจากการผ่าตัดดังกล่าวได้ไม่บ่อยแต่ควรลดภาวะแทรกซ้อนให้น้อยที่สุดโดยเฉพาะเรื่องการได้ยินแย่ลงและกล้ามเนื้อใบหน้าอ่อนแรงหลังผ่าตัด*