Review Article

Doo-Sik Kong, M.D.

Kwan Park, M.D., Ph.D.

Department of Neurosurgery
Samsung Medical Center
Sungkyunkwan University
School of Medicine, Seoul, Korea

Hemifacial Spasm : A Neurosurgical Perspective

Hemifacial spasm (HFS) is characterized by tonic clonic contractions of the muscles innervated by the ipsilateral facial nerve. Compression of the facial nerve by an ectatic vessel is widely recognized as the most common underlying etiology. HFS needs to be differentiated from other causes of facial spasms, such as facial tic, ocular myokymia, and blepharospasm. To understand the overall craniofacial abnormalities and to perform the optimal surgical procedures for HFS, we are to review the prevalence, pathophysiology, differential diagnosis, details of each treatment modality, usefulness of brainstem auditory evoked potentials monitoring, debates on the facial EMG, clinical course, and complications from the literature published from 1995 to the present time.

KEY WORDS : Hemifacial spasm · Microvascular decompression · Craniofacial abnormalities.

INTRODUCTION

Hemifacial spasm (HFS) is characterized by unilateral, intermittent contractions of the muscles of facial expression, which typically begins in the orbicularis oculi and spreads to the other muscles of expression over several years⁴⁾. At early period, it usually affects the periorbital muscles unilaterally. With time, other ipsilateral facial muscles become affected and brief clonic twitching may gradually lead to more sustained tonic contractions¹⁷⁾. There is now considerable evidence that primary HFS is in almost all cases related to a vascular compression of the facial nerve at its Root Exit Zone (REZ) from brainstem^{18,31,33)}, and that microvascular decompression (MVD) constitutes its curative treatment⁶⁹⁾.

Here, in an attempt to understand the overall craniofacial abnormalities and to perform the optimal surgical procedures for HFS, published articles from 1995 to the present time were reviewed along with our accumulated experiences.

EPIDEMIOLOGY

Valid prevalence estimates of HFS are elusive because of underdiagnosis, misdiagnosis, and the absence of population based data. To our knowledge, there have been few studies documenting the prevalence of hemifacial spasm^{3,55)}. These studies demonstrated the total prevalence of HFS in range of 9.8 to 11 per 100,000 in the total population. Unfortunately, little is not known about the accurate prevalence in Asia, although HFS is known to be more prevalent than trigeminal neuralgia in northeastern Asia.

PATHOPHYSIOLOGY

Since Dandy proposed first that cranial nerves by an ectatic vessel can cause clinical syndromes in 1934¹³⁾, Jannetta further explored and popularized MVD as an effective treatment for HFS and trigeminal neuralgia³⁴⁾. He assumed that the mechanical effect of a pulsating blood vessel was the cause of the disease and that the compression must be at the root entry zone (REZ) of the cranial nerve to cause symptoms³¹⁾.

However, several authors suggested that the compression can occur at any point along the CNS segment of cranial nerve^{43,47,63)}. The length of the CNS segment of the trigeminal nerve is longer than that of the facial nerve^{39-42,71)}. Assuming that the CNS segment of cranial nerves is more vulnerable to microtrauma than the PNS segment, more people would be expected to have trigeminal neuralgia³⁶⁾ than hemifacial spasm^{3,14)}. This study was inconsistent with Jannetta's

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- Address for reprints :
Kwan Park, M.D.
Department of Neurosurgery
Samsung Medical Center
Sungkyunkwan University
School of Medicine, 50 Irwon-dong
Gangnam-gu, Seoul 135-710, Korea
Tel : +82-2-3410-3496
Fax : +82-2-3410-0048
E-mail : kwanpark@skku.edu

hypothesis that symptoms are caused by vascular compression of the REZ, the length of which is similar in all cranial nerves. De Ridder et al. supposed that the incidence of trigeminal neuralgia, hemifacial spasm, and glossopharyngeal neuralgia is clearly related to the length of their CNS segments¹⁴. Consequently, the authors hypothesized that the site for a vascular compression to become symptomatic should be at the CNS segment and not only at the REZ. As an evidence of this suggestion, Sindou et al. described that in his 579 patients with trigeminal neuralgia, the neurovascular compression was at the root entry/exit zone in only 52.3%, while compression was in the mid portion of the root in 54.3%⁶⁷.

Although etiology of HFS is not well-known yet, a possible association between HFS and arterial hypertension has been suggested although not clearly evident. Three recent case-control studies suggested that primary HFS may be associated with arterial hypertension^{15,16,56}. Compression of the ventrolateral medulla (VLM) at the root entry zone of cranial nerves IX and X may induce hypertension. Because the rostral VLM is close to the origin of the facial nerve, vessel loops originating from the vertebral/basilar arteries or their branches might be responsible for multiple neurovascular compressions, inducing both hemifacial spasm and hypertension^{35,44}. Alternatively, hypertension could be a risk factor for HFS because it may produce vascular anomalies resulting in compression of the seventh cranial nerve. Under this

hypothesis, one would expect that hypertension would precede hemifacial spasm.

DIFFERENTIAL DIAGNOSIS

Craniofacial dyskinesias encompass a variety of abnormal spontaneous craniofacial movements that often appear similar in morphology. Physicians including neurosurgeons, neurologists, and other medical practitioners (including those involved in oriental medicine) should be aware of a variety of abnormal spontaneous craniofacial movements (Table 1).

Blepharospasm

Blepharospasm is a bilateral condition characterized by focal, excessive involuntary closure of the eyelids generally caused by spasm of the orbicularis oculi muscles^{2,21,22} and it may sometimes lead to functional blindness⁸. Despite having different pathophysiology, both HFS and blepharospasm produce involuntary eyelid closure due to contractions of the orbicularis oculi muscle. Blepharospasm is known to be due to hyperexcitability of brainstem interneurons, as a result of organic dysfunction of the basal ganglia⁵. Patients may complain of photophobia, ocular irritation, or dry eyes. Hallett et al. demonstrated in their case-control study that blepharospasm was more associated with generalized anxiety disorder than hemifacial spasm²¹.

Table 1. Differential diagnosis of craniofacial dyskinesias¹⁶

	Hemifacial spasm	Blepharospasm	Tic disorder	Myokymia	Synkinesis
Involvement	Unilateral	Bilateral	Unilateral/ bilateral	Unilateral	Unilateral
Characteristics	Intermittent contractions of facial muscles. Typically begins in the orbicularis oculi. It may persist during sleep. On EMG, with lateral spread response (LSR) and variable synkinesis on blink reflex	Focal, excessive involuntary closure of the eyelids. It may accompany photophobia, ocular irritation, or dry eyes. Closely associated with generalized anxiety disorder.	Brief, repetitive, stereotyped, and involuntary movements or sounds. Usually begins in the face, spreads caudally. Resemble voluntary movements such as blink, wink, head movement, raised eyebrow, mouth movement.	Undulating, vermicular, rippling and wavelike movements across the muscle surface. Usually resolve spontaneously in a few days or weeks.	Intermittent contraction of facial muscle atypical contraction sometimes post-traumatic
Aggravation	Fatigue, anxiety, stress, and driving	Anxiety, fatigue, bright lights, driving, self-consciousness	Stress, relaxation after stress, excitement, boredom, heat	Fatigue	
Treatment	1. Botulinum toxin-A injection 2. Microvascular decompression	1. Benzodiazepine, 2. Anti-convulsants 3. Anti-cholinergics 4. Baclofen 5. Botulinum toxin-A injection	1. Benzodiazepines, 2. Calcium channel blockers 3. Dopamine depletors	1. Reassurance 2. Relaxation	1. Botulinum toxin A injection

Craniofacial tics

Tics are brief, repetitive, stereotyped, and involuntary movements or sounds¹⁷. The most severe type of tic disorder is Gilles de la Tourette's syndrome. It usually begins in the face, spreads caudally, and fluctuates in frequency, intensity and distribution. Treatment includes benzodiazepines, calcium channel blockers, and dopaamine depleters.

Ocular myokymia

Myokymia is characterized by undulating, vermicular, rippling and wavelike movements across the muscle surface⁷⁶. Most cases are idiopathic and may constitute a generalized benign or essential myokymia syndrome¹⁷. Almost all of idiopathic cases resolve spontaneously in a few days or weeks. Eyelid myokymia occurs in many people who are tired and fatigued⁷⁶. Unfortunately, this disease entity was not clearly defined and not widely accepted as a unique disease entity despite high incidence.

TREATMENT

As is already well known, widely-accepted treatment modalities of HFS include injections of Botulinum toxin as less invasive procedure and microvascular decompression (MVD) as the definite treatment⁶⁹.

Botulinum toxin A (BTX-A)

Botulinum toxin type A (BTX-A) injections have been widely accepted as a safe and efficacious modality for the treatment of both blepharospasm and HFS^{6,10,17,25,26,30,37,59,73,74}. BTX-A, which has been approved by Food and Drug Administration for HFS in U.S., can be injected subcutaneously or intramuscularly into the involved facial muscles. At the early state of HFS, BTX-A injection is very attractive method because it is less invasive and easily approached by physicians. The onset of action is about 3 to 5 days and the mean duration of benefit is 3 to 6 months. Thus, patients require repetitive injections two to four times per year. Complications of BTX-A injection include ptosis, blurred vision, and diplopia that usually improve in days to weeks. Most common injection sites are pretarsal and preseptal portions. Cakmur et al. showed that pretarsal injections of BTX-A in patients with involuntary eyelid closure due to contractions of the orbicularis oculi muscle are associated with higher efficacy and lower frequency of complications than preseptal injections⁸. Although BTX-A injection shows high success rate of treatment of HFS and blepharospasm, its substantial limitation is that it required repeat injection, which eventually lead to charge of high economic cost because of uncoverage under current national health insurance policy.

Microvascular decompression (MVD)

Whereas Botulinum toxin injection does not provide long-term relief because of its temporary benefit, surgical treatment using MVD results in long-term relief of the symptoms in the majority of patients^{3,60}. The detailed surgical procedures are as follows.

Opening

The operation technique for MVD is variable according to the surgeon's preference. For example, some authors favor supine position^{29,70} and others lateral decubitus position (it is so called "park-bench position")^{34,57}. In our series, all of the surgical procedures were performed via a lateral retrosigmoid suboccipital approach. The patient is placed in a lateral park bench position with the head rotated approximately 10 degrees away from the affected site and the vertex dropped 15 degrees toward the floor. A 20 × 25 mm sized craniectomy is performed with extension to the flat occipital squama inferiorly and the sigmoid sinus laterally, below the posterior end of the digastric notch on the mastoid process.

Intradural procedure

After the cisterna magna or lateral cerebellomedullary cistern is opened, the cerebellum gives way so that the VIIth to VIIIth cranial nerve complex can be approached with minimal use of brain retractors. Some drainage of

Fig. 1. Variable patterns on microanatomical findings of neurovascular conflicts. (Park et al. in processing). (A : thick arachnoid bands, B : branching vessels, C : sandwich vessels, D : small perforating vessels).

CSF is necessary to minimize potential injury by placing the retractor on the cerebellum. After CSF is drained, a tapered retractor blade is placed over the previously placed rubber dam and cottonoid. After careful dissection of the arachnoid membrane and gentle retraction of the flocculus, the root exit zone (REZ) of the facial nerve can be visualized. A good access from below is of most importance, because most conflicts are located ventrocaudal to the facial exit zone at the brainstem⁶¹⁾. Jannetta et al. and Sindou et al. indicated that reaching the facial nerve along an infra-floccular route was important for two reasons; 1) Neurovascular conflicts are usually located ventro-caudally at the REZ, 2) A lateral-to-medial retraction of the cerebellar hemisphere would exert stretching of the VIIIth nerve and lead to hearing loss^{46,61,69)}.

To reduce the cerebellar retraction, according to Nakanishi's approach, the operator's head and the patient's head are at the same height, and the operator can look at the surgical field through the microscope in a horizontal direction²⁹⁾. The compressing vessel, so called offender, can be identified near to the REZ. Several pieces of Teflon sponge are placed between the compressing vessel and the REZ.

Microanatomical observations

In our accumulated experience of about 900 cases, we found that the patterns of neurovascular conflicts by offending vessels are so variable in each case. As anatomical variation of neurovascular conflicts could be observed in trigeminal neuralgia⁶⁷⁾, we found that a variety of anatomical compressive patterns contributed to the neurovascular conflicts. In some instances, focal thick arachnoid band was often a major contributing factor to compression. In others, two or even three compressing vessels were often observed with a type of sandwich, branching vessels, or multiple perforating vessels (unpublished data, Fig. 1). These findings were already mentioned by Sindou et al.⁶⁷⁾ and it could be the source of insufficient decompressive surgery and consequently surgical failure⁶⁹⁾. However, no statistical significance was shown between variable neuro-vascular conflicts and clinical outcome.

Closure

After the neurovascular decompression, the dura mater can be closed in the following fashion which was newly designed⁵⁷⁾. Four to five interrupted stitches are made at intervals of 6-7 mm. Several pieces of muscle that have been obtained from the adjacent muscles can be interposed into the space between the stitches. The muscle piece should be small enough to enter the slit but large enough to fill the defect between the stitches⁵⁷⁾. In cases where the mastoid cavity is opened, bone wax should be applied to completely

seal the opened surface of the cavity. Cranioplasty is performed using polymethyl methacrylate (PMMA) bone cement according to the surgeon's preference. No fixation device is necessary because of the bulky muscle groups placed over the lesion. Irrigation with droplets of papaverine solution (10% concentration) is used in all cases as routine during surgical dissection of the neurovascular conflict and before closing of the dura mater.

BRAINSTEM AUDITORY EVOKED POTENTIALS (BAEP) MONITORING; ITS IMPORTANCE FOR HEARING PRESERVATION

Microsurgical maneuvers involved in relieving the neurovascular conflict may endanger the cranial nerve VIII. The use of intraoperative monitoring of brainstem auditory evoked potentials (BAEPs) is generally acknowledged to reduce the risk of hearing impairment. It can be estimated that Peaks I and II are generated in the distal and proximal parts of the cochlear nerve, respectively, and Peaks III to V along the brainstem auditory pathways from the cochlear nucleus up to the inferior colliculus. Damage to the VIIIth nerve results in a delay in latency and reduction in amplitude of Peak V. When this delay becomes significant, the neurophysiologist must inform the surgeon to discontinue the procedure until the cause is identified, and the dangerous maneuvers corrected. The main critical situations are the following : stretching of the VIIIth nerve when retracting the cerebellum; manipulation of the labyrinthine artery and/or the anteroinferior cerebellar artery; direct trauma to the nerve by instruments or a nearby coagulation; and at the end of surgery, neo-compression of the nerve by the prosthesis interposed between the conflicting vessel and the VIIth-VIIIth nerve complex^{53,68)}. For most clinical neurophysiologists, latency of Peak V is considered the best electrophysiological indicator for signaling cochlear nerve damage by operative manipulations^{7,51,66)}. Grundy et al.²⁰⁾ used the value of 1.5 milliseconds delay in latency of Peak V to warn the surgeon. Hatayama and Møller²³⁾ paired latency delay of Peak V greater than 1 millisecond with amplitude modifications of this wave. Polo et al. demonstrated that latency of Peak V was the most frequently observed and the most significant phenomenon, especially during cerebellar retraction and the decompression step of the microvascular decompression procedure⁶¹⁾. The main practical contribution of this study consists in neurophysiological parameters identified by the authors as "warning values" to avoid an irreversible Injury to the hearing function. Conversely, in our series, decrease of amplitude of Peak V wave was independently predicting value for hearing loss during MVD rather than the delayed latency of Peak V (unpublished). In clinical

practice, we are using these amplitude changes of Peak V wave as the treatment guideline. In conclusion, we strongly believe that close collaboration between neurosurgeon and neurophysiologist is very important to guarantee the safety of MVD.

INTRA-OPERATIVE MONITORING OF THE FACIAL EMG RESPONSES

In the 1980s, Møller and Jannetta showed that lateral spread responses (LSR) could be recorded from one muscle innervated by the superior branch of the facial nerve when the inferior branch was stimulated or inversely^{48-50,52}. These phenomena were presumed to be related to ephaptic transmission at the lesion site alone or in combination with motor nucleus hyperactivity. They advocated routine intraoperative EMG recording of LSR during MVD, to ensure that adequate decompression was achieved. Mooij et al. demonstrated that a guiding role of Intraoperative monitoring was apparent in 33.8% of patients, and a confirming role was demonstrated in 52.7% of patients, which showed that facial EMG can be useful as an aid in the “learning curve” as well as in guiding even the experienced neurosurgeon to obtain the best results⁵⁴. However, Hatem and Sindou et al. documented that the persistence of LSR did not necessarily indicate postoperative residual spasm. In their study of 30 cases²⁴. The authors suggested that the practical value of their intraoperative disappearance as control-test of an effective decompression remains controversial⁶⁹. Based on our series of 300 cases, facial EMG monitoring of the LSR was proved to be useful in predicting the outcomes and might be an effective assistant tool to accomplish complete decompression if exploration is incomplete³⁸. Patients with persistent LSR after MVD showed poor one-year outcome, as compared with patients with absence of LSR after MVD. In the future, a well-organized and controlled study will be required for the evaluation of efficacy of LSR on the facial EMG monitoring.

CLINICAL OUTCOME; IMMEDIATE OR GRADUAL IMPROVEMENT

MVD of the facial nerve for idiopathic HFS has a long-term success rate ranging from 83 to 97%^{1,4,12,18,28,32,49}. On the statistical analysis, no prognostic factor such as gender, age, side, and nature of neurovascular conflict showed any significant influence on outcome⁶⁹. In our series of 455 cases, cure rate after one year was established at 95.7%.

It is of note that resolution of HFS is often gradual, whereas dramatic resolution is expected by patients and surgeons in most of cases^{3,27-29}. Understanding of precise course of

postoperative HFS is very crucial to make decision when the second reoperation should be done²⁹. Ishikawa et al. observed that approximately one-half of their patients (50.3%) who underwent MVD for HFS continued to experience the condition to some degrees after the operation. Goto et al. demonstrated that the longer the symptom duration of HFS, the longer the time interval until complete resolution¹⁹. The immediate resolution of HFS could result from the disappearance of the spontaneous or ectopic excitation by the pulsatile compressive force of the offending vessel⁶⁵. Saito et al. interpreted that delayed resolution could be attributed to the complete regeneration of the micro-injury of the facial nerve or the gradual stabilization of the facial motor nucleus⁶⁴. According to their suggestion, reappearance of HFS within 4 days after microvascular decompression (MVD) does not necessarily suggest a failed procedure, and patients should be observed for at least a few months before another procedure is considered. On the analysis of our 236 series during recent two years, we found that 76 of a total patients (32.2%) who were eventually cured had residual symptoms for a variable period of time (from several months to a year), while 144 patients (61%) improved immediately after MVD (unpublished). Based on these results, we believe that we could predict the result after at least postoperative 3 months of MVD and the postoperative one-year is the optimal time to judge the final outcome.

For the evaluation of success rate of MVD, Chang et al. investigated the role of postoperative 3D-TOF MRA in predicting clinical outcomes and complications after MVD treatment of HFS⁹. Their study revealed that postoperative 3D-TOF MRA for patients with HFS might provide surgeons with anatomic information regarding neurovascular changes of the facial nerve REZ, although its correlation with clinical outcome was not proven yet.

COMPLICATIONS OF MVD

In the numerous reported series^{4,11,34}, deafness accounted in a range of 1.6 to 2.6% and moderate hearing loss 0.6 to 0.7%. Permanent facial weakness was seen 3.4 to 4.8% following operations. Among other complications, CSF leakage affected 2.7% of the patients. In our series, hearing loss after MVD was in 1.8% of patients and rate of facial palsy was 0.8%. On an account of above mentioned muscle-plugging method⁵⁷, CSF leakage rate was only 2 cases (0.29%) in 678 patients. Other than acute facial weakness, postoperative delayed facial palsy was seen during the follow-up period. Delayed facial palsy can be found in 2.8-8.3% of patients following vestibular schwannoma surgery. In 21 of 410 patients (5.4%), we could find the delayed facial palsy

at the postoperative 7-23 days (average 12.1 days)⁶². Our study revealed that the incidence of delayed facial palsy after MVD was not low as was previously reported in the literature but almost all patients achieved complete recovery without any further special treatment.

REOPERATION OF MVD

With recent advances in the diagnostic technology using high resolution MRI, offending vessels leading to neurovascular conflicts can be easily detected. Unlike trigeminal neuralgia, for which many medical and surgical options besides a second MVD are available if pain recurs, for patients with recurrent hemifacial spasm, botulinum toxin is the only other treatment option. Accordingly, Park et al. documented that repeated MVD could be applied for residual or recurrent HFS⁵⁸. However, time to re-operate MVD for recurrent disease should be carefully determined. Because spasm is often gradually improved after MVD (e.g. several months to even a few years), Sindou et al. did not recommend early re-operation in patients with initial failure or early poor results until one year at least has passed⁶⁹. Based on our limited experience of recurrent HFS, careful decision making on reoperation should be appreciated, considering high risk of morbidity on second MVD.

CONCLUSION

By Tan et al. in 2004, an interesting finding was reported that among 203 family physicians who participated in a "video" test on HFS, only 9.4% were able to diagnose HFS and 46.3% of them did not know how to manage the condition⁷². It implies that specialists need to play a greater role in implementing educational programs to increase the awareness of the common movement disorders in the primary care setting. This will reduce misdiagnosis and inappropriate treatment for disorders such as HFS, which can be effectively treated.

Recently, Lunsford et al. mentioned that many patients who are eligible for MVD have a tendency of favoring a lesser invasive procedure for trigeminal neuralgia⁴⁵. This current trend toward less invasive technique may be similar in the treatment of HFS, because less invasive technique such as BTX-A injection clearly has less impact on their quality of life and allows rapid return to work. Accordingly, we should be aware of this trend and overcome this limitation of invasive technique such as microsurgical procedures. By experts' hands under the guidance of intraoperative electrophysiological monitoring of BAEP, MVD procedure can be done with minimal morbidity and long-benefit. At the same time, more

enthusiastic efforts and ongoing endeavor should be made to improve surgical technique toward the best outcome

References

1. Acevedo JC, Sindou M, Fischer C, Vial C : Microvascular decompression for the treatment of hemifacial spasm. Retrospective study of a consecutive series of 75 operated patients--electrophysiologic and anatomical surgical analysis. *Stereotact Funct Neurosurg* 68 : 260-265, 1997
2. Anderson RL, Patel BC, Holds JB, Jordan DR : Blepharospasm : past, present, and future. *Ophthalm Plast Reconstr Surg* 14 : 305-317, 1998
3. Auger R : Hemifacial spasm in Rochester and Olmsted County, Minnesota, 1960 to 1984. *Arch Neurol* 47 : 1233-1234, 1990
4. Barker FG 2nd, Jannetta PJ, Bissonette DJ, Shields PT, Larkins MV, Jho HD : Microvascular decompression for hemifacial spasm. *J Neurosurg* 82 : 201-210, 1995
5. Berardelli A, Rothwell JC, Day BL, Marsden CD : Pathophysiology of blepharospasm and oromandibular dystonia. *Brain* 108 : 593-608, 1985
6. Bihari K : Safety, effectiveness, and duration of effect of BOTOX after switching from Dysport for blepharospasm, cervical dystonia, and hemifacial spasm dystonia, and hemifacial spasm. *Curr Med Res Opin* 21 : 433-438, 2005
7. Broggi G, Scaioli V, Brock S, Dones I : Neurophysiological monitoring of cranial nerves during posterior fossa surgery. *Acta Neurochir Suppl (Wien)* 64 : 35-39, 1995
8. Cakmur R, Ozturk V, Uzunel F, Donmez B, Idiman F : Comparison of preseptal and pretarsal injections of botulinum toxin in the treatment of blepharospasm and hemifacial spasm. *J Neurol* 249 : 64-68, 2002
9. Chang JW, Chang JH, Choi JY, Kim DI, Park YG, Chung SS : Role of postoperative magnetic resonance imaging after microvascular decompression of the facial nerve for the treatment of hemifacial spasm. *Neurosurgery* 50 : 720-725; discussion 726, 2002
10. Chen R, Karp BI, Hallett M : Botulinum toxin type F for treatment of dystonia : long-term experience. *Neurology* 51 : 1494-1496, 1998
11. Chung SS, Chang JH, Choi JY, Chang JW, Park YG : Microvascular decompression for hemifacial spasm : a long-term follow-up of 1,169 consecutive cases. *Stereotact Funct Neurosurg* 77 : 190-193, 2001
12. Chung SS, Chang JW, Kim SH, Chang JH, Park YG, Kim DI : Microvascular decompression of the facial nerve for the treatment of hemifacial spasm : Preoperative magnetic resonance imaging related to clinical outcomes. *Acta Neurochir (Wien)* 142 : 901-907, 2000
13. Dandy W : Concerning the cause of trigeminal neuralgia. *Am J Surg* 24 : 447-495, 1934
14. De Ridder D, Moller A, Verlooy J, Cornelissen M, De Ridder L : Is the root entry/exit zone important in microvascular compression syndromes? *Neurosurgery* 51 : 427-433; discussion 433-424, 2002
15. Defazio G, Berardelli A, Abbruzzese G : Primary hemifacial spasm and arterial hypertension : a multicenter case-control study. *Neurology* 54 : 1198-1200, 2000
16. Defazio G, Martino D, Aniello MS, Masi G, Logroscino G, Manobianca G, et al : Influence of age on the association between primary hemifacial spasm and arterial hypertension. *J Neurol Neurosurg Psychiatry* 74 : 979-981, 2003
17. Evidente VG, Adler CH : Hemifacial spasm and other craniofacial movement disorders. *Mayo Clin Proc* 73 : 67-71, 1998
18. Gardner JW, Sava GA : Hemifacial spasm : A reversible pathophysiologic state. *J Neurosurg* 19 : 240-247, 1962
19. Goto Y, Matsushima T, Natori Y, Inamura T, Tobimatsu S : Delayed effects of the microvascular decompression on hemifacial spasm : a retrospective study of 131 consecutive operated cases. *Neurol Res* 24 : 296-300, 2002
20. Grundy BL, Procopio PT, Jannetta PJ, Lina A, Doyle E : Evoked potential changes produced by positioning for retromastoid craniectomy. *Neurosurgery* 10 : 766-770, 1982
21. Hallett M : Blepharospasm : recent advances. *Neurology* 59 : 1306-1312, 2002
22. Hallett M, Daroff RB : Blepharospasm : report of a workshop. *Neurology* 46 : 1213-1218, 1996

23. Hatayama T, Møller AR : Correlation between latency and amplitude of peak V in the brainstem auditory evoked potentials : Intraoperative recordings in microvascular decompression operations. *Acta Neurochir (Wien)* 140 : 681-687, 1998
24. Hatem J, Sindou M, Vial C : Intraoperative monitoring of facial EMG responses during microvascular decompression for hemifacial spasm. Prognostic value for long-term outcome : a study in a 33-patient series. *Br J Neurosurg* 15 : 496-499, 2001
25. Holds JB, Alderson K, Fogg SG, Anderson RL : Motor nerve sprouting in human orbicularis muscle after botulinum A injection. *Invest Ophthalmol Vis Sci* 31 : 964-967, 1990
26. Holds JB, Fogg SG, Anderson RL : Botulinum A toxin injection. Failures in clinical practice and a biomechanical system for the study of toxin-induced paralysis. *Ophthalm Plast Reconstr Surg* 6 : 252-259, 1990
27. Huang CI, Chen IH, Lee LS : Microvascular decompression for hemifacial spasm : analyses of operative findings and results in 310 patients. *Neurosurgery* 30 : 53-56; discussion 56-57, 1992
28. Illingworth RD, Porter DG, Jakubowski J : Hemifacial spasm : a prospective long-term follow up of 83 cases treated by microvascular decompression at two neurosurgical centres in the United Kingdom. *J Neurol Neurosurg Psychiatry* 60 : 72-77, 1996
29. Ishikawa M, Nakanishi T, Takamiya Y, Namiki J : Delayed resolution of residual hemifacial spasm after microvascular decompression operations. *Neurosurgery* 49 : 847-854; discussion 854-856, 2001
30. Jankovic J, Schwartz K, Donovan DT : Botulinum toxin treatment of cranial-cervical dystonia, spasmodic dysphonia, other focal dystonias and hemifacial spasm. *J Neurol Neurosurg Psychiatry* 53 : 633-639, 1990
31. Jannetta PJ : The cause of hemifacial spasm : definitive microsurgical treatment at the brainstem in 31 patients. *Trans Sect Otolaryngol Am Acad Ophthalmol Otolaryngol* 80 : 319-322, 1975
32. Jannetta PJ : Neurovascular compression in cranial nerve and systemic disease. *Ann Surg* 192 : 518-525, 1980
33. Jannetta PJ : Observations on the etiology of trigeminal neuralgia, hemifacial spasm, acoustic nerve dysfunction and glossopharyngeal neuralgia. Definitive microsurgical treatment and results in 117 patients. *Neurochirurgia (Stuttg)* 20 : 145-154, 1977
34. Jannetta PJ, Kassam A : Hemifacial spasm. *J Neurol Neurosurg Psychiatry* 66 : 255-256, 1999
35. Jannetta PJ, Segal R, Wolfson SK : Neurogenic hypertension : etiology and surgical treatment. I. Observations in 53 patients. *Ann Surg* 201 : 391-398, 1985
36. Katusic S : Incidence and clinical features of trigeminal neuralgia : Rochester, Minnesota. *Ann Neurol* 27 : 89-95, 1990
37. Kenney C, Jankovic J : Botulinum toxin in the treatment of blepharospasm and hemifacial spasm. *J Neural Transm*, 2007
38. Kong DS, Park K, Shin BG, Lee JA, Eum DO : Prognostic value of the lateral spread response for intraoperative electromyography monitoring of the facial musculature during microvascular decompression for hemifacial spasm. *J Neurosurg* 106 : 384-387, 2007
39. Lang J : [Anatomy, length and blood vessel relations of "central" and "peripheral" paths of intracisternal cranial nerves]. *Zentralbl Neurochir* 43 : 217-258, 1982
40. Lang J, Reiter U : [Intracisternal length of cranial nerves 7-12]. *Neurochirurgia (Stuttg)* 28 : 153-157, 1985
41. Lang J, Reiter U : [Intracisternal length of the brain path and nerve tracts of the I to IV cranial nerves]. *Neurochirurgia (Stuttg)* 27 : 125-128, 1984
42. Lang J, Reiter U : [Intracisternal length of the trigeminal nerve]. *Neurochirurgia (Stuttg)* 27 : 159-161, 1984
43. Ledercq TA, Hill CL, Grisoli F : Retromastoid microsurgical approach to vascular compression of the eighth cranial nerve. *Laryngoscope* 90 : 1011-1017, 1980
44. Levy EI, Scarrow AM, Jannetta PJ : Microvascular decompression in the treatment of hypertension : review and update. *Surg Neurol* 55 : 2-11, 2001
45. Lunsford L, Niranjana A, Konziolka D : Surgical management options for trigeminal neuralgia. *J Korean Neurosurg Soc* 41 : 359-366, 2007
46. McLaughlin MR, Jannetta PJ, Clyde BL, Subach BR, Comey CH, Resnick DK : Microvascular decompression of cranial nerves : lessons learned after 4400 operations. *J Neurosurg* 90 : 1-8, 1999
47. Møller AR : Vascular compression of cranial nerves : II : pathophysiology. *Neurol Res* 21 : 439-443, 1999
48. Møller AR, Jannetta PJ : Hemifacial spasm : results of electrophysiologic recording during microvascular decompression operations. *Neurology* 35 : 969-974, 1985
49. Møller AR, Jannetta PJ : Microvascular decompression in hemifacial spasm : intraoperative electrophysiological observations. *Neurosurgery* 16 : 612-618, 1985
50. Møller AR, Jannetta PJ : On the origin of synkinesis in hemifacial spasm : results of intracranial recordings. *J Neurosurg* 61 : 569-576, 1984
51. Møller AR, Jannetta PJ : Physiological abnormalities in hemifacial spasm studied during microvascular decompression operations. *Exp Neurol* 93 : 584-600, 1986
52. Møller AR, Jannetta PJ : Synkinesis in hemifacial spasm : results of recording intracranially from the facial nerve. *Experientia* 41 : 415-417, 1985
53. Møller MB, Møller AR, Jannetta PJ : Brain stem auditory evoked potentials in patients with hemifacial spasm. *Laryngoscope* 92 : 848-852, 1982
54. Mooij JJ, Mustafa MK, van Weerden TW : Hemifacial spasm : intraoperative electromyographic monitoring as a guide for microvascular decompression. *Neurosurgery* 49 : 1365-1370; discussion 1370-1371, 2001
55. Nilsen B, Le KD, Dietrichs E : Prevalence of hemifacial spasm in Oslo, Norway. *Neurology* 63 : 1532-1533, 2004
56. Oliveira LD, Cardoso F, Vargas AP : Hemifacial spasm and arterial hypertension. *Mov disord* 14 : 832-835, 1999
57. Park JS, Kong DS, Lee JA, Park K : Intraoperative management to prevent cerebrospinal fluid leakage after microvascular decompression : dural closure with a "plugging muscle" method. *Neurosurg Rev* 30 : 139-142; discussion 142, 2007
58. Park YS, Chang JH, Cho J, Park YG, Chung SS, Chang JW : Reoperation for persistent or recurrent hemifacial spasm after microvascular decompression. *Neurosurgery* 58 : 1162-1167; discussion 1162-1167, 2006
59. Patrinely JR, Whiting AS, Anderson RL : Local side effects of botulinum toxin injections. *Adv Neurol* 49 : 493-500, 1988
60. Payner TD, Tew JM Jr. : Recurrence of hemifacial spasm after microvascular decompression. *Neurosurgery* 38 : 686-690; discussion 690-691, 1996
61. Polo G, Fischer C, Sindou MP, Marneffe V : Brainstem auditory evoked potential monitoring during microvascular decompression for hemifacial spasm : intraoperative brainstem auditory evoked potential changes and warning values to prevent hearing loss--prospective study in a consecutive series of 84 patients. *Neurosurgery* 54 : 97-104; discussion 104-106, 2004
62. Rhee DJ, Kong DS, Park K, Lee JA : Frequency and prognosis of delayed facial palsy after microvascular decompression for hemifacial spasm. *Acta Neurochir (Wien)* 148 : 839-843; discussion 843, 2006
63. Ryu H, Yamamoto S, Sugiyama K, Nishizawa S, Nozue M : Neurovascular compression syndrome of the eighth cranial nerve. Can the site of compression explain the symptoms? *Acta Neurochir (Wien)* 141 : 495-501, 1999
64. Saito S, Møller AR, Jannetta PJ, Jho HD : Abnormal response from the sternocleidomastoid muscle in patients with spasmodic torticollis : observations during microvascular decompression operations. *Acta Neurochir (Wien)* 124 : 92-98, 1993
65. Sanders DB : Ephaptic transmission in hemifacial spasm : a single-fiber EMG study. *Muscle Nerve* 12 : 690-694, 1989
66. Sekiya T, Iwabuchi T, Kamata S, Ishida T : Deterioration of auditory evoked potentials during cerebellopontine angle manipulations. *J Neurosurg* 63 : 598-607, 1985
67. Sindou M, Howeydi T, Acevedo G : Anatomical observations during microvascular decompression for idiopathic trigeminal neuralgia (with correlations between topography of pain and site of the neurovascular conflict) : Prospective study in a series of 579 patients. *Acta Neurochir (Wien)* 144 : 1-13, 2002
68. Sindou M, Fobe JL, Ciriano D, Fischer C : Hearing prognosis and intraoperative guidance of brainstem auditory evoked potential in microvascular decompression. *Laryngoscope* 102 : 678-682, 1992
69. Sindou MP : Microvascular decompression for primary hemifacial spasm. Importance of intraoperative neurophysiological monitoring.

- Acta Neurochir (Wien)** 147 : 1019-1026; discussion 1026, 2005
70. Sindou MP, Polo G, Fischer C, Vial C : Neurovascular conflict and hemifacial spasm. **Suppl Clin Neurophysiol** 58 : 274-281, 2006
 71. Skinner H : Some histologic features of the cranial nerves. **Arch Neurol Psychiatry** 25 : 356-360, 1931
 72. Tan NC, Tan EK, Khin LW : Diagnosis and misdiagnosis of hemifacial spasm : a clinical and video study. **J Clin Neurosci** 11 : 142-144, 2004
 73. Thusu A, Barman CR, Prabhakar S : Botulinum toxin treatment of hemifacial spasm and blepharospasm : objective response evaluation. **Neurol India** 47 : 206-209, 1999
 74. Wan XH, Vuong KD, Jankovic J : Clinical application of botulinum toxin type B in movement disorders and autonomic symptoms. **Chin Med Sci J** 20 : 44-47, 2005
 75. Watanabe E, Schramm J, Strauss C, Fahlbusch R : Neurophysiologic monitoring in posterior fossa surgery : Part II-BAEP-waves I and V and preservation of hearing. **Acta Neurochir (Wien)** 98 : 118-128, 1989
 76. Wilkins RH : Hemifacial spasm : a review. **Surg Neurol** 36 : 251-277, 1991