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Clinical Article

Infranuchal Infrafloccular Approach to the More Vulnerable Segments of the Facial Nerve in Microvascular Decompressions for the Hemifacial Spasm

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Objective : We investigated the locations of compressing vessels in hemifacial spasm. To approach compression sites, we described and evaluated the efficacy of the infranuchal infrafloccular (INIF) approach.
Methods : A retrospective review of 31 consecutive patients who underwent microvascular decompression (MVD) through INIF with a minimum follow-up of 1 year was performed. Along the intracranial facial nerve, we classified the compression sites into the transitional zone (TRZ), the central nervous system (CNS) segment and the peripheral nervous system (PNS) segment. The INIF approach was used to inspect the CNS segment and the TRZ. Subdural patch graft technique was used in order to achieve watertight dural closure. The cranioplasty was performed using polymethylmethacrylate. The outcome and procedure-related morbidities were evaluated.
Results : Twenty-nine patients (93%) showed complete disappearance of spasm. In two patients, the spasm was resolved gradually in 2 and 4 weeks, respectively. Late recurrence was noted in one patient (3%). The TRZ has been identified as the only compression site in 19 cases (61.3%), both the TRZ and CNS segment in 11 (35.5%) and the CNS segment only in 1 (3.2%). There was no patient having a compressing vessel in the PNS segment. Infection as a result of cerebrospinal fluid leak occurred in one patient (3%). Delayed transient facial weakness occurred in one patient.
Conclusion : The TRZ and the CNS segment were more vulnerable area to the compression of vessels. We suggest that surgical avenue with the INIF approach provides early identification of this area.

KEY WORDS : Hemifacial spasm · Microvascular decompression · Root exit zone.

INTRODUCTION

Hemifacial spasm (HFS) represents a segmental myoclonus of muscles innervated by the facial nerve. It is generally accepted that HFS is caused by compression of the facial nerve by blood vessels at root entry/exit zone (REZ)[13-15,20,23,29,39]. The REZ or the transitional zone (TRZ) has been regarded as the area at which the central glial myelin extending into the facial nerve from the brainstem meets the peripheral myelin composed of Schwann cell[6,12-15,22,23,29,35,37]. Janetta and colleagues hypothesized that vascular compression must be at the REZ which should be the target point of microvascular decompression (MVD)[13,14]. These terms such as the REZ and the TRZ, however, have often been confused with each other. Recently, more detailed anatomical description of the relationship between these terms and new hypothesis of vascular compression have been suggested[6,35]. Based on the recent report, the CNS segment and the TRZ are known to be the target area of MVD for HFS[6]. However, this hypothesis lacks clinical evidences. On the other hand, in order to reach this area, more basal bony opening is useful to get an early access and after opening the dura, infrafloccular approach can provide an early overview of this area especially on the CNS segment. Para-condylar fossa approach and lateral subocci-

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pital infrafloccular approach have been proposed to reach the REZ more efficiently[11,19]. We adopted these approaches and made a small modification for the purpose of more efficient decompression of the CNS segment and a closure of the dura. We performed MVDs for HFS using this approach in 31 consecutive patients. Retrospective analysis on the compression sites, procedure-related complications and clinical outcomes were carried out. In this report, we discussed which segment was more responsible for the occurrence of HFS. In addition, we described the surgical steps briefly and discussed the usefulness of INIF approach with an emphasis on the CNS segment decompression.

MATERIALS AND METHODS

Patient population

Thirty-one patients with HFS underwent MVD using this approach between March 2003 and November 2005. The patient population was composed of 21 women and 10 men with mean age of 47.5 years ranging from 25 to 75 years. Clinical symptoms are summarized in Table 1.

Surgical procedures

Following induction of endotracheal anesthesia, a three-point head fixation was applied. The patients with the thin and long neck were placed in the supine position and the patients with the short and thick neck in the lateral decubitus position with appropriate padding of pressure points. The head was rotated to the contralateral side, the vertex was inclined about 15 degrees toward the floor and the shoulder was taped down toward the foot in an attempt to expose easily the occipital bone below the inferior nuchal line. The inferior nuchal line, mastoid notch and tip of the mastoid process should be identified by palpation. For skin incision, a slightly curved line 5 cm in length is drawn at a position 1.5 cm posterior to the mastoid notch, which begins from the point 1 cm superior to the inferior nuchal line (Fig. 1). After exposure of the inferolateral occipital bone, a small key hole is made at the portion between the inferior nuchal line and the posterior condylar canal and then enlarged with a drill and a rongeur to the medial margin of the sigmoid sinus (Fig. 2). After dural opening, lower cranial nerves in the cistern should be identified, and then arachnoids dissection proceeds to the CNS segment and the root detachment point of the facial nerve. Through the infrafloccular avenue with gentle retraction of the flocculus in a caudorostral direction (infrafloccular approach), the root exit point and the CNS segment can easily be

Table 1. Clinical summary

Variables	No. of cases (%)
Side	
Sx. on right side	17 (55)
Sx. on left side	14 (45)
Types	
Typical	28 (90.4)
Atypical	3 (9.6)
Spasm	
Predominantly tonic	3 (9.6)
Predominantly clonic	28 (90.4)

Sx. : symptom

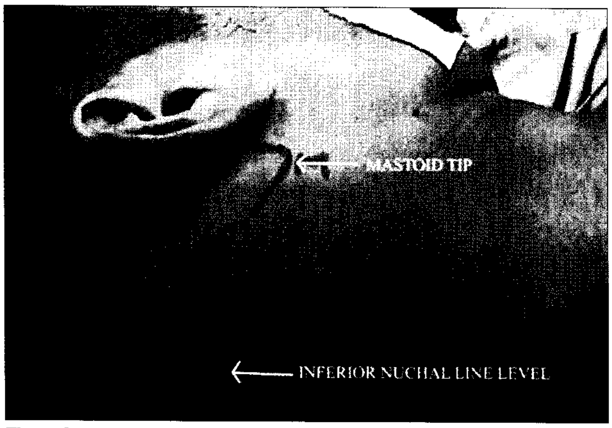

Fig. 1. Skin incision in the retroauricular region.

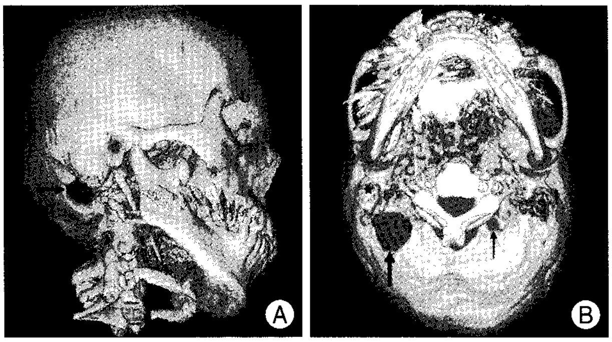

Fig. 2. Skull reconstruction 3D computed tomography obtained postoperatively. A : Lateral oblique view showing a bony opening below the inferior nuchal line and posterior to the mastoid process. Arrow : craniectomy. B : Basal view showing a bony opening. Star; mastoid process, thick arrow : craniectomy, thin arrow : contralateral posterior condylar canal.

Table 2. Operative findings

Compressing vessels and sites	No. of cases
Compressing vessels	
AICA	19
PICA	8
AICA + arteriole	2
AICA + small vein	1
PICA + small vein	1
Sites (%)	
TRZ	19 (61.3)
TRZ + CNS segment	11 (35.5)
CNS segment	1 (3.2)

AICA: anterior inferior cerebellar artery, CNS: central nervous system, PICA: posterior inferior cerebellar artery, TRZ: transitional zone

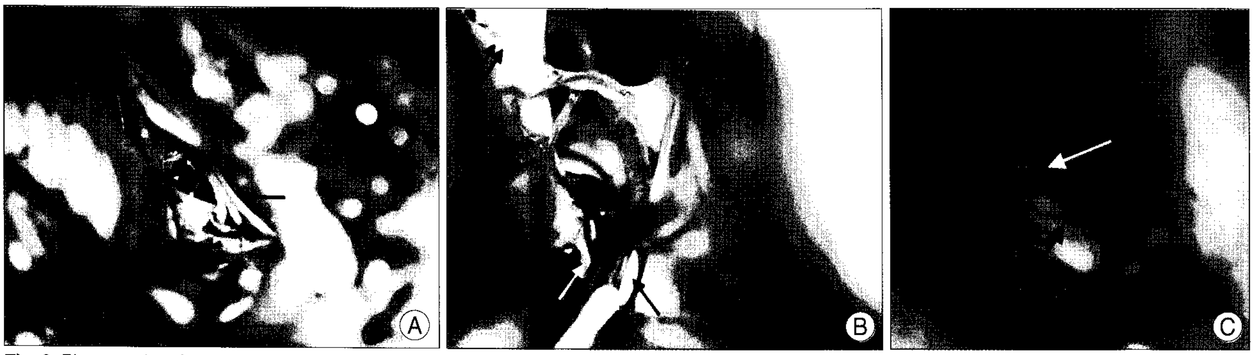

Fig. 3. Photographs of compressing vessels and sites. A : The anterior inferior cerebellar artery (AICA) loop (white arrows) compressing the facial nerve from the proximal area just above the ninth nerve to the nerve detaching point from the stem. Thick black arrow : lower cranial nerves, Thin black arrow : facial nerve. B : Photograph demonstrating two vessels located between the root detachment point and the ninth cranial nerve. Single arrow : glossopharyngeal nerve, double arrows : acoustic nerve. C : Photograph showing an AICA loop crossing the facial nerve below the root detachment point. Black arrow : AICA, white arrow : Root Detachment Point.

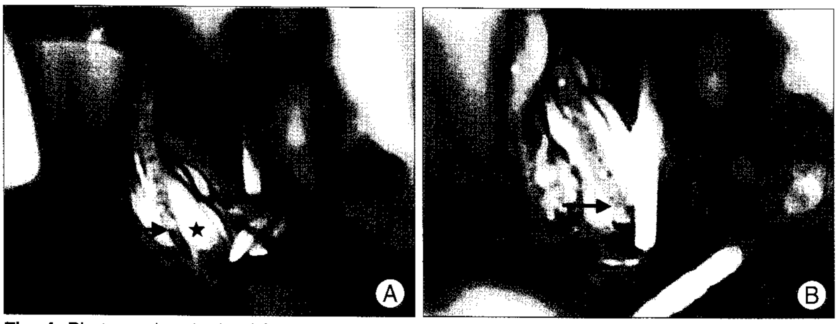

Fig. 4. Photographs obtained from the patient with an atypical hemifacial spasm. A : Photograph demonstrating the central nervous system segment (★) of the facial nerve. The glossopharyngeal nerve and the anterior inferior cerebellar artery (AICA) (black arrows) is identified. The loop is placed on the pontomedullary sulcus. B : After insertion of a piece of teflon felt beneath the AICA loop (black arrows), the compressing segment of the AICA can be observed which is displaced outwardly from the pontomedullary sulcus.

identified. The location of neurovascular compression was categorized based on the previous reports[4]. We defined the exit point of the facial nerve at which the facial nerve exited the brainstem as the upper end of the supraolivary fossette around the pontomedullary sulcus. During the operation, this site was easily observed because this portion is the most concave area below the facial nerve. The portion at which the facial nerve is detached from the pons was defined as root detachment point (RDP). As the previous report presented, the exact location of the RDP could almost always be determined and the point should offer a good landmark during procedures for HFS to locate the TRZ. The CNS segment of the facial nerve is located between the root exit point and the RDP. This segment shows white glistening color. The PNS segment with pale slightly gray color is distal to the RDP. The TRZ is located around the RDP where the central myelin is converted to the peripheral myelin. We used this classification as Campos-Benitez described.

Careful inspection should be performed keeping in mind that vascular compression can be present along the whole CNS segment and/or the TRZ. Two to three pieces of shredded Teflon felt are inserted between the nerve and the compressing vessel or vessels. The fascial patch graft or Neuro-Patch (B. Braun Medical S. A., Boulogne, France) is used for a dural closure. The patch is inserted subdurally in order to be pushed toward the dura by CSF pressure. For additional sealing, Tisseel fibrin glue (Biotek Pharma, Baxter Healthcare Ltd., Thetford, Norfolk, UK) was used over the epidural area and then the closure is reinforced with a piece of Gelfoam. Cranioplasty is performed using polymethylmethacrylate (PMMA) to prevent muscle adhesion to the dura. The wound is closed layer by layer.

RESULTS

The compressing vessels and sites are listed in Table 2. The AICA was the most common compressing vessel. In 4 cases, we identified more than 2 vessels compressing either the CNS segment or the TRZ (Fig. 3C). In terms of the compression sites (Fig. 3, 4), the TRZ has been identified as the only compression site in 19 (61.3%), both the TRZ and CNS segment in 11 (35.5%) and the CNS segment only in 1 (3.2%). There was no patient having a compressing vessel in the distal segment of the facial nerve. Among three cases with atypical HFS, two patients showed the TRZ compressed by the crossing vessel and the other one revealed compression at the pontomedullary junction that is the portion of the proximal CNS segment (Fig. 4). Twenty-nine patients (93.5%) showed complete disappearance of the HFS within one week after the operation. Five patients (16.1%) complained of spasm attacks once or twice in a day, which showed a shorter duration than those

Table 3. Results and postoperative complications

Results and complications	No. of cases
Results	
Spasm-free within a week	29
Spasm-free gradually	2
Spasm persisted	0
Recurrence	1
Complications	
CSF leak	1
Infection	1
Meningitis	1
Facial paresis	
Temporary	1
Permanent	0
Hearing impairment	0

CSF : cerebrospinal fluid

observed preoperatively. In two patients (6.5%), their spasm persisted for more than a week but ceased completely within four weeks. One of them showed recurrence of HFS with less severity seven months after the operation. She declined re-exploration. Operative complications are presented in Table 3. In one patient with diabetes mellitus, CSF rhinorrhea occurred 11 days after the operation. The patient eventually developed infection of the operation site and meningitis. In one patient (3.2%), facial paresis on the operation side occurred 2 months later but resolved completely through 3 weeks without specific treatment.

DISCUSSION

Compression sites

HFS has been reported to be caused by compression of the facial nerve on the REZ[13-15,20,23,29,39]. Therefore, it has been generally accepted that MVD of the compressing vessels from the REZ is an effective treatment option for HFS[3,12-17,19,22,23,26,29,33,39]. The REZ has been regarded as the area in which the sheath of the facial nerve is converted to the peripheral myelin composed of Schwann cells from the central glial myelin[6,13,35]. Some investigators have called this area as the Obersteiner-Redlich zone[24] or the TRZ, the region of transition from central to peripheral myelin[6,9,14]. On occasion, these terms have been confused with each other. The facial nerve exits from the stem at the upper end of the supraolivary fossette which is located just around the pontomedullary sulcus[6,35]. After exiting the brainstem, the facial nerve goes upward adhered strongly to the pons through the pia mater and connective tissue and then detached from the pons. Based on their anatomical study, Tomii et al.[35] proposed new terms such as the root exit point (RExP), the root detachment point (RDP) and the TRZ. They have clearly described differences between the RExP and the RDP. They also described the location of the TRZ which was observed between 8 to 9.9 mm distant from the RExP in the lateral portion of the facial nerve which had a mean length of 1.9 mm[35]. The RDP of the facial nerve at the medial side was located very close to the beginning of the medial TRZ in their report. The TRZ has been a target area to be decompressed in MVD. De Rider et al.[6], however, suggested that vascular compression syndromes can be caused by vascular contact occurs along the CNS segment of a cranial nerve and not specifically at the TRZ, as previously thought. In this report, we investigated the location of compression according to the anatomical descriptions proposed by the above mentioned authors. Our results revealed that the most common compression site was the TRZ not the CNS segment. In eleven cases, vascular compressions were observed at both the CNS segment and the TRZ by a single or multiple vessels (Fig. 3, 4). We usually decompressed all vessels involving the TRZ and the CNS segment except one patient. In that patient, a small vein was crossing the CNS segment and the AICA was crossing the TRZ. We decompressed the anterior inferior cerebellar artery alone. Postoperatively, the spasm disappeared. In one patient with atypical HFS, we identified the only compression site was the CNS segment around the pontomedullary sulcus. There was no case that showed compression on the PNS segment. Even in the cases showing vascular contacts with the facial nerve distal to the TRZ, we can identify compressing vessel or vessels around the TRZ. Our results showed that the TRZ was the compression site in almost all cases except one. But, in the cases showing compression in both the CNS segment and the TRZ, we could not distinguish which site is more responsible for occurrence of the HFS. Several authors with large clinical series have indicated that the lower segment to the pons should be decompressed[11,19,33]. Conversely, the lower segment that is the CNS segment may be a causative site of the HFS. In our report, there was a patient in whom only the CNS segment was compressed. Recently, there have been reports that not only the TRZ/CNS but also the other segments of the facial nerve can cause the HFS[8,10,18,28,31,36]. The distal cisternal segment and even the extracranial portion of the facial nerve have been reported to be sites causing HFS[2,7,8,10,18,28,31]. In patients with the HFS, both partial demyelination and axonal degeneration of the seventh cranial nerve may be essential to produce the hyperactivity of the facial motor nucleus. In an experimental animal study by Kuroki and Møller[20], they showed that close contact between a peripheral branch of the facial nerve and an artery also facilitated the development of an abnormal muscle response, but only if the facial nerve had previously been slightly injured (by a chromic suture) at the location of the arterial contact. Based on the previous reports[1,2,7,8,18,27,28,30,31,34,36] and our results, HFS may be caused along any segment of the facial nerve by a stimulus which can cause injury such as demyelination. The stimulus should be enough to cause demyelination, which may be the reason why arteries are more frequently reported to be an offending vessel than veins or capillaries. Conversely, if the intensities of stimuli are within a constant range such as an arterial pulsation the occurrence of HFS may be dependent on the site. The PNS segment ensheathed with Schwann cells is generally accepted to be more resistant to vascular compression causing demyelination[6,35]. But, if there is an injury, the HFS may be developed as shown in the reports[31,36]. The TRZ may be the weakest point and the CNS segment may be more vulnerable to the mechanical irritation than the PNS segment. Ryu et al.[28] insisted that the REZ is the primary site of vascular compression causing HFS in most cases and so this portion should be decompressed initially and then the distal portion of the seventh cranial in cases in which neurovascular compression at the REZ was not confirmed during surgery. To achieve successful outcomes in MVD for HFS, attention should be placed firstly on more vulnerable segments and then on the other segments.

We have experienced three cases with atypical HFS in which spasm began in the buccal muscles, with progressive spread upward to involve the orbicularis oculi muscle. It has been suggested that compression of the facial nerve at the posterorostral side could have caused atypical HFS[27]. In our patients with atypical HFS, however, the TRZ was the compression site in two patients and the CNS segment was in one patient. Further clinical study in a larger population would be needed to seek differences between typical and atypical HFS.

Infra-nuchal infra-floccular approach

To inspect compressing vessels thoroughly at the TRZ and the CNS segment of the facial nerve, it may be easier to approach those areas from the inferolateral portion of the posterior fossa. The proximal CNS segment started from the upper edge of the supraolivary fossette around the pontomedullary sulcus. The ninth nerve is a good landmark to identify the RExP after opening the cerebellomedullary cistern. We usually try to inspect carefully from the RExP to the RDP because this area is more vulnerable segment. The lateral suboccipital retrosigmoid approach is commonly used to treat lesions in the cerebellopontine angle. This approach has been modified according to the specific surgical anatomy required for certain operations including cerebrovascular, tumor surgery and also MVD[3,5,19,21,25,32,38]. Advancement of surgical instruments, operating microscope and endoscope has made minimally invasive modifications of this approach. Furthermore, to prevent or reduce the procedure-related morbidity, some technical tips have been added. Among several modified approaches, the lateral suboccipital infrafloccular approach or the paracondylar fossa approach is the one which provide direct access to the more vulnerable segment of the facial nerve[11,19]. In these approaches, bony opening is located below the inferior nuchal line. The occipital bone in this area is usually thin so it is easy to open. While extending the bony opening to the margin of the sigmoid sinus which courses medially to the jugular foramen, we seldom face the mastoid bone overhanging the line of surgical view. The bony opening usually located in the occipital bone itself, which make it possible not to open the mastoid air cells. Consequently, we can reduce the risk of CSF leak. In addition, these are more useful in cases with the large flocculus. Sindou[33] described why this infrafloccular route is important. First, the compressing vessels are usually located ventrocaudally at the REZ which may mean, in other words, compressing usually occurs at the more vulnerable segments. The other reason he suggested is that using this route can avoid a lateral-to-medial retraction of the cerebellar hemisphere which would exert stretching of the eighth nerve, causing a hearing loss[33]. Hitotsumatsu et al.[11] indicated that the REZ could be easily observed with gentle retraction of the flocculus in a caudorostral direction perpendicular to the VIIIth cranial nerve during this approach. The direction of retraction perpendicular to the VIIIth cranial nerve can minimize the risk of hearing impairment. In more than 50% of our cases, we were able to perform MVDs without a routine use of retractors which resulted in no hearing impairment. To expose more basal occipital bone over the cistern magna, caudomedial extension of skin incision may be helpful as described by Hitotsumatsu et al.[11]. However, we usually have made a curvilinear skin incision of slight extension to the medial side which was sufficient for the bony opening (Fig. 1). We also usually used the inferior nuchal line as the bony landmark so named it INIF approach. This is not a new one but just an integrative modification of several authors' procedures. This approach facilitates both view and access to the more vulnerable segments of the facial nerve.

CONCLUSION

In our investigation, almost all compressing vessels in HFS are identified at the TRZ alone and at both the TRZ and the CNS segment. In one case, compression was identified around the pontomedullary junction that is the CNS segment. We suggest that the TRZ and the CNS segment may be more vulnerable to the mechanical stimulus such as the pulsation of vessels than the PNS segment. To inspect this region and perform MVD more safely, the INIF approach is more effective. Further study on the compressing sites should be necessary to confirm and further evaluations of applications of this approach are needed.

References

1. Altinörs N, Kars Z, Cepoğlu C : Rare causes of hemifacial spasm. Report of two cases. **Clin Neurol Neurosurg 93** : 155-158, 1991
2. Asaoka K, Sawamura Y, Tada M, Abe H : Hemifacial spasm caused by a hemangioma at the geniculate ganglion : case report. **Neurosurgery 41** : 1195-1197, 1997
3. Badr-El-Dine M, El-Garem HF, Talaat AM, Magnan J : Endoscopically assisted minimally invasive microvascular decompression of hemifacial spasm. **Otol Neurotol 23** : 122-128, 2002
4. Campos-Benitez M, Kaufmann AM : Neurovascular compression findings in hemifacial spasm. **J Neurosurg 109** : 416-420, 2008
5. Cueva RA, Mastrodimos B : Approach design and closure techniques to minimize cerebrospinal fluid leak after cerebellopontine angle tumor surgery. **Otol Neurotol 26** : 1176-1181, 2005
6. De Ridder D, Møller A, Verlooy J, Cornelissen M, De Ridder L : Is the root entry/exit zone important in microvascular compression syndromes? **Neurosurgery 51** : 427-433; discussion 433-434, 2002
7. Fukuda M, Kameyama S, Honda Y, Tanaka R : Hemifacial spasm resulting from facial nerve compression near the internal acoustic meatus -- case report. **Neurol Med Chir (Tokyo) 37** : 771-774, 1997
8. Gálvez-Jiménez N, Hanson MR, Desai M : Unusual causes of hemifacial spasm. **Semin Neurol 21** : 75-83, 2001
9. Gardner WJ : Concerning the mechanism of trigeminal neuralgia and hemifacial spasm. **J Neurosurg 19** : 947-958, 1962
10. Han IB, Chang JH, Chang JW, Huh R, Chung SS : Unusual causes and presentations of hemifacial spasm. **Neurosurgery 65** : 130-137; discussion 137, 2009
11. Hitotsumatsu T, Matsushima T, Inoue T : Microvascular decompression for treatment of trigeminal neuralgia, hemifacial spasm, and glossopharyngeal neuralgia : three surgical approach variations : technical note. **Neurosurgery 53** : 1436-1441; discussion 1442-1443, 2003
12. Hopf HC, Lowitzsch K : Hemifacial spasm : location of the lesion by electrophysiological means. **Muscle Nerve 5** : S84- S88, 1982
13. 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
14. Jannetta PJ, Abbasy M, Maroon JC, Ramos FM, Albin MS : Etiology and definitive microsurgical treatment of hemifacial spasm. Operative techniques and results in 47 patients. **J Neurosurg 47** : 321-328, 1977
15. Jannetta PJ, McLaughlin MR, Casey KF : Technique of microvascular decompression. Technical note. **Neurosurg Focus 18** : E5, 2005
16. Kabil MS, Eby JB, Shahinian HK : Endoscopic vascular decompression versus microvascular decompression of the trigeminal nerve. **Minim Invasive Neurosurg 48** : 207-212, 2005
17. Kalkanis SN, Eskandar EN, Carter BS, Barker FG 2nd : Microvascular decompression surgery in the United States, 1996 to 2000 : mortality rates, morbidity rates, and the effects of hospital and surgeon volumes. **Neurosurgery 52** : 1251-1261; discussion 1261-1262, 2003
18. Kiley MA, Voyvodic F, Burns RJ : An unusual cause of hemifacial spasm. **J Clin Neurosci 6** : 349-351, 1999
19. Kim EY, Park HS, Kim JJ, Lee SC, Ha CK, Park HC : A more basal approach in microvascular decompression for hemifacial spasm : the para-condylar fossa approach. **Acta Neurochir (Wien) 143** : 141-144; discussion 145, 2001
20. Kuroki A, Møller AR : Facial nerve demyelination and vascular compression are both needed to induce facial hyperactivity : a study in rats. **Acta Neurochir (Wien) 126** : 149-157, 1994
21. Li N, Zhao WG, Pu CH, Shen JK : Clinical application of artificial dura mater to avoid cerebrospinal fluid leaks after microvascular decompression surgery. **Minim Invasive Neurosurg 48** : 369-372, 2005
22. 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
23. Møller AR : Hemifacial spasm : ephaptic transmission or hyperexcitability of the facial motor nucleus? **Exp Neurol 98** : 110-119, 1987
24. Obersteiner H, Redlich E : Uber Wesen und Pathogenese der tabischen Hinterstrangsdegeneration. **Arb Neurol Inst Univ Wien 1-3** : 158-172, 1894
25. Quiñones-Hinojosa A, Chang EF, Lawton MT : The extended retrosigmoid approach : an alternative to radical cranial base approaches for posterior fossa lesions. **Neurosurgery 58** : ONS208-ONS214; discussion ONS214, 2006
26. Rak R, Sekhar LN, Stimac D, Hechl P : Endoscope-assisted microsurgery for microvascular compression syndromes. **Neurosurgery 54** : 876-881; discussion 881-883, 2004
27. Ryu H, Yamamoto S, Miyamoto T : Atypical hemifacial spasm. **Acta Neurochir (Wien) 140** : 1173-1176, 1998
28. Ryu H, Yamamoto S, Sugiyama K, Uemura K, Miyamoto T : Hemifacial spasm caused by vascular compression of the distal portion of the facial nerve. Report of seven cases. **J Neurosurg 88** : 605-609, 1998
29. Samii M, Günther T, Iaconetta G, Muehling M, Vorkapic P, Samii A : Microvascular decompression to treat hemifacial spasm : long-term results for a consecutive series of 143 patients. **Neurosurgery 50** : 712-718; discussion 718-719, 2002
30. Sandberg DI, Souweidane MM : Hemifacial spasm caused by a pilocytic astrocytoma of the fourth ventricle. **Pediatr Neurol 21** : 754-756, 1999
31. Shimizu S, Tanaka R, Sato S, Fujii K : Hemifacial spasm following a blow to the mandible causing blunt injury to the peripheral facial nerve. **Neurol Med Chir (Tokyo) 45** : 192-195, 2005
32. Silverman DA, Hughes GB, Kinney SE, Lee JH : Technical modifications of suboccipital craniectomy for prevention of postoperative headache. **Skull Base 14** : 77-84, 2004
33. Sindou MP : Microvascular decompression for primary hemifacial spasm. Importance of intraoperative neurophysiological monitoring. **Acta Neurochir (Wien) 147** : 1019-1026; discussion 1026, 2005
34. Tanaka A, Tanaka T, Irie Y, Yoshinaga S, Tomonaga M : Elevation of the petrous bone caused by hyperplasia of the occipital bone presenting as hemifacial spasm: diagnostic values of magnetic resonance imaging and three-dimensional computed tomographic images in a bone anomaly. **Neurosurgery 27** : 1004-1009, 1990
35. Tomii M, Onoue H, Yasue M, Tokudome S, Abe T : Microscopic measurement of the facial nerve root exit zone from central glial myelin to peripheral Schwann cell myelin. **J Neurosurg 99** : 121-124, 2003
36. Wang HC, Lu CH, Lee RJ, Yang TM, Hung KS : Post-traumatic hemifacial spasm. **J Clin Neurosci 13** : 681-683, 2006
37. Yamakami I, Oka N, Higuchi Y : Hyperactivity of the facial nucleus produced by chronic electrical stimulation in rats. **J Clin Neurosci 14** : 459-463, 2007
38. Yamashima T, Lee JH, Tobias S, Kim CH, Chang JH, Kwon JT : Surgical procedure "Simplified Retrosigmoid Approach" for C-P angle lesions. **J Clin Neurosci 11** : 168-171, 2004
39. Yuan Y, Wang Y, Zhang SX, Zhang L, Li R, Guo J : Microvascular decompression in patients with hemifacial spasm: report of 1200 cases. **Chin Med J (Engl) 118** : 833-836, 2005