

Analysis of Microvascular Decompression for the Treatment of Trigeminal Neuralgia and Hemifacial Spasm

Koreaki Mori, Masanori Morimoto, Masahiro Kurisaka, Yasufumi Uchida, and Patrick Eghwrudjakpor

> Department of Neurosurgery Kochi Medical School Received for Publication, July. 4, 1986.

Abstract

Results of the treatment of 61 patients with trigeminal neuralgia (TN) and 65 patients with hemifacial spasm (HES) by microvascular decompression (MVD) and their problems are here reported. In atypical TN, the results were less favorable. Based on the degree of abnormal vascular contact on the trigeminal nerve, the root entry zone of the trigeminal nerve in TN may be more extensive than the root exit zone of the facial nerve in HFS. In cases of HFS, MVD should be limited to typical cases with care being taken to thoroughly examine the root exit zone in order not to miss any offending vessel. It is also necessary to avoid undue manipulation to prevent operative complications.

Introduction

Based on Dandy's³⁾, Sunderland's²⁵⁾ and Gardner's^{5,6)} observations of abnormal vascular contacts with cranial nerves in patients with trigeminal neuralgia (TN) and hemifacial spasm (HFS), a new concept of "cranial nerves and brain stem vascula rcompression syndrome" was developed by Jannetta^{14,16)}.

With the development of microsurgical techniques it has become quite common in many neurosurgical institutes to perform microvascular decompression^{4,7,17)} which was established by JANNETTA^{10,11,12,13)}; and this has become a definite treatment for TN and HFS.

Between 1982 and 1985, we performed MVD on a total of 117 patients at the Kochi Medical School. In this paper, the patients are analyzed and problems in the diagnosis and treatment of TN and HFS are discussed.

Key words: Cranial nerve vascular compression syndrome, Hemifacial spasm, Microvascular decompression, Trigeminal neuralgia

索引語:脳神経血管圧迫症候群,顔面痙攣,微小血管減圧術,三叉神経痛.

Present address: Department of Neurosurgery Kochi Medical School, Kohasu Oko-cho, Nankoku City, Kochi 781-51, Japan.

Summary of Patients

1. Trigeminal neuralgia

Sixty-one patients including 9 cases of "symptomatic" TN were operated upon. Microvascular decompression of the trigeminal nerve was performed in 52 of 61 cases-21 males and 31 females-with ages ranging from 29 to 77 years to give an average of 56.5 years. The operative findings and results are shown in Table 1 and postoperative complications in Table 2.

On the basis of clinical symptoms, 33 of 52 patients were considered to be typical. In these patients compression of the trigeminal nerve by a vessel loop was found in 88%. Complete relief of neuralgia was achieved in all of them following MVD. In the remaining nineteen patients considered to be atypical, the frequency of neurovascular compression was less than in the typical cases and complete relief of neuralgia was achieved only in 47%. It was observed that arterial compression of the trigeminal nerve was not necessarily confined to the area of the nerve immediately adjacent to the brain stem in all patients; but that in some, compression was seen distal to this area. The superior cerebellar artery (SCA) and its branches was most frequently incriminated, followed by the anterior inferior cerebellar artery (AICA), basilar artery (BA) and vertebral artery (VA) respectively. In 5 patients only a tributary of the petrosal vein or a pontine vein compressed the trigeminal nerve.

Post-operative complications were attributable mainly to either operative approach or manipulation. There was no operative mortality.

nature of neuralgia	neurovascular compression		outcome	
typical 33	(+)	29	complete relief	28
			incomplete relief	1
	(-)	4	complete relief	1
			incomplete relief	3
atypical 19	(+)	10	complete relief	5
			incomplete relief	5
	(-)	9	complete relief	4
			incomplete relief	5

Table 1. Summary of 61 cases of trigeminal neuralgia

Table 2	,	Postoperative	complications	in	trigeminal	neurale	ria.
Table 4	2.	Postoperative	complications	ın	trigeimmai	neurais	2.

sensory disturbance in the face	5
hearing disturbance	4
ataxia	4
transient diplopia	3
csf otorrhea	2
herpes	2
tinnitus	1
meningitis	1

case No	age	sex	nature of pain	V nerve sign	other c. n. sign	previous treatment	radiological abnormality	operative findings	results
1	71	M	typical	(-)	(-)	medication	(-)	epidermoid	pain free, no deficit
2	49	F	atypical	(+)	(-)	med., block	(+)	epidermoid	recurrence-rhizotomy
3	54	M	atypical	(+)	(+)	med., block	(+)	neurinoma	pain free, facial palsy
4	50	M	typical	(-)	(-)	med., block	(+)	AVM	pain free, no deficit
5	53	F	typical	(+)	(+)	med., block	(+)	neurinoma	pain free, facial palsy
6	51	F	atypical	(-)	(+)	med.	(+)	aneurysm	pain free, no deficit
7	54	M	typical	(+)	(+)	med., block	(+)	epidermoid	pain free, 9th n. palsy
8	52	M	atypical	(-)	(+)	med.	(+)	aneurysm	pain free, no deficit
9	68	M	atypical	(+)	(-)	med.	(-)	metastasis	anesthesia dolorosa

Table 3. Symptomatic trigeminal neuralgia

V: trigeminal nerve, c. n.: cranial nerve

Nine of 61 patients (15%) had "symptomatic" TN (Table 3). Their average age was 50.4 years which is a little less than that of patients with idiopatic TN. Six of these 9 patients showed symptoms typical of TN. In 2 patients diagnosis was not made preoperatively. The SCA was found to be sandwiched between a cerebello-pontine angle tumor and the trigeminal nerve at its entry zone in some patients. Complete relief of pain was obtained in 7 patients; but 2 required rhizotomy for relief.

2. Hemifacial spasm

65 patients were treated by MVD—22 males and 43 females with ages ranging from 17 to 74 years (average age—53.4 years). The operative findings and results in these patients is shown in Table 4. In 82% complete disappearance of spasm was obtained. In 7 of ten referal cases who had an initial operation without satisfactory results, we found the offending arterial loop at the root exit zone of the facial nerve at the second operation. We believe that it may have been missed at the earlier operation. Of 18 patients in whom spasm didn't disappear immediately following MVD, 4 subsequently showed spontaneous disappearance. Eight of 65 patients were condidered to be atypical on clinical basis—with 3 postparalytic, 3 young patients, 1 bilateral and 1 blepharospasm-like. In these patients, operation was exploratory. Arterial compression on the facial nerve was found in three of them. Of the remaining five, who didn't have any vascular compression, one had spontaneous disappearance of his symptom subsequently.

The offending artery in most cases was the AICA and its branches, followed by the PICA and VA.

Hearing disturbance of varying degrees was the only postoperative complication of significance being noted in 18% of the cases. In 75% of patients with this complication, it was believed to be a result of spasm of the labyrinthine artery which probably occurred as a result of manipulation during surgery.

In both TN and HFS, the effect of MVD seemed not to be influenced by such previous destructive operations as nerve block in our series¹⁾.

arterial compression initial operative results follow-up results at the root exit zone complete disappearance (+) 57 cases (88%) complete disappearance 52 cases (91%) 45 cases (79%) partial improvement partial improvement 12 cases (21%) 5 cases (9%) (7 cases reoperated) (-) 8 cases (12%) complete disappearance complete disappearance none 1 cases (12%) partial improvement partial improvement 8 cases (100%) 7 cases (88%) (3 cases reoperated)

Table 4. Summary of 65 cases of hemifacial spasm

Table 5. Postoperative complications in hemifacial spasm

	•
transient facial weakness	20
hearing disturbance	12

Discussion

1. Preoperative evaluation

It is not always easy to distinguish between "symptomatic" and "idiopathic" TN on basis of clinical symptomatology alone. The typical pain characteristic of "idiopathic" neuralgia may be seen in "symptomatic" neuralgia. In our series, 6 of 9 patients with "symptomatic" neuralgia presented the typical pain picture. This underscores the need for performance of angiography and/or CT cisternography when routine CT examinations—plain and contrast enhanced—reveal evidence of such cerebellopontine angle tumors as epidermoid^{9,19,27,29)} and neurinoma; and such vascular anomalies as aneurysm and arteriovenous malformation. The possibility of symptomatic TN should always be considered in young patients.

Although there was no single case of "symptomatic" facial spasm in this series, it does occur albeit infrequently. Symptomatic spasm due to aneurysm of the vertebrobasilar system and arteriovenous malformation has been reported in the literature^{18,22})

Prospective analysis of the offending vessel was carried out by vertebral angiography in the initial 100 cases. Cerebral angiograms contributed to the diagnosis of symptomatic TN caused by vascular malformations and to evaluations of the degree of arteriosclerosis where present. However, they did not necessarily predict the presence or absence of compressing vessels. In atypical cases of TN and HFS, there was generally a less likelihood of neurovascular compression, and so less favorable results from MVD. Consequently, we believe, indications for MVD should be carefully determined in all atypical cases.

2. Operative procedures

MVD was performed by Jannettas's method¹²). The patient is placed in the contralateral lateral decubitus position and a small unilateral retromastoid craniectomy is performed. MVD of the appropriate cranial nerve entry or exit zone is then carried out using mircosugical

techniques. This involves mobilization of the vessel away from the nerve and insertion of teflon felt between them.

a) Identification of the offending vessel and its separation from the nerve.

Among cases in whom MVD was performed, neurovascular compression was identified in 75% of trigeminal neuralgia and 88% of hemifacial spasm respectively. Approximately 10% of the vessels compressing on the trigeminal nerve were veins²³, and 13% were combination of artery and vein. The site of adhesion and/or compression varied from the area of the trigeminal nerve adjacent to the brainstem to its distal portion. Where the offending vessels were found to be veins, these were carefully coagulated since their collaterals develop easily and these may even be a cause of early recurrence in TN¹⁵. Even small veins around the nerve root should be obliterated by meticulous electrocoagulation. In patients in whom no offending vessel is found at the entry zone, a partial rhizotomy may be effective.

In an operation on "symptomatic" TN, any vessel sandwiched between a cerebellopontine angle tumor and the trigeminal nerve should be decompressed as the offending vessel. Marked indentation of the nerve by such vessels was observed in several of our patients.

In HFS, all the vessels compressing on the facial nerve were arteries and were identified at the exit zone.

(These results show that the "root entry zone" of the trigeminal nerve in TN may more extensive



Fig. 1(a). Intraoperative photograph of the trigeminal nerve compressed by the branch of the superior cerebellar artery from below at the root entry zone.

The trigeminal nerve is flattened and elevated by the offending artery close to the pons.

than the "root exit zone" of the facial nerve in HFS.)

Patients who showed no improvement of symptoms after two weeks of the initial operation were re-operated upon. In seven of them a small arterial loop was identified at the exit zone of the facial nerve. In some of these patients only one of two branches of AICA or PICA had been decompressed at the initial operation. It is our policy in MVD for HFS to reoperate whenever no improvement in facial spasm is observed two weeks after the first operation.

In the earlier operations in our series, a piece of plastic sponge was used as the implant. This however had the potential drawback of slipping out or causing the arterial loop to kink following seperation from the nerve. After introduction of teflon felt¹⁵, small wisps of this material have been inserted between offending vessel and nerve without significant complication (Fig. 1). By this procedure even sclerotic arterial loops can be separated from the nerve without kinking. An elongated and ectatic basilar artery and vertebral artery^{2,26,28} can be decompressed without difficulty.

b) Prevention of complications in MVD

Undue retraction of the cerebellum and cranial nerve can be avoided if the direction of approach is appropriate. The lateral margin of the retromastoid craniectomy should be drilled away sufficiently and any exposed mastoid air cells should be plugged with bone wax. It will be helpful to have tapered brain spatulas of varying sizes in order to ensure adequate visualization



Fig. 1(b). Intraoperative photograph of the placement of small wisps of teflon felt between the trigeminal nerve and the branch of the superior cerebellar artery.

of the root entry or exit zone. Proper adjustment of the operation table and positioning of the operation microscope are invaluable in ensuring a satisfactory approach.

Approach to the trigeminal nerve is preferably from a cranial to caudal direction, not between the 5th and 8th nerves. When petrosal veins are in the way, they can be safely coagulated and divided. Injury to these veins during mobilization of the offending vessel often results in very troublesome bleeding which may render decompression incomplete.

In HFS, the approach to the facial nerve and its entry zone is usually between the 8th nerve and vagus group and not from above the 8th nerve. An approach to the facial nerve from above would require that undue traction be applied to the 8th nerve, and considerable retraction of the cerebellum would be needed for visualization of the exit zone. Hearing disturbance may occur as a result of either prolonged 8th nerve retraction or injury or spasm resulting from manipulation of the labyrinthine artery^{20,24}. Intraoperative monitoring using auditory evoked brainstem response (AEBR) may be helpful in prevention of hearing disturbance^{8,21}. We however do not routinely carry out intraoperative monitoring of AEBR in all cases in our department. Reliability is sometimes questioned and slight abnormalities like prolongation of latency of the fifth wave may not be clinically significant in cases where MVD is properly performed. Facial weakness due to operative manipulation during MVD usually recovers within two months after operation.

c) MVD for syndromes other than TN and HFS

Experience in MVD for other neurovascular compression syndromes is limited.

When we perform MVD for TN and HFS on the left side in patients with essential hypertension, we usually explore the lateral aspect of the medulla for possible compression of the vertebral artery or the PICA on the medulla. If any compressing vessel is present, it is decompressed in addition to the MVD for TN or HFS.

It is however too early in this series to discuss the effect of MVD on essential hypertension. Further follow-up of the cases may proove its usefulness or otherwise.

Summary and Conclusion

- 1. Results of the treatment for TN-61 cases and HFS-65 cases by MVD and their problems were reported.
- 2. Operative results were less favorable in atypical neuralgia. We suggest that care be taken to avoid mixing up symptomatic and atypical cases as the ideal treatment would differ for each one. MVD should be limited to typical cases in HFS and it is of utmost importance to thoroughly examine the root exit zone in order not to miss the offending artery (or arteries).
- 3. Based on the extent of abnormal vascular contact, the trigeminal nerve root "entry zone" may be more extensive than the facial nerve root "exit zone"
- 4. Operative complications will be largely prevented by avoiding unnecessary manipulation, and undue retraction. Intraoperative monitoring of AEBR may be helpful for the protection of hearing acuity.

A part of this paper was presented at the 44th Annual Meeting of Japan Neurosurgical

Society held in Nagasaki in 1985.

Acknowledgment

The authors wish to thank Dr. PPETER J. JANNETTA, Division of Neurosurgery, University of Pittsburgh who kindly reviewed their English text and gave us valuable comments on the paper.

References

- Barba D, Alksne JF: Success of microvascular decompression with and without prior surgical therapy for trigeminal neuralgia. J Neurosurg 60: 104-207, 1984.
- 2) Carella A, Caruso G, Lamberti P: Hemifacial spasm due to elongation and ectasia of the distal segment of the vertebral artery. Report of two cases. Neuroradiology 6: 233-6, 1973.
- 3) Dandy WE: Concerning the cause of trigeminal neuralgia. Am J Surg 24: 447-455, 1934.
- 4) Fukushima T: Posterior fossa microvascular decompression in the management of hemifacial spasm and trigeminal neuralgia. Neurological Surgery (Tokyo) 10: 1257-1261, 1982.
- Gardner WJ: Concerning the mechanism of trigeminal neuralgia and hemifacial spasm. J Neurosurg 19: 947-957, 1962.
- Gardner WJ, Sava GA: Hemifacial spasm. A reversible pathophysiologic state. J Neurosurg 19: 240-7, 1962.
- Goya T, Kinoshita K, Yamakawa Y, et al: Hemifacial spasm. Analysis of 40 cases of neurovascular decompression. Neurol Med Chir (Tokyo) 23: 651-658, 1983.
- 8) Grundy BL, Lina A, Procopio PT: Reversible evoked potential changes with retraction of the eight cranial nerve. Anesth Analg 60: 835-838, 1981.
- 9) Hori T, Numata H, Hokama Y, et al: Trigeminal pain caused by a parapontine epidermoid cyst. Surg Neurol 19: 517-519, 1983.
- Jannetta PJ: Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia.
 J Neurosurg 26: 1159-1162, 1967.
- 11) Jannetta PJ: Microsurgical exploration and decompression of the facial nerve in hemifacial spasm. Curr Topics Surg Res 2: 217-220, 1970.
- 12) Jannetta PJ, Abbasy M, Maroon JC, et al: Etiology and definitive micosurgical treatment of hemifacial spasm, operative techniques and results in 47 patients. J Neurosurg 47: 321-328, 1977.
- 13) Jannetta PJ: Microsurgery of the cranial nerve crosscompression. Clin Neurosurg 26: 607-615, 1979.
- 14) Jannetta PJ: Neurovascular compression in cranial nerve and systemic disease. Ann Surg 192: 518-525, 1980.
- 15) Jannetta PJ: Personal communication. 1982.
- 16) Jannetta PJ: Posterior fossa neurovascular compression syndromes other than neuralgia. In Wilkins RH. Rengachary SS (eds). Neurosurgery 1985; vol 2, McGraw-Hill, New York, p. 1901-1906.
- 17) Kondo A, Ishikawa J, Yamasaki T, et al: Microvascular decompression of cranial nerve, particularly of the 7th cranial nerve. Neuol Med Chir (Tokyo) 20: 739-751, 1980.
- 18) Maroon JC, Lunsford LD, Deeb ZI: Hemifacial spasm due to aneurysmal compression of the facial nerve. Arch Neurol 35: 545-546, 1978.
- 19) Miyazaki S, Fukushima T, Takusagawa Y: Epidermoid presenting as trigeminal neuralgia. Clinical significance and surgical approach. Neurol Med Chir (Tokyo) 24: 774-781, 1984.
- 20) Møller MB, Møller AR: Loss of auditory function in microvascular decompression for hemifacial spasm. J Neurosurg 63: 17-20, 1985.
- 21) Møller AR, Jannetta PJ: Microvascular decompression in hemifacial spasm. Intraoperative electrophysiological observations. Neurosurgery 16: 612-618, 1985.
- 22) Pierry A, Cameron M: Clonic hemifacial spasm from posterior fossa arteriovenous malformation. J Neurol Neurosurg Psychiatry 42: 670-672, 1979.
- 23) Sato O, Kanazawa I, Kokunai T: Trigeminal neuralgia caused by compression of trigeminal nerve by pontine vein. Surg Neurol 11: 285-286, 1979.
- 24) Scoville WB: Hearing loss following exploration of cerebellopontine angle in treatment of hemifacial spasm.

- I Neurosurg 31: 47-49, 1969.
- 25) Sunderland S: Neurovascular relationships and anomalies at the base of the brain. J Neurol Neurosurg Psychiatry 11: 243-247, 1948.
- 26) Takamiya Y, Toya S, Kawase T, et al: Trigeminâl neuralgia and hemifacial spasm caused by a tortuous vertebrobasilar system. Surg Neurol 24: 559-562, 1985.
- 27) Ueda T, Goya T, Kinoshita K: Epidermoid cyst in the cerebellopontine angle cistern presenting as trigeminal neuralgia. Diagnosis values of the orbicularis oculi reflex and metrizamide CT cisternography—case report—. Neurol Med Chir (Tokyo) 23: 375–380, 1983.
- 28) Waga S, Morikawa A, Kojima T: Trigeminal neuralgia. Compression of the trigerminal nerve by an elongated and dilated basilar artery. Surg Neurol 11: 13-16, 1979.
- 29) Wakabayashi T, Tamaki N, Satoh H: Epidermoid tumor presenting as painful tic convulsif. Surg Neurol 19: 244-246. 1983.

和文抄録

三叉神経痛および顔面けいれんに対する 微小血管減圧術の分析

高知医科大学脳神経外科 森 惟明,森本 雅徳,栗坂 昌宏,内田 泰史 Patrick Eghwrudjakpor

三叉神経痛61例,顔面けいれん65例の治療として微小血管減圧術を施行した結果とその問題点につき報告する. 非典型的三叉神経痛においては,その効果は典型的なものに比し劣った. 責任血管の三叉神経への異常接触の範囲から,三叉神経における "entry zone" は顔面けいれんにおける顔面神経の "exit zone" よりも

広範であると考えられる。顔面けいれんにおいては微小血管減圧術の適応となるのは典型例に限られ、手術に際しては root exit zone を丹念にしらべ責任血管を見逃さぬようにしなければならない。また、合併症を防止するためには不適切な手術操作を避けなければならない。