*Neurol Med Chir (Tokyo) 20, 739~751, 1980*

# *Microvascular Decompression of Cranial Nerves, Particularly of the 7th Cranial Nerve*

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**Summary**

Fifty-three patients with hemifacial spasm, six patients with trigeminal neuralgia and five patients with unilateral tinnitus were treated by a "microvascular decompression" method and follow-up results for more than one year were evaluated. Follow-up operative results of 44 patients with hemifacial spasm were: 1) excellent—24, 2) good—12, 3) fair—7 and 4) poor—1. The operative results of hemifacial spasm were evaluated according to Jannetta's classification[17] combined with the degree of postoperative hearing reduction. Reduction of hearing acuity occurred in nine out of 53 patients. No recurrence of hemifacial spasm was encountered during follow-up period. It was interesting enough that the ipsilateral facial paresis which existed preoperatively gradually improved after decompressive surgery for hemifacial spasm. Eight out of 10 patients who developed facial paresis without having previous traumatizing surgical treatment and six out of 13 patients who had undergone previous peripheral surgical treatment for hemifacial spasm gradually improved after this decompressive surgery. All six patients with trigeminal neuralgia were completely cured and they noticed no sensory changes on the affected side of the face postoperatively. Four out of five patients with tinnitus were improved by the surgery.

The most characteristic forms of compressing arteries were that the arteries cross-distorted the cranial nerves usually at their exit zones from the brain-stem and the arteries were angled and sclerotic focally at the compressing sites. Vertebral angiographic studies provided a key to solve the problem of why these arteries were sclerotic at the angular part and why hemifacial spasm affected one side of the face. Although the etiology of these hyperactive dysfunctions of the cranial nerves is still controversial, vascular compression-distortion seems very likely to be responsible for these syndromes. The "microvascular decompression" of cranial nerves is a refined and nontraumatic technique which seems to promise a stable and permanent cure of the hyperdysfunction of cranial nerves.

**Key words:** Microvascular decompression, hemifacial spasm, trigeminal neuralgia, tinnitus, arteriosclerosis

## Introduction

Hyperactive dysfunction of cranial nerves, such as hemifacial spasm, tinnitus, trigeminal neuralgia and glossopharyngeal neuralgia are all mainly benign, but very distressing disorders characterized by subacute, intermittent and involuntary occurrences. Surgical treatment has long been performed for hemifacial spasm and trigeminal neuralgia, but not for tinnitus. Treatments for such hyperactive dysfunctions of cranial nerves have usually consisted of application of mild to severe trauma to the facial or trigeminal nerves extracranially or intracranially in the cerebellopontine angle. Dandy[6] first noticed the trigeminal root compressed by vessels in 66 out of 215 patients with trigeminal neuralgia operated upon through the posterior fossa, but he did not remove the compressing arteries from the trigeminal nerves. Scoville[28] successfully treated hemifacial spasm in two patients by moving a small artery from the facial nerve in the cerebellopontine

angle after posterior fossa craniectomy. The posterior fossa approach to this region to decompress cranial nerves was improved by Jannetta et al.[17] with the aid of advanced microsurgical instruments and also by an excellent microsurgical technique.

The authors have had an opportunity to treat 53 patients with hemifacial spasm, six patients with trigeminal neuralgia, and five patients with tinnitus by the method of "microvascular decompression" from 1976 to 1979. In this paper, the authors report the details of operative method and results or complications of microvascular decompressive surgery for the syndrome of hyperactive dysfunction of cranial nerves and also discuss the pathogenesis or etiology of "vascular compression" of cranial nerves.

## Summary of Cases

### I. Hemifacial spasm

Fifty three patients were operated on and the ages of the patients ranged from 22 to 67 years and females outnumbered males by 39 to 14. All of them showed typical, persistent and intractable hemifacial spasm. Neither side of the face was affected more commonly. The spasm had been present for from 1 to 20 years. Twenty three patients had undergone prior operative procedures one to four times, such as partial resection or crushing of the facial nerve in the peripheral region, resulting in either no relief or a recurrence of symptoms within approximately 6 months postoperatively. Forty eight patients underwent preoperative vertebral angiography and electromyographic studies were performed in almost all patients before and after surgery.

*Other symptoms accompanying hemifacial spasm before surgery*

a) Peripheral facial motor weakness

Among 53 patients with hemifacial spasm, 23 had mild to moderate facial weakness on the affected side of the face preoperatively. Among these 23 patients, 14 patients had undergone previous extracranial traumatizing surgery for peripheral facial nerves and the remaining nine patients had received no previous surgical treatments. Facial weakness was noted both clinically and electromyographically.

b) Hyperlacrimation

Seven out of 53 patients with hemifacial spasm also had hyperlacrimation on the affected side. This symptom seems to be caused by hyperfunction of N. intermedius.[25]

c) Tinnitus

Ten patients complained of tinnitus on the ipsilateral side simultaneously with an attack of hemifacial spasm. Four of them noticed tinnitus starting just before the attack of hemifacial spasm.

d) Reduced hearing acuity

Sixteen patients revealed mild to moderate hypofunction of the cochlear nerve, mainly 30–40 dB reductions at 6,000–8,000 Hz.

### II. Trigeminal neuralgia

Six patients, two men and four women, aged 40 to 75 years showed typical, intractable trigeminal neuralgia. Attacks of this neuralgia had been present for a couple of months to about 10 years. All patients except one had undergone previous surgical treatment in the peripheral region, but the treatment resulted in only transient effects on relieving of pain.

### III. Tinnitus

The eighth cranial nerve, like the 5th and 7th cranial nerves, is subject to symptoms of disordered hyperactivity which may be gradually accompanied by progressive loss of functions of the cochlear nerve. Five patients with tinnitus, three men and two women, ranging in age from 37 to 58 years, have been operated on. In all cases, the nature of the tinnitus was "pulsating", which was synchronous to their radial pulses. One of them showed clinical findings of Ménière's variants. All patients except for one showed mild to moderate hearing difficulties preoperatively.

*Preoperative angiographic studies*

Preoperative vertebral arteriographies were performed in 48 out of 64 patients on whom microvascular decompression surgery was carried out. Angiographic studies were performed to clarify the possible compressing artery and to rule out anomalous vessels or a mass lesion. All antero-posterior, lateral and Stenvers projections of arteriograms were taken. In almost all patients with hemifacial spasm and tinnitus, anterior inferior or

posterior inferior cerebellar arteries or vertebral and basilar arteries were found dilated, elongated and redundant, particularly around the internal auditory meatus and these findings were most clearly noticed in Stenvers projection. The vasculatures of vertebral or basilar arteries and anterior inferior or posterior inferior cerebellar arteries were all closely examined and the possible compressing artery was conjectured before surgery. Finally both angiographic and operative findings were studied together to decide the "compressing artery" of the cranial nerves. The characteristic findings of vasculatures of vertebro-basilar arteries and their branches will be commented on later in the "Discussion".

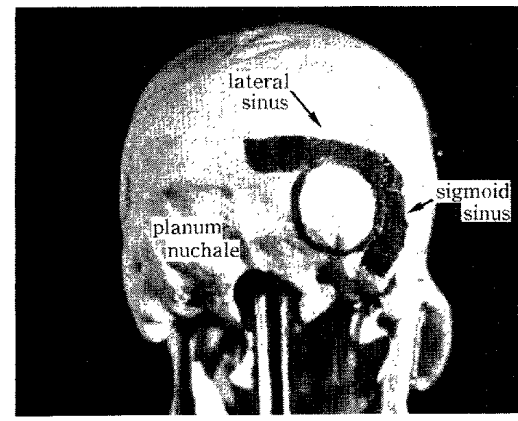


Fig. 1 The broad line on the skull shows the extension of sinuses. The encircled area of the planum nuchale was resected.

## Operative Method

All patients were operated on in the semiprone or lateral decubitus position through a retromastoid craniectomy approach. The ipsilateral hair is shaved posteriorly to the ear. Head is fixed with a Mayfield head rest and an ipsilateral part of the planum nuchale is placed parallel to the floor. The head may be tilted or rotated during surgery if necessary. A vertical or slightly curved skin incision of about 5–6 cm long, parallel to the hair line, is made about 1.5 or 2.0 cm medial to the mastoid eminence. Soft tissues are separated from the bone by means of a periosteal elevator and retractor using electrocautery. The medial half of the mastoid eminence should be cleared of subcutaneous tissue. A retromastoid craniectomy, approximately 3 cm in diameter is made only to expose the parts of the lateral and sigmoid sinuses. (Fig. 1) Mastoid air cells might be exposed partially during this procedure and bone wax must be inserted. An angled dural incision is made along the sigmoid and lateral sinuses. The microsurgical self-retaining retractor is put into the place after covering the cerebellar cortex by thin rubber plates with spongel. The cerebellar cortex is then gently retracted medially and cerebrospinal fluid is suctioned to obtain a sufficient operative field. Arachnoid membranes around the 5th and 7th cranial nerves and surrounding vessels are resected by sharp dissection. Preparation then proceeds and retraction of the cerebellar cortex is progressed gradually to expose the exit zone of these cranial nerves. During these procedures, coagulation of tissues near these cranial nerves should be avoided as much as possible. More than one small ectated or redundant arteries are frequently found touching or coursing parallel to these cranial nerves or running between the 7th and 8th cranial nerves between brain-stem and porus acusticus.

The first important point of the operation is to identify the artery which is actually cross-compressing the cranial nerves. The most characteristic finding of the artery which cross-compresses these cranial nerves is that this artery is almost at right angles at the compression site and is whitish in appearance and hardened when touched by a microvascular forceps. (Fig. 2) When an artery is found which is only "touching" the cranial nerves and is not angled or does not appear arteriosclerotic, such an artery is not considered as an "compressing artery" and does not cause symptoms. The second point of this operative procedure is how to decompress the cranial nerves at their exit zone from the brain-stem, since these "compressing parts" of arteries are frequently concealed behind the cerebellar flocculonodular lobe or choroid plexus of the lateral recess of the fourth ventricle. To decompress the cranial nerves, the artery is first separated and removed sufficiently away from the nerve by changing the axis of the arterial loop after partial resection of the cerebellar structures for 7th and 8th nerve decompression. (Fig. 3) A small prosthesis

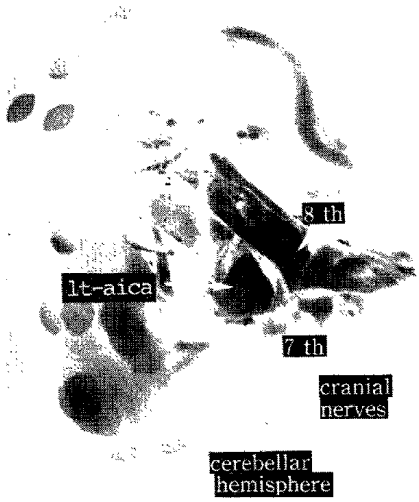


Fig. 2 Aica is seen compressing the 7th cranial nerve. Note the site of compression where the artery is almost right-angled and appears whitish and hardened when touched by a vascular forceps. Other parts of the artery do not show such pathoanatomical changes.

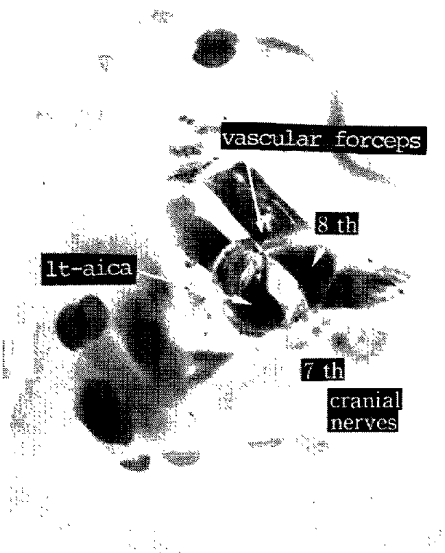


Fig. 3 The 7th cranial nerve is freed from the compressing artery. The compressing site of the artery is seen retracted by a vascular forceps.

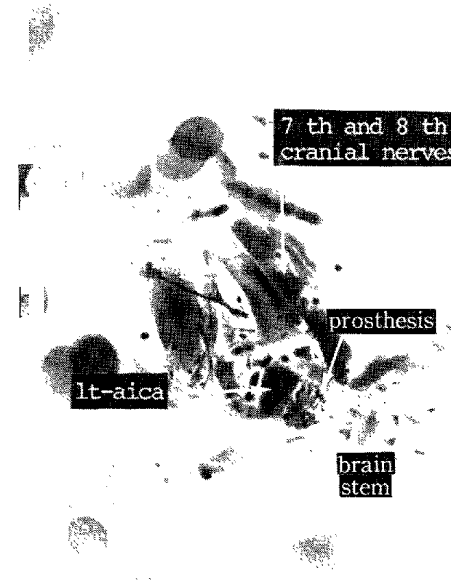


Fig. 4 A small piece of vinyl sponge is interposed as a prosthesis between the artery and the brain-stem.

is then inserted between the artery and the brain-stem. (Fig. 4) Precautions should be taken, during these procedures, to avoid manipulating or traumatizing these nerves, particularly the 8th cranial nerve. The size of the prosthesis is variable, but should be small enough to hold the artery in position and be irregularly shaped to fit the surrounding tissue easily and avoid slip-out. More than one prosthesis may be necessary to hold the artery satisfactorily depending on the tortuosity or position of the artery. Compressing vessels which were verified during operative procedure and also by vertebral angiogram are shown in Tables 1, 2 and 3. The dura is closed tightly and a dural graft may be used on occasion. Before closing the wound, mastoid air cells should be rechecked. The epidural drainage may be indwelling. When the patients complain of postoperative headache, the drainage might be occluded intermittently to avoid excessive lowering of the intracranial pressure.

## Operative Results

The results of operations on 53 patients with

classical hemifacial spasm, six patients with trigeminal neuralgia and five patients with tinnitus were evaluated (Tables 1, 2 and 3).

**I. Hemifacial spasm**

Among 53 patients with hemifacial spasm who had been operated on using this decompressive surgery, the results of 44 patients who were followed up for more than one year postoperatively were evaluated according to Jannetta's classification combined with consideration of postoperative changes in hearing acuity. Jannetta et al.[17] classified the results of the operation as follows;

1) Excellent: no clinical or electrical facial spasm or motor weakness, 2) Good: trace of electrical facial spasm or trace of motor weakness, 3) Fair: mild evident facial spasm or mild motor weakness, 4) Poor: persistent clinical spasm, although improved. We considered that postoperative hearing reduction is the most severe complication, although this hearing reduction is usually improved to some extent within 3 months postoperatively. Postoperative hearing disorders are likely to occur in elderly patients or in patients with a long history of hemifacial spasm. Postoperative hearing reduction continuing more than one year is considered a factor which brings down the classification of surgical results, for example, from "Excellent" to "Good". According to our classification of operative results, the results were: Excellent—24, Good—12, Fair—7, and Poor—1. Almost all patients awoke from the anesthesia without any evidence of facial spasm, but some patients awoke with mild persistent facial spasm which gradually disappeared within one week after the operation. Eighteen patients awoke with ipsilateral reduction of hearing, but nine of them gradually improved within 3 months after the surgery and complained of no difficulties in conversation and talking over the telephone. Nine patients, however, revealed permanent hearing difficulties or loss. One patient with a "Poor" result had undergone two operations early in the series of this operative procedure. In the first operation, a small artery which did not appear arteriosclerotic at its compressing site was removed from the 7th nerve but the operative result was poor, although the facial spasm was as not severe as it had been. In the second operation, however, there was no artery found even after removal of the cerebellar flocculus and choroid plexus of the 4th ventricle and also around the entrance of the internal auditory meatus. Prosthesis previously inserted had not slipped out. No recurrence of hemifacial spasm occurred in the 44 patients who had been followed-up for more than one year in our series. Postoperative cerebrospinal fluid rhinorrhea occurred in two patients postoperatively and they were cured either by reopening the wound and refilling of bone wax into the mastoid air cells or by a ventriculoperitoneal shunt. A characteristic postoperative finding is an improvement in facial weakness after the decompressive surgery. Six patients out of 13 with preoperative facial motor weakness induced by prior destructive surgery to the peripheral facial nerve recovered well after this decompressive surgery, whereas recovery time was much prolonged and sometimes incomplete depending on the duration and severity of preoperative facial paresis in the remaining cases. On the other hand, it is striking that eight patients out of 10 with facial weakness without undergoing prior destructive surgery showed better and quicker recovery after this operative procedure.

**II. Trigeminal neuralgia**

All six patients who were treated by this decompressive surgery revealed complete relief of pain after the operation. The follow-up periods ranged from 6 months to 2 years. Compressing vessels verified were superior cerebellar arteries which were focally angled and arteriosclerotic at their compressing sites in all patients except for one whose trigeminal nerve was strangled by a thickened arachnoid membrane and superior petrosal vein. The most characteristic finding in the treatment of trigeminal neuralgia by this operative method is that none of the patients showed postoperative sensory changes on the affected area of the face.

**III. Tinnitus**

Two out of five patients with tinnitus revealed complete disappearance of tinnitus, whereas in the other two, tinnitus decreased considerably although a minimum grade of occasional tinnitus was still noted. The remaining patient still complained of mild, but lessened tinnitus

Table 1 Operative cases and results of hemifacial spasms.

| Cases | Sex Age | Other symptoms | Compressing vessel(s) | Operative results | Complications & other findings |
|---|---|---|---|---|---|
| 1. | 22, M. | | petrosal vein | excellent | |
| 2. | 27, F. | facial paresis | aica | excellent | facial paresis (−) |
| 3.* | 28, F. | hyperlacrimation reduced hearing | aica | excellent | |
| 4. | 32, F. | reduced hearing facial paresis | va | excellent | facial paresis (−) |
| 5.* | 48, F. | | aica | excellent | |
| 6. | 47, M. | reduced hearing, facial paresis, tinnitus, hyperlacrimation | aica** | excellent | facial paresis (−) |
| 7.* | 50, F. | facial paresis, tinnitus | pica** | excellent | facial paresis (−) |
| 8.* | 53, F. | facial paresis | aica** | excellent | facial paresis (−) |
| 9. | 55, F. | reduced hearing facial paresis | aica | fair | mild spasm (+) facial paresis (−) |
| 10.* | 56, F. | facial paresis | va | good | facial paresis (+) |
| 11.* | 65, M. | reduced hearing facial paresis | aica | fair | reduced hearing (+) facial paresis (+) |
| 12. | 31, M. | tinnitus | aica** | poor | persistent spasm (+) |
| 13.* | 56, F. | facial paresis hyperlacrimation | aica | fair | reduced hearing (+) facial paresis (+) |
| 14. | 34, F. | facial paresis, tinnitus | aica | excellent | facial paresis(−) |
| 15. | 59, F. | reduced hearing, facial paresis, tinnitus | pica** | excellent | facial paresis (−) |
| 16. | 67, F. | facial paresis | aica | good | facial paresis (+) |
| 17. | 36, F. | facial paresis | aica | fair | trace of spasm (+) reduced hearing (+) facial paresis (−) |
| 18. | 52, M. | reduced hearing, tinnitus, hyperlacrimation | aica | good | tinnitus (+) |
| 19.* | 61, M. | reduced hearing facial paresis | pica | good | facial paresis (+) |
| 20.* | 49, F. | facial paresis | aica** | fair | facial paresis (+) |
| 21. | 51, F. | tinnitus | aica | excellent | |
| 22.* | 57, F. | reduced hearing facial paresis | aica | good | reduced hearing (+) nasal liquorrhea (+) |
| 23. | 54, F. | reduced hearing hyperlacrimation | pica | excellent | |
| 24. | 55, F. | | pica | good | reduced hearing (+) |
| 25. | 41, F. | tinnitus | aica pica | good | reduced hearing (+) |
| 26.* | 59, F. | reduced hearing | aica | fair | mild facial paresis (+) |
| 27.* | 46, M. | | va | excellent | |
| 28. | 45, F. | hyperlacrimation | pica | excellent | |
| 29. | 53, M. | facial paresis reduced hearing | aica | excellent | facial paresis (−) |
| 30. | 65, F. | reduced hearing facial paresis | pica | fair | reduced hearing (+) facial paresis (+) |
| 31. | 54, F. | | pica | excellent | |
| 32. | 30, M. | | pica | excellent | |
| 33. | 54, F. | | va | good | reduced hearing (+) |
| 34. | 37, F. | | pica | excellent | |
| 35.* | 50, F. | reduced hearing hypolacrimation | pica | good | reduced hearing (+) |
| 36.* | 47, F. | facial paresis | pica** | excellent | facial paresis (−) |

| | | | | | |
|---|---|---|---|---|---|
| 37.* | 41, F. | reduced hearing facial paresis | pica | good | facial paresis (+) |
| 38. | 58, F. | reduced hearing | pica | excellent | |
| 39. | 57, F. | reduced hearing | pica | good | trace of spasm (+) |
| 40. | 32, F. | tinnitus | pica | excellent | trace of spasm 3 days postop. (+) |
| 41. | 50, F. | | petrosal vein** | excellent | |
| 42.* | 50, F. | | pica | excellent | |
| 43.* | 42, M. | facial paresis | pica | excellent | facial paresis (−) |
| 44.* | 45, F. | | aica | excellent | |
| 45. | 35, M. | tinnitus | pica | (good) | facial paresis (+) |
| 46.* | 29, F. | | aica | (excellent) | slight spasm 1 week after op. (+) |
| 47.* | 36, F. | facial paresis | aica | (excellent) | facial paresis (−) |
| 48.* | 51, F. | facial paresis | pica | (excellent) | facial paresis (−) |
| 49.* | 23, M. | | aica | (excellent) | |
| 50. | 60, F. | tinnitus hyperlacrimation | pica | (good) | reduced hearing (+) |
| 51. | 52, M. | tinnitus hyperlacrimation | pica | (good) | facial paresis (+) |
| 52. | 46, M. | | aica | (excellent) | |
| 53. | 40, F. | | pica | (excellent) | |

* Patient was treated previously by other methods.
** Compressing vessel running between the 7th and 8th cranial nerves.
va: vertebral artery, aica: anterior inferior cerebellar artery, pica: posterior inferior cerebellar artery.

Table 2 Operative cases and results of trigeminal neuralgia.

| Cases | Sex Age | Other symptoms | Compressing vessel(s) | Operative results | Complications & other findings |
|---|---|---|---|---|---|
| 1.* | 51, F. | | sca | excellent | |
| 2.* | 41, M. | | sca | excellent | |
| 3.* | 56, F. | tinnitus | supeior petrosal vein | excellent | |
| 4. | 66, M. | | sca | excellent | |
| 5. | 75, F. | | sca | excellent | |
| 6. | 62, F. | | sca | excellent | |

*: treated previously by other methods.
sca: superior cerebellar artery.

Table 3 Operative cases and results of tinnitus.

| Cases | Sex Age | Other symptoms | Compressing vessel(s) | Operative results | Complications & other symptoms |
|---|---|---|---|---|---|
| 1. | 41, M. | vertigo, nausea, nystagmus, reduced hearing | pica** | excellent | |
| 2. | 58, F. | reduced hearing | aica | good | trace of tinnitus |
| 3. | 37, M. | reduced hearing | aica | good | trace of tinnitus |
| 4. | 42, F. | | pica | excellent | |
| 5. | 38, M. | reduced hearing | pica | fair | mild tinnitus |

**: compressing vessels run between the 7th and 8th cranial nerves.

postoperatively. Reduced hearing noted in four out of five patients preoperatively did not become worse atfer surgery except in one patient who developed a variant of Ménière's syndrome preoperatively, although attacks of tinnitus or vertigo almost completely stopped. All patients with tinnitus were followed-up for more than one year.

## Discussion

Here, "microvascular decompression of cranial nerves" for the treatment of syndromes of hyperactive dysfunction is discussed mainly with respect to hemifacial spasm.

Treatment of hemifacial spasm has long consisted of the application of mild to severe trauma to the peripheral facial nerve; this includes extracranial blocking of the facial nerve by procaine chloride, crushing and partial resection of the facial nerve with or without reversed fashioned resuture, or partial rhizotomy with accessory facial anastomosis. Details of these methods are omitted here.[4,9,14] In 1960, Bragdon[2] first mentioned a method of intracranial crushing of the facial nerve by a hemostat as a treatment of hemifacial spasm. Gardner and Gardner and Sava[11,12] performed gentle manipulation of facial nerves, irrigation by Ringer solutions and also insertion of a Gelfoam prosthesis between the compressing vessel and facial nerve. Scoville[28] first treated facial spasm by moving an artery compressing the facial nerve in the cerebellopontine angle without using an operative microscope. The above treatments for hemifacial spasm have, however, disadvantages in that the operative effects are still uncertain and also these operative procedures are likely to cause permanent facial paresis. In 1970, Jannetta et al.,[17] pioneers of "decompressive microsurgery" of cranial nerves, first decompressed facial nerve from vessels with the aid of new technical adjuvants such as microsurgical instruments and the binocular operative microscope and also with fine microsurgical techniques without traumatizing the facial nerve.

There are still some controversial opinions on whether arterial compression of the facial nerve is really etiological of hemifacial spasm or whether, on the other hand, all facial spasm can be cured by vascular decompression and also why almost all hemifacial spasm occurs on one side of the face. First we will proceed with a discussion about these problems.

*Organic lesions described in the past as causing hemifacial spasm*

In 1875, Schultze[27] found a small vertebral aneurysm compressing the 7th and 8th cranial nerves in a patient with hemifacial spasm who died of pulmonary tuberculosis and was autopsied, and in 1917 Cushing[5] reported four cases of hemifacial spasm among 30 patients with acoustic tumors. In 1945, Ehni et al.[9] described the existence of arteriosclerosis in 37 out of 106 patients with hemifacial spasm and suggested that the arteriosclerosis was a possible causative lesion of hemifacial spasm. Campbell et al.,[3] Laine,[19] McKenzie[22] and Gardner[11] found cirsoid aneurysms of the basilar artery, anomalies of the auditory artery, arteriovenous malformations and redundant anterior and posterior inferior cerebellar arteries in patients with hemifacial spasm. Furthermore, there have recently been many reports describing the existence of arteries which are found compressing facial nerves in hemifacial spasm.[17,23,24] Maroon,[20] reviewing the literature, described 16 communications reporting a total of 107 cases of hemifacial spasm probably caused by vascular compression of the facial nerve. Confirmation in these cases was by surgery (92 cases), angiography (12 cases), and autopsy (three cases). These reports are strongly suggestive of organic lesions existing between the facial nerve nucleus and internal auditory meatus as causative factors of hemifacial spasm. Hemifacial spasm is a "motor counterpart of trigeminal neuralgia" and these two clinical syndromes are reported to be caused by the same organic etiological factor. These hyperactive dysfunctions of the cranial nerves may then share common pathophysiological factors.[3]

There have been many different studies concerning how organic lesions can act as a trigger causing hemifacial spasm. In 1958, Greenfield[15] suggested that myelin was more vulnerable both to mechanical pressure and to slight degrees of anoxia than nerve cells and axons. Granit et al.[13] experimentally created an "artificial synapse" between afferent and efferent fibers by placing a ligature around the

sciatic nerve so gently that it did not interrupt the passage of the nerve impulse. They then abolished this synapse by removal of the ligature and irrigation of the compressed portion of the nerve with Ringers solution. Subsequently they explained that the resulting squeezing together of the insulating myelin sheaths permitted a transaxonal "short circuit" of the action current. In 1962, Gardner et al.[12] mentioned that the 5th, 7th, 9th and 10th nerves were mixed nerves in which afferent and efferent axons came into contact with each other proximal to the site where the oligodendroglial covering of the myelin sheath was replaced by the tougher neurilemma (junction zone). Jannetta et al.[17] also described from surgical and autopsy findings that the point at which irritable lesions are most likely to produce disordered nerve function was at the exit zone of the seventh nerve from the brain-stem, and they hypothesized that at this anatomically discernible junction zone, a physiological threshold to mechanical deformation and pressure might exist and these stimuli could easily interfere with axonal metabolism and transport, causing a hyperirritable condition to ensue. Ruby et al.[26] biopsied the facial nerves following relief of compression and an electronmicroscopic study revealed that there were significant changes in many of the myelinated fibers, such as proliferation or hypermyelinations of myelin sheaths with a concomitant disorganization of the myelin lammelar structure. However, they neither clearly mentioned which part of the facial nerve was studied nor described that these structural changes are really found at the junction zone. In conclusion, the "peripheral hypothesis" of the cause of hemifacial spasm is explained by "fiber interaction" at the place of "injury", and the compressing site, that is a junctional zone, is thought to give rise to potentials which cause spontaneous movements and to act as a false synapse (ephapse).

Ferguson,[10] however, has a theory which opposes these "peripheral hypotheses". He pointed out that the experimental studies done by Granit et al.[13] were all acute and the duration of the effects was not known. He also cited a report of an electromyographic study by Wigand et al.[30] in which differing latencies between the periocular, chin and lip motor units during "synchronous" spasms were noted. This difference between latencies was constant and stood as evidence against impulse initiation from a common point, as required by the ephapse or slit-fiber hypothesis. He noted that "the unique central organization of the facial motor system" was the basis not only for a wide range of voluntary movements, but for diverse "involuntary" or "automatic" (emotional, repetitive, associated and reflexive) movements as well. He concluded that following injury of any degree to the facial motor nerve, partial and selective deafferentiation would occur on motor neurons in the nucleus. He also proposed that nuclear reaction to "axotomy" was more severe with lesions close to the parent cell, and that this was why lesions close to the brain-stem might result in more severe facial hyperkinesia than more distal lesions such as those causing Bell's palsy. We partly agree with the opinion of Ferguson since we realized that pathoanatomical and pathophysiological bases of the "peripheral hypothesis" of the cause of hemifacial spasm do not thoroughly explain the emotional or reflexive effects on eliciting severe hyperkinetic movements of facial muscles. On the other hand, we found a paper in which Habel[16] reported an interesting patient whose hemifacial spasm was not influenced at all when the patient developed ipsilateral hemiplegia due to cerebrovascular disease, and also we noticed not infrequently the persistence of hemifacial spasm during surgery even after the patients were under deep anesthesia. These facts partly oppose the opinion of Ferguson[10] who mentioned the existence of a unique central organization able to unmask and augument automatic, reflexive movements resulting in facial hyperkinesia, including corticobulbar fibers and motor neurons in the facial nerve nucleus.[8,18] Trigeminal neuralgia and glossopharyngeal neuralgia which were all found to be caused by the same mechanism of "neurovascular compression" from the operative findings could not be explained by the hypothesis of Ferguson,[10] since he stressed the unique or peculiar character of the facial nerve as the cause of the hyperkinetic syndrome, although the other cranial nerves are mainly sensory nerves.

The first important point in performing such surgery is confirmation of the compressing

vessel. According to Sunderland,[29] anterior inferior cerebellar arteries ran close to 7th and 8th cranial nerves around the internal auditory meatus in 64% of his autopsied cases, and we also noted many arteries or veins ran close to or touched in parallel these cranial nerves (Fig. 5). Jannetta et al.[17] emphasized that vascular compression must be a "cross-compression" at right angles to the nerve and must at the root exit zone of these cranial nerves from the brain-stem. He also added that peripheral parts of vessels crossing the facial nerve and vessels running parallel to and only distorting the nerve are not responsible for hemifacial spasm. We noticed in our series, however, not only the same findings, but also noted in a few patients that these cranial nerves were compressed near the internal auditory meatus as well. Jannetta et al.[17] did not mention the nature of these compressing arteries, whereas we will emphasize that all compressing arteries were arteriosclerotic at this angled and compressing site, i.e. they are whitish and hardened when touched by a vascular forceps. These specific arterial changes are very characteristic, since such focal arteriosclerosis is noted without any relation to the age of the patients and is also noted at the angled compressing site, and not at other proximal or peripheral parts of the artery.

We have performed vertebral angiography on 48 out of 64 patients who have undergone decompressive surgery. The most characteristic finding is that the vertebral artery is frequently larger in diameter on the ipsilateral side of the facial spasm, making a sharp, hair-pin like curve in the peripheral region just before uniting to become a basilar artery and from this angled part a posterior inferior cerebellar artery, which is commonly more ectated, elongated and more redundant than the contralateral side is given off in the direction of the internal auditory meatus. The other type of peculiar vasculature is that the basilar artery makes an unusual S-shaped curve and gives off an anterior inferior cerebellar artery towards the affected side, accompanied by unequal size of the vertebral artery. (Fig. 6, 7) There are a number of hemodynamic theories explaining the mechanism of atherogenesis, such as the pressure-related hypothesis, wall-shear hypothesis, turbulance hypothesis and wall stress concentration hypothesis.[21] We however, supposed from our vertebral angiogram studies that the intraluminal blood stream of the larger vertebral artery is likely to subject to the peripheral part of anterior or posterior inferior

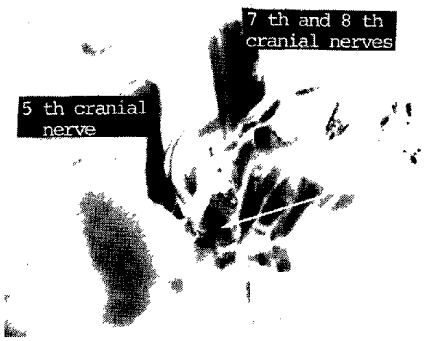


Fig. 5 Operative photograph showing several arteries running close to or touching the 7th and 8th cranial nerves. These arteries are not arteriosclerotic except for the part where the artery really compresses the cranial nerves. The arrow shows that this compressing part of the artery is angled and sclerotic.

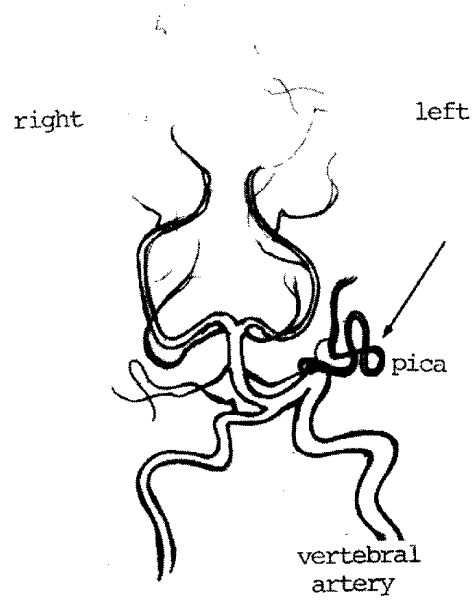


Fig. 6 Schematic drawing of a typical A-P view of a vertebral arteriogram of a patient with left hemifacial spasm. Note the larger vertebral artery on the left side making a sharp, hair-pin like curve in the peripheral region and from which an ectated and redundant posterior inferior cerebellar artery (arrow) is given off in the direction of the left internal auditory meatus.

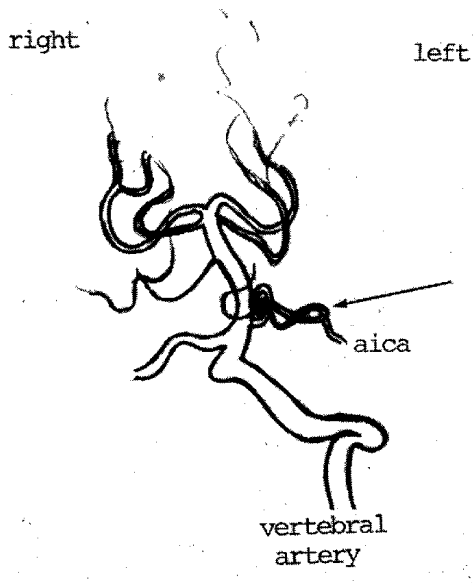


Fig. 7 Schematic drawing of a typical A-P projection of a vertebral arteriogram a patient with left hemifacial spasm. Note the S-shaped basilar artery giving off a more ectated and redundant anterior inferior cerebellar artery (arrow) on the left side, running toward the internal auditory meatus, with no ipsilateral pica.

cerebellar arteries to stronger hemodynamic factors resulting in elongation or redundancy of these arteries and also in arteriosclerotic changes at the angled part of the arteries where cranial nerves are compressed. Both these presumably developmental, peculiar anatomical changes of the vertebro-basilar arteries and their branches, and the resulting physiological factors suggest a key to solve the problem of why the compressing artery is arteriosclerotic at its angled site and why facial spasms are elicited very frequently on one side of the face.

The second point of this surgery is that the hyperfunction of the cranial nerves is really relieved by "vascular decompression" and not by manipulation of cranial nerves. As far as our operative results are concerned, patients were cured by this decompression methods except for one patient with facial spasm in whom no evident compressing artery was found. From our operative results, therefore, we are confident that vascular decompression is really a curative method, since there were no recurrences among 44 patients who have been followed up for more than one year and this follow-up interval is longer than the average period of recurrence with other methods of treatment. Slight manipulation of cranial nerves during surgery can not really be ignored, especially when the compressing artery is well concealed behind the cranial nerve in the operative field. The fact, however, that some patients awake from the anesthesia with some facial spasms and these mild spasm gradually disappear in less than one week after surgery strongly suggests that surgical intervention is possible without traumatizing the facial nerve.

As mentioned in "Operative results", some patients showed improvements in facial paresis after decompressive surgery. These are very interesting and advantageous findings for this operative procedure. Facial paresis is more likely to be cured when patients have had no previous traumatizing treatment and the recovery time is well correlated with the duration of persistent facial spasm; the shorter the duration, the shorter the recovery time. These results suggest that facial muscles may be wasted as a result of the hyperfunction of facial nerves, probably due to inappropriate use.

## Complications

Since this microsurgical decompressive surgery for hyperdysfunction of cranial nerves is exclusively functional surgery, no complications should occur from this surgical procedure. As far as postoperative complications are concerned, hearing reduction is the most serious and should be avoided at all costs. There are many possible causes to take into account as etiological factors for hearing dysfunctions. Minor trauma to the cochlear nerve should be considered first, as well as vascular insufficiency of the internal auditory arteries due to trauma or prolonged vasospasms. The cochlear nerve is more likely to be vulnerable to all kinds of traumas because the vestibular nerve is less likely to be affected even when hearing acuity is reduced. We do not agree with the opinions of Dandy and Anastasio[1,6] who partially coagulated the internal auditory artery with partial resection of the 8th cranial nerve or cut the internal auditory artery for the treatment of Ménière's disease and conclude that no hearing

disturbance can result from these methods. Davey et al.[7] experimentally manipulated small arteries which supplied the 8th cranial nerve and noticed prolonged vasospasms of these arteries in cats. Rhoton, Jr. et al.[25] reported that arteries entering the internal auditory meatus were "end arteries" and they, therefore, were very vulnerable to occlusion. In our operative results, postoperative hearing disturbance occurred in the early series of this surgery and this complication has lessened recently. The most important point in avoiding this complication is that bipolar coagulation should not be used near the cochlear nerve or internal auditory artery and all kinds of trauma in these structures should be avoided by the best microsurgical techniques.

## Conclusion

"Microvascular decompression" for the treatment of hyperdysfunction of cranial nerves presents the following problems. These problems are partly solved but some remain unsolved. They are as follows:
1) Is this operative procedure superior to other treatment? 2) Is the effect of the surgery not actually due to a trauma or manipulation to the cranial nerves? 3) Are there no recurrences with this surgical method? 4) Are there any complications? 5) What is the etiology of hyperfunction of cranial nerves and why do these dysfunctions occur unilaterally?

The first question is answered in the "Discussion." Concerning question 2), the problem of manipulating cranial nerves during surgery is not really denied, but the fact that the mild facial spasm which is persistent after the surgery gradually disappears without recurrence strongly suggests the satisfactory effects of this surgery. There were no recurrences encountered in our surgical series over more than one year of follow-up and this result coincides with that of Jannetta.[17] Postoperative reduction of hearing acuity is the most severe complication, but this can be lessened by meticulous technical care using fine microsurgical techniques. Characteristic findings of vertebral arteriograms suggest a key to solve the problem why the compressing site of the artery appears arteriosclerotic and why hyperdysfunction of cranial nerves occurs unilaterally.

## Acknowledgments

The authors wish to thank Drs Isao Matsuda, Department of Neurosurgery, Shiga Medical College and Kouzo Moritake, Department of Neurosurgery, Kyoto University Medical School for their advice on hemodynamic studies of vertebral arteriograms.

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