ORIGINAL ARTICLE

Angiographic manifestations and operative findings with 70 cases of hemifacial spasm: relation of common trunk anomalies

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Abstract. The relations between angiographic manifestations and operative findings of hemifacial spasm were studied in 70 cases between 1988 and 2001. Vertebral angiography was performed, and Towne, straight AP, and lateral projections were routinely examined. The dominant anterior inferior cerebellar artery (AICA) directly compressed the facial nerve root exit zone in 26 cases, the dominant posterior inferior cerebellar artery (PICA) in 20, the AICA in 13, the PICA in 2, and the vertebral artery (VA) in 9. Compression by multiple vessels was observed in 11 cases. Anatomical variations of the affected AICA and PICA were classified into 3 groups according to their origins and distributions of blood supply: normal distribution of AICA and PICA in 18%, common trunk anomaly with dominant AICA (basilar artery origin) in 48% and common trunk anomaly with dominant PICA (vertebral artery origin) in 34%. Analyses of the angiograms revealed significantly increased numbers of common trunk anomalies compared with cases with normal angiograms. In 18 of the 20 cases of unilateral common trunk anomalies, facial nerves were compressed by the dominant artery. Preoperative vertebral angiograms may clarify the offending vessels and their sites in most hemifacial spasm cases, thus increasing the safety of surgical interventions. (Keio J Med 52 (3): 189–197, September 2003)

Key words: hemifacial spasm, microvascular decompression, vertebral angiogram, common trunk anomaly

Introduction

Hemifacial spasm is a movement disorder characterized by intermittent, involuntary, irregular, unilateral, tonic or clonic contractions of muscles innervated by the ipsilateral facial nerve. In a typical case, the spasm starts from the orbicularis oculi muscle, buccinator.

Cushing[1] first suggested the palsies of cranial nerves could be caused by vascular compression. Vascular arterial compression of the VIIth cranial nerve in patients with hemifacial spasm was first reported by Campbell Keedy.[2] In 1962, Gardner and Sava[3] proposed vascular decompression for treatment of trigeminal neuralgia and hemifacial spasms. Fifteen years later, the pioneering work of Jannetta *et al*.[4] provided a great contribution to understanding the pathophysiological mechanism of this rhizopathy.

The concept of neurovascular decompression for the treatment of hemifacial spasm is now widely accepted. Recently, in cases of microvascular decompression, some hospitals performed preoperative cerebral angiography. In our hospital, most patients with hemifacial spasm undergo cerebral angiography to exclude vascular diseases, and to estimate the operative findings. In the present study, we understook an anatomical analysis of compressing arteries, especially for common trunk anomalies in comparison with operative findings.

Patients and Methods

Microvascular decompressions and preoperative cerebral angiography were performed between 1988 and 2001 in 70 patients with hemifacial spasm. The patient group included 45 females (64.3%) and 25 males

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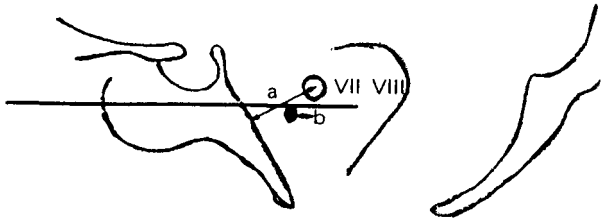
a) distance from the clivus 14.0 ± 1.6 mm (M ± SD)

b) perpendicular line 5 mm posteriorly from EAC

c) radius of its area 2.4 ± 0.3 mm



d) the most medial part from the midline 13.1 ± 1.6 mm

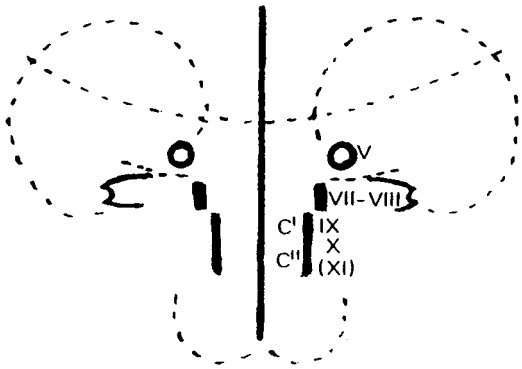


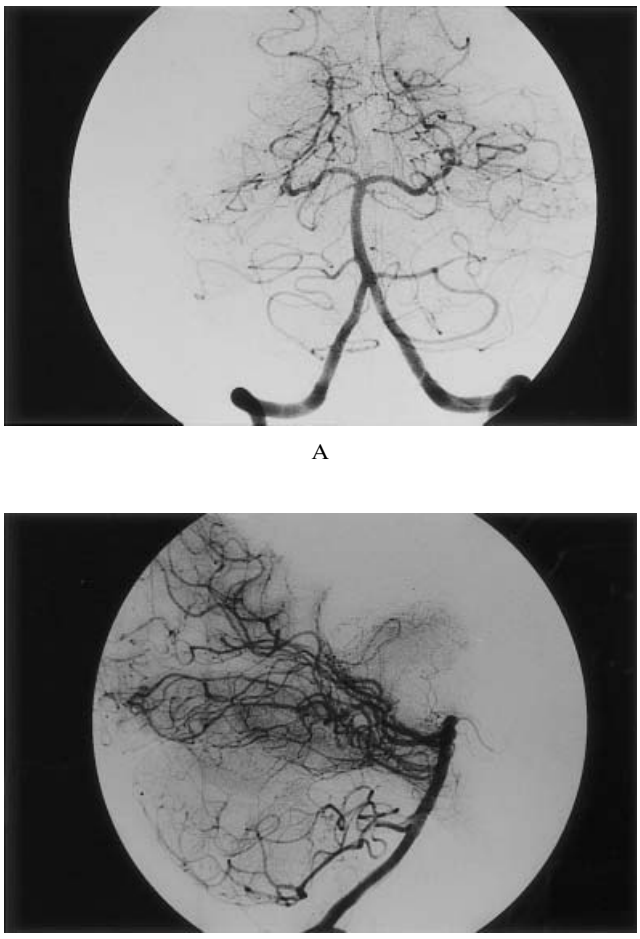
Fig. 1 Schematic illustration for measurements around the VIIth root exit zone.

(35.8%), ranging in age from 25 to 74 years (mean age, 50.9 yr). The symptoms were on the left side in 41 cases (58.6%) and right side in 29 cases (41.4%). The mean duration of symptoms was 5.4 years (range, 1–26 yr).

Cerebral angiography of bilateral internal carotid arteries and bilateral vertebral arteries was performed by the Seldinger method with stereographic straight anterior-posterior and lateral views. Digital subtraction was performed in most cases. There were no complications associated with cerebral angiography.

The facial nerve exit zone is present in a circle with a radius of 2.5 mm, and center at the point 14 mm from the clivus on the perpendicular drawn from the site 5 mm posterior to the opening of the external acoustic meatus to the anthropologic base line on lateral images. On frontal images, this zone is present 13 mm from the median line (Fig. 1).[5] Therefore, on vertebral angiograms, the sites of the facial nerve exit zone and the compressing vessel, and surrounding angioarchitecture were estimated before operation.

Microvascular decompression was performed in all cases, and the facial nerve exit zone was decompressed by removing the compressing vessel.[6,7] After the operation, the anatomical relations of the compressing vessels and area were compared with the angiographic findings.



A

B

Fig. 2 Right vertebral angiogram in a right hemifacial spasm patient. (A) Straight AP view. (B) Lateral view.

As an example, in the patient shown in Fig. 2, the right side was affected, and the above-described method and the estimation of the below-described angioarchitecture before operation suggested compression of the facial nerve exit zone by the metal loop of the dominant AICA. Fig. 3 shows the operative findings. As preoperatively estimated, the site of the bifurcation of the internal auditory artery from the metal loop of the right dominant AICA compressed the facial nerve exit zone. As shown in Fig. 4, the site of the compressing vessel could be determined on vertebral angiograms.

Anatomical Relation of the Cerebropontine Angle Artery

We defined the anterior inferior cerebellar artery (AICA) as the artery originating from the basilar artery, and the posterior inferior cerebellar artery (PICA) as the artery originating from the vertebral artery (VA).[8]

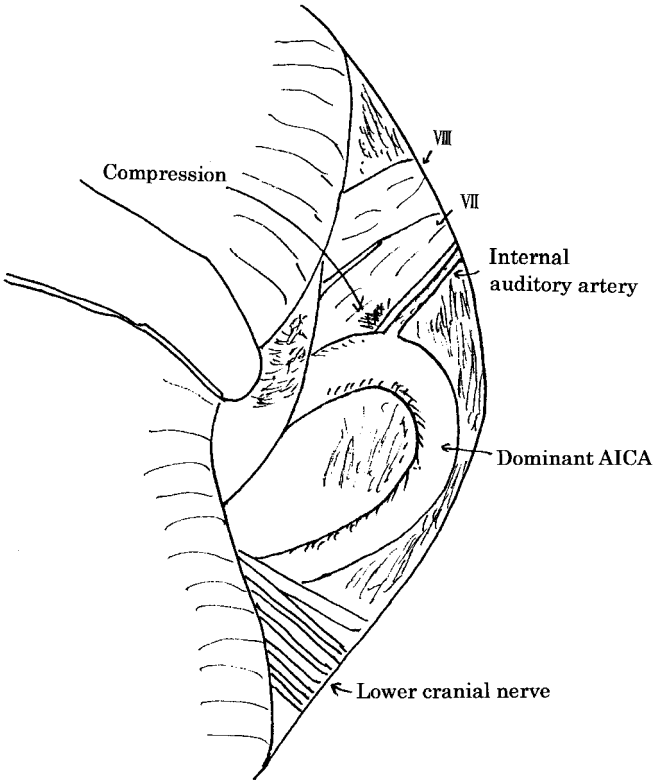


Fig. 3 Schematic illustration of an operation in the case of right hemifacial spasm.

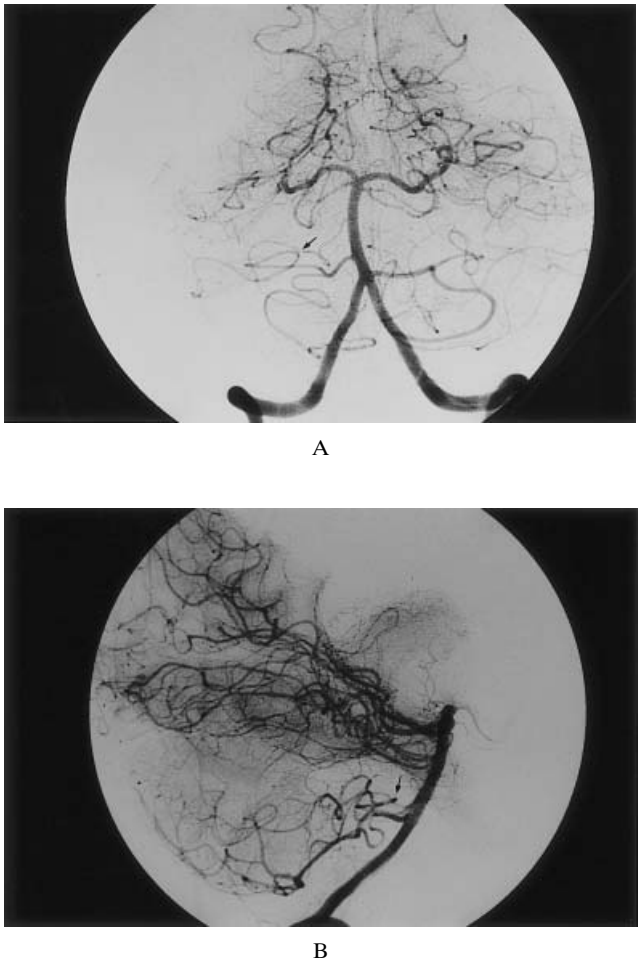


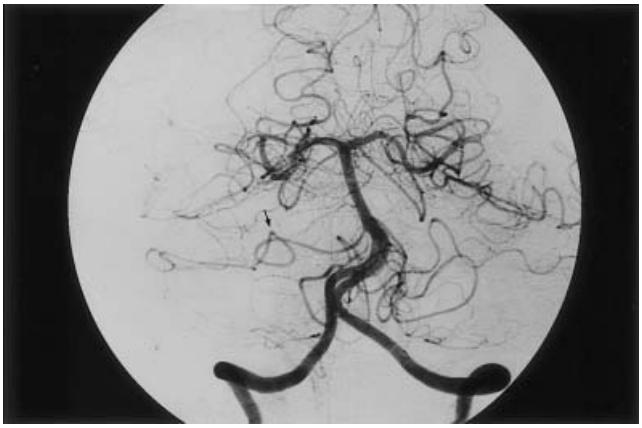
Fig. 4 Right vertebral angiogram. (A) Straight AP view. Anatomical classification with bilateral dominant AICA. Compressing portion of right dominant AICA is indicated by an arrow. (B) Lateral view. Compressing portion is indicated by an arrow.

The AICA generally arises from the basilar artery, runs lateroinferiorly, forming a meatal loop at the lateral end of the pontomedullary sulcus, i.e., around the VIIth·VIIIth cranial nerves, and subsequently divides into the rostral and caudal branches. The rostral branch runs along the flocculus, reaching the great horizontal fissure, and mainly supplies the cerebellar petrosal surface above the fissure. The caudal branch runs in the direction lateroinferior to the VIIth·VIIIth cranial nerves, supplying the cerebellar petrosal surface.[9]

The PICA originates from the VA, but its subsequent direction varies. When its origin level is high and near the vertebrobasilar junction, the PICA is associated with the facial nerves in some cases. The PICA ascends, forms an upward convex loop at the lateral end of the pontomedullary sulcus, approaches the VIIth·VIIIth cranial nerves, and posterosuperiorly runs as the lateral medullary segment, reaching the dorsal surface of the medulla oblongata.

The distributions of the AICA and PICA in the cerebellum are generally complementary. When the PICA is hypoplastic, the AICA is well developed, supplying the original PICA territory. When the PICA is completely absent, the well-developed AICA originates as a common trunk (dominant AICA) from the basilar artery, feeding both the original AICA territory (rostral branch) and PICA territory (caudal branch). In contrast, when the AICA is hypoplastic, the PICA is well developed. When the AICA is completely absent, the well-developed PICA originates as a common trunk from the VA (dominant PICA).[10–13]

Therefore, with these anatomical considerations, images were interpreted, and the site of the facial nerve origin was estimated based on the positional relation to the internal acoustic foramen, and the compressing vessel and vessels running near the compressing vessel were also evaluated before operation.[14,15] In addition, the presence or absence of common trunk anomalies, the levels of the origins of the AICA and PICA, their calibers at the origin, bifurcation site, and the level of the cranial loop and the largest caliber of the fourth segment of the VA were evaluated.



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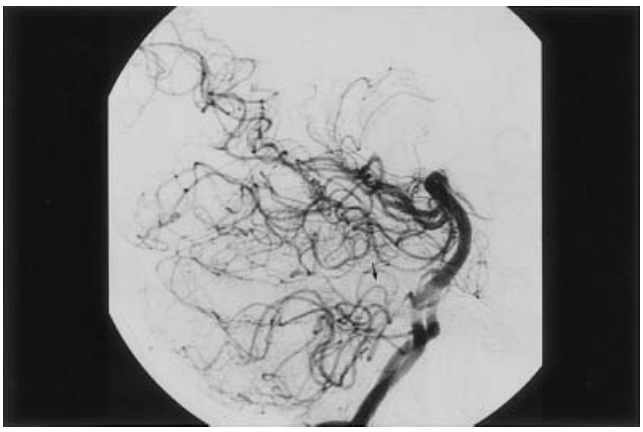

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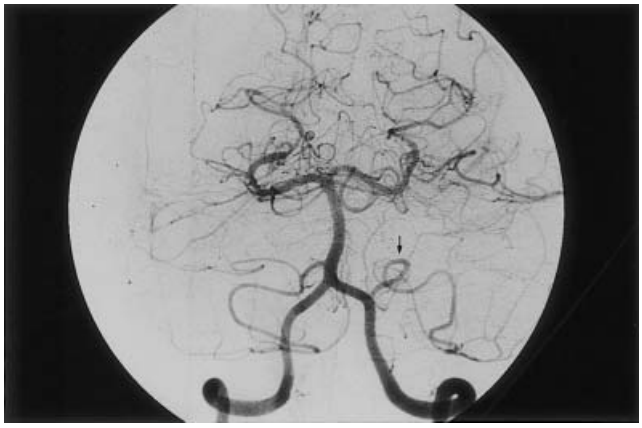
Fig. 5 Right vertebral angiogram. (A) Straight AP view. Anatomical classification with normal distribution of AICA and PICA. Compressing portion of right AICA indicated by an arrow. (B) Lateral view. Compressing portion indicated by an arrow.



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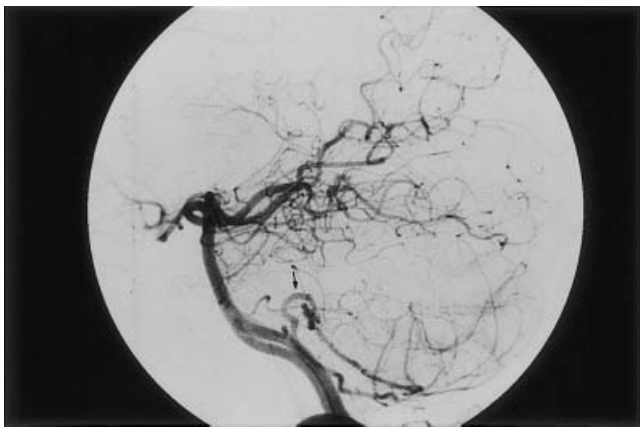

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Fig. 6 Left vertebral angiogram. (A) Straight AP view. Anatomical classification with left dominant PICA and right dominant AICA. Compressing portion of left dominant PICA indicated by an arrow. (B) Lateral view. Compressing portion indicated by an arrow.

Results

The nerve-compressing vessel was the AICA, PICA or VA. Patients with compression by veins or without compressing vessels were excluded.

Originating patterns of the AICA and PICA

The originating patterns of the affected AICA and PICA were classified with consideration of their distribution into 3 types: normal distribution in which the AICA is present independently of the PICA (Fig. 5); dominant AICA showing a common trunk of the two arteries originating from the basilar artery (Fig. 4); dominant PICA showing a common trunk of the two arteries originating from the VA (Fig. 6).

Types II and III were defined as common trunk anomalies. The AICA was present as multiple vessels in 2 patients, of whom 1 with the PICA independent from the AICA vessels was considered to have a normal distribution, and the other without a normal PICA who had one of the AICA vessels as a common trunk was considered to have a dominant AICA. There were 3 patients with the PICA as multiple vessels. All of them were considered to have the dominant PICA because a normal AICA was absent, and one of the PICA vessels was present as a common trunk.

Compressing vessel

The compressing vessel was the dominant AICA in 26 patients (37%), dominant PICA in 20 (29%), AICA in 13 (19%), PICA in 2 (3%), and VA in 9 (13%). Indirect compression by the VA was observed in 2 patients with a dominant AICA, 3 with a dominant PICA,

Table 1 Operative Findings

Compressing vessel	No. of cases
dominant AICA	26 (37%)*
dominant PICA	20 (29%)**
AICA	13 (19%)***
PICA	2 (3%)
VA	9 (13%)

Including indirect VA compression * 2 cases, ** 3 cases, *** 6 cases.

Table 2 Distribution of HFS and Normal Cases by Common Trunk Anomaly (Without direct or indirect VA compression)

	50 cases of HFS	100 cases of normal[16]
Normal distribution	9 (18%)	48%
dominant AICA	24 (48%)	40%
dominant PICA	17 (34%)	10%

Table 3 Common Trunk Anomaly and Compression Vessel (Without direct or indirect VA compression)

a) Normal Distribution on One Side and Common Trunk Anomaly on the other Side

	Rate of compression vessel
dominant AICA	13/15 (87%)
dominant PICA	5/5 (100%)

b) Cases of Bilateral Common Trunk Anomaly

	Rate of compression vessel with a large caliber
bilateral dominant AICA	6/8 (75%)
bilateral dominant PICA	6/6 (100%)
dominant AICA and dominant PICA	9/9 (100%)*

* dominant AICA, 3 cases: dominant PICA, 6 cases.

and 6 with the AICA as the compressing vessel (Table 1). Indirect compression by the VA means as follows: the AICA or PICA is between the VA and the nerve, they compress the nerve. Furthermore the VA compresses the nerve on that vessel. Direct compression by the VA means that only the VA compresses the nerve directly.

Compression vessel in patients excluding those with direct·indirect compression by the VA

After excluding 20 patients with direct·indirect compression by the VA, the compressing vessel was a normal vessel in 9 patients (18%), a dominant AICA in 24 (48%), and a dominant PICA in 17 (34%).

One hundred cases of healthy individuals showed normal vessels in 48 patients (48%), dominant AICAs in 40 (40%), and dominant PICAs in 10 (10%) (Table 2).[16]

Involvement of common trunk anomalies

Of the above 50 patients, 15 had normal distribution on one side and a dominant AICA on the other. The compressing vessel was a dominant AICA in 13 (87%) of the 15 patients and the AICA in normal distribution in the other 2. In all 12 patients in whom the caliber at the origin of the dominant AICA was larger than that of the contralateral AICA/PICA, the dominant AICA was the compressing vessel.

In all 5 patients with normal distribution on one side and a dominant PICA on the other side, the dominant PICA was the compressing vessel (Table 3-a), and the caliber at the origin of the dominant PICA was larger than that of the contralateral AICA/PICA.

When there were bilateral common trunk anomalies, the compressing vessel was present on the side showing a larger caliber at the origin in 6 (75%) of 8 patients with a bilateral dominant AICA, all 6 with bilateral dominant PICAs, all 9 with dominant AICAs on one side and dominant PICAs on the other side (compressing vessel: dominant AICA, 3 patients; dominant PICA, 6) (Table 3-b).

Caliber at the origin of the compressing vessel

The compressing vessel was classified as the dominant AICA, AICA, dominant PICA, and PICA excluding those associated with indirect VA compression, VA causing direct compression, and VA causing indirect compression. After excluding patients without a corresponding vessel on the normal side, the caliber at the origin of the compressing vessel (largest caliber of the fourth segment for the VA) was larger than that of the corresponding vessel on the unaffected side in 18 (86%) of 21 with dominant AICAs as a compressing a vessel, 5 (71%) of 7 with AICAs, 13 (93%) of 14 with dominant PICA, all 2 with PICAs, 8 (89%) of 9 with VA, and 10 (91%) of 11 with VAs causing indirect compression (Table 4).

Levels of the origin and bifurcation of the compressing vessel

For the AICA, dominant AICA, PICA, and dominant PICA excluding those associated with indirect VA compression, the levels of the origin and bifurcation were compared between the compressing vessel and the

Table 4 Caliber of Compression Vessel and Normal Vessel

Compression vessel	No. of large caliber	
	Compression side	Normal side
dominant AICA*	18 (86%)	3 (14%)
AICA	5 (71%)	2 (29%)
dominant PICA**	13 (93%)	1 (7%)
PICA	2 (100%)	0
VA (Largest caliber of fourth segment)	8 (89%)	1 (11%)
VA of indirect compression	10 (91%)	1 (9%)

* Excluding 3 cases without AICA on the normal side. ** Excluding 3 cases without PICA on the normal side.

Table 5 The Higher Side of Compression Vessel and Normal Vessel

Compression vessel	Stem		Bifurcation	
	Compression side	Normal side	Compression side	Normal side
dominant AICA*	11 (52%)	10 (48%)	15 (71%)	6 (29%)
AICA	4 (57%)	3 (43%)	5 (71%)	2 (29%)
dominant PICA**	12 (86%)	2 (14%)	13 (93%)	1 (7%)
PICA	2 (100%)	0	2 (100%)	0

* Excluding 3 cases without AICA on the normal side. ** Excluding 3 cases without PICA on the normal side.

Table 6 Age and Duration

Compression vessel	No. of cases	Mean age $\pm$ SD	Mean duaration $\pm$ SD (month)
dominant AICA	24	51.0 $\pm$ 11.7	61.4 $\pm$ 40.5
AICA	7	53.3 $\pm$ 9.4	14.1 $\pm$ 89.7
dominant PICA	17	46.1 $\pm$ 11.7	56.8 $\pm$ 56.6
PICA	2	36.5 $\pm$ 16.3	36.0 $\pm$ 0.0
VA*	20	55.4 $\pm$ 8.2	53.0 $\pm$ 29.3

* include indirect compression.

Table 7 Post-Operative Evaluation

Authors (year reported)	No. of patients	No. of MVD	Excelent	Good	Poor
Oizumi (2003)	70	70	96%	1%	3%
Barker (1995)[14]	612	672	84%	7%	9%
Illingworth (1996)[17]	83	86	92%	3%	5%
Nagahiro (1997)[18]	101	106	85%	8%	7%
Samii (2002)[19]	143	145	91%	2%	8%

corresponding vessel on the unaffected side. When the compressing vessel was the dominant AICA, its origin level was higher than that of the corresponding vessel on the unaffected side in 11 (52%) of 21 patients, and its bifurcation level was higher than that of the corresponding vessel in 15 (71%). When the compressing vessel was the AICA, its origin level was higher than that of the corresponding vessel in 4 (57%) of 7 patients, and its bifurcation level was higher in 5 (71%). When the compressing vessel was the dominant PICA, its origin level was higher than that of the corresponding vessel in 12 (86%) of 14 patients, and its bifurcation level was higher in 13 (93%). Both patients with the PICA as the compressing level showed higher origins and bifurcation levels than those of the corresponding vessel on the unaffected side (Table 5). All 9 patients with the VA as a compressing vessel and all 11 with the VA as an indirect compressing vessel showed a higher level of its cranial loop than that of the corresponding vessel on the unaffected side.

Mean age and duration until surgery according to the compressing vessel

In patients with the AICA, dominant AICA, PICA, or dominant PICA as the compressing vessel excluding those associated with indirect VA compression, the mean age and mean duration until surgery were 51.0 $\pm$ 11.7 years (mean $\pm$ SD) and 61.4 $\pm$ 40.5 months (mean $\pm$ SD), respectively, in 24 patients with dominant AICAs, 53.3 $\pm$ 9.4 years and 14.1 $\pm$ 89.7 months in 7 with AICAs, 46.1 $\pm$ 11.7 years and 56.8 $\pm$ 56.6 months in 17 with dominant PICAs, 36.5 $\pm$ 16.3 years and 36.0 $\pm$ 0.0 months in 2 with PICAs, and 54.4 $\pm$ 8.2 years and 53.0 $\pm$ 29.3 months in 20 with VAs including VAs causing indirect compression (Table 6). The mean age significantly differed ($p < 0.01$) between patients with the dominant PICA and VA (including VA causing indirect compression).

Surgical results

Surgical results were classified according to the postoperative state into: (1) excellent, disappearance of symptoms without a decrease in facial muscle power; (2) good, disappearance of symptoms despite a transient decrease in facial muscle power; and (3) poor, negligible improvement. Excellent results were obtained in 67 (96%) of 70 patients, good results in 1 (1%), and poor results in 2 (3%). The 2 patients with poor results underwent re-operation and showed complete disappearance of spasms (Table 7).[14,17–19]

Discussion

Estimation of the compressing vessel and the incidence of each vessel causing compression

Preoperative vertebral angiography in patients with hemifacial spasm allows the exclusion diagnosis of aneurysms and arteriovenous anomalies, and the estimation of the site of the compressing vessel site. Recntly, MRI has often been used for diagnosis of hemifacial spasm.[20–23] However oblique sagittal gradient echo MRI discriminated between the AICA and PICA, with a true positive rate of 75.9%.[23] The diagnosis of common trunk anomaly is difficult using MRI.

Towne, straight AP, and lateral images were obtained in all patients, and straight AP images were optimal for diagnosis. On Towne images, there were many overlapping vessels in the cerebellopontine angle, and the estimation of compressing sites was difficult. On the other hand, magnified stereographic imaging and film subtraction on straight AP images further facilitated reading. However, for estimating compression sites, not only subtraction films but also evaluation of the positional relation between the vessels and internal acoustic foramen in negative plates was very useful.

As Table 8 shows, previous studies reported various incidences of each vessel as the compressing vessel.[17–19,24] However, the incidence of the AICA was frequently the highest, followed in order by the PICA and the VA, which was similar to the present results.

Involvement of common trunk anomalies in hemifacial spasm

Takahashi,[16] who evaluated vertebral angiograms in 100 normal subjects, reported the total, dominant AICA in 40% and dominant PICA in 10% (50%). In the present patients with hemifacial spasm excluding VA compression, the dominant AICA accounted for 48% and dominant PICA for 34% (82%). These percentages were higher than those reported in the normal subjects by Takahashi. In particular, the dominant PICA was observed in 10% of the normal subjects but in 34% of the compressing vessels.

Table 8 Compression Vessels

Authors (year reported)	No. of cases	AICA	PICA	VA	Others
Oizumi (2003)	70	56%	31%	13%	0%
Huang (1992)[24]	310	56%	39%	4%	1%
Illingworth (1996)[17]	89	16%	55%	9%	20%
Nagahiro (1997)[18]	101	51%	19%	29%	1%
Samii (2002)[19]	145	66%	19%	4%	11%

When the unilateral dominant AICA/PICA and normal distribution were observed, the compression vessel was the dominant AICA in 87% of patients with a dominant AICA and the dominant PICA in 100% of patients with a dominant PICA. In particular, when the caliber of the a dominant AICA/PICA was larger than that of the AICA/PICA on the unaffected side, the compression vessel was the dominant AICA/PICA in 100%.

When there were bilateral common trunk anomalies, the vessel showing a larger caliber at the origin was the compressing vessel in 75% of patients with a bilateral dominant AICA and 100% in those with a bilateral dominant PICA, and 100% in those with a dominant AICA on one side and dominant PICA on the other, suggesting the close involvement of the caliber at the origin.

Therefore, in patients with hemifacial spasm in whom vertebral angiography excludes the involvement of the VA, when a unilateral dominant AICA/PICA is present, there is a strong possibility that this dominant vessel is the compressing vessel. This possibility further increases when the caliber of this dominant vessel is larger than that of the normal AICA/PICA on the unaffected side.

When bilateral common trunk anomalies are present, it is possible that the vessel with a larger caliber at its origin is the compressing vessel, particularly in patients with a bilateral dominant PICA and those with a dominant AICA on one side and dominant PICA on the other side.

Considering the presence of a compressing vessel on the affected side together with the above findings, the compressing vessel can be estimated at a relatively high probability when there is a common trunk.

Calibers of the origin of the compressing vessel and the corresponding vessel on the unaffected side and the levels of their origin and bifurcation

When the compressing vessel was the dominant AICA, AICA, dominant PICA, PICA, VA causing direct compression, and VA causing indirect compression, the caliber of the origin of the compressing vessel (largest caliber of the fourth segment for the VA) was larger than that of the corresponding vessel on the unaffected side in 86%, 71%, 93%, 100%, 89%, and 91%, respectively. Thus, the caliber of the origin of each type of compressing vessels was larger than that of the corresponding vessel on the unaffected side in a very high percentage of patients.

The origin level of the compressing vessel was higher than that of the corresponding vessel on the unaffected side in 52% and 57%, and the bifurcation level of the compressing vessel was higher than that of the corre-

sponding vessel in 71% and 71% of patients with dominant AICAs and AICAs, respectively, as the compressing vessel. Of patients with a dominant PICA and PICA, 86% and 100%, respectively, showed a higher origin level of the compressing vessel, and 93% and 100%, respectively, showed a higher bifurcation level of the compressing vessel than that of the corresponding vessel on the unaffected side. In the VA, the level of the bent portion was high on the affected side in both direct and indirect compression.

This may be because vessels with a large caliber at the origin have lower vascular resistance than narrow vessels and therefore, receive more blood flow, more readily undergo bending·stretching·dilation, and more often compress the origin of the facial nerves.[25]

Comparison of the levels of the origin and bifurcation between the affected and unaffected sides showed only slight differences for the dominant AICA and AICA as compressing vessels, which may be due to their original distribution near the origin of the facial nerves. However, there was a spatial gap between the origin of a dominant PICA or PICA and the origin of the facial nerves above. A dominant PICA or PICA with its origin and bifurcation at higher levels reduced the distance to the nerve and may tend to be a compressing vessel. When the compressing vessel was the VA, its bent portion was often present at a high level, which may be because the bent portion compresses the origin of the facial nerves, and its high level reduces the normally long distance to the facial nerves.

Mechanism of hemifacial spasm and the involvement of common trunk anomalies

In hemifacial spasm, the responsible vessel is normal or a normal variant in principle. Aged-related vascular bending, stretching, and dilation were suggested to be involved in the development of nerve compression. Therefore, hemifacial spasm often occurs after middle age. In the present study, the mean age of the subjects was 51 years.

The present results suggested that the vascular architecture itself in addition to age-related arterial changes is an important factor for the development of hemifacial spasm. The high incidence of common trunk anomalies may be due to the complicated arrangement of the main trunk, rostral branch, and caudal branch near the origin of the facial nerves. Age-related changes in addition to vascular anomalies may induce nerve compression.

The mean age significantly differed ($p < 0.01$) between patients with a dominant PICA as the compressing vessel and those with a VA (including VA causing indirect compression). This may be because of the difference in the distance between the compressing vessel and the origin of the facial nerves. Due to the considerable distance between the VA and the origin of the facial nerves, a long period is necessary for the development of age-related VA changes that induce spasm. In contrast, the trunk and branches of a dominant PICA are present near the origin of the facial nerves and may more readily induce hemifacial spasm than the VA even in relatively young people.

Based on the present results, the preoperative estimation of the responsible artery and compression site may be possible using vertebral angiography in most patients. However, in general, cerebral angiography is not always a safe examination method though no complications were observed in our subjects. When lesions such as cerebral aneurysm, arteriovenous anomalies, and tumors are completely excluded by high resolution CT, 3D CT Angio, MRI, or MRA, cerebral angiography is not always necessary between operation. However, when an operation is performed without vertebral angiography, whether the compressing vessel is the AICA or PICA can not be determined in some patients with complicated vasculature. Janetta *et al.*[4] reported the highest incidence of compression by the PICA while we observed the highest incidence of compression by the dominant AICA. The results obtained by Janetta may have been associated with vertebral angiography performed only in some of the patients. Many previous studies have shown the highest incidence of AICA as the compressing vessels, followed in order by the PICA and VA, but some showed the highest incidence of the PICA (Table 8). Therefore, we reported the results of preoperative vertebral angiography that had been performed at our institution to clarify the pathology of vascular compression of nerves in hemifacial spasm.

Compared with previous findings, the postoperative results in the present study were excellent (Table 7).

We evaluated the association between findings of preoperative vertebral angiography and operative findings in 70 patients with hemifacial spasm who underwent neurovascular decompression. Straight AP images were the most useful for interpreting the images. The compressing vessel was most frequently the group with AICA, followed in order by the group with PICA and VA. After excluding VA-associated cases, dominant AICAs and dominant PICAs accounted for 82% of the compressing vessels. In the group with the AICA as the compressing vessels, a dominant AICA was observed in 78%. In the group with the PICA as the compressing vessels, a dominant PICA was observed in 89%. When the unaffected side showed a normal vascular distribution, the compressing vessel was a dominant AICA in 87% of patients with a dominant AICA and 100% of patients with a dominant PICA. In most patients, the

responsible vessel and compression site could be determined by vertebral angiography.

References

1. Cushing H: Strangulation of the nervi abducentes by lateral branches of the basilar artery in case of brain tumor. Brain 1911; 33: 204–235
2. Campbell E, Keedy C: Hemifacial spasm: a note on the aetiology in two cases. J Neurosurg 1947; 4: 342–347
3. Gardner WJ, Sava GA: Hemifacial spasm: a reversible pathophysiologic state. J Neurosurg 1962; 19: 240–247
4. 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 1977; 47: 321–328
5. Okamura T, Aoki H, Yanai K, Abe T, Oh M: Radiographic positions of the cranial nerve root (V, VII–XI) proximal zones with special reference to microvascular decompression. Neurol Med Chir (Tokyo) 1983; 23: 776–782 (in Japanese)
6. Jannetta PJ: Microsurgery of cranial nerve cross-compression. Clin Neurosurg 1979; 26: 607–615
7. Jannetta PJ: Neurovascular compression in cranial nerve and systemic disease. Ann Surg 1980; 192: 518–525
8. Martin RG, Grant JL, Peace D, Theiss C, Rhoton AL Jr: Microsurgical relationships of the anterior inferior cerebellar artery and the facial-vestibulocochlear nerve complex. Neurosurgery 1980; 6: 483–507
9. Matsushima T, Rhoton AL Jr, Lenkey C: Microsurgery of the fourth ventricle: Part 1. Microsurgical anatomy. Neurosurgery 1982; 11: 631–667
10. Sunderland S: Neurovascular relations and anomalies at the base of the brain. J Neurosurg Psychiatry 1948; 11: 243–257
11. Lister JR, Rhoton AL Jr, Matsushima T, Peace DA: Microsurgical anatomy of the posterior inferior cerebellar artery. Neurosurgery 1982; 10: 170–199
12. Carlos R, Fukui M, Hasuo K, Uchino A, Matsushima T, Tamura S, Kudo S, Kitamura K, Matsuura K: Radiological analysis of hemifacial spasm with special reference to angiographic manifestations. Neuroradiology 1986; 28: 288–295
13. Baba T, Matsushima T, Fukui M, Hasuo K, Yasumori K, Masuda K, Kuromatsu C: Relationship between angiographical manifestations and operative findings in 100 cases of hemifacial spasm No Shinkei Geka 1988; 16: 1355–1362 (in Japanese)
14. Barker FG 2nd, Jannetta PJ, Bissonette DJ, Shields PT, Larkins MV, Jho HD: Microvascular decompression for hemifacial spasm. J Neurosurg 1995; 82: 201–210
15. Okamura T, Aoki H, Yanai K, Abe T, Oh M: Radiographic positions of the cranial nerve root (V, VII–XI) proximal zones with special reference to microvascular decompression. Neurol Med Chir (Tokyo) 1983; 23: 776–782 (in Japanese)
16. Takahashi M: The anterior inferior cerebellar artery. In: Newton TH, Potts DG, eds, Radiology of the Skull and Brain: Angiography, Book 2, St. Louis, Mosby, 1974; 1796–1808
17. Illingworth RD, Porter DG, Jakubowski J: Hemifacial spasm: a prospective long-term follow up of 83 cases treated by microvascular decompression at two neurosurgical centres in the United Kingdom. J Neurol Neurosurg Psychiatry 1996; 60: 72–77
18. Nagahiro S: Pathophysiology and surgical treatment of hemifacial spasm. No Shinkei Geka 1998; 26: 101–111 (in Japanese)
19. Samii M, Gunther 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 2002; 50: 712–718
20. Harsh GR 4th, Wilson CB, Hieshima GB, Dillon WP: Magnetic resonance imaging of vertebrobasilar ectasia in tic convulsif. Case report. J Neurosurg 1991; 74: 999–1003
21. Nagaseki Y, Horikoshi T, Omata T, Ueno T, Uchida M, Nukui H, Tsuji R, Sasaki H: Oblique sagittal magnetic resonance imaging visualizing vascular compression of the trigeminal or facial nerve. J Neurosurg 1992; 77: 379–386
22. Nagaseki Y, Omata T, Ueno T, Uchida M, Ohhashi Y, Kase M, Nukui H, Tsuji R: Prediction of vertebral artery compression in patients with hemifacial spasm using oblique sagittal MR imaging. Acta Neurochir (Wien) 1998; 140: 565–571
23. Pego Reigosa R, Pulpeiro Rios JR: Hemifacial spasm. J Neurol Neurosurg Psychiatry 1998; 64: 687
24. Huang CI, Chen IH, Lee LS: Microvascular decompression for hemifacial spasm: analyses of operative findings and results in 310 patients. Neurosurgery 1992; 30: 53–56
25. Kondo A, Ishikawa J, Konishi T, Yamasaki T: Mechanism of vascular compression of cranial nerves: role of changes of vertebro-basilar vasculatures. Neurol Med Chir (Tokyo) 1981; 21: 287–293 (in Japanese)