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## Hemifacial Spasm: MR Imaging Features

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MR imaging was used to evaluate the relationship of the root exit zone of the seventh cranial nerve to surrounding vascular structures in 13 patients with clinically documented hemifacial spasm and in 70 asymptomatic patients. MR imaging clearly demonstrated the course of the seventh nerve from the root exit zone of the brainstem to the internal auditory canal and its relationship to the surrounding vertebrobasilar system. The presence of a vascular structure at the root exit zone of the seventh nerve was identified in all 13 patients with hemifacial spasm. In the 70 asymptomatic patients, examination of 140 seventh nerves revealed that 21% had contact by a vascular structure at the root exit zone of the seventh nerve.

Our results indicate that although neurovascular contact may be asymptomatic, MR demonstration of a vascular structure at the root exit zone of the seventh cranial nerve in a patient with hemifacial spasm may implicate neurovascular compression as the cause of symptomatology. This finding may alter therapeutic management. Because of the inherent limitations of CT in the visualization of posterior fossa structures, MR imaging should be considered the initial screening procedure in the assessment of patients with hemifacial spasm.

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Hemifacial spasm is characterized by unilateral hyperactive facial nerve dysfunction that is commonly reflected in intermittent, painless spasm of the orbicularis oculi muscle, and may progress in frequency and severity to include the other muscles involved with facial expression [1]. There is extensive evidence supporting the hypothesis that hemifacial spasm is caused by compression of the seventh cranial nerve at its anterior caudal root exit zone (REZ) by vascular loops of either the posterior inferior cerebellar artery (PICA), anterior inferior cerebellar artery (AICA), vertebral artery, or cochlear artery [2]. In addition, aneurysms of these arteries, arteriovenous malformations, veins, and cerebellopontine angle masses occasionally can compress the facial nerve at its REZ to cause hemifacial spasm. Although this theory is controversial [3], this mechanism has been invoked as the cause of other hyperactive dysfunction syndromes, including trigeminal neuralgia, hyperactive dysfunction of the eighth nerve, and glossopharyngeal neuralgia [4].

Conservative medical treatment of patients with hemifacial spasm includes carbamazepine or baclofen [5, 6]. The surgical procedure of choice in patients who do not respond to conservative treatment consists of microvascular decompression. This procedure provides complete resolution or improvement of symptoms in the majority of cases. Whether surgical success is due to removal of the offending agent or to trauma to the nerve itself is a controversial point [3]. Despite the controversy over the cause and surgical treatment of hemifacial spasm, demonstrable compression of the REZ of the seventh nerve may alter therapeutic management.

We used MR imaging in 13 patients with hemifacial spasm to determine whether neurovascular compression of the seventh nerve at the REZ could be detected. In

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addition, we retrospectively studied the brainstem area on MR images of 70 asymptomatic patients with vascular compression of the REZ of the seventh nerve to determine if vascular compression can be seen in the asymptomatic individual.

### Patients and Methods

Thirteen patients with a diagnosis of hemifacial spasm were referred to our institution for MR imaging. This study group comprised six men, 40–80 years old, and seven women, 31–75 years old. Examinations were performed on a superconductive magnet operated at 1.5 T (GE Medical Systems, Milwaukee). T1-weighted axial and coronal images were obtained with imaging parameters of 400/20/6 (TR/TE/excitations), 3-mm section thickness with no intersection gap, matrix size of 256 × 128 or 256 × 192, and 16-cm field of view. On the coronal T1-weighted images, all investigators independently attempted to trace the vessel found at the REZ to its origin from the vertebrobasilar system. Linear thin or contiguous focal areas of signal void were interpreted as blood vessels, either arterial or venous. Naming the exact vessel was considered to be less important, since this would not alter the surgical technique if microvascular decompression were to be performed. In all 13 patients with hemifacial spasm, a vascular structure was found at the REZ of the seventh cranial nerve corresponding to the side of the patient's symptomatology. Of the 13 patients, one had microvascular decompression, at which time a tortuous vertebral artery was found at the REZ of the seventh nerve. The other patients refused surgery despite facial spasms that were resistant to medical treatment.

Our control group consisted of 70 patients referred for evaluation of the pituitary gland because of a suspected endocrine abnormality. This group comprised 18 men and 52 women, with an average age of 37 years (range, 16–73 years). T1-weighted (600/20/2) coronal images were obtained with a 3-mm slice thickness with no intersection gap, 256 × 256 matrix size, and 16-cm field of view. Despite the slight differences in imaging techniques, there was excellent visualization of the brainstem as well as its relationship to surrounding vascular structures with both protocols.

The relationship between the REZ of the seventh cranial nerve and vertebrobasilar system was divided into three major categories: no contact, contact with the REZ, and deformity of the REZ. The category "contact with the REZ" comprised those cases in which the REZ of the seventh nerve had a vascular structure adjacent to it but in which the course of the nerve was normal, without deformity. The category "deformity of the REZ" consisted of those cases in which both neurovascular contact with the REZ and secondary deformity of the REZ were seen. Because of the angle that the seventh nerve takes with the brainstem, deformity of the REZ also included cases in which a vascular structure was noted to clearly deform the adjacent pons.

In each case, two investigators independently evaluated the relationship of the vessels to the seventh nerve and assigned it to one of the three categories. Results were then compared, indeterminate cases were reviewed, and a consensus was reached.

### Results

Brief case histories and MR findings for all 13 study patients are summarized in Table 1. Representative images from selected cases are shown in Figures 1 and 2.

Of the 140 seventh nerves examined in the control group, 30 (21%) had contact between the REZ and a vascular structure, while two (1%) had contact and deformity of the REZ (Fig. 3).

### Discussion

Hemifacial spasm is a symptom complex comprising involuntary, painless spasms of the orbicularis oculi muscle. It may progress to involve all the facial muscles. It usually afflicts middle-aged women and is generally unilateral, affecting the left more often than the right [1]. Hemifacial spasm is an exceptionally distressing symptom that can be quite disabling, interfering with important daily activities because of forced eye closure. It is thought that hemifacial spasm is related to compression of the facial nerve at the anterior caudal aspect of the REZ from the brainstem by vascular loops or aneurysms, veins, arteriovenous malformations, and cerebellopontine angle masses [2, 7]. At the REZ, the motor root of the facial nerve is medial to both the nervus intermedius and the acoustic nerve. Medially located, redundant arterial loops will preferentially impinge upon the motor fibers causing hemifacial spasm.

Pathologically, defects exist in the REZ of cranial nerves at points where central myelin has been replaced by peripheral myelin. It is thought that continued pulsatile pressure at the seventh nerve REZ junctional area results in disordered conduction and "short-circuiting" of impulses, resulting in hemifacial spasm [4].

Although no medical treatment has proved consistently effective, some patients may respond dramatically to carbamazepine or baclofen [5, 6]. In patients refractory to medical therapy, microvascular decompression is among the surgical procedures of choice. This procedure entails moving the causative vessel away from the REZ and interposing a nonresorbable spongy material between the vessel and the brainstem. It provides complete resolution or improvement in the majority of cases [2, 8].

In the past, assessment of patients with hemifacial spasm consisted of CT and/or angiography. Since both CT and angiography failed to demonstrate the relevant portions of the seventh nerve, the results have been mixed. In the series reported by Sobel et al. [9] of 24 patients with hemifacial spasm studied by both CT and vertebral angiography, the vessel responsible for compression was not identified before surgery. Digre et al. [10] have been more successful. Of 46 patients with typical hemifacial spasm studied with CT, 38 (83%) had detectable abnormalities. Thirty-six had dolichoectasia of the vertebrobasilar system, with an artery pointing to the side of spasm in 92% of the cases. The other two had surgically documented tumors. Carlos et al. [11] studied 51 patients with hemifacial spasm who had undergone angiography prior to surgery and who had facial nerve compression by arteries at the time of surgery. Although there was a significant increase in the number of common trunk anomalies and variations of the AICA, PICA, and vertebral arteries as compared with the normal population, these researchers sug-

TABLE 1: Findings in 13 Patients with Hemifacial Spasm

Case No.	Age (years)	Sex	Clinical History	MR Findings	Case No.	Age (years)	Sex	Clinical History	MR Findings
1	75	F	Intermittent twitching of right eye and cheek of recent onset; no medical treatment	Contact and deformity of REZ on right, probably by AICA	6	42	M	Intermittent twitching of left eye with subsequent involvement of cheek and forehead for approximately 1 yr; no medical treatment	Contact and deformity of REZ on left, probably by PICA
2	40	M	Intermittent twitching of left eye and cheek with subsequent involvement of corner of mouth and angle of jaw of 6 mo duration; no medical treatment	Contact and deformity of REZ on left by a tortuous vertebral artery	7	31	F	Intermittent twitching of right eye of at least several months duration; no medical treatment	Contact and deformity of REZ by a vascular structure
3	73	M	Intractable left hemifacial spasm of 5 yr duration; inconstant relief while on medical regimens, including carbamazepine and baclofen; spasms markedly improved 6 wk after MVDC; recurrent symptoms 3 mo after surgery prompted reexploration	Contact and deformity of REZ on left by a tortuous vertebral artery (Fig. 1) <sup>a</sup>	8	80	M	Intermittent twitching of left eye of several years duration; inconstant relief with medical treatment	Contact and deformity of REZ on left by a vascular structure
4	46	F	Intermittent twitching of right eye with subsequent involvement of the cheek of 4 yr duration; some improvement on baclofen, 10 mg three times daily	Contact at REZ on right by a vascular structure	9	49	M	Intermittent twitching of left eye with subsequent involvement of cheek of 4 yr duration; no improvement with medical treatment	Contact and deformity of REZ on left by a vascular structure
5	47	F	Intermittent twitching of left eye with subsequent involvement of cheek of 4–5 yr duration; some improvement on baclofen, three times daily	Contact and deformity of REZ on left, probably by AICA (Fig. 2)	10	63	F	Intermittent twitching of right eye for 2 yr; no medical treatment	Contact and deformity of REZ on right, probably by a large vertebral artery and PICA
					11	55	F	Intermittent twitching of right eye with subsequent involvement of cheek of 4 yr duration; no medical treatment	Contact and deformity of REZ on right by a vascular structure
					12	64	F	Intermittent twitching of right eye; no medical treatment	Contact and deformity of REZ on right by a vascular structure
					13	42	M	Intermittent twitching of left eye, extending to forehead; no medical treatment	Contact and deformity of REZ on left by a vascular structure

Note.—REZ = root exit zone, AICA = anterior inferior cerebellar artery, MVDC = microvascular decompression, PICA = posterior inferior cerebellar artery.

<sup>a</sup> At surgery, a tortuous vertebral artery was found. A second operation, after recurrence of symptoms, revealed fibrous adhesion of vertebral artery to seventh nerve.

gest that the angiographic findings would usually be interpreted as normal or as variants of normal in the asymptomatic individual.

MR imaging directly depicts the course of the seventh nerve from the brainstem to the internal auditory canal. This portion of the seventh nerve is more easily evaluated with MR imaging than with other imaging methods. Compression of the nerve by vascular structures at the REZ is also readily identified.

In the symptomatic patient, compression of the REZ by a vascular structure suggests causation. The rate of occurrence of neurovascular compression in the asymptomatic population, however, has not received as much attention as it has in patients with trigeminal neuralgia in which both anatomic and radiographic studies have been performed. In the 70 asymptomatic patients we examined, 21% of the facial nerves had contact at the REZ and 1% had compression or distortion of the REZ. The average age of these patients was less than that of the patients with symptoms. Presumably, the percentage of asymptomatic patients with contact at the REZ and with compression or distortion of the REZ would rise even

higher if the control population could be exactly age-matched. Because of the high rate of occurrence of contact at the REZ, and even the occurrence of deformity of the REZ in the asymptomatic individual, a definitive diagnosis of neurovascular compression as a cause of symptomatology in patients with hemifacial spasm cannot be made with complete certainty.

In conclusion, CT has limited utility in the evaluation of the posterior fossa and the first portion of the seventh nerve; angiography is invasive and fails to show the exact relationship between nerves and blood vessels. Because MR imaging clearly depicts the course of the seventh nerve from the REZ to the internal auditory canal and the relationship of the seventh nerve to the vertebrobasilar system, it should be considered the initial screening procedure of choice in all patients with hemifacial spasm. Because this study is limited by the number of surgically proved cases, larger prospective studies with surgical correlation are still necessary to confirm our findings as long as the origin and surgical treatment of hemifacial spasm remain controversial.

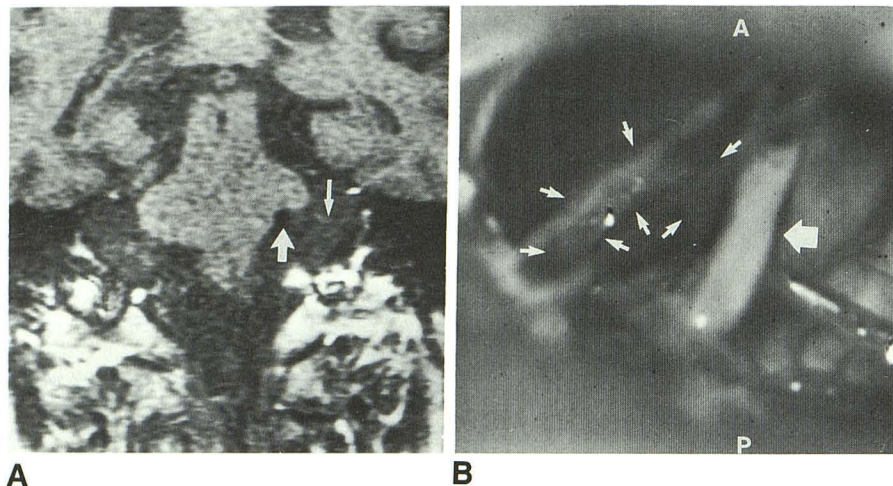


Fig. 1.—Case 3. 73-year-old man with left-sided hemifacial spasm.

A, Coronal T1-weighted MR image shows focal area of signal void (large arrow) deforming left seventh nerve (small arrow) at root exit zone (REZ).

B, Intraoperative micrograph shows ectatic vertebral loop (small arrows) compressing left seventh nerve (large arrow) at REZ. A = anterior, P = posterior.

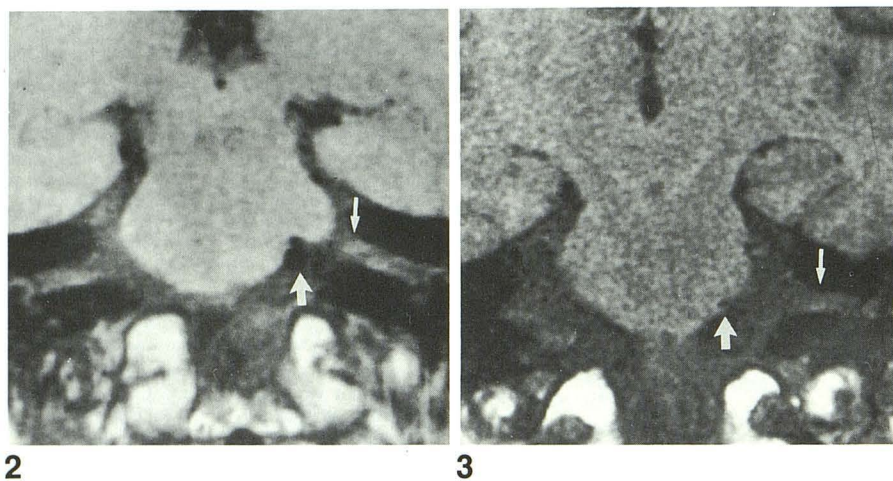


Fig. 2.—Case 9. 49-year-old man with left-sided hemifacial spasm. Coronal T1-weighted MR image shows focal area of signal void (large arrow) deforming left seventh nerve (small arrow) at root exit zone.

Fig. 3.—37-year-old asymptomatic man. Coronal T1-weighted MR image shows focal area of signal void (large arrow) deforming left seventh nerve (small arrow) at root exit zone.

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