The cause of trigeminal neuralgia and hemifacial spasm is multifactorial, and surgery has been reserved for severe cases that have ceased to respond to medical treatment and are considered to be physically fit enough to undergo surgery.1,19,24 With improved anesthetic capabilities and the protective benefits of intraoperative monitoring of brain-stem function, surgery has become more readily recommended.9 However, it has been reported that surgical exploration fails to reveal vascular compression in up to 16% of cases;1,19,24 thus, there is an increasing need to identify preoperatively the site of compression.

We report the case of a woman with tic convulsif20,21 (hemifacial spasm with trigeminal neuralgia) and hypertension in whom a novel magnetic resonance (MR) angiography sequence demonstrated multiple neurovascular compressions, which were subsequently confirmed and successfully treated at surgery.

Case Report

This 60-year-old woman was referred to our institution for assessment of left-sided trigeminal neuralgia and left hemifacial spasm. The pain was lancinating, triggered by touch in the maxillary division, and spread to involve all three divisions of the trigeminal nerve. The pain was exacerbated by eating and exposure to cold. The spasm involved the entire left side of the face excluding the distribution of the temporal branch of the facial nerve. There was an associated residual spastic facial weakness. The patient had been treated for 2 years with local injections of botulinum toxin which had produced good initial relief from the hemifacial spasm; however, over the 5 months preceding her present admission, their efficacy had decreased. Imipramine and carbamazepine had been ineffective in controlling her hemifacial spasm and trigeminal neuralgia.

Examination. The patient had obvious left facial spasms and a persistent fixed spastic deformity. There was a trigger point in the left maxillary area that provoked pain over all three divisions of the trigeminal nerve in the left side of the face. Quantitative sensory testing revealed a marginal impairment of pinprick sensation in the maxillary division of the fifth cranial nerve on the left side of the face. Hearing was normal in both ears.

The patient had been treated for hypertension over
the past 10 years and was currently receiving 50 mg atenolol and 12.5 mg chlorthalidone daily. Her blood pressure during the preceding 2 years had been stable at 160/90 mm Hg and on admission was 160/100 mm Hg. In view of the failure to control her symptoms by pharmacological means, the patient was considered for posterior fossa exploration and microvascular decompression of the left trigeminal, facial, and vagus nerves.

Radiological Studies. Computerized tomography (CT) revealed mild cerebellar atrophy but no evidence of a mass lesion in the cerebellopontine angle. Magnetic resonance angiography with a 1.5-tesla system utilizing an FISP (fast imaging with steady-state precision) three-dimensional sequence was performed. Although the axial slices from an MR angiography sequence are designed to suppress the signal from brain parenchyma while maximizing the intravascular signal, at our institution we have established that the axial slices provide adequate delineation of the cranial nerves to allow accurate assessment of the exact relationships of the nerves to adjacent vessels. The technique produces slices 0.9 mm thick; thus, reconstruction in any plane is possible without loss of resolution. Angiograms were reconstructed from the base data by means of a maximum intensity projection algorithm. Using a combination of the axial images and operator-defined reformatted images, we inspected the fifth, seventh, ninth, and 10th cranial nerves and left ventrolateral medulla. Sagittal reformatted images along the left trigeminal nerve revealed that the nerve was sandwiched between a vessel that descended to contact the nerve from above and a large anterior inferior cerebellar artery (AICA) touching it from beneath (Fig. 1 upper left). The axial images showed that the vessel in contact with the nerve from above continued forward along the nerve (Fig. 1 right) before passing into Meckel’s cave. This was thought to represent a persistent trigeminal artery variant. The elongated and looping left AICA made a prominent 180° medial turn before tucking into the root entry zone of the facial nerve (Fig. 1 lower left). On this side, the posterior inferior cerebellar artery (PICA) arose from the AICA (Fig. 2 left). From its origin, the PICA passed almost vertically downward to the level of the medulla before turning posteriorly to contact the medulla at the root entry zone of the vagus nerve (Fig. 2 right).

Operation. The trigeminal nerve was found to be compressed anteriorly by a loop of a persistent trigeminal artery variant (Fig. 3). The trigeminal nerve was successfully decompressed by displacing the loop anterosuperiorly with the interposition of a polyvinyl sponge. The facial nerve bundle was found to be de-
Preoperative MR angiography for neurovascular compression

Fig. 2. Left: Maximum intensity projection algorithm angiogram showing the horizontally aligned anterior inferior cerebellar artery (AICA) and the prominent loop that compresses the facial nerve. The sites of compression of the fifth nerve (arrow) and seventh nerve (arrowhead) are indicated. The origin of the posterior inferior cerebellar artery (PICA) from the AICA is visible. The persistent trigeminal artery variant is not demonstrated by the angiogram because of its small size. Right: Axial image at the level of the medulla showing the vagus nerve on the right side (arrow). The PICA appears in the root entry zone on the left side (arrowhead).

Fig. 3. Operative view of the left trigeminal nerve with the artery variant loop lying on its anterior margin (arrow). The anterior inferior cerebellar artery loop (asterisk) is compressing the facial nerve anteriorly.

Manipulation of the PICA and this fasciculus, the patient’s blood pressure rose abruptly to 220/120 mm Hg with an associated bradycardia of 40 beats/min. There were marked fluctuations in blood pressure for the remainder of the operation. An attempt was made to displace the offending vessel from the medulla, but this was not totally successful due to the fine perforating vessels that supplied the medulla. Nevertheless, a sponge was interposed between the PICA and the lower rootlets of the 10th cranial nerve, displacing the PICA posteriorly.

Postoperative Course. On regaining consciousness, the patient had some left facial weakness but both her facial spasm and trigeminal neuralgia had resolved. Apart from some nausea, her postoperative course was uneventful. Her blood pressure remained mildly elevated, and she required her preoperative dosage of antihypertensive medication to control it. At her 3-month follow-up examination, she was free of pain and had no hemifacial spasm or added sensory loss, but she suffered some mild residual left facial weakness. Her blood pressure was 150/90 mm Hg. Hypotensive medication was withdrawn, and in the subsequent 4 months her blood pressure has been monitored monthly and has remained at 150/90 mm Hg.

Discussion

A descending loop of the superior cerebellar artery is most commonly found to be the compressing vessel in trigeminal neuralgia. A persistent trigeminal artery variant is the most common carotid-basilar anastomosis and is seen angiographically in 0.1% of cases. This variant has been reported as a rare cause of trigeminal neuralgia, and the artery or one of its variants
was considered responsible for eight of 1257 cases of trigeminal neuralgia treated by microvascular decompression.\textsuperscript{15} Neurovascular compression of the facial nerve at its root entry zone is similarly thought to be responsible for hemifacial spasm.\textsuperscript{11} Although facial spasm may occur as a non-specific reaction to facial pain,\textsuperscript{10} it appears that there is a true association between the two entities; however, less than 60 surgical cases have been reported in the literature.\textsuperscript{2,6,8,12} The cause may be ectasia of the vertebrobasilar system, arteriovenous malformation, posterior fossa tumor, or compression by multiple vessels.\textsuperscript{20,21}

There is a recorded association between hypertension and hemifacial spasm but not in combination with trigeminal neuralgia.\textsuperscript{8} The etiology of hypertension remains unknown in the majority of cases, although an association between hypertension and neurovascular compression of the left ventrolateral medulla at the root entry zone of the ninth and tenth cranial nerves has been proposed.\textsuperscript{12} The mechanism may be a response to local brain-stem ischemia.\textsuperscript{4} Jannetta, et al.,\textsuperscript{12} have reported the presence of neurovascular compression of the left ventrolateral medulla at the root entry zone of the ninth and tenth cranial nerves in 51 of 53 hypertensive patients who underwent left-sided exploration of the posterior cranial fossa for a variety of reasons. The majority of patients in whom decompression of the offending vessel was successfully performed were noted to be normotensive at short-term follow-up examination. A recent postmortem study lends support to this theory.\textsuperscript{17} We are currently conducting a prospective study to establish the neurovascular relationships around the medulla in hypertensive patients and normotensive controls.

Until recently, for various reasons, neuroimaging has been of limited value in the preoperative demonstration of neurovascular compression.\textsuperscript{2,3,16,22,28} Both CT and standard spin-echo MR imaging routinely display slice thicknesses in excess of 3 mm; it is impractical to demonstrate exact neurovascular anatomical relationships with either diagnostic approach when the size of the vessel to be imaged is frequently less than 1 mm. In addition, beam hardening and streak artifact from surrounding bony results in poor delineation of structures in the posterior fossa by CT. Arteriography has also been employed in the preoperative assessment of patients with trigeminal neuralgia to look for a looping vessel on the affected side,\textsuperscript{14} but because this modality does not depict the actual nerves, the presence of a looping vessel in the region of the root entry zone does not necessarily indicate contact between the vessel and the nerve.

Magnetic resonance angiography offers several advantages over conventional MR imaging in the assessment of neurovascular compression syndromes,\textsuperscript{2} and a recent paper has reported promising results with this technique.\textsuperscript{2} In MR angiography, flowing blood produces an intense signal; to further enhance the vascular component, the sequence is designed to suppress any signal from the brain parenchyma to facilitate reconstruction of the angiograms. We have established, however, that delineation of the cranial nerves and brain parenchyma suffices to demonstrate the exact relationships of the nerves to the surrounding arteries, as illustrated by our case. Using this technique, the images can be reconstructed in any plane without loss of detail, and visualization of the entire subarachnoid course of the lower cranial nerves and their vascular relationships is possible. If neurovascular compression is confirmed, the vessel and its origin can be identified from the maximum intensity projection algorithm angiogram. Accurate assessment of the presence and site of vascular compression can thus be made.

**Conclusions**

Vascular compression has been accepted as a causative factor for trigeminal neuralgia and hemifacial spasm in most studies, and has been proved valid with effective treatment by decompression, even if the pathophysiology is not satisfactorily explained. A case has been made that so-called essential hypertension may be due to compression of the root entry zone of the ninth and 10th cranial nerves on the left.

This case illustrates the potential role of MR angiography in assessing the presence of neurovascular compression preoperatively. We know of no similar case in which neurovascular compression was demonstrated preoperatively at all three sites with subsequent surgical validation and successful treatment.

**References**


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