Repositioning of the vertebral artery as treatment for neurovascular compression syndromes

Technical note

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Neurovascular compression syndromes are usually treated by interposing Teflon felt or padding or some other implant between the offending vessel and the nerves. However, this cannot be done in some cases in which ectatic vertebrobasilar arteries are involved. In these instances, alternative techniques must be used.

The authors report the use of a sling made of Prolene to reposition the vertebral artery in two patients with neurovascular compression disorder. The clinical results were gratifying, with complete resolution of the patients’ symptoms.

Compression by large vessels is an uncommon but important source of neurovascular compression in patients with trigeminal neuralgia, hemifacial spasm, disabling positional vertigo, and, possibly, hypertension. The technique described may be useful to surgeons treating these problems.

Key Words • facial nerve • hemifacial spasm • vestibulocochlear nerve • disabling positional vertigo • neurovascular compression syndrome • neurovascular decompression

Illustrative Cases

Case 1

This 51-year-old man presented with a 3-year history of worsening left-sided hemifacial spasm. The spasms originated around the left eye, but extended to involve the entire face, and were present even when the man was at rest. The patient had received multiple nonsurgical treatments including administration of Tegretol, baclofen, and botulinum toxin, without any success. His medical history was significant for hypertension.

Examination. A magnetic resonance (MR) image of the patient’s brain showed an aberrant VA compressing the brainstem near the REZ of the seventh cranial nerve (Fig. 1). On physical examination, spasms were observed whenever the patient talked. They started in the upper face and spread to involve the rest of the face. Mild weakness was observed in the left orbicular muscle of the eye and in the levator muscle of the angle of the mouth. Microvascular decompression of the facial nerve was proposed to the patient and was accepted.

Operation. The patient underwent surgery while in the...
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right lateral decubitus position. Neurophysiological monitoring of the facial nerve’s spontaneous electromyographic activity and of its lateral spread was performed. The cerebellopontine cistern was explored via a left retrosigmoid craniotomy and further craniectomy.

The left VA was seen in an aberrant position, prominently compressing the REZs of the seventh and eighth cranial nerves as well as the proximal portion of the ninth cranial nerve. The vertebrobasilar junction was located anterior to the left seventh and eighth cranial nerves. The proximal portion of the anterior inferior cerebellar artery (AICA) was also compressed against the REZ of the facial nerve by the VA (Fig. 2 left).

An initial attempt was made to mobilize the VA by placing a number of Teflon felt pads between the VA and the brainstem, starting inferiorly in front of the 10th cranial nerve and moving superiorly. However, this was unsuccessful in relieving the lateral spread. The pulsatile pressure continued to be transmitted to the facial nerve, and therefore, we decided to reposition the VA. A No. 7-0 Prolene suture was passed around the VA and stitched to the dura overlying the left jugular tubercle. The left VA was then repositioned laterally, but the suture produced some kinking of the vessel. A relatively large padding of Teflon felt was placed between the artery and the suture, relieving the kinking completely. Additional Teflon padding was placed between the AICA and the REZ of the seventh cranial nerve to decompress that nerve (Fig. 2 right). This abolished the lateral spread and the facial spasms completely. Care was taken to ensure that there was no kinking or excessive stretching of perforating vessels. The wound was closed in the usual fashion.
Postoperative Course. Postoperatively, the patient had complete relief of his hemifacial spasms and has remained free of spasm throughout a follow-up period of 11 months. A postoperative MR angiogram showed normal flow through the artery and MR imaging revealed repositioning of the vessel (Fig. 3).

Case 2

This 59-year-old woman presented with a history of progressively worsening episodic dizziness as well as a bad taste on the left side of her tongue. Oscillopsia, nausea, and vomiting were also present. The patient’s symp-
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Fig. 7. Case 2. Pre- (left) and postoperative (right) MR images shown side by side. The VA has been moved away from the REZ of the eighth cranial nerve (curved arrow in right).

toms worsened to the point that she felt like “jumping off a cliff.”

Examination. Magnetic resonance imaging and MR angiography (Fig. 4) revealed compression of the brainstem and the REZs of the seventh and eighth cranial nerves by a VA that was abnormally situated on the left side. Both VAs displayed an aberrant curve to the left side and into the brainstem.

The dysgeusia was believed to be due to compression of the nervus intermedius and the patient was admitted to our institution to undergo microvascular decompression of the seventh and eighth cranial nerves.

Operation. A left retromastoid craniotomy was performed and the cerebellopontine cistern was explored. The right VA curved toward the left side where it joined the left VA to form the BA, which curved back to the opposite side. The seventh and eighth cranial nerves were markedly compressed by the right VA loop (Fig. 5). The VA loop also caused the AICA to compress these nerves. The AICA and the VA were carefully mobilized without damage to the perforating vessels. A No. 6-0 Prolene suture was passed through the dura dorsal to the ninth and 10th cranial nerves and then brought around the VA and the AICA. As it was tied the VA moved to its new position. There was no stretching of the perforating vessels. A piece of shielded Teflon felt was placed between this suture and the VA to make sure that there was no kinking of the VA and no direct contact between the suture and the VA wall. Some Teflon felt was also placed between the AICA and the VA and the REZ of the seventh cranial nerve (Fig. 6). A transient delay in the brainstem auditory evoked responses was noticed but resolved with the removal of the retractor. The wound was closed in the usual fashion.

Postoperative Course. Postoperatively, the patient’s dizziness, vertigo, nausea, vomiting, and dysgeusia completely disappeared. She has been totally asymptomatic throughout a follow-up period of 5 months. Postoperative MR imaging showed decompression of the REZ of the eighth cranial nerve (Fig. 7).

Discussion

In most patients with vascular compression of the cranial nerves, decompression can be performed by using Teflon felt padding in the case of offending arteries or by coagulation and division in the case of veins. However, when large arteries such as the VA or BA are involved, Teflon padding may not achieve adequate decompression. In these cases, repositioning of the artery or nerve may be necessary. In a patient with trigeminal neuralgia that was caused by an ectatic BA severely compressing the fifth cranial nerve, we repositioned the trigeminal root by opening Meckel’s cave widely via a small presigmoid petrosal approach. A good result was obtained in that patient (unpublished data). A number of authors have proposed alternative means of decompressing the cranial nerves, including using a hard implant to isolate the cranial nerves from the vessel, repositioning the vertebrobasilar complex, and even sectioning the VA.

Takamiya, et al., used a fenestrated aneurysm clip to mobilize the trigeminal nerve away from a hard and immobile BA that could not be decompressed in the usual manner. Laws and colleagues reported nine patients in whom a vascular clip graft (Sundt clip) was placed around the sensory root of the trigeminal nerve to isolate it from the offending vessel. Yoshimoto, et al., encircled the trigeminal nerve with a silastic ring to decompress it.

Hongo and associates reported a case of compression of the medulla oblongata by a tortuous VA. Initial decompression using a sponge failed to provide long-term success. The VA was subsequently sectioned to decompress the brainstem.

Rawlinson and Coakham used a thin silastic rubber sling stitched to the dura with No. 4-0 Prolene in two patients with hemifacial spasm. In one patient the causative vessel was too large to allow effective decompression of the brainstem, and in the other patient, the interposition of Ivalon sponge placed the brainstem perforating vessels at risk. Ogawa and colleagues reported the use of a sling made from a synthetic vascular graft to move the verteobasilar complex away from the trigeminal, facial, and vestibulocochlear nerves. The sling was then sutured to the clival dura. Stone, et al., used a small sling made from fine silicone tape to reposition the BA away from the trigeminal nerve. The sling was sewn to the adjacent petrous dura.

Fukushima also reported the use of Teflon slings to mobilize the offending vessel. These loops were affixed to the dura using fibrin glue and pieces of Surgicel to promote fibrous adhesions of the Teflon tape to the dura. However, these slings were not strong enough to hold large vessels such as the VA and BA.

We report the use of a Prolene suture as a sling to transpose the offending vessel. The suture was combined with Teflon felt to avoid kinking of the artery. This is a simple technique, needing only one properly positioned suture. The patency of the artery and perforating vessels was not compromised and the clinical results were gratifying. This technique can be very helpful to the neurosurgeon who treats patients with neurovascular compression that is caused by large ectatic arteries—patients in whom the traditional method of interposing a soft prosthesis may prove to be less than optimum.
References


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