MR Angiography in Diagnosing Vascular Compression of Brainstem Structures as the Etiology of Certain Disease States

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Purpose
Vascular compression of cranial nerves is a known cause of several syndromes including trigeminal neuralgia, hemifacial spasm, facial pain, and glossopharyngeal neuralgia. Recently much data has shown that vascular compression of the rostral ventrolateral medulla near the root entry zones of cranial nerves 9 and 10, or the retro-olivary sulcus, is the cause of reversible or neurogenic hypertension. Surgical microvascular compression is a proved treatment in cases that are refractory to noninvasive therapies. The results of most previous efforts to demonstrate that MR imaging is a sound diagnostic test for detecting vascular compression as the cause of these syndromes and disease states have been equivocal. It has been suggested that three dimensional time-of-flight (3D TOF) MR angiography (MRA), might improve diagnostic power of MR imaging in cases of compression of the ventrolateral medulla as the cause of hypertension. One study showed 3D TOF MRA to be relatively successful in diagnosing compression of cranial nerve V preoperatively in patients with refractory trigeminal neuralgia. Thus, the purpose of this retrospective study was to validate the role of MR imaging, and specifically MRA, as a competent tool in diagnosing neurovascular compression. Another goal was to demonstrate that MRA will correctly identify patients who will benefit from surgical microvascular decompression. Also, we attempted to clarify if position and degree of the vascular compression are needed preoperatively.

Materials & Methods
3D TOF MRA using a 1.5 T superconducting magnet, quadrature head coil, and spoiled gradient echo pulse sequence was obtained preoperatively on 33 patients with the clinical diagnoses of trigeminal neuralgia, hemifacial spasm, facial pain, otalgia, glossopharyngeal neuralgia, hypertension or combinations thereof. Contrast enhancement with gadolinium was used in 29 cases. Acquisition and resolution parameters were as follows; TR = 23 ms, TE = 4.4 ms, flip angle = 20 degrees, 256 x 192 matrix, FOV = 16 cm, slice thickness = 1mm, NEX = 2. These patients had been referred for microvascular decompression of the cranial nerve(s) and/or brainstem structures corresponding to their clinical diagnoses via suboccipital craniectomy. One investigator (WHB), blinded to the preoperative diagnoses and operative results, reviewed the axial source images of the MRA through the brainstem to detect vascular compression. Images were classified for right and/or left-sided compression or no compression. Contrast enhancement with gadolinium was used in 29 cases. Acquisition and resolution parameters were as follows; TR = 23 ms, TE = 4.4 ms, flip angle = 20 degrees, 256 x 192 matrix, FOV = 16 cm, slice thickness = 1mm, NEX = 2. These patients had been referred for microvascular decompression of the cranial nerve(s) and/or brainstem structures corresponding to their clinical diagnoses via suboccipital craniectomy. One investigator (WHB), blinded to the preoperative diagnoses and operative results, reviewed the axial source images of the MRA through the brainstem to detect vascular compression. Images were classified for right and/or left-sided compression or no compression. Another investigator (SJP), blinded to the MRA results, reviewed the operative notes for the presence or absence of neurovascular compression. When present, the identity and position of the offending vessel(s) were noted. A positive finding was defined as vascular signal enhancement in contact with the
cisternal or root entry zone portions of cranial nerves V-IX or left retro-olivary sulcus on the side ipsilateral to that observed during surgery.

**Results**

MRA showed vascular compression concordant with the side and neural structure observed intraoperatively in 28 of 31 patients (sensitivity = 90%, positive predictive value = 97%). For 4 of these 28 patients, MRA showed bilateral neurovascular compression. One patient had compression via MRA but none seen intraoperatively, and one patient had no compression on MRA or observed at surgery (specificity 50%). Three patients had venous compression only one of which was detected by MRA. Three patients had vascular compression of multiple structures observed intraoperatively of which two were detected correctly by MRA. The superior cerebellar artery was the most frequent compressing vessel (22 cases). In the one case where MRA results were falsely positive, bilateral compression was indicated by MRA.

**Conclusion**

Given the quantitative results of our data, 3D TOF MRA using these or similar imaging parameters will likely prove to be the diagnostic test that identifies patients with neurovascular compression who will benefit from decompression surgery. The diagnostic utility from our results are equal or superior to any study to date assessing the ability of MR imaging to detect neurovascular compression of ponto-medullary structures. The very small number of false negative results between MR imaging and surgery limits the measurement of specificity. We believe that vessel identification, position, and degree of compression are not essential in preoperative imaging diagnosis since we found that patients were sometimes symptomatic when the offending vessel was touching but not mechanically compressing the neural structures. More cases in which venous compression proves at surgery to be the cause of symptoms are needed before a consensus can be reached on the need for contrast enhancement. Vessels compressing cranial nerves often lie parallel to the nerve and in plane with an axial FOV resulting in vascular signal reduction in TOF MRA. Acquiring images coronally might improve diagnosis of neurovascular compression in this orientation.

**References**

2. Janetta, PJ. **Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia.** J Neurosurg 1967;26:159–162
5. Patel SJ. Vascular compression of the rostral ventrolateral medulla in sympathetically mediated hypertension. submitted to Hypertension