

Magnetic Resonance Imaging Assessment of Vascular Contact of the Facial Nerve in the Asymptomatic Patient

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Abstract

Objective The objective of this study was to determine the prevalence of facial nerve vascular contact on magnetic resonance imaging (MRI) in patients without hemifacial spasm (HFS).

Study Design Our radiology database was queried to identify consecutive adult patients without a history of HFS, intracranial tumor, brain radiation therapy, intracranial surgery, traumatic brain injury, or trigeminal nerve vascular compression. One hundred high-resolution MRIs of the posterior fossa were independently reviewed by two neuroradiologists for facial nerve vascular contact (200 sides).

Main Outcome Measures The prevalence of vascular nerve contact in the non-HFS patient, the location of contact along the facial nerve, the culprit vessel, and severity of compression was recorded.

Results The presence of vascular contact in the non-HFS patient may be as high as 53%. It is typically mild to moderate in severity, most commonly involves the cisternal portion, and usually caused by the anterior inferior cerebellar artery.

Conclusion Vascular contact of the facial nerve is frequently identified in asymptomatic individuals but tends to be more peripheral and mild compared with previous descriptions of neurovascular contact in HFS patients. These results should be considered in assessing the candidacy of HFS patients for microvascular decompression.

Keywords

- ▶ neurovascular compression
- ▶ microvascular decompression
- ▶ hemifacial spasm
- ▶ asymptomatic
- ▶ MRI
- ▶ facial nerve

Introduction

Vascular contact of the facial nerve is widely believed to be the most common cause of hemifacial spasm (HFS). HFS affects 1 in 100,000 people per year and manifests with unilateral spasms of the facial musculature that can lead to significant reduction in quality of life.^{1,2} Microvascular decompression has been established as an effective and potentially curable treatment for HFS, with success rates exceeding 90% for the initial operation.^{3–5}

Cranial nerve compression is most vulnerable at the nerve's root entry/exit zone (REZ) where the central glial myelin transitions into the peripheral myelin created by Schwann cells.³ Therefore, microvascular decompression targets this transition zone (TZ). The facial nerve is unique in that the segment proximal to the TZ is also an area of vulnerability. When the facial nerve emerges from the brain stem within the pontomedullary sulcus at a point called the root exit point (REXP), it courses along the undersurface of the pons for 8 to

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10 mm. This segment is called the attached segment (AS). The facial nerve then separates from the pons at the root detachment point (RDP). The TZ extends 2 to 4 mm from the RDP before extending anterolaterally to the porus acusticus in a segment known as the cisternal portion (CP).^{6,7} Vascular contact in this area from the RExP to the end of the TZ has been found to be associated with HFS and inadequate decompression of this proximal segment has been associated with persistent symptoms following surgery.^{6,8}

Although investigators have studied the accuracy of different types of magnetic resonance imaging (MRI) sequences for identifying vascular loop compression preoperatively as compared with the gold standard of intraoperative findings during microvascular decompression for HFS, there is much less data to address the frequency with which a vessel is contacting or compressing the facial nerve as an incidental finding in the absence of a history of HFS.⁹⁻¹⁵ The purpose of this study was to determine the prevalence of facial nerve vascular contact on MRI in patients without HFS.

Methods

This retrospective study was approved by the Institutional Review Board at Mayo Clinic, and the need for informed consent was waived. Our radiology database was queried to identify adult (age ≥ 18 years) patients who underwent MRI that included high-resolution volumetric T2 and T1 postcontrast sequences of the posterior fossa. Patients with a history of HFS, intracranial tumor, brain radiation therapy, intracranial surgery, imaging evidence of traumatic brain injury, trigeminal nerve vascular compression, or poor image quality were specifically excluded. As such, the predominant indications for the imaging were audio-vestibular symptoms and headache. The MRI scans of 100 consecutive patients (81 at 1.5 T and 19 at 3.0 T) who met eligibility criteria were independently reviewed by two board-certified neuroradiologists with 29 and 18 years of experience, respectively, for facial nerve vascular contact (200 sides). The volumetric high-resolution MRI sequences of the posterior fossa were acquired with one of two scanner platforms as follows:

1. 3.0 T MAGNETOM Skyra (Siemens Healthcare, Erlangen, Germany)
 - a. T2-weighted spatial and chemical-shift encoded excitation (repetition time 1,020 milliseconds, echo time 209 milliseconds, flip angle 120 degrees, field of view 15 cm, matrix size 320 \times 320, 1 mm axial slice thickness with 1 mm coronal and sagittal reformatted images)
 - b. Contrast-enhanced T1-weighted magnetization-prepared rapid acquisition gradient echo (repetition time 1,900 milliseconds, echo time 2.6 milliseconds, inversion time 900 milliseconds, flip angle 9 degrees, field of view 18 cm, matrix size 192 \times 192, 1 mm axial slice thickness with 1 mm coronal and sagittal reformatted images)
2. 1.5 T Signa HDx (GE Healthcare, Waukesha, Wisconsin, United States)

- a. T2-weighted fast imaging employing steady-state acquisition (repetition time 9.3 milliseconds, echo time 2.6 milliseconds, flip angle 65 degrees, field of view 14 cm, matrix size 448 \times 256, 1 mm axial slice thickness with 1 mm coronal and sagittal reformatted images)
- b. Contrast-enhanced T1-weighted spoiled gradient recalled (repetition time 38 milliseconds, echo time 13 milliseconds, flip angle 45 degrees, field of view 18 cm, matrix size 256 \times 192, 1 mm axial slice thickness with 1 mm coronal and sagittal reformatted images).

Intravenous contrast was administered as a standard dose of 0.1 mmol/kg gadobutrol (Gadavist, Bayer HealthCare LLC, Whippany, New Jersey, United States).

The reviewing neuroradiologists were aware of the study objectives for this non-HFS cohort of patients. They were asked to review all of the MRI pulse sequences and multiplanar reconstructions and then synthesize the imaging features with their knowledge and experience to make a final determination as to the presence and nature of facial nerve vascular contact. When vascular contact was identified, the facial nerve segment contacted was recorded and, if identifiable, the vessel responsible for the contact was documented. Because no intraoperative gold standard exists for these subjects, the neuroradiologists were instructed to only specify a culprit vessel when they had a high level of certainty and to otherwise indicate "uncertain" in lieu of making an educated guess. The location along the facial nerve where vascular contact was identified was classified as "brain stem," "REZ," or "CP" based on anatomic landmarks that are reliably visualized with MRI. **►Fig. 1** demonstrates how these areas correlate to the anatomic terms initially proposed by Tomii et al in 2003 and expanded upon by Campos-Benitez and Kaufmann.⁶ Compression at the "brain stem" is defined as anywhere in the region from the RExP at the pontomedullary sulcus to the RDP. Therefore, this includes the segment known as the AS where the facial nerve adheres to the undersurface of the pons for 8 to 10 mm. These brain stem regions were treated in aggregate as vascular loops often have a broad point of contact making it difficult to delineate compression of the RExP from the adjacent AS. We define the REZ as the segment from the RDP to 4 mm distal to include the entire length of the TZ where the central glial myelin transitions to the peripheral myelin created by Schwann cells. Finally, the CP is the segment distal to the TZ to the porus acusticus. Vascular contact was graded using the scale proposed by Campos-Benitez and Kaufmann which defined mild as "contact without indentation of the nerve," moderate as "indentation of the nerve without deviation of its course," and severe as "deviation of the natural course of the facial nerve."⁶

Descriptive statistics were used to describe the patient demographics. Interobserver agreement for the presence or absence of nerve contact was calculated using the kappa coefficient of agreement index, which establishes the extent of agreement compared with chance. Kappa coefficient was interpreted according to the guidelines proposed by Fleiss et al and Landis and Koch.^{16,17} Kappa values of less than 0.40 indicate poor reliability, 0.40 to 0.75 indicate fair to good

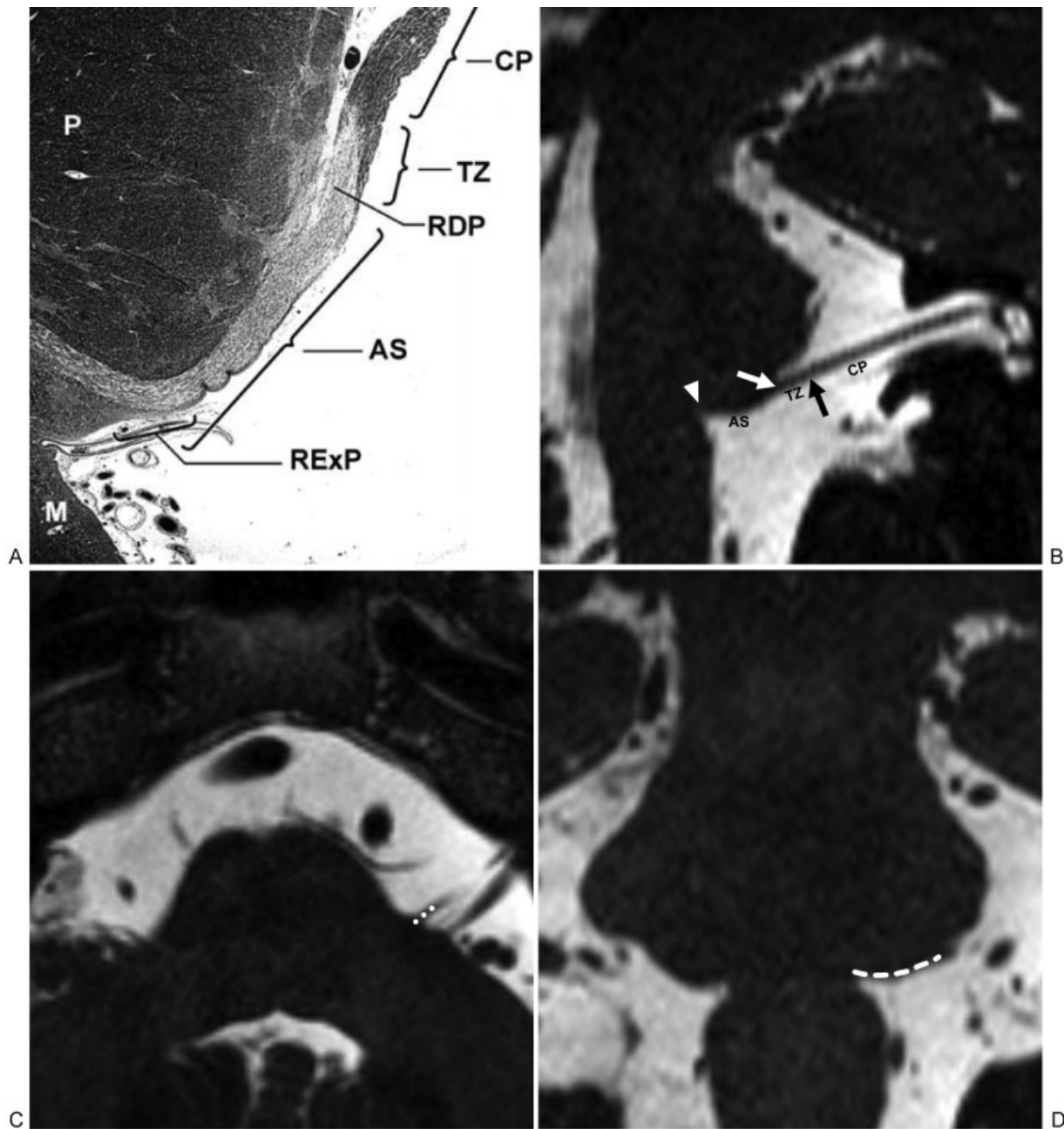


Fig. 1 (A) Photomicrograph of the facial nerve as it leaves the brain stem and the anatomic terms used by Campos-Benitez and Kaufmann to describe its segments. (Reprinted with permission from Campos-Benitez and Kaufmann.⁶) (B) For illustration purposes only (i.e., not performed as part of the study), an oblique coronal high-resolution T2-weighted MRI sequence was reconstructed parallel to the facial nerve to demonstrate the corresponding anatomy on a single image. RExP (white arrowhead), RDP (white arrow), and estimated lateral border of TZ (black arrow). (C) Axial high-resolution T2-weighted MRI illustrates the “REZ” (dotted white line) which corresponds to RDP and TZ as seen in the photomicrograph by Campos-Benitez and Kaufmann. (D) Coronal high-resolution T2-weighted MRI demonstrates the “brain stem” segment (dashed white line) which corresponds to the RExP and AS as defined in the photomicrograph by Campos-Benitez and Kaufmann. AS, attached segment; CP, cisternal portion; M, medulla; MRI, magnetic resonance imaging; P, pons; RExP, root exit point; REZ, Root Entry/Exit Zone; RDP, root detachment point; TZ, transition zone

reliability, and 0.75 to 1.00 indicate excellent reliability. The *p*-values < 0.05 were considered statistically significant.

Results

The cohort had a mean age of 56 years (range, 22–89 years) and included 38 men and 62 women. The results of the assessment for neurovascular compression of the facial nerve

in these patients are reported in ► **Table 1**. The first neuroradiologist identified vascular contact in 88 of 200 facial nerves (44%), with the CP being most commonly affected (48.9%, ► **Fig. 2**), followed by the REZ (31.8%, ► **Fig 3**), and lastly the brainstem (19.3%, ► **Fig. 4**). Vascular contact was most commonly caused by the anterior inferior cerebellar artery (AICA) which occurred in 59.1%. The severity of vascular contact was mild in most cases (84.1%). The second neuroradiologist

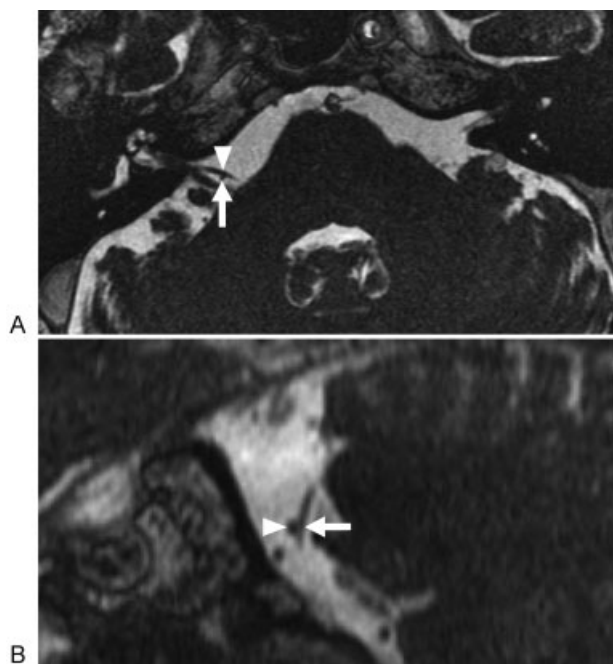


Fig. 2 (A) Axial and (B) sagittal high-resolution T2-weighted images illustrate indentation (moderate neurovascular contact) of the cisternal portion of the right facial nerve (white arrowhead) by the anterior inferior cerebellar artery (white arrow).

identified vascular contact in 75 of 200 facial nerves (37.5%) with the CP being the most commonly affected segment (54.7%). Vascular contact was most commonly caused by the AICA, which occurred in 52.0%. The severity of vascular contact was mild in 73.3%. No cases of severe neurovascular

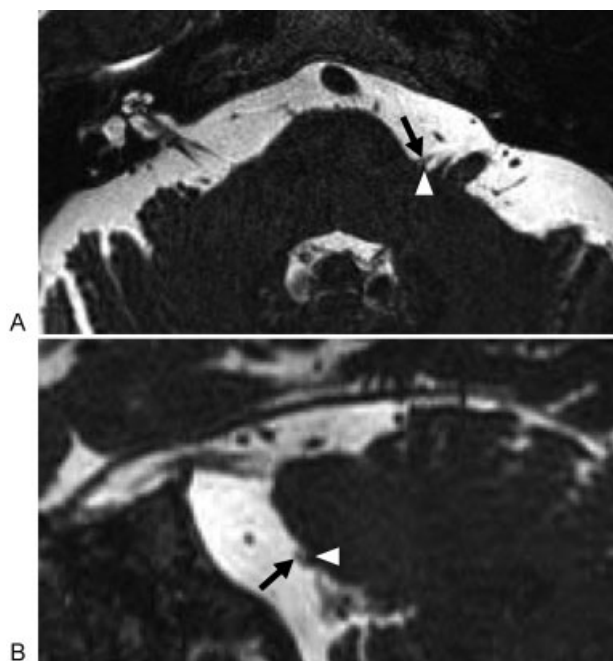


Fig. 3 (A) Axial and (B) sagittal high-resolution T2-weighted images depict mild neurovascular contact between the left facial nerve (black arrow) and a vein (white arrowhead) at the level of the root entry/exit zone.

compression were identified by either neuroradiologist. In 94 of 200 nerves (47%), both neuroradiologists agreed that there was no vascular contact. Therefore, the presence of vascular contact in the asymptomatic patient may be as high as 53%. **Table 2** details the interobserver agreement between the two neuroradiologists. The kappa coefficient of agreement for the presence/absence of vascular contact was 0.50 (95% confidence interval = 0.37–0.62), indicating fair to good agreement between the two observers. All differences in opinion between the two neuroradiologists varied by only one grade of severity (i.e., no contact vs. mild or mild vs. moderate; **Fig. 5**).

Discussion

Although vascular contact of the facial nerve is widely believed to be a cause of HFS, the prevalence of facial nerve vascular contact on MRI in patients without HFS has not been well described in the literature. A few prior studies have examined vascular contact on the contralateral, asymptomatic side of patients with HFS and determined that there is about a 15% rate of vascular contact on the asymptomatic side.^{10,11,18} In another study, Tash et al studied the MRI findings of facial nerve vascular contact in the REZ in 13 patients with HFS and 70 patients without HFS.¹⁹ They found the presence of a vascular contact in all 13 patients with HFS and in 21% of the asymptomatic patients.¹⁹ However, this study was published in 1991 using early generation MRI technology such that the imaging performed on the control group only included 3 mm coronal T1-weighted images. Consequently, these results likely underestimated the prevalence of facial nerve vascular contact.¹⁹ Recently, Sekula et al evaluated the presence of vascular compression of the centrally myelinated portion of the facial nerve in a cohort of HFS patients.¹³ They included a brief discussion of their control group of 28 asymptomatic patients in which there was imaging evidence of neurovascular compression of this vulnerable zone by an artery or vein in three and one control subjects, respectively.¹³ Kakizawa et al performed an anatomical study of the facial nerve in 110 asymptomatic individuals using 3 T MRI and described a high rate of contact (78.6%) between the facial nerve and other structures (arteries, veins, dura mater, and other cranial nerves).²⁰ However, their contact points were recorded based on distance from the TZ, as opposed to stratifying based on anatomic landmarks. In addition, their high frequency of neurovascular contact is at least partially secondary to inclusion of the meatal segment, which has a high rate of contact with the AICA.

Our study is unique in that we used contemporary MRI technology to image a larger cohort of non-HFS patients and assessed neurovascular compression severity for multiple anatomic segments of the facial nerve. We demonstrate that facial nerve vascular contact may be identified on MRI in approximately 37 to 53% of patients who are asymptomatic. The findings of vascular contact almost always go unnoticed or unreported when the indications for the study do not alert the radiologist to specifically evaluate for facial nerve

Table 1 Neurovascular compression of the facial nerve

		MRI findings in asymptomatic patients			Surgically confirmed findings in hemifacial spasm patients
		Neuroradiologist 1	Neuroradiologist 2	Mean	Campos-Benitez and Kaufmann (2008)
Presence of vascular contact	No	112 (56%)	125 (62.5%)	59.3%	0%
	Yes	88 (44%)	75 (37.5%)	40.8%	100%
Location	Brain stem (REXP + AS)	17 (19.3%)	5 (6.7%)	13.0%	74%
	Root entry/exit zone (RDP + TZ)	28 (31.8%)	29 (38.7%)	35.3%	22%
	Cisternal portion	43 (48.9%)	41 (54.7%)	51.8%	3%
Vessel	AICA	52 (59.1%)	39 (52.0%)	55.6%	43%
	PICA	7 (8.0%)	8 (10.7%)	9.6%	31%
	Vertebral artery	0 (0.0%)	0 (0.0%)	0.0%	23%
	Vein	14 (15.9%)	20 (26.7%)	21.3%	3%
	Vessel of uncertain origin	15 (17.0%)	8 (10.7%)	13.9%	0%
Severity	Mild	74 (84.1%)	55 (73.3%)	78.7%	27%
	Moderate	14 (15.9%)	20 (26.7%)	21.3%	61%
	Severe	0 (0.0%)	0 (0.0%)	0.0%	12%

Abbreviations: AICA, anterior inferior cerebellar artery; AS, attached segment; PICA, posterior inferior cerebellar artery; RDP, root detachment point; RExp, root exit point; TZ, transition zone.

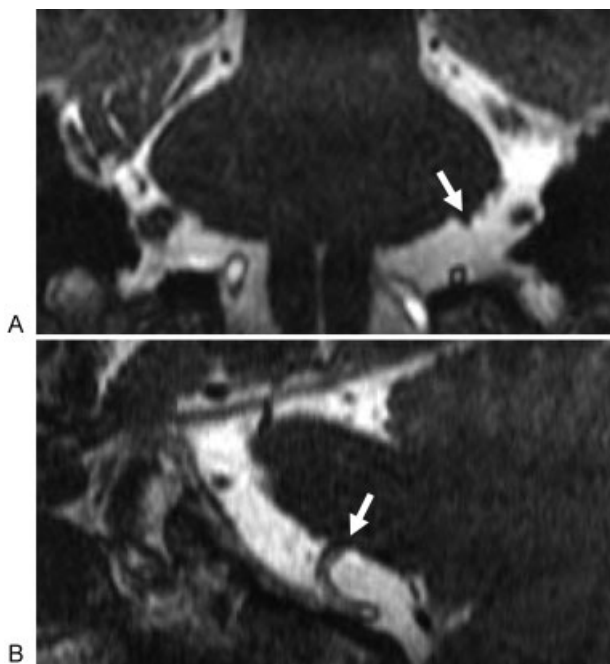


Fig. 4 (A) Coronal and (B) sagittal high-resolution T2-weighted images demonstrate a loop of the anterior inferior cerebellar artery (white arrow) indenting the attached segment of the facial nerve along the undersurface of the left pons (moderate neurovascular contact).

vascular compression. The results of this study may be helpful to a surgeon who is evaluating a patient preoperatively for HFS better ascribe significance to the MRI findings, particularly in mild or equivocal cases of vascular contact.

In an important article by Campos-Benitez and Kaufmann, patients with HFS undergoing microvascular decompression were analyzed according to the location of neurovascular compression, the primary culprit vessel, and severity.⁶ For reference, **Table 1** juxtaposes their results in patients with HFS with the results from our study of neurovascular compression in patients without HFS.⁶ Although the cross-study comparison of MRI findings in asymptomatic patients with intraoperative observations in HFS patients has some limitations, several important conclusions can nevertheless be drawn when comparing these results.

First, mild compression was more prevalent in our asymptomatic group, whereas moderate or severe compression was more common in patients with symptoms of HFS. The prevalence of asymptomatic contact or compression of other cranial nerves such as the trigeminal nerve is known to be high.²¹⁻²⁴ Therefore, it follows logically that mild contact, which is defined in this study as “contact without indentation of the nerve,” would be less likely to cause symptoms.

Second, when vascular compression is seen in an asymptomatic patient, it is more likely to occur in the distal segments, such as in the CP. This is in contrast to the patients with HFS who more commonly demonstrate compression in the proximal segments, from the RExp at the pontomedullary sulcus

Table 2 Interobserver agreement

	Neuroradiologist 2			Total
		N	Y	
Neuroradiologist 1	N	94 (47.0%)	18 (9.0%)	112 (56.0%)
	Y	31 (15.5%)	57 (28.5%)	88 (44.0%)
	Total	126 (62.5%)	74 (37.5%)	200 (100%)
Simple kappa coefficient	0.50 (95% CI = 0.37–0.62)			

Abbreviation: CI, confidence interval.

medially to the end of the TZ, which is roughly 3.5 to 4 mm distal to the RDP.^{6,8} Recent research on patients with a history of failed prior microvascular decompression for HFS have identified persistent neurovascular contact along the more proximal portion of the facial nerve in most patients.^{8,25} Their findings highlight the importance of addressing the proximal vascular compression of the facial nerve where compression of the nerve is more likely to cause symptoms as compared with the more resistant, peripherally myelinated segment of the nerve located more distally. It is not definitively understood why compression of segments covered with central myelin is more likely to cause symptoms when compared with peripheral myelin. It is hypothesized that the central nervous system segment of cranial nerves is structurally weaker and more vulnerable to microtrauma than the peripheral portion.²⁶ The hypothesis is supported by histologic studies on the trigeminal, facial, glossopharyngeal, and vagus nerves which demonstrate that the longer the length of the central myelin portion of the nerve, the greater the incidence of the corresponding disease (trigeminal neuralgia, HFS, and vagoglossopharyngeal neuralgia).^{26,27} Overall, these studies conclude that compression of the proximal, centrally myelinated segment is more likely to manifest with symptoms as compared with compression of the distal, peripherally myelinated segments. That having been said, our study still identified a not insignificant number of non-HFS patients with proximal neurovascular contact.

Third, in patients with HFS, the vertebral artery is the third most common culprit vessel (after the AICA and posterior inferior cerebellar artery [PICA]) to compress the facial nerve.⁶

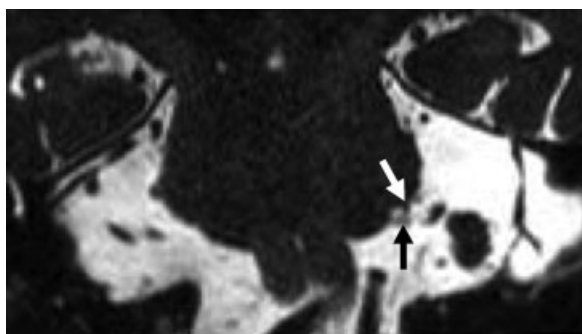


Fig. 5 High-resolution coronal T2-weighted image demonstrates an example of interobserver variability. One observer felt that mild contact was present between the left facial nerve (black arrow) and the anterior inferior cerebellar artery (white arrow), while the second neuroradiologist thought that a small cleft of cerebrospinal fluid was visible separating the two structures.

In our study on asymptomatic patients, however, the vertebral artery was not readily identified as an offending vessel. Instead, the AICA was implicated in over half of the patients found to have vascular contact. This asymptomatic contact can be explained by understanding the anatomy of the AICA, which makes a normal “hairpin” loop within the internal auditory canal in the cerebellopontine cistern, at which point it may come into contact with the resistant, peripherally myelinated segment of the facial nerve at that location.²⁵ When the vertebral artery, on the other hand, makes contact with the facial nerve, it typically occurs more proximally, along the more vulnerable centrally myelinated facial nerve fibers, and therefore is more likely to cause symptoms of HFS.⁶

Knowledge of the location of the vulnerable segment of the facial nerve as well as the prevalence of contact in the asymptomatic patient is crucial for preoperative planning, particularly for the surgeon confronted with the patient who has a history of a failed microvascular decompression. Surgical candidacy depends on the complete clinical picture, including history, physical, electromyography results, and MRI findings. Patients presenting with symptoms of HFS and evidence on MRI of distally based (CP) vascular compression should also be scrutinized carefully for proximal contact (within the pontomedullary sulcus and the AS), since the prevalence of asymptomatic vascular contact may be as high as 53% and contact occurring in the distal, peripherally myelinated portion of the nerve is more likely to be asymptomatic. Current research supports the notion that the entire portion of the centrally myelinated facial nerve should be decompressed for best surgical outcome.^{6,8,25,28,29}

The primary limitation of this study is the lack of surgical confirmation of neurovascular contact as a gold standard, which is obviously not attainable in a cohort without HFS. Interobserver agreement fell into the “fair to good” range, and this understandably stems from the inherently subjective assessment of intricate neurovascular structures. When discrepant, the assessment of neurovascular contact severity in our study only differed by one grade. Previous reports evaluating preoperative MRI in patients undergoing microvascular decompression for HFS have shown varying degrees of interobserver variability as well.^{9,12,13} However, it is important to make note that comparing studies is limited by the variability in the patient group being studied, MRI pulse sequences used, type and field strength of the MRI scanner, as well as the subjective nature of neurovascular compression rating by the radiologist or surgeon. Nevertheless, when reconciling the disagreement between neuroradiologists, the

prevalence of facial nerve vascular contact in the non-HFS patient may be best estimated as a range of 37 to 53% in the current study. In addition, because the study subjects were undergoing clinically indicated MRI scans, they cannot be considered as truly asymptomatic, but criteria were set in a manner to limit the impact of potentially confounding variables on posterior fossa anatomy.

Conclusion

Vascular contact of the facial nerve is frequently identified in the non-HFS patient (as high as 53%), typically mild to moderate in severity, most commonly involves the CP, and usually caused by the AICA. This is in contrast with studies done on patients with HFS, who more commonly demonstrate moderate to severe vascular compression which is located proximal to the CP of the facial nerve, and is frequently caused by the vertebral artery, AICA, and/or PICA. These results should be considered in assessing the candidacy of HFS patients for microvascular decompression.

Conflict of Interest

None.

Acknowledgments

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