

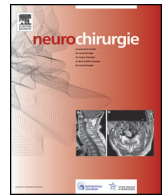


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Original article

## Imaging of hemifacial spasm

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### ABSTRACT

Almost all primary hemifacial spasms are associated with one or more neurovascular conflicts, most often at the root exit zone in the immediate vicinity of the brainstem. Imaging has first to exclude a secondary hemifacial spasm and secondly to search for and characterize the responsible neurovascular conflict(s). Magnetic resonance imaging should include high-resolution anatomical hyper T2-weighted sequences and magnetic resonance angiography by using 1.5 or even better 3 Tesla magnets. The most frequent vascular compressions are from the anterior-inferior cerebellar artery, the posterior-inferior cerebellar artery and the vertebralbasilar artery; venous conflicts are very rare. Conflicts are often multiple; also, the same vessel may compress the facial nerve in two places. Also, conflicts may be aided by particular anatomical circumstances, including arterial dolichoectasia, posterior fossa with a small volume or bony malformations.

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### 1. Introduction

The vast majority of organic hemifacial spasms (HFS) are primitive and associated with one or more neurovascular conflicts (NVC) [1–3]. Microvascular decompression (MVD) is highly effective in HFS and symptoms disappear after surgery in 90–95% of cases [4,5]. Magnetic resonance imaging (MRI) has become an essential tool [6–9] with the aim of excluding a spasm of secondary etiology and to search for NVC and characterize it. In addition, MRI may be useful when surgery has not provided symptom relief, mainly to search for missed NVC.

#### 1.1. Magnetic resonance imaging technique

##### 1.1.1. Field strength

MRI should be performed when possible using a 3T magnet [10]. In fact, signal-to-noise and contrast-to-noise ratios are better at 3T compared to lower field strengths, as well as anatomic conspicuity, including delineation of cranial nerves (CNs) and nerve branches and also assessment of small vessels. In some patients, NVCs that were not identifiable with certainty at 1.5T revealed a better diagnosis at 3T [10,11].

##### 1.1.2. Sequences

A combination of high-resolution 3D T2-weighted imaging with 3D time-of-flight angiography and 3D T1-weighted gadolinium-enhanced sequences is considered the standard of reference for the detection of the neurovascular compression [12]. This type of combination can successfully guide neurosurgical treatment and may help to predict treatment response.

A variety of high-resolution 3D T2-weighted sequences is currently available, depending on the manufacturer, including constructive interference in steady state (CISS), steady-state free precession (SSFP), T2WI-driven equilibrium radiofrequency reset pulse (DRIVE), three-dimensional fast imaging employing steady-state acquisition (FIESTA) and sampling perfection with application-optimized contrasts by using different flip angle evolutions (SPACE). 3D T2-weighted sequences provide “cisternographic” images of cranial nerves and surrounding vessels in their parts surrounded by cerebrospinal fluid, with high spatial resolution. Slice thickness should be adapted to the small caliber of the facial nerve and vessels, typically 0.3 to 0.4 mm. Multiplanar oblique reconstructions should be obtained, especially in the transverse and coronal planes following the course of the facial nerve. Coronal MR views are of special value to assess the proximal part of facial nerve, where most NVCs occur [13].

High-resolution 3D time-of-flight (TOF) magnetic resonance angiography (MRA) covering the posterior fossa is obligatory to assess vascular conflicts. We advocate performing this acquisition without any contrast injection and a presaturation band positioned up to the MRA field of view in order to depict only the arterial

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vessels. It should be taken into account that due to the TOF technique, the vessel signal may decrease in vessels with a cranio-caudal trajectory, especially at intermediate field strength.

Thin-section gadolinium-enhanced T1-weighted images are helpful in differentiating small veins from arteries, by comparison to unenhanced 3D TOF arterial MRA, which only shows arterial vessels [12]. Post-contrast T1 sequences are useful to screen for and characterize cerebellopontine angle masses that may cause HFS.

Image fusion of 3D T2-weighted sequences with corresponding TOF images or 3D T1-weighted gadolinium-enhanced images can be useful in a preoperative context [9,14].

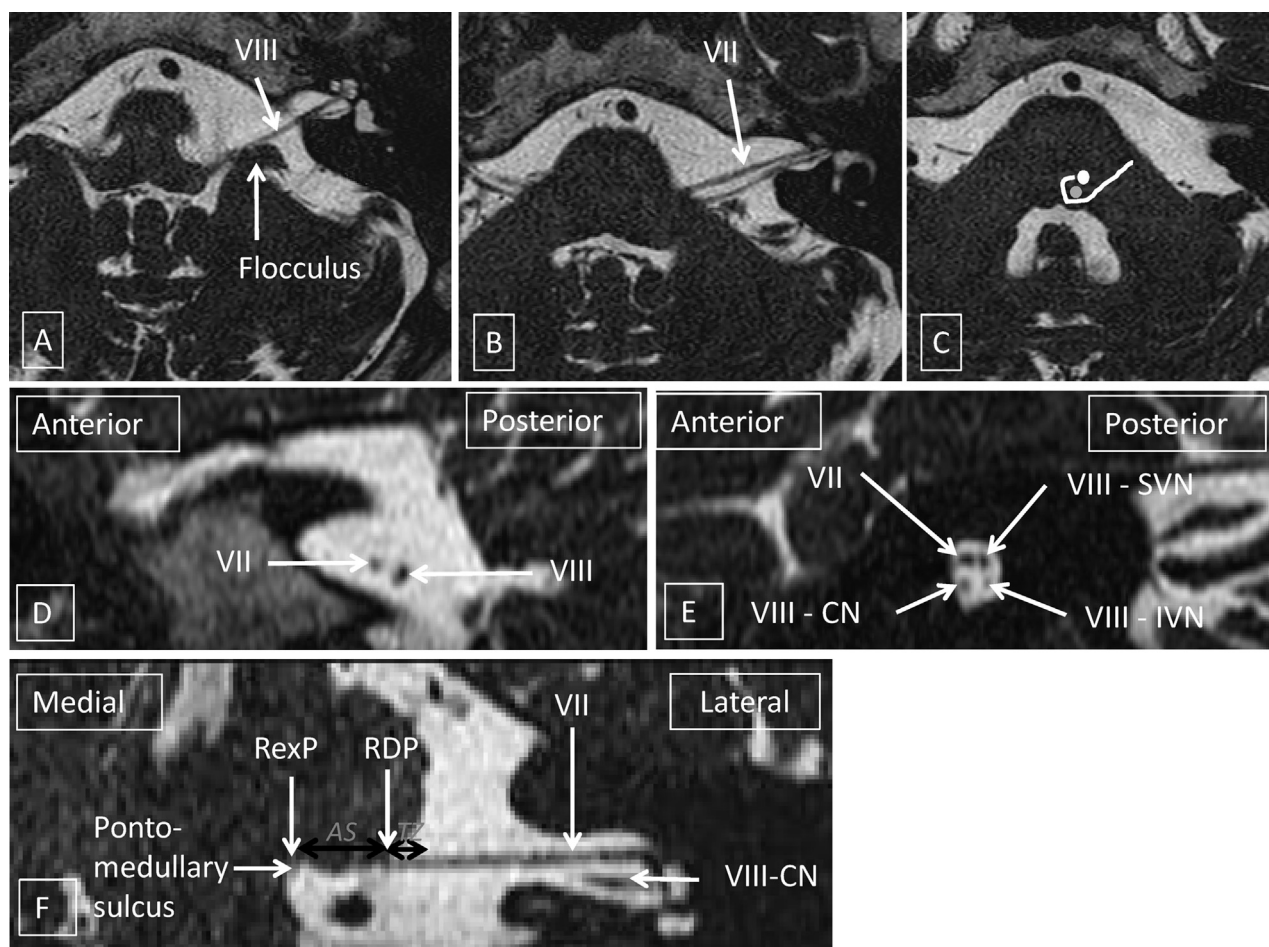
Other sequences may provide useful information. A T2-weighted sequence covering the brain should be performed to rule out intra-axial lesions as the source of HFS. In cases of neurovascular compression, there is usually no signal abnormality at T2-weighted imaging on the intra-axial course or facial nuclei. It is quite uncommon to observe nerve atrophy even in cases with severe, long-standing, compression.

Diffusion-weighted imaging is useful for the differential diagnosis of cystic lesions that may compress the facial nerve. Epidermoid cysts appear as bright signal expansive lesions on diffusion-weighted images whereas arachnoid cysts do not. Diffusion tensor imaging and tractography have been advocated for the exploration of trigeminal neuralgia, but are still of limited value in HFS resulting

from neurovascular compression (NVC), due to the small diameter of the facial nerve and limited spatial resolution of these sequences in clinical routine. Tractography may however be an adjunct to anatomical sequences to assess the course of cranial nerves when displaced by large cerebellopontine angle tumors [15,16].

### 1.2. Normal MRI findings

MRI provides high-resolution anatomic details of the facial nerve outside the brainstem (Fig. 1). Imaging data should always be interpreted taking into account clinical symptoms, since neurovascular relationships can be observed in asymptomatic subjects. Most asymptomatic neurovascular contacts are not associated with nerve indentation; however a significant displacement of the course of facial nerve by dolichoectatic vertebral arteries at some distance from the root exit zone (REZ) is sometimes observed in asymptomatic subjects. A study of 100 MRIs in subjects without HFS in search of neurovascular contact by two independent observers was recently reported. Contact between the facial nerve and neighboring vessels was retained by the two observers in 37% and 53% of the subjects respectively [17]. These contacts tended to be more peripheral, sparing the REZ, and more moderate than in patients with HFS.



**Fig. 1.** Normal anatomy of the facial nerve (FN) at MRI. A, B, C: submillimeter axial slices of a heavily 3D T2-weighted sequence (DRIVE). D, E: sagittal oblique reconstructions (perpendicular to the axis of VII-VIII) of the same sequence showing the position of the VIIth and VIIIth nerves (D, in the cistern; E, in the internal auditory canal). F: coronal reconstruction through the axis of the cisternal part of the VIIIth nerve. In Fig. 1C, the motor nucleus of the FN as is represented by a white dot; its motor tract (white line) courses around the abducens nucleus (grey dot) at the level of the facial colliculus which appears as a small bump on the floor of the fourth ventricle; then FN continues towards the inferior aspect of the pons. The cisternal parts of the nerves are clearly seen, delineated by the hyperintense cerebrospinal fluid. As shown in Fig. 1F, the VIIIth nerve is first attached to the inferior border of the pons just above the level of pontomedullary sulcus at root exit point (RexP) then detaches at RDP (root detachment point). CN: cochlear nerve; IVN: inferior vestibular nerve; SVN: superior vestibular nerve.

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