# How to face the hemifacial spasm: challenges and misconceptions

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**Word count:**
**Total:** 1803 words
**Abstract:** 158 words
**Legends:** 363 words
**Figures:** 2 figures, 5 videos
**Tables:** 2
**References:** 58

**Key words:**
Hemifacial Spasm - Facial Nerve - Movement Disorders - Differential Diagnosis - Botulinum Toxin

**Statements and Declarations**

**Financial disclosure:** For each author,
FD received funds from the Belgian Fund for Scientific Research (F.R.S-FNRS)
DA has no financial conflicts of interest to disclose

**Funding sources:** No funding was received to assist with the preparation of this manuscript.

**Competing interests:** The authors have no competing interests to declare that are relevant to the content of this article.

**Patients consent for publication:** Available

## Abstract
Hemifacial spasm (HFS) is characterised by intermittent, brief or sustained, repetitive contractions of the muscles innervated by one facial nerve. It is one of the most frequent movement disorders affecting the face. However common and allegedly straightforward to diagnose, it might reveal as a challenge for clinicians in various situations. Indeed, it often needs prior exclusion of many other movement disorders affecting the face, with frequent phenomenological overlaps with blepharospasm, post-facial palsy, facial motor tics, etc. The clinical diagnosis shall be supported by modern brain imaging techniques, and sometimes electromyography, as some particular aetiologies may require specific treatment. Primary forms are associated with vascular compression of the ipsilateral seventh cranial nerve, whereas secondary forms can be caused by any injury occurring on the facial nerve course. This article proposes a global and organised approach to the diagnosis, and the ensuing therapeutic options, as many practitioners still use some inefficient medications when they encounter a case of facial spasm.

## Introduction
Hemifacial spasm (HFS) is a movement disorder characterised by subtle or prolonged contractions of muscles innervated by the seventh cranial nerve. It often results from a neurovascular conflict in the root-exit zone in the brainstem but can also be secondary to various types of lesions in this particular zone. Various aetiologies can mimic HFS, but the diagnosis is mostly clinical. However, in some specific cases, magnetic resonance (MR) scan of the brain and electromyography (EMG) may help in the differential diagnosis.

## 1. Clinical aspects
HFS corresponds to a movement disorder affecting the muscles innervated by the facial nerve. It generally starts as unintentional, myoclonic, succinct contractions in the region of the orbicularis oculi muscle, progressively spreading to the lower part of the face, affecting the zygomatic, orbicular oris, mentalis, or even the platysma muscles (**video 1**). Sometimes, curious presentations can be observed, and the patient may complain about movement disorders of the ears, because of the contraction of peri-auricular muscles (**video 2**).
It sometimes occurs in more sustained muscle contractions, mostly automatically, but sometimes in response to deliberate or lengthened hemifacial contractions[1–3].

Besides the facial involvement, the HFS may lead to various concomitant symptoms, like dysarthria, vision impairment (due to the constant eye closure), or tinnitus, mostly because of a spasm of the middle ear muscles (e.g., stapedius). Another manifestation, called *"tic convulsif"* from French publications, has been described when HFS is accompanied by trigeminal neuralgia [4,5]. Furthermore, it can lead to social discomfort, or even participate as an independent psychiatric comorbidity. The spasms can remain during sleep, and are often exacerbated by anxiety, stress, mastication, etc[6–8].

Regarding epidemiology, HFS affects mostly women (1,7 – 1 ratio) around 40-50 years of age (but sometimes involve the older or younger ranges). The Asian population seems to be more concerned[9].
HFS are mostly sporadic; nevertheless, some rare familial cases have been described. For instance, HFS have been observed in some patients presenting with AIFM1 variant (**video 3**) [10].

The evolution is characterised by a diffusion of the spasms, followed by a stabilisation. There are only rare cases of disappearance, but it is then mostly transitory[11–13].

Patients usually describe HFS as a "tremor" or a "twitching" affecting half of the face, but in some rare cases, it can occur bilaterally. Nevertheless, the spasms start unilaterally, and the contractions are asynchronous and milder in the contralateral hemifacial region[3]. The other Babinski's sign is typically present, but the sensibility is not high. This sign manifests when "orbicularis oculi contracts and the eye closes, the internal part of the frontalis contracts at the same time, the eyebrow rises during eye occlusion", as described by Babinski himself in his publication dedicated to HFS (**fig. 1**)[14–16].


*Fig. 1 (a) The other Babinski sign, as seen in a typical case of HFS. The patient is a 77-year-old woman with an idiopathic (primary) form of HFS. (b) Charcot's sign, as seen in a case of blepharospasm. These examples show the difference between the elevation of the ipsilateral eyebrow in HFS, as opposed to the blepharospasm which leads to a lowering of the eyebrow during the spasm.*

## 2. Aetiologies

The aetiologies of HFS are divided in two broad categories, primary and secondary causes (**Table 1**) [3,17].

*Table 1 Aetiologies of the hemifacial spasms*

| Primary HFS | Secondary HFS |
|---|---|
| • Idiopathic<br>• Rare genetic causes<br>• Neurovascular compression of the facial nerve at the root-exit zone in the brainstem<br>  ○ Compression by an artery (AICA, PICA, etc)<br>  ○ Compression by a vein<br>  ○ Compression by multiple vessels | • Meningioma<br>• Acoustic neuroma<br>• Glioma<br>• Schwannoma<br>• Epidermoid, arachnoid cyst<br>• Lipoma<br>• Arteriovenous malformations<br>• Brainstem lesions (stroke, trauma, demyelinating disorders)<br>• Infections (otitis media, tubercular meningitis)<br>• Structural abnormalities of the posterior cranial fossa (Paget's disease, Chiari malformation)<br>• Parotid tumours |

The primary causes, also referred to as idiopathic, result from a compression of the facial nerve in the root-exit zone in the brainstem, by an aberrant or ectatic blood vessel, usually the antero-inferior cerebellar artery (AICA) or the postero-inferior cerebellar artery (PICA), but it can also be a vein, or even multiple vessels in general (**fig. 2**) [18,19].
It is important to note that a neurovascular compression (NVC) is not constantly linked to a pathological presentation, and it can sometimes be coincidental. Furthermore, it can sometimes be present on the contralateral side in patients with HFS[20]. Regarding this aetiology, some aggravating factors have been described, such as arterial hypertension, or low posterior cranial fossa volume as often seen in Asian populations[21,22].

Still in the category of primary HFS, some genetic factors have been incriminated, with a few familial cases reported in the literature[12,23,24].

The secondary causes are numerous. Among them are benign and malignant growing lesions such as gliomas, meningiomas, schwannomas, arachnoid cysts, but also vascular abnormalities (telangiectasia, etc.), demyelinating lesions, brainstem infarctions, etc. These lesions are generally located in the brainstem, ponto-cerebellar angle or the fourth ventricle[1,3].

In the context of secondary HFS, the associated spasm is generally more sustained or even fixed[25]. These examples of secondary causes show that HFS might reveal, along with other neurological signs, malign and preoccupying aetiologies deserving thorough investigations and sometimes specific therapeutic intervention.

## 3. Investigations

Besides a well-conducted neurological examination, the use of MR scan of the brain can be useful to demonstrate a vascular compression or to rule out rare causes as mentioned above in the differential diagnosis process (**fig. 2**). The different sequences that are recommended are high resolution 3D-T2 weighted sequences [(Constructive Interference Steady State (CISS) and Fast Imaging Employing Steady State Acquisition (FIESTA)] with angiography or 3D-TOF[26,27]. MR scan is also essential as a pre-operative assessment (see point 5)[28].

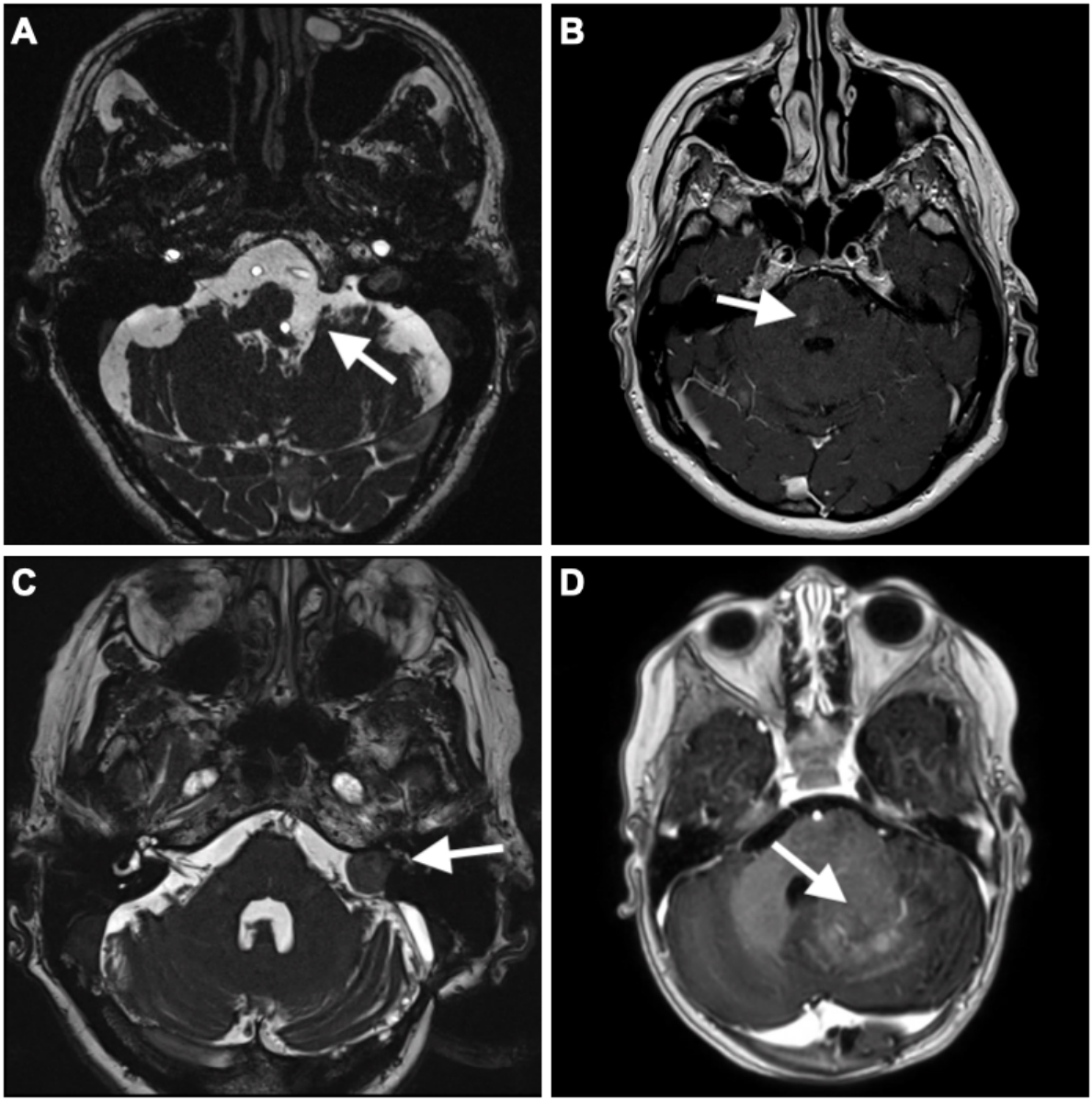


*Fig. 2 Conventional MR scan of the brain in HFS. (A) 3D-T2 weighted sequence (CISS), axial slices at the level of the cerebellum, of a 67-year-old man presenting a compression of the bulbo-pontic junction by an ectatic left vertebral artery. (B) T1-weighted sequence post-gadolinium contrast, axial slices at the level of the cerebellum, of a 65-year-old man presenting a telangiectasia in the right paramedial part of the pons. (C) CISS axial slices at the level of the cerebellum, of an 80-year-old woman presenting a compression in the left cerebellopontine angle by an acoustic neuroma. (D) T1-weighted sequence post-gadolinium contrast, axial slices at the level of the cerebellum, of a 5-year-old girl presenting a cerebello-pontine ganglioglioma (mutation BRAF V600E).*

Besides, EMG examination might also be useful to differentiate HFS from other movement disorders affecting the face, particularly synkinesis. Indeed, the recording of muscle activity in the lower part of the face while contracting one muscle of the upper part (or the other way round) may be able to demonstrate spontaneous activity. In the context of HFS, when a patient voluntarily contracts his facial muscles, the recording should be strictly normal. However, at rest, some contractions may be found as a sign of nerve hyperexcitability[6,8].

## 4. Differential diagnosis

There are many phenomenological overlaps between HSF and other movement disorders and the differential diagnosis is mostly suggested by the clinical characteristics of the muscle contractions (**table 2**).

*Table 2 Differential diagnosis of the facial movement disorders*

| | Movement characteristic | Localisation | Particularities | Associated signs |
|---|---|---|---|---|
| **Hemifacial spasm** | ○ Clonic / Tonic movements<br>○ Presence of the "other Babinski sign" | ○ Muscles innervated by the ipsilateral 7th nerve<br>○ Starting around the eye followed by a spreading to the inferior part of the face<br>○ Unilateral (rarely bilateral) | ○ May remain during sleep<br>○ Exacerbated by anxiety, mastication | ○ Dysarthria<br>○ Tinnitus<br>○ Vision impairment |
| **Focal dystonia** | ○ Stereotyped pattern of movements<br>○ Brief or sustained<br>○ Bilateral (rarely unilateral) | Blepharospasm:<br>○ Orbicularis oculi and other periorbital muscles (blepharoclonus, sustained closing of the eye)<br>Oromandibular dystonia:<br>• Medial pterygoids, masseters and temporalis (closing dystonia or bruxism)<br>• Lateral pterygoids and digastric muscles (opening and/or protrusion) | ○ Exacerbated by light, anxiety<br>○ *Geste antagoniste*<br>○ Absence during sleep | ○ Vision impairment<br>○ Bruxism, trouble eating, vocalizations<br>○ Extra-facial signs may be associated |
| **Focal motor tic** | ○ Sudden, repetitive, non-rhythmic movements<br>○ Simple or complex | ○ Any muscle group may be involved<br>○ Mostly bilateral | ○ May persist during sleep<br>○ Frequently associated with urge to move and premonitory sensations<br>○ Can be suppressed voluntarily<br>○ Exacerbated by stress, excitement and during relaxation after stress | ○ Vocal tics<br>○ Limbs tics |
| **Post-facial palsy** | ○ Involuntary muscle contractions (e.g., chin and neck contraction) during voluntary facial movements (e.g., smile, eye closure) | ○ Muscles innervated by the 7th nerve | ○ Absence at rest | ○ Residual facial palsy |
| **Functional spasm** | ○ Asymmetric spasm of orbicularis oculi<br>○ Eyebrow rising contralateral to closed eye | ○ Any muscle group may be involved<br>○ Variability<br>○ Bilateral | ○ Resistance to passive eyelid openinig<br>○ Absence during sleep<br>○ Distractible | ○ Lip-pulling sign<br>○ Depression, anxiety |



## 4.1. Post-facial palsy

The manifestation of post-facial palsy, also known as synkinesis, usually occurs after three to six months of a serious case of Bell's palsy, but also after an infectious or auto-immune aetiology leading to facial palsy. The process of reinnervation can sometimes be chaotical, leading to the manifestation of synkinesis. The diagnosis is brought by the clinical history and, if needed, EMG data, revealing signs of synkinetic activity, along with needle-EMG evidence of muscle denervation, which correlates to a remaining palsy (**video 4**)[29,30].

## 4.2. Focal dystonia

Various types of focal dystonias may affect the face, sometimes with surprising phenomenology. Blepharospasm is one of the most frequent adult-onset focal dystonia, affecting the muscles of the upper part of the face (e.g., orbicularis oculi, corrugator supercilia, frontalis, etc.) and inducing unintentional tonic contracture of periocular muscles resulting in abnormal blinking ("eye fluttering") or tonic spasm of the eyelids[31–33].
Both eyelids are generally involved; however, this condition is often asymmetrical at the beginning. The Charcot's sign (the spasms of the eyes are associated with the lowering of the eyebrow, below the superior orbital rim) can be a hint for the diagnosis (**fig. 1**) [34].

Blepharospasm tends to increase with the activity of oral muscles (e.g., during speech) and with light overexposure, and is often alleviated by voluntary movements, such as pursing the lips or applying fingers around the eyes. This corresponds to a *"geste antagoniste"* or sensory trick, a typical sign found in dystonic conditions[35,36].

Oromandibular dystonia and Meige syndrome (characterised by the association of blepharospasm and oromandibular dystonia) may also mimic HFS but these conditions are generally bilateral and tend to involve muscles innervated by other cranial nerves[37–39].

### 4.3. Facial motor tics

Motor tics are – as described in the established definition – "sudden, rapid, recurrent, non-rhythmic motor movements or vocalizations usually appearing in bouts while waxing and waning in frequency, intensity and kind of tics"[40].

Facial tics may sometimes mimic HFS, but the movements are usually less stereotyped and tics generally take place after a premonitory feeling with the compulsion to release the tics ("urge"). General neurologic examinations might reveal vocal manifestations or some movement disorders affecting the limbs (**video 5**) [41,42].

### 4.4. Functional spasm

Sometimes, facial spasms might be functional (formerly "psychogenic"). Typically, patients presenting with functional spasm display an elevation of the eyebrow contralateral to the closing eye, which is the opposite of what is observed with the other Babinski sign (see section 1). Besides, it is characterised by variable and mostly distractible movements affecting one or both sides of the face [15,43,44].

### 4.5. Other rare causes

Whipple's disease, an infectious disease due to *Tropheryma whipplei*, may provoke an oculomasticatory myorhythmia, which consists of rhythmical, tremor-like involuntary movements that usually occur in the face but might also affect the limbs. When compared to HFS, myorhythmia appears as more rhythmic and continuous[45,46].

Facial myoclonus can also be observed in Rassmussen encephalitis, a rare autoimmune condition responsible of a cortical inflammation leading to focal epilepsy. The subsequent muscle contraction may sometimes mimic HFS, but the diagnosis is usually suggested by EEG and imaging[47–49].

Facial myokymia is a benign entity associated with asthenia or caffeine consumption and consists of undulating movements of the muscles underneath the skin. Rarely, it is associated with a pontine lesion of the facial nerve, and when it occurs, the "rippling" movements are persistent. The needle-EMG examination may help in the differential diagnosis[50–52].

Hemimasticatory spasm is the result of the contraction of muscles innervated by the trigeminal nerve and is a rare idiopathic condition affecting mostly mastication. It is sometimes correlated with a compression or a lesion in the pontine area. It is also associated with facial hemiatrophy[53,54].

## 5. Treatment

There is not much evidence of a successful medication treatment, as it is sometimes prescribed in general practice. Some clinicians have proposed antiepileptic drugs or benzodiazepines. Adverse effects supplant the efficacy of these molecules, and it is not recommended to use them[7].

Botulinum toxin type A (BoNT-A) is currently considered as the first-line therapy for symptomatic treatment of HFS. BoNT-A prevents the release of acetylcholine in the cholinergic motor endings, eventually leading to a chemodenervation of the muscles and *in fine* to a focal paresis. The injections must be repeated every three

months by a specialised neurologist. The adverse effects include palpebral or labial ptosis, lagophtalmia, xerophtalmia. The long-term use of BoNT-A has been demonstrated as safe and efficient in the treatment of HFS assuming that the antigenicity phenomenon decreases with the use of the lowest effective dose, as recommended by Atassi and Oshima[55–57].

Microvascular decompression of the seventh nerve represents another therapeutic option to treat primary HFS. This surgical intervention consists of introducing a Teflon sponge between the nerve and the vessel responsible for the compression. The monitoring is insured by intraoperative EMG and auditory evoked potentials[1,58].

## Conclusion

HFS is a common movement disorder affecting territories innervated by the seventh cranial nerve and is often mistaken for other pathological mimickers. The diagnosis is based on the clinical examination but may sometimes be comforted by paraclinical investigations, such as MR scan of the brain or EMG, essentially when there are concomitant atypical signs. In routine clinical practice, the problem dwells in the diagnosis, which is often missed or delayed, and may unveil rare and treatable causes.
The therapeutic options are limited to BoNT-A injections and microvascular decompression surgery, whereas other medications have been demonstrated as inefficient or even deleterious.



## Legend of the figures

- **Fig. 1**: These examples show the difference between the elevation of the ipsilateral eyebrow in HFS, as opposed to the blepharospasm which leads to a lowering of the eyebrow during the spasm.
  - (A) The other Babinski sign, as seen in a typical case of HFS. The patient is a 77-year-old woman with an idiopathic (primary) form of HFS.
  - (B) Charcot's sign, as seen in a case of blepharospasm.

- **Fig. 2**: Conventional MR scan of the brain in HFS.
  - (A) 3D-T2 weighted sequence (CISS), axial slices at the level of the cerebellum, of a 67-year-old man presenting a compression of the bulbo-pontic junction by an ectatic left vertebral artery.
  - (B) T1-weighted sequence post-gadolinium contrast, axial slices at the level of the cerebellum, of a 65-year-old man presenting a telangiectasia in the right paramedial part of the pons.
  - (C) CISS axial slices at the level of the cerebellum, of an 80-year-old woman presenting a compression in the left cerebellopontine angle by an acoustic neuroma.
  - (D) T1-weighted sequence post-gadolinium contrast, axial slices at the level of the cerebellum, of a 5-year-old girl presenting a cerebello-pontine ganglioglioma.

- **Video 1**: Various cases of primary HFS
  - (A) 77-year-old woman presenting an idiopathic HFS with a typical other Babinski sign.
  - (B) 67-year-old man presenting a compression of the pons by an ectatic left vertebral artery, responsible for a primary HFS. During voluntary contraction, the spasms disappear.
  - (C) 63-year-old man presenting an idiopathic HFS, with the demonstration of an increase of the frequency of the spasms with dorsal decubitus. This video is recorded during orthostatism.
  - (D) The same patient as in video 1-C, but during dorsal decubitus.

- **Video 2**: Atypical primary HFS
  - Atypical presentation of a primary HFS affecting the right peri-auricular muscles.

- **Video 3**: Primary genetic HFS
  - Rare case of a right primary HFS in a 7-year-old boy affected by AIFM1 variant.

- **Video 4**: Post-facial palsy
  - This video highlights the occurring of synkinesis in the right side of the face of a 47-year-old woman suffering from residual facial palsy (Ramsay-Hunt syndrome).

- **Video 5**: Facial motor tics
  - This 38-year-old patient with Tourette's syndrome presents facial motor tics which are present bilaterally and are non-stereotyped.

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