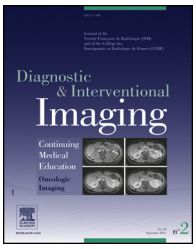
Diagnostic and Interventional Imaging (2013) **94**, 1033—1042



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# Facial nerve: From anatomy to pathology

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**KEYWORDS**
Cranial nerves;
Pathology;
Facial nerve (CN VII)

**Abstract** The facial nerve (CN VII) emerges from the facial nerve nucleus in the pons. It is accompanied by CN VIII along its cisternal pathway, as well as at the internal auditory meatus. Its petrous pathway includes a labyrinthine segment, a horizontal tympanic segment and a vertical mastoid segment until the stylomastoid foramen. It then continues to the parotid gland. Pontine impairment is usually associated with other neurological symptoms. Lesions of the cerebellopontine angle (most often meningioma and schwannoma) initially result in impairment of CN VIII. The impairment of CN VII takes second place. Peripheral impairment (outside of a traumatic context) is most often due to Bell's palsy.


## Functions, clinics

The facial nerve (seventh pair of cranial nerves) is a mixed nerve with efferent (motor and vegetative) and afferent (sensitive and sensory) nerve fibres. It consists of the facial nerve properly speaking (CN VII), pure motor and the glossopalatine nerve (CN VIIb) [1]. It is the nerve from the second branchial arch with a very early formation within the acoustico-facial complex. It consists of the juxtaposition of somitic and branchial elements of the cranial nerve nuclei, in particular accounting for trigeminal and facial nerve anastomosis [2].

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2211-5684/$ — see front matter 
http://dx.doi.org/10.1016/j.diii.2013.06.016

### Motor function (efferent pathway)

The symptoms associated with the loss of this function are in general those motivating the consultation. It innervates the platysma muscles of the face and neck except for the levator palpebrae superioris (third pair of cranial nerves). Impairment thereby results in facial asymmetry, as the healthy side is responsible for a contralateral attraction. In case of peripheral impairment, it is homogenous on the upper face and lower face but is not accompanied by automatic-voluntary dissociation. In case of central facial nerve paralysis, the deficiency is predominate on the lower face and is accompanied by automatic-voluntary dissociation. Bell's sign corresponds to an automatic upward and outward movement of the eye (mechanism of corneal protection) when the subject tries to close his eye. Souques' sign is only found in cases of moderate facial nerve paralysis and is manifested by the eyelashes being more visible on the pathological side when the patient is asked to close his eyes.

The severity of the facial impairment may be assessed according to the extent of the motor deficiency with clinical scales, also allowing it to be monitored. The House-Brackmann scale is most often used. It corresponds to a global score ranging from I (normal) to V (total paralysis). The motor fibres via the collateral branches of the facial nerve are: stapedial nerve, posterior auricular nerve, digastric nerve and stylohyoid nerve while the terminal fibres are destined to the platysma muscles of the face and neck (temporal, zygomatic, buccal, mandibular and cervical branches).

### Visceral function (secretory efferent pathway)

On the one hand, it comprises the parasympathetic fibres destined to the lacrimal glands, the oronasal mucous membranes. This is clinically manifested by a dry eye. On the other hand, the facial nerve innervates the sublingual and submandibular salivary glands. Impairment of this portion thereby is manifested by a reduction in the flow of saliva, except for that derived from the parotid gland (glossopharyngeal nerve).

### Sensitivity function

This corresponds to the Ramsay-Hunt zone, including the ear canal, the auricle and the retroauricular region.

### Sensory function

This involves the taste sensitivity of the anterior two-thirds of the tongue and the palate by means of the chorda tympani.

## Topography

### Brain stem nuclei

#### Motor nucleus of facial nerve

It is located at the level of the pons in front and lateral to the nucleus of CN VI. There is a certain somatotopic arrangement at this level that then disappears. From here, the fibres run towards the back creating a loop behind the nucleus of CN VI and emerge from the stem just above the bulbopontine junction between CN VIII (above) and CN VI [3]. Supranuclear control is provided by the corticospinal tract. It is bilaterally projected on the upper part of the nucleus (controlling the muscles of the upper face) and only contralaterally for the lower part of the face (accounting for the predominant impairment on the lower face in case of facial nerve paralysis of central origin). Central impairment may therefore be located from the pre-central gyrus up to the pontine nucleus.

Projections also exist from the facial nuclei to the extrapyramidal tract, the cerebellum, other nuclei from the brain stem (nucleus of CN V, superior olivary complex, nucleus of CN VIII...).

#### Visceral nuclei

They mainly comprise the superior salivary nucleus, the lacrimal nucleus and the solitary nucleus [4]. The corresponding fibres group along part of their pathway within the glossopalatine nerve. There are then several divisions involving trigeminal-facial nerve anastomoses.

The efferent fibres correspond to the secretory parasympathetic contingent. They arise from the superior salivary nucleus and the lacrimal nucleus. On the one hand, they are destined to the lacrimal gland and the rhinopharyngeal mucous membrane. They correspond to the greater petrosal nerve, extending by the pterygoid nerve creating a synapse at the pterygopalatine ganglion. In addition, they innervate the submandibular and sublingual glands via the chorda tympani, distally rejoining the lingual nerve (trigeminal branch).

The afferent fibres are on the one hand sensitive from the Ramsay Hunt zone, also running along the lingual nerve and then the chorda tympani towards the spinal tract (CNV) and gustative perception by the same pathways towards the solitary nucleus. The latter also receives the gustatory afferences from CN IX, CN X and CN XI.

### Cisternal segment

The motor fibres from CN VII, after adhering to the pons for 2 to 3 mm, take an ascendant and lateral direction in the cerebellopontine angle. CN VIIb is theoretically separated although in close contact with the facial nerve in the cisterne and separation is not possible, including in post-surgery. The REZ is found about 2 mm after the emergence of the brain stem (although this may go up 21 mm). This corresponds to the theoretical zone where the oligodendrocytes (central nervous system) turn into Schwann cells (peripheral nervous system) [5].

### Intracanalicular segment (internal auditory meatus)

Fibres from CN VII and CN VIIb are grouped at the anterior-superior part of the internal auditory meatus (IAM), motor fibres covering the sensitive fibres. An arachnoid layer invaginates at the bottom of the IAM (or even up to the geniculate ganglion according to certain authors).

## Petrous segment (Fallopian canal)

It corresponds to the facial canal and is separated in three portions [1].

### First portion: labyrinthine

It runs from the fundus of the IAM to the geniculate ganglion. The facial nerve runs outside and in front, between the cochlear and the vestibular.

### Geniculum of the facial nerve: geniculate ganglion

It is located in the petrous bone posterior-laterally with respect to the internal carotid artery, posterior-median with respect to the spinosum foramen, anterior with respect to the superior semicircular canal. It contains the cell bodies derived from the nerve of the chorda tympani (taste and sensitivity). However, the motor fibres, including the visceral fibres, pass without creating synapse as their cell bodes are located in the brain stem.

The greater superficial petrosal nerve arises at this level. It exits from the base of the skull through the foramen of the greater petrosal. It then passes by the trigeminal cave to join the pterygoid canal where it is joined by the deep petrosal nerve (sympathetic contingent derived from CN IX) to form the nerve from the pterygoid canal. The latter passes through the pterygopalatine fossa to end at the pterygopalatine ganglion, then gives rise to branches anastomosing with the branches of trigeminal V1 (lacrimal) and V2 (oronasal).

### Second portion: horizontal or tympanic

Its path is posterior from the geniculate ganglion, under the lateral semicircular canal between the vestibule and the eardrum. There is no collateral anastomosis at this level.

### Third portion: vertical or mastoidal

The nerve descends vertically between the eardrum and the ear canal. Three collateral branches arise at this level:

- acoustic (stapedius muscle);
- chorda tympani (efferent fibres for the submandibular and sublingual glands and afferent fibres for taste of the anterior two-third of the tongue). It crosses the middle ear at the level of the eardrum then passes by the petrotympanic fissure to join the lingual nerve (branch of CN V3);
- sensitive branch for the auricular region.

## Stylomastoid foramen, parotid

It corresponds to the emergence of the facial nerve at the bottom of the skull. It is located outside of the styloid. Immediately after its passage, the facial nerve leaves branches for the cervical muscles (auricular, digastric, stylohyoid. . .) and then enters the parotid to give rise to muscular temporofacial and cervicofacial branches in contact with the posterior side of the external jugular.

# Complementary examinations outside of imaging

## Electroneuromyography

It can confirm the existence of a facial nerve lesion, characterise it (axon impairment or demyelinising, conduction block), quantify the motor denervation and provide prognostic information. It comprises two parts:

- neurography (or stimulodetection), during which the stimulation of the facial nerve in front of the ear generates a muscle contraction recorded by surface electrodes on different facial muscles (orbicularis oculi muscle, nasalis muscle, orbicularis oris muscle). Comparison between the impaired side and the healthy side helps quantify the extent of the impairment and follow the evolution. Study of the blink reflex is used to assess the reflex arc between the trigeminal nerve (arc related by branches of the supraorbital nerve) and facial nerve (efferent arc by its motor fibers). Relays exist in the pons and the medulla, this review thereby providing complementary information about the brain stem [2]. In case of facial nerve paralysis, its very early persistence in the initial phase is an element indicating a good prognosis;
- the electromyography properly speaking (or needle detection), during which a needle is successively inserted in the orbicularis oculi muscle and then the orbicularis oris muscle, in order to study the appearance of the line when at rest and in contraction. The abnormal presence of spontaneous activity when at rest such as fibrillations or slow potentials is a sign of denervation, although these activities only appear with a delay (8—10 days for the facial). During the voluntary contraction, the appearance of a number of motor unit potentials as well as their mode of recruitment provide both quantitative and prognostic elements as to current or future reinnervation.

The value of the electroneuromyography (ENMG) depends on the time after the occurrence of the facial nerve paralysis: during the first days, the blink reflex is certainly the best prognostic tool, before a full PFP. As of the eighth day, the classic neurograph and needle EMG provide information about the possible existence of active denervation and quantify it. On a longer-term basis, the ENMG helps predict whether or not the reinnervation will continue.

## Paraclinical examinations carried out by the ORL

In addition to the systematic otoscopy, vestibular testing and pure-tone audiometry, certain tests may be carried out to determine the location.

### Schirmer's test

If the result is pathological (unilateral lacrimal deficiency), it locates the impairment upstream from the origin of the greater superficial petrosal nerve (origin at the geniculate ganglion) or along its path (isolated impairment of this contingent without impairment of the rest of the functions, in particular motor functions of the facial nerve).

### Stapedial reflex: acoustico-facial

The clinical translation is painful hyperacousis. After a sound stimulus, the afferent arc passes via the cochlear and olivary nuclei. The efferent arc passes via the ipsi and contralateral accessory facial motor nuclei towards the stapedius muscle. Its alteration may therefore be related to the facial nerve, auditory nerve or impairment of the middle ear. It is abolished if there is a lesion located upstream or on the stapedius nerve (origin on the mastoid portion of the facial nerve). About 50% of all patients presenting a spasm of the hemiface have anomalies on this reflex arc and are normalised after surgery.

### Electrogustometry

It is abnormal with impairment upstream or on the chorda tympani.

## Imaging

### Indications

Imaging is not systematic with peripheral facial nerve paralysis (although it is with central facial nerve paralysis). It is indicated when confronted with a progressive or recurrent installation, a serious non-regressive form or association with other symptoms suggesting impairment of other cranial nerves.

### Exploration protocols

#### Orientation towards a ''central'' origin

It is suspected in case of impairment of other cranial nerves such as CN VI or impairment of the long pathways. The minimum protocol consists of: Flair, T2*, diffusion, if necessary completed by T2 in thin sections, MR angiography, and an injection.

#### Topographic orientation at the cerebellopontine angle or internal auditory meatus

It is suspected in case of impairment associated with CN VIII.

Sequences centred at this level are obtained: CISS or FIESTA T2-very weighted sequences, fine high resolution T1 sections without and with gadolinium. A full exploration of the brain, in particular with injection, is required in case of meningeal disease.

#### Topographic orientation of the petrosal bone

The CT-scan of the petrosal bones is examined in first intention in a traumatic context. In other situations, the MRI and CT-scan are most often complementary. MRI exploration at the temporal bone is carried out in high resolution T2 and high-resolution thin section T1 with fat saturation without and after the injection of gadolinium. The diffusion sequence may be useful in certain situations, in particular when a cholesteatoma is suspected. The parotid gland should be included during the exploration of isolated peripheral facial nerve paralysis.

### Normal appearance in sections

The facial colliculus is prominent in the floor of the fourth ventricle (corresponding to the curve of the facial nerve around the nucleus of CN VI). It is easily identified on the axial sections by MRI (Fig. 1).

The facial nerve, along its cisternal pathway and at the IAM, is easily detected by MRI, in particular on the CISS or FIESTA sequences (Fig. 2). CN VII and CN VII b cannot be differentiated.

Within the petrosal bone, the facial nerve is contained in a bone canal, indirectly enabling its localisation at this level by CT-scan by locating the canal (Fig. 3). In axial section, the muscle of the tensor tympani should not be confused with the tympanic segment (continuing towards other segments on the over and underlying segments). In MRI, the facial nerve may be seen in an inconstant manner on the three portions in particular in T1 after injection or in 3D-Flair. The collateral branches are usually not identifiable.

The facial nerve cannot be detected within the parotid gland with conventional imaging. The use of a diffusion tensor imaging sequence may sometimes help delimit it and specify its relationship with a parotid lesion.

### Normal enhancement

Certain portions of the facial nerve present a close anatomical relationship with vascular structures. Therefore, enhancement is often found, in particular in the facial canal, whose normal or pathological nature is sometimes difficult to determine [6,7]. Nevertheless, intense enhancement in the first and third portion is not usually observed in normal subjects. The segments outside of the facial canal are normally not or are barely enhanced. Moreover, enhancement of the internal acoustic meatus involving CN VII and CN VIII should be considered as pathological. Recently, certain 3T

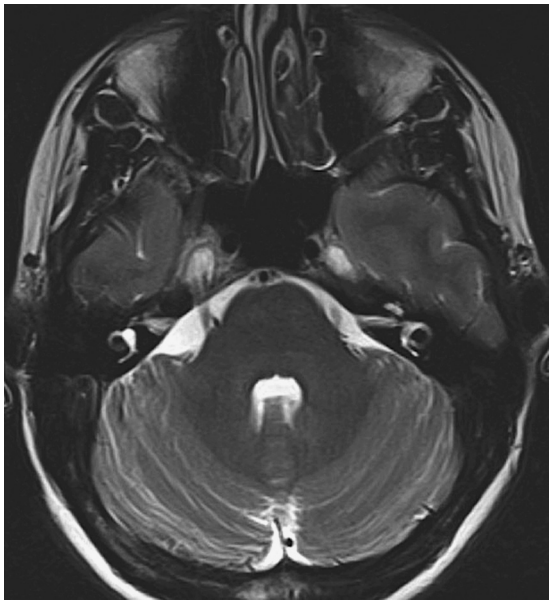

**Figure 1.** MRI in axial section of the brain stem in T2. Protrusion of the facial colliculi at the floor of the fourth ventricule (tip of arrow) corresponding to the curve of the facial nerve around the nucleus of CN VI.

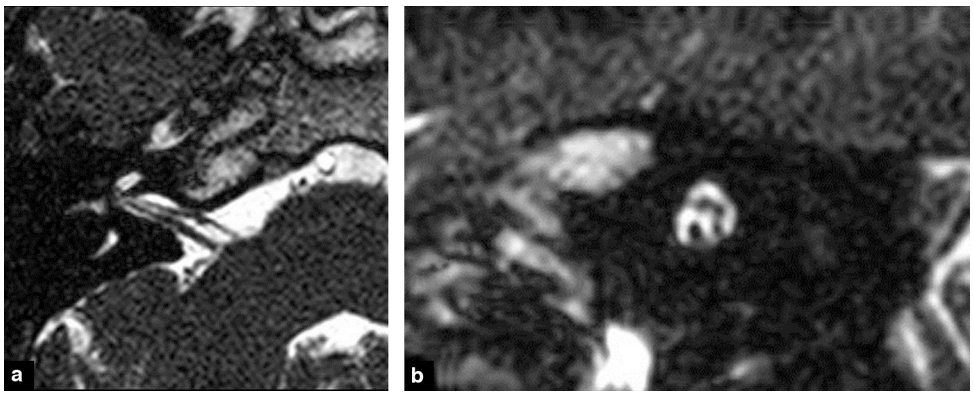

Figure 2. MRI in CISS sequence centred on the internal acoustic meatus. a: axial section revealing the facial nerve in anterior situation within the acoustico-facial; b: oblique sagittal section perpendicular to the axis of the IAM, the facial nerve is in anterior-superior position the cochlear nerve in anterior-inferior position, the superior and inferior vestibular nerves behind.

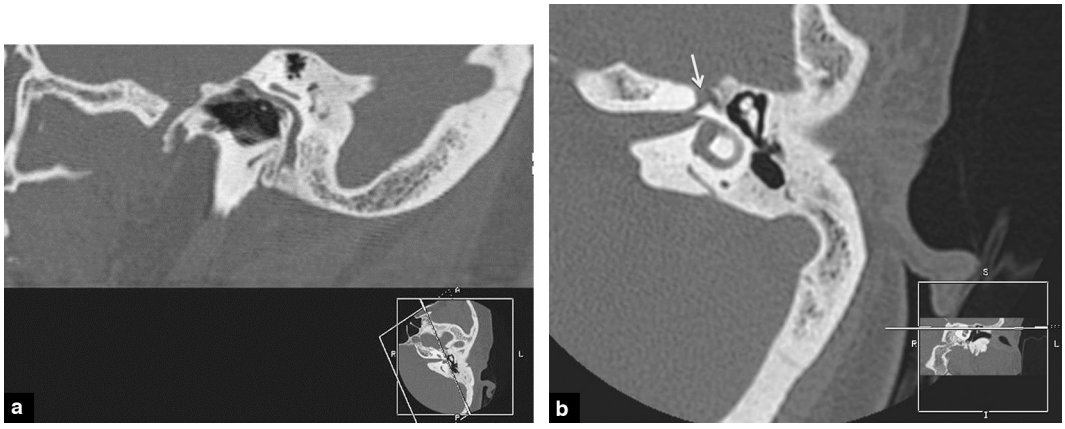

Figure 3. CT-scan of the petrosal bone. a: reconstruction in oblique sections revealing the tympanic and mastoid section of the facial nerve in the petrosal bone; b: axial section revealing the labyrinthic portion and the geniculate ganglion.

MRI studies have reported the superiority of 3D-FLAIR after injection compared with T1 after injection with fat saturation in the interpretation of the enhancement of the facial nerve, in particular in terms of specificity [8].

## Congenital anomalies affecting the pathway of the facial nerve

### Dehiscence of the facial canal

It is frequent in anatomopathological studies and only involves focal zones. The tympanic segment is the segment preferentially affected. In cases of inflammatory/infectious impairment of the middle ear, this defect in bone covering accounts for a zone of weakness [9].

### Variants of the intrapetrous pathway

They should be reported in a pre-surgical assessment, especially since they are often associated with ear malformations (stenosis or atresia of the ear canal, hypoplasia or atresia of the fenestra vestibuli...).

*Labyrinth portion variants*
It may be bifid.

*Tympanic portion variants*
The tympanic portion is caracterised as below:
- bifid;
- absent (the third portion is very anterior in this case);
- procident (in front of the fenestra vestibuli), often accompanied by agenesis of the fenestra vestibuli, this picture being responsible for transmission deafness;
- passage between the branches of the stapes, also responsible for transmission deafness.

*Mastoid portion variants*
Mastoid portion:
- bifid;
- situation very anterior in case of major aplasia or even absent.

## Pathologies responsible for peripheral facial nerve paralysis

The main aetiology is Bell's palsy (60%) followed by a traumatic origin (17%), inflammatory or infectious causes (10%), and tumoral causes (6%). The frequency of the different aetiologies depends on the lesion topography.

## Nuclear impairment

Even if we, in the strict sense, speak of central topography, the clinical translation will be peripheral. Nevertheless, in view of the proximity of other nuclei and tracts belonging to other long pathways, facial impairment is rarely isolated.

The most frequent aetiologies are tumoral (primary or secondary), inflammatory (Fig. 4) or ischemic. The mode of installation as well as the MRI characteristics of the signal anomaly of the brain stem most often suggest an orientation.

With ischemia, two syndromes have in particular been distinguished:

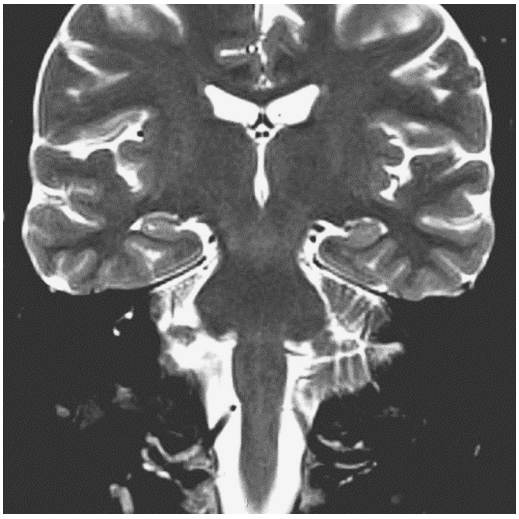

**Figure 4.** T2 TIRM in coronal section on the brain stem revealing a protuberance hypersignal along the pathway of the facial nerve just before it emerges in the cerebellopontine angle in a patient presenting peripheral facial nerve paralysis. Diagnosis of inflammatory disease of the central nervous system after a full assessment.

- Millard-Gubler syndrome: homolateral impairment of CN VII and contralateral motor deficiency respecting the face;
- Foville syndrome: homolateral impairment of CN VII, homolateral impairment of CN VI, paralysis of the laterality towards the lesion and contralateral motor deficiency respecting the face.

## Cerebellopontine angle and internal acoustic meatus

In view of the proximity of CN VIII, the latter is in general symptomatic, most often dominating the picture (deafness, vertigo, hyperacousis, tinnitis...).

Certain more voluminous processes may also be responsible for impairment associated with CN V or CN VI or even long pathways at the brain stem. The aetiologies may be divided into two groups:

- bilateral impairment with diffuse meningeal enhancement: carcinomatous meningitis (Fig. 5), hemopathies (lymphoma, leukaemia), infectious meningitis (Lyme's disease, tuberculosis...) or even inflammatory meningitis (sarcoidosis...), Miller-Fischer syndrome (form of Guillain-Barré syndrome);
- mass syndrome: meningioma, schwannoma (of CN VIII or VII — Fig. 6).

A vascular conflict on the cisternal pathway of CN VII (Fig. 7) may be manifested by a spasm of the hemiface (conflict on the motor branch) or a neurlgia of CN VII (conflict on CN VII b — paroxysmal otalgia). The spasm of the hemiface is attested to by clonus of the hemiface beginning at the lower eyelid and then progressing to the entire hemiface. In this situation, a time of flight MR angiography is helpful. The vessible responsible for the conflict is in general an artery. A vein or a vascular malformation is rarely involved. The artery most often involved in the PICA, impairment related to the AICA, the homo or even contralateral vertebral is possible [5,10]. In view of the frequency of vasculo-nervous contacts

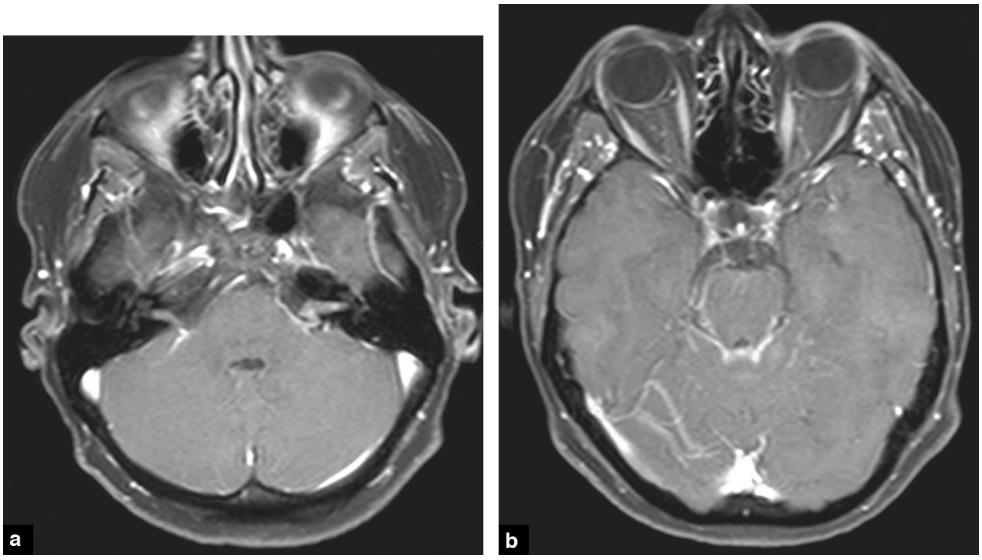


**Figure 5.** Axial T1 after injection of gadolinium revealing a symmetrical bilateral enhancement of the IAM (a), a diffuse leptomeningeal enhancement in posterior fossa (b). Carcinomatous meningitis confirmed with the lumbar puncture.

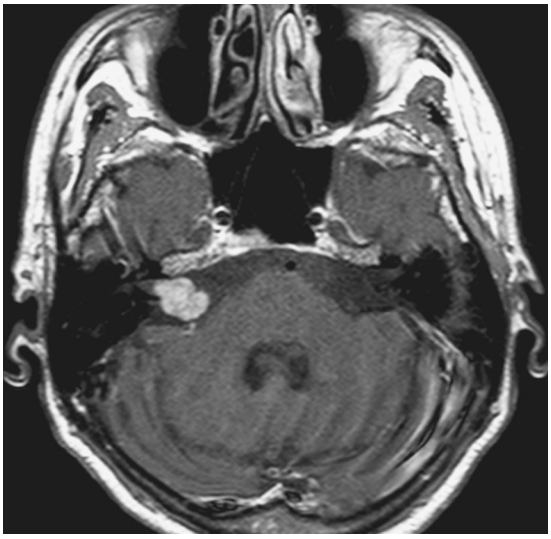

**Figure 6.** Axial T1 2 mm after injection of gadolinium. Enhanced tissue lesion of the cerebellopontine angle invading the IAM. Schwannoma.

in the healthy subject, the conflict is considered to be pathological only at the REZ level (2 to 3 mm after the emergence of the nerve from the brain stem), since this contact is perpendicular and involves a deformation of the nerve. The role of the vasculo-nervous conflict in other cases is discussed. A tumoral aetiology when confronted with a spasm of the hemiface is suspected in case of facial nerve paralysis and/or another associated neurological disorder. Injections of botulinum toxins in the muscles involved may be beneficial. Surgical decompression may be proposed when the vasculo-nervous conflict is detected.

## Facial canal

### Infectious aetiologies

#### *Viral aetiologies*

The most common as well as the primary cause of peripheral facial nerve paralysis is Bell's palsy. However, this involves a diagnosis by elimination. The installation is sudden, sometimes preceded by mastoid pain. Dysgeusia is often associated. The aetiology may be a viral reactivation of herpes (most often HSV1). The prognosis is most often good. The imaging does not indicate the isolated, typical forms (sudden onset, benign evolution). However, an atypical presentation (progressive onset or slow or even no recovery, impairment of other cranial pairs or considerable associated pain), the total lack of recovery after 6 months or a recurrence should lead to a revision of the diagnosis and indicates the necessity of an MRI. In cases of Bell's palsy [11], it detects the enhancement of the facial nerve with regular enhancement (Fig. 8). All segments from the internal acoustic meatus right to the mastoid portion may be involved. Nevertheless, enhancement is often found at the bottom of the IAM, from the first portion of the facial nerve and/or geniculate ganglion. This enhancement is aspecific. A thick, irregular or nodular aspect is atypical for Bell's palsy and should lead to an enlargement of the aetiological assessment.

Geniculate herpes zoster is also a common viral aetiology. It actually corresponds to a resurgence of VZV in the geniculate ganglion. The otalgia preceding the facial nerve paralysis is often very intense as opposed to Bell's palsy. The facial nerve paralysis is sudden and very quickly total. The vesicular eruption in the Ramsay Hunt zone is pathognomonic but inconstant. Signs of neuritis of CN VIII are frequently associated, more rarely other cranial nerves.

The other infectious aetiologies most often found are HSV, MNI, Lyme's disease and syphilis.

#### *Complications of infectious impairment of the middle ear*

Acute otitis media may be complicated by peripheral facial nerve paralysis. The evolution is generally favourable after antibiotic therapy. Dehiscence of the tympanic segment of the facial canal is a favouring factor. In case of chronicity of the otitis media, the occurrence of facial impairment will most often be due to a cholesteatoma or a cholesterol granuloma, even if it remains a rare complication in these cases. Tuberculosis should be tested for in case of complication of a chronic otitis media without cholesteatoma [12]. Malignant external otitis may also be complicated by facial impairment within the petrosal bone with a pseudo-tumoral appearance in the imaging.

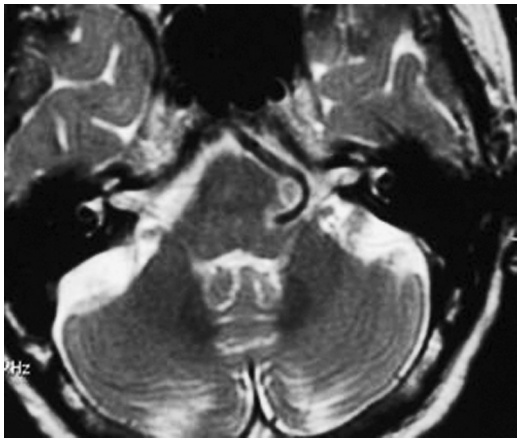

**Figure 7.** Axial T2 3 mm. Unrolled appearance of the left vertebral responsible for a vasculo-nervous conflict with CN VII revealed by a spasm of the hemiface.

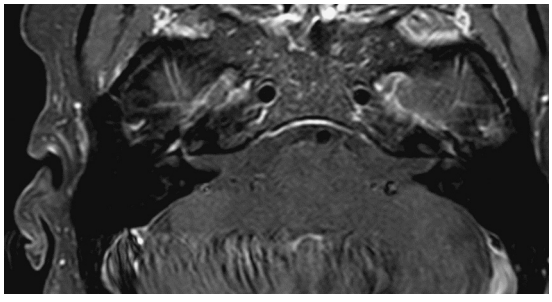

**Figure 8.** Axial T1 2 mm after injection of gadolinium and fat saturation. Fine but intense enhancement of the facial nerve in the bottom of the internal acoustic meatus, continuing in the first portion of the facial nerve and geniculate ganglion. Bell's palsy.

### Traumatic aetiology

It is observed in 50% of all transverse fractures (accounting for less than 20% of all fractures of the petrosal bone) as opposed to 20% in longitudinal fractures [13]. It is mainly related to impairment of the geniculate ganglion or the mastoid segment. Trans-labyrinth impairment is searched for in case of associated vertigo and/or total deafness, (Fig. 9). Sometimes, the fracture line is not visible, an indirect sign being the filling of the mastoid cells. The mechanism and care differ according to the time before the installation after the trauma:

- secondary: its origin is inflammatory with a favourable prognosis under corticoids;
- immediate and complete: the mechanism is a section or a compression. Early surgery may be indicated.

In view of a traumatic facial nerve paralysis without elements pointing to the petrosal bone, it is necessary to search for impairment on another segment (brains stem, parotid).

An iatrogenic origin may also be found, in particular in case of a non-detected anatomical variant.

### Tumoral aetiology

The possibility is raised with incomplete, fluctuating, recurrent or progressive peripheral facial nerve paralysis, preceded or accompanied by a spasm of the hemiface. The petrosal bone is the most frequent topography, in particular with:

- facial schwannoma: it is possible along the entire pathway although it is most often found at the bottom of the IAM, on the geniculate ganglion or in its tympanic segment (Fig. 10). In case of impairment of the geniculate ganglion, the appearance of pseudomeningioma of the middle fossa is detected [14];
- hemangioma of the facial nerve: it is located at the geniculate ganglion. The characteristic MRI elements include a very hyper T2 as well as intense enhancement. The honeycomb appearance on the CT-scan highly suggests it [15];
- jugulotympanic paraganglioma: the diagnosis may be suspected along with the otoscopy in view of a retrotympanic bluish mass. In the imaging, it is characterised by bone lysis, very intense enhancement of the images with flow void corresponding to dilated vascular structures.

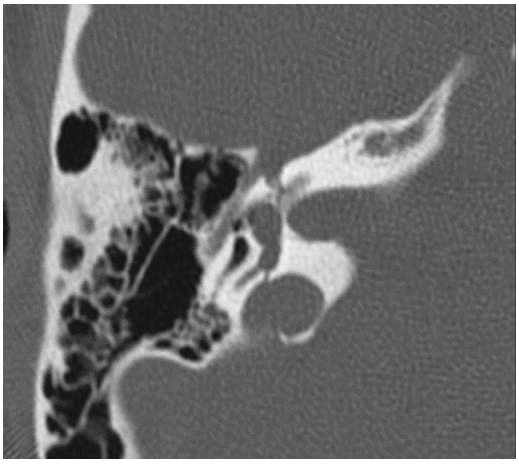

**Figure 9.** Transverse fracture of the petrosal bone passing by the jugular bulb, the labyrinth and exhausting itself at the geniculate ganglion.

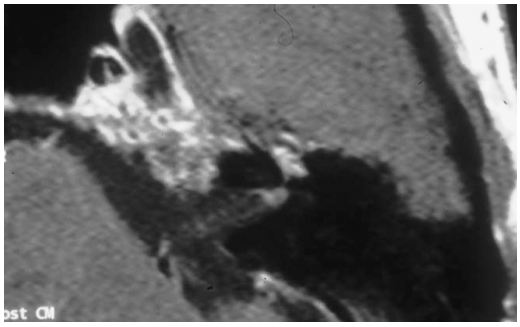

**Figure 10.** Axial T1 2 mm arter injection of gadolinium and fat saturation. Thick enhancement of the facial nerve at the bottom of the internal acoustic meatus, continuing in the first portion of the facial nerve and geniculate ganglion. Schwannoma of the facial nerve.

### Inflammatory aetiologies

They are responsible for nervous infiltration. The most common are sarcoidosis and Wegener's granulomatosis.

## Parotid

The tumoral lesions at the level, responsible for facial impairment, are in general malignant [16]. It may consist of primary tumours (adenoid cystic carcinoma), of intraparotid metastasis (Fig. 11).

Facial schwannoma may also develop on its parotid segment. The signal characteristics in MRI are close to those of a pleomorphic adenoma. Therefore, a tumoral extension is searched for along the facial nerve, in particular along the stylomastoid foramen with foraminal enlargement without osteolysis helping in the diagnosis.

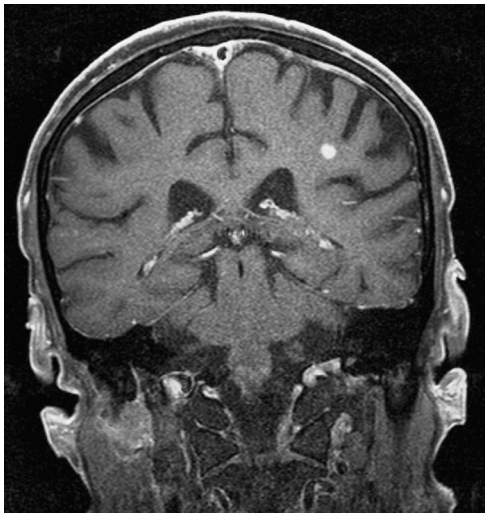

**Figure 11.** Coronal T1 3 mm after injection of gadolinium and fat saturation, centred on the parotids. Intraparotid nodular lesion with enhancement continuing along the facial nerve in its third portion. Enhanced nodular lesion of the left parietal intracebral. Metastatic dissemination within renal cell carcinoma.

## Perineural impairment

This corresponds to the dissemination of tumoral cells along the nerve sheath. In view of the richness of the nerve anastomoses between CN V and CN VII (greater petrosal nerve), there are numerous possible pathways for propagation.

The main neoplasms found consist of carcinoma of the head and neck, melanoma and lymphoma [17]. The signs to look for include:

- an increase in the calibre of the nerve with irregular, nodular enhancement;
- enlargement of the foramens of the base of the skull;
- effacement of the perineural fat, to look for more specifically in the pterygopalatine fossa;
- signs of denervation on the muscle groups involved.

It is important to search for this dissemination, especially since it may be asymptomatic, in view of the modifications in the therapeutic care (passage from curative to palliative in certain cases).

## Paediatric aetiologies [9]

### Newborn

It may involve an obstetrical trauma or a congenital anomaly. In the latter, the main aetiologies are facial hypoplasia (identified by a small canal on the CT-scan of the petrosal bone), Möbius syndrome (facial diplegia associated with impairment of CN VI with, in the MRI, hypoplasia of the brain stem and absence of the nerve detected in the cistern) and ear aplasia (most often a malposition of CN VII, rarely with facial nerve paralysis).

### Infant and child

Although the leading aetiology also remains Bell's palsy, it is less frequent than in the adult. Post-otitic impairment is frequent in view of the prevalence of otitis in the paediatric population. Viral causes are also found, more particularly with facial diplegia. Like in the adult Lyme's disease is not exceptional.

The most common tumoral causes in paediatrics are rhabdomyosarcoma of the petrosal bone, histiocytosis and the hemopathies. Schwannomas are much more rare and most often are included with neurofibromatosis type 2.

***TAKE-HOME MESSAGES***

- The exploration of peripheral impairment of the facial nerve is carried out from the brain stem to the parotid;
- Enhancement of the facial nerve is pathological outside of the facial canal, if it is intense on the second and third portions or if it extends to CN VIII or is located outside of the petrosal bone (acoustic meatus, after the stylomastoid foramen);
- Bell's palsy is the most frequent aetiology. However, MRI, the first intention examination, is not systematic except in case of atypies, recurrent or aggravating symptoms.

## Clinical case

A 40-year old man presenting progressive moderate facial nerve paralysis for 2 years without other associated, in particular audio-vestibular signs. A CT-scan of the petrosal bone is first carried out (Fig. 12). A complementary MRI is carried out (Fig. 13).

### Questions

1. What anomaly is found in this examination?
2. What do you suspect are the main aetiologies?
3. What will be your minimum protocol?
4. What anomalies are seen in this sequence?
5. What element with a high diagnostic orientation may have been found in the CT-scan?

### Answers

1. Osteolysis centres on the geniculate ganglion.
2. Facial hemangioma, schwannoma, metastasis.
3. Axial T1 2 mm without and with gadolinium (± with fat saturation). Coronal T1 2 mm with injection of gadolinium (± with fat saturation). High resolution axial T2 2 mm.

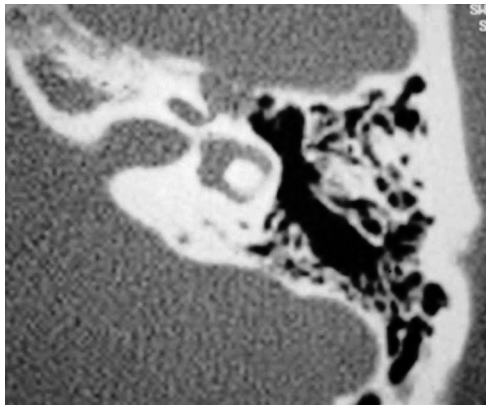

**Figure 12.** Scanner of the petrosal bone in axial sections, passing by the geniculate ganglion. Osteolysis at this level, with hazy and irregular edges.

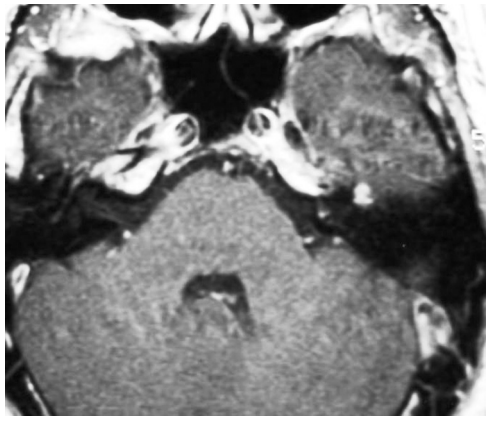

**Figure 13.** T1-weighted MRI after injection of gadolinium in axial sections. Intense and homogenous enhancement corresponding to the zone of osteolysis seen in the CT-scan.

4. Intense and homogenous enhancement in the osteolytic zone. The diagnosis retained is hemangioma of the facial nerve (confirmed with surgery).
5. Appearance of the bone framework at the geniculate ganglion in honeycomb matrix.

## Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

## References

[1] Binder DK, Sonne DC, Fischbein NJ. Facial nerve. In: Cranial nerves: anatomy, pathology imaging. New York: Thieme; 2010. p. 82—110.
[2] Lacombe H. Anatomie fonctionnelle du nerf facial. Neurochirurgie 2009;55(2):113—9.
[3] Leblanc A. Imagerie anatomique des nerfs crâniens. In: Méthodes d'investigation par imagerie par résonance magnétique et scanner. Berlin: Springer Verlag; 1989. p. 1—277.
[4] Harnsberger HR. Handbook of head and neck imaging. 2e ed St Louis: Mosby; 1995.
[5] Mercier P, Brassier G, Fournier HD, Delion M, Papon X, Lasjaunias P. Anatomie morphologique des nerfs crâniens dans leur portion cisternale. Neurochirurgie 2009;55(2):78—86.
[6] Martin-Duverneuil N, Sola-Martínez MT, Miaux Y, Cognard C, Weil A, Mompoint D, et al. Contrast enhancement of the facial nerve on MRI: normal or pathological. Neuroradiology 1997;39(3):207—12.
[7] Hong HS, Yi BH, Cha JG, Park SJ, Kim DH, Lee HK, et al. Enhancement pattern of the normal facial nerve at 3.0 T temporal MRI. Br J Radiol 2010;83(986):118—21.
[8] Lim HK, Lee JH, Hyun D, Park JW, Kim JL, Lee Hy, et al. MR diagnosis of facial neuritis: diagnostic performance of contrast-enhanced 3D-FLAIR technique compared with contrast-enhanced 3D-T1-fast-field echo with fat suppression. AJNR Am J Neuroradiol 2012;33(4):779—83.
[9] Elmaleh-berges M, Van Den Abbeele T. Nerf facial. In: Doyon D, editor. Nerfs crâniens : anatomie, clinique, imagerie. Paris: Masson; 2006. p. 137—60.
[10] Girard N, Poncet M, Caces F, Tallon Y, Chays A, Martin-Bouyer P, et al. Three-dimensional MRI of hemifacial spasm with surgical correlation. Neuroradiology 1997;39(1):46—51.
[11] Kress B, Griesbeck F, Stippich C, Bähren W, Sartor K. Bell palsy: quantitative analysis of MR imaging data as a method of predicting outcome. Radiology 2004;230(2):504—9.
[12] Makeham TP, Croxson GR, Coulson S. Infective causes of facial nerve paralysis. Otol Neurotol 2007;28(1):100—3.
[13] Zayas JO, Feliciano YZ, Hadley CR, Gomez AA, Vidal JA. Temporal bone trauma and the role of multidetector CT in the emergency department. Radiographics 2011;31(6):1741—55.
[14] Wiggins 3rd RH, Harnsberger HR, Salzman KL, Shelton C, Kertesz TR, Glastonbury CM. The many faces of facial nerve schwannoma. AJNR Am J Neuroradiol 2006;27(3):694—9.
[15] Lo WW, Shelton C, Waluch V, Solti-Bohman LG, Carberry JN, Brackmann DE, et al. Intratemporal vascular tumors: detection with CT and MR imaging. Radiology 1989;171(2):445—8.
[16] Veillon F, Ramos-Taboada L, Abu-Eid M, Charpiot A, Riehm S. Imaging of facial nerve. Eur J Radiol 2010;74(2):341—8.
[17] Chang PC, Fischbein NJ, McCalmont TH, Kashani-Sabet M, Zettersten EM, Liu AY, et al. Perineural spread of malignant melanoma of the head and neck: clinical and imaging features. AJNR Am J Neuroradiol 2004;25(1):5—11.