Imaging Anatomy and Pathology of the Intracranial and Intratemporal Facial Nerve

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KEYWORDS

• Facial nerve • Facial nerve palsy • Bell's palsy • Hemifacial spasm • Facial nerve canal

KEY POINTS

- The facial nerve has a complex course from the brainstem to the extracranial soft tissues, with a tortuous and long intratemporal segment.
- A variety of congenital, inflammatory, vascular, and neoplastic processes may affect each segment.
- Knowledge of the complex anatomy of the facial nerve is essential to localize the site of the disorder.
- Computed tomography (CT) and magnetic resonance (MR) studies are complementary but MR imaging is the modality of choice for most segments, whereas CT is reserved to assess the petrous facial nerve canal.

INTRODUCTION

The facial nerve is one of the most complex cranial nerves (CNs) and is responsible for facial expression, lacrimation, salivation, and taste. It is also one of the most commonly paralyzed nerves in the body and its palsy gets early attention because of its role in facial expression. Facial neuropathy has numerous and varied causes, including congenital, traumatic, vascular, inflammatory, and neoplastic processes. Many of those disorders can now be identified on imaging with the increased resolution of imaging studies and new magnetic resonance (MR) sequences. In addition, there are congenital anomalies and variations that may or may not be symptomatic but play a crucial role in the setting of surgical planning to avoid complications.

Appropriate imaging of the facial nerve requires detailed knowledge of its anatomy and recognition of the imaging features of the wide spectrum of pathologic processes that may affect this nerve.

NORMAL ANATOMY

The CN VII, also known as the facial nerve, is one of the most complex CNs with motor, sensory, and parasympathetic fibers (**Fig. 1**).[1,2]

The nerve is composed of a large motor root, containing motor fibers, and a smaller somatosensory root (containing sensory and parasympathetic fibers) called the nervus intermedius (NI).[1,3] The facial nerve has several different parts with multiple connections, but the overall course can be simplified and summarized as in **Fig. 2**.[4]

Intra-axial Segment

The fibers arising from 3 different nuclei, located in the pontine tegmentum, form the motor root and the NI (**Fig. 3**, **Table 1**).[1,5–8] The facial nerve fibers exit the pontomedullary sulcus at the root exit point (RExP) but they remain strongly adherent to the surface of the pons (as the attached segment), before separating from the brainstem. After this

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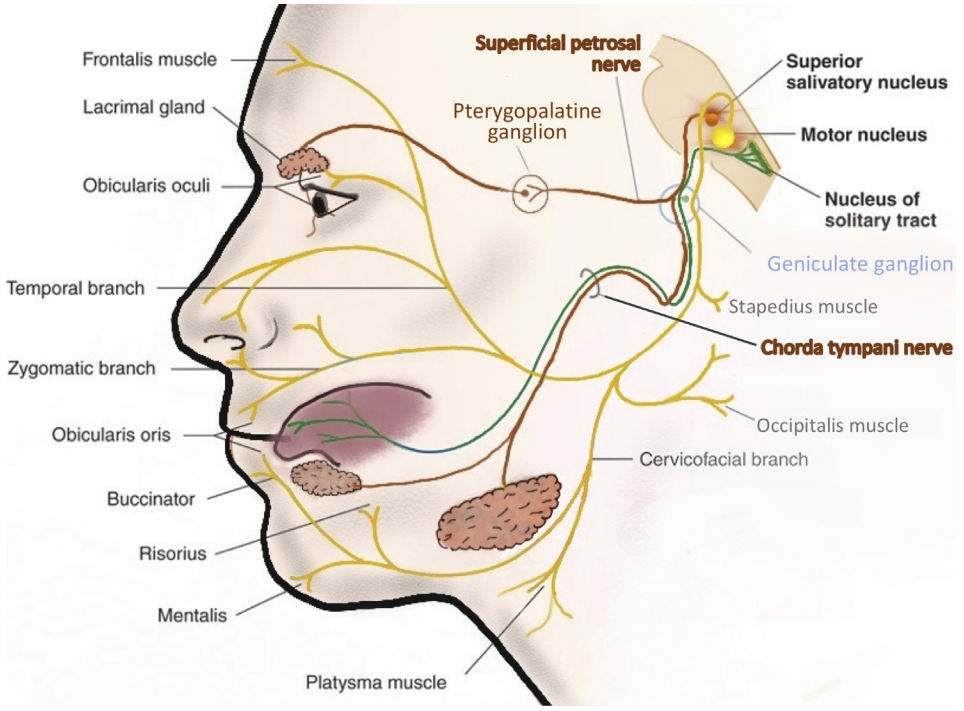

Fig. 1. The facial nerve components and its terminal branches.

root detachment point, a transition zone (TZ) is seen (where oligodendrocyte-derived central myelin is replaced by the peripheral Schwann cell–derived myelin). All the segments from the RExP to the TZ form the root exit zone (REZ) of the facial nerve.[9]

Cisternal Segment

After the REZ, the motor root (facial nerve proper) and the NI course through the cerebellopontine angle (CPA), toward the porus of the internal auditory canal (IAC) (see Fig. 3; Fig. 4). The NI (thinner than the motor part because it has fewer fibers)

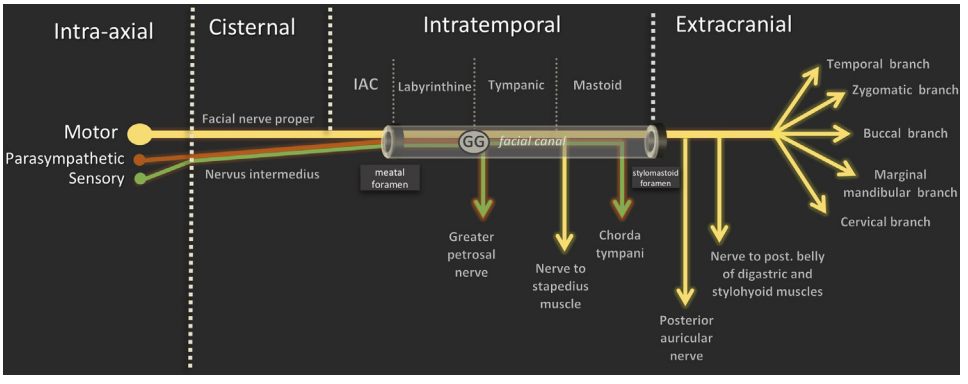

Fig. 2. Facial nerve segments. The nerve originates from the brainstem nuclei (intra-axial segment), traverses the cerebellopontine angle (cisternal segment) to enter the temporal bone (intratemporal segment). Within the temporal bone, it courses within the internal auditory canal (IAC) (canalicular/meatal segment), and then enters the facial/fallopian canal where it has a very convoluted, Z-shaped course (consisting of labyrinthine, geniculate, tympanic, and mastoid segments). It then exits through the stylomastoid foramen into the extracranial soft tissues (extracranial segment). GG, geniculate ganglion. (*Adapted from* Chung R, Dorros S, Mafee MF. Imaging of facial nerve pathology. *Operative Techniques in Otolaryngology-Head and Neck Surgery.* 2014;25(1):58-65; with permission.)

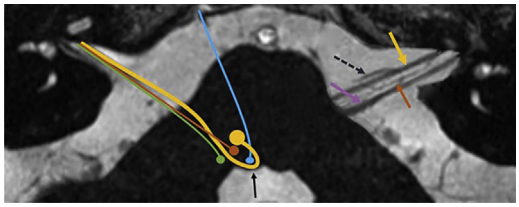

Fig. 3. The facial nuclei and components of the facial nerve in the cisternal and canalicular segments. The estimated location of the motor nucleus (*yellow*), superior salivatory nucleus (*orange*), and nucleus tractus solitarius (*green*) on the axial DRIVE (driven equilibrium radio frequency reset pulse) sequence. The facial nerve proper (*yellow arrow*) and the NI (*orange arrow*) are seen anterior to the cochleovestibular nerve (*purple arrow*). The motor fibers of facial nerve loop around the nucleus of the abducens nerve (*blue*), producing a small bulge at the floor of the fourth ventricle known as the facial colliculus (*black arrow*). An anterior and inferior cerebellar artery loop (*dashed arrow*) is seen extending into the left IAC. (*Adapted from* Harnsberger, H. R., Hudgins, P. A., Koch, B. L., Hamilton, B. E. (2016). Diagnostic Imaging: Head and Neck. United States: Elsevier Health Sciences; with permission from Elsevier.)

can be identified as it courses between the facial nerve proper and CN VIII (see **Fig. 3**).[8] The anterior and inferior cerebellar artery (AICA) usually loops more or less deeply in the CPA and can extend into the IAC (see **Fig. 3**).

Intratemporal Segment

The different segments of the intratemporal facial nerve are summarized in **Table 2**.[10]

Canalicular segment

This is the segment of the nerve within the IAC (see **Fig. 4**).[4] At the fundus of the IAC, the facial nerve enters a bony canal called the facial canal/fallopian canal. The zone of transition between the IAC and the facial canal is the narrowest part of the bony facial canal.[11] Furthermore, at this location the nerve is only covered by pial and arachnoid membranes because the dural lining terminates at the fundus of the IAC.[3] This focal narrowing and thin covering is the reason the meatal segment is the most common site of facial nerve injury when the nerve is swollen from an inflammation such as Bell's palsy (BP) or Ramsay Hunt syndrome.[3]

Labyrinthine segment

This first segment of the facial canal is the shortest and the narrowest segment of the canal (**Fig. 5**A, D).

Geniculate ganglion

The term geniculate (from the Latin meaning knee-like) refers to the abrupt posterior turn of the facial canal (**Fig. 5**B).[4] The upper bony covering of the geniculate fossa can be dehiscent in up to 15% of temporal bones, which makes the facial nerve vulnerable to injury during anterior epitympanic recess or middle cranial fossa surgeries.[3,5]

Tympanic segment

This segment descends obliquely along the medial wall of the tympanic cavity (**Fig. 5**B, D). It is located above the oval window and can be easily identified on the coronal images as a small dot, below the lateral semicircular canal (**Fig. 5**E).

Posteriorly the nerve enters the facial recess, lateral to the pyramidal eminence, where it turns

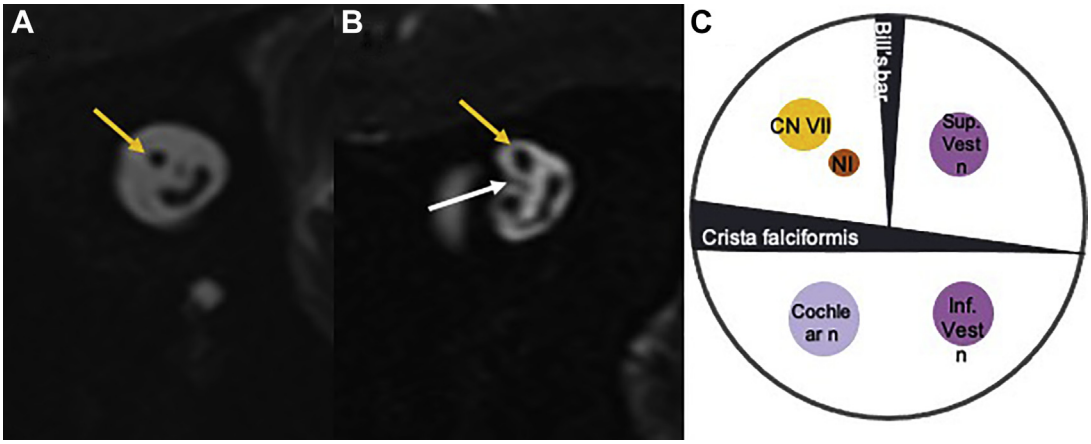

Fig. 4. The facial nerve (*yellow arrow*) is located at the anterosuperior aspect of the canal as demonstrated on the sagittal oblique DRIVE sequence (*A* and *B*) and on the diagram illustrating the IAC (*C*). Crista falciformis (*white arrow*) separates the facial nerve from inferiorly located cochlear nerve and a vertical bone crest, the Bill bar, separates it from the superior vestibular nerve, located posteriorly. n, nerve; Sup. Vest. N., superior vestibular nerve; Inf. Vest. N, inferior vestibular nerve.

Table 1
Facial nerve nuclei: locations and innervation

	Location	Nerve Fiber Type	Innervated Structures/Functions	Exit From Brainstem
Main motor nucleus	Ventrolateral pontine tegmentum, within the reticular formation	Motor fibers	• Muscles of facial expression • Stylohyoid muscle • Posterior belly of the digastric muscle • Stapedius muscle	Inferior lateral border of the pons, medial to CN VIII
Superior salivatory nucleus/lacrimal nucleus	Pontine tegmentum, posterolateral to motor nucleus	Parasympathetic secretory fibers	• Lacrimal glands • Submandibular glands • Sublingual glands	Nerve fibers join to form the nucleus intermedius, which exits the pontomedullary sulcus between the motor root and CN VII
Nucleus of tractus solitarius/ gustatory nucleus	Dorsolateral medulla oblongata and lower pons, lateral to other nuclei	Special afferent sensory fibers	Taste sensation from the anterior two-thirds of the tongue	
		Somatic sensory fibers	Sensation from the pinna and external auditory canal	

inferiorly to form the second genu (**Fig. 5**C) and this part is the most susceptible portion for iatrogenic injury during surgery, especially for cholesteatoma.[10]

Mastoid segment

This segment is the longest but also the largest part of the canal; the nerve filling only half to 25% of the lumen, which is why inflammatory entrapment of the facial nerve is rare in this segment (see **Fig. 5**E).[12]

Extracranial Segment

The facial nerve exits the facial canal via the stylomastoid foramen. It then courses anterolaterally around the lateral aspect of the styloid process, to penetrate the parotid gland, where it gives off its terminal branches, providing motor innervation to the muscles of facial expression.

Vascularization of the Facial Nerve

Three main arteries supply the different segments of the facial nerve:

- The AICA supplies the facial nucleus: the cisternal, intracanalicular, and labyrinthine segments via the labyrinthine artery.
- The superficial petrosal artery (SPA), a branch of the middle meningeal artery.
- The stylomastoid artery (SMA), from the occipital artery in two-thirds of patients or from the postauricular artery.

The SMA anastomoses with the SPA at the level of the second genu and forms an arterial arcade called the facial arch, which supplies the tympanic and mastoid segments of the nerve.[13] The venous drainage parallels the arterial blood supply.[6] The nerve segments distal to the geniculate ganglion (GG) are surrounded by a venous network surrounded by a strong connective tissue sheath. The normal enhancement of the GG and distal nerve segments on postcontrast MR imaging is the result of this circumneural venous plexus.[1]

IMAGING TECHNIQUE AND PROTOCOLS

Computed tomography (CT) and MR imaging represent the primary imaging modalities for evaluating facial nerve disorder (**Table 3**). Although they may provide complementary information, MR imaging is the modality of choice in most circumstances and is preferred to evaluate the intra-axial, cisternal, canalicular, as well as extracranial segments of the facial nerve.[5,11,14,15]

Computed Tomography

CT imaging is usually performed without contrast, and, for facial nerve disorders, contrast-enhanced

Table 2
Intratemporal facial nerve segments

Segments	Location	Course	Branches	Important Imaging Points	Clinically Relevant Points
Canalicular/meatal	From porus to fundus of IAC	Anterior and laterally	—	The nerve is located in the anterosuperior quadrant of the canal	The lateral aspect is the most common site of injury from inflammatory swelling
Labyrinthine	From the meatal foramen to the GG	Anterior and laterally	—	Shortest and the narrowest segment of the facial canal	—
GG	In the geniculate fossa	Acute angled, reverse V-shaped posterior turn	GPSN • Secretory fibers to the lacrimal glands • Sensory innervation to the NC and palate mucosa	In addition to GG, it contains veins and arteries, which results in physiologic contrast enhancement	Weakest zone of canal, most common location for nerve injury in temporal bone fractures
Tympanic/horizontal/ second segment	From GG to the pyramidal eminence at the posterior wall of the middle ear cavity	Posterior and inferiorly	—	Frequently dehiscent	Most common site for congenital anomalies
Mastoid/vertical/third segment	From the posterior genu to the stylomastoid foramen inferiorly	Vertical	Nerve to the stapedius muscle, responsible for the stapedial reflex Chorda tympani nerve • Secretomotor innervation to the SM and SL glands • Sensory innervation to the anterior two-thirds of the tongue	Branching off at the level of second genu Branching off about 5–6 mm above the stylomastoid foramen	This segment is a frequent site of iatrogenic injury during posterior tympanotomy and mastoidectomy

Abbreviations: GG, geniculate ganglion; GPSN, greater superficial petrosal nerve; NC, nasal cavity; SM, submandibular; SL, sublingual.

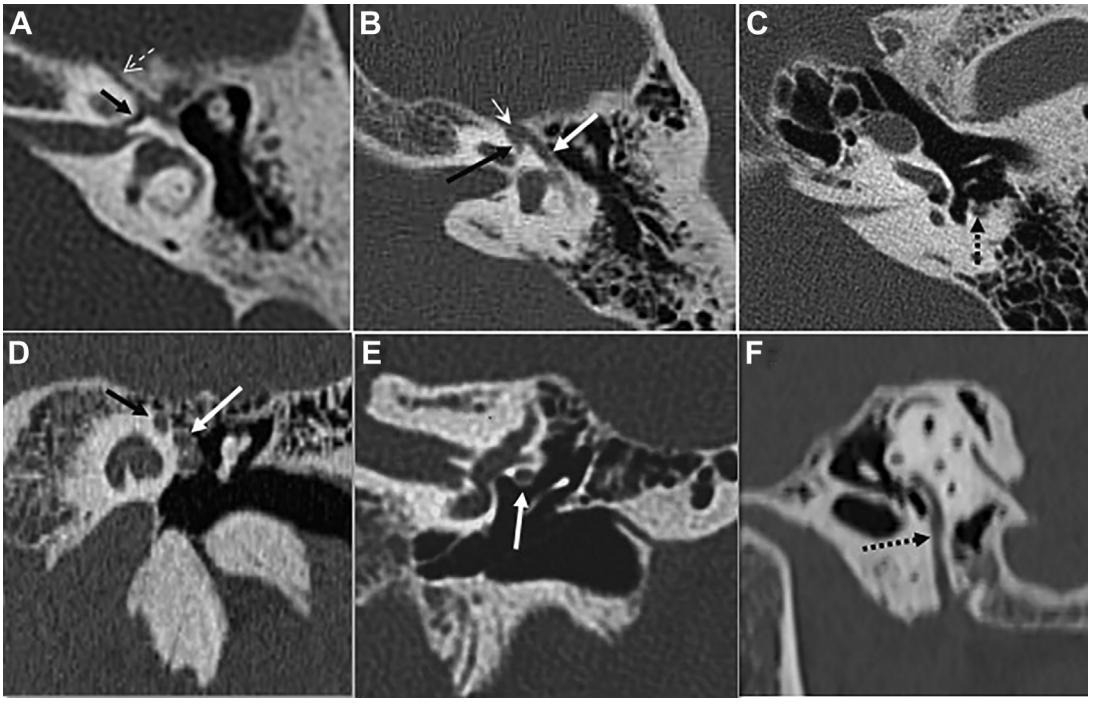

Fig. 5. Segments of the facial canal seen on axial (*A–C*), coronal (*D*, *E*), and Poschl (*F*) reformatted CT images. The labyrinthine segment (*black arrows*), the tympanic segment (*white arrows*), and the mastoid segments (*dotted arrow*) with the GG (*short open arrow*). Note the greater superficial petrosal nerve (GSPN) exiting anteriorly from the GG (*dashed arrow in A*).

CT should be reserved for patients who have contraindications for MR.[5] Although multidetector CT is used worldwide, cone-beam CT using flat-panel detector technology is slowly taking over for detailed evaluation of the temporal bone structures.

Magnetic Resonance Imaging

Tables 4 and **5** summarize the recommended protocol and sequences for facial nerve imaging.[2,16,17] There is building evidence that three-dimensional (3D) fluid-attenuated inversion recovery (FLAIR) MR imaging, especially after contrast, can reveal subtle changes in facial nerve disorders and should replace the standard axial contrast-enhanced T1-weighted sequences.[18,19] Similarly, balanced steady-state free precession (b-SSFP) sequences such as fast imaging employing steady-state acquisition–constructive interference (FIESTA-C) acquired after contrast can provide additional information in subtle cases.[20]

PATHOLOGY

Developmental Lesions and Variants

Aplasia/hypoplasia

Congenital facial paralysis may be seen in several syndromes, including Mobius, CHARGE (coloboma, heart defects, atresia choanae, growth retardation, genital abnormalities, and ear abnormalities), Goldenhar, and also in brain malformations such as pontine tegmental cap dysplasia.[21]

Mobius sequence/syndrome is characterized by congenital, often bilateral, CN VII and CN VI palsies. The underlying CN VII and VI nuclei aplasia result in absence of the facial colliculus with flattening of the floor of the fourth ventricle and nonvisualization of the ipsilateral abducens and facial nerves (**Fig. 6**).[22,23]

Unilateral congenital facial nerve hypoplasia may also occur as an isolated abnormality or accompanied by inner ear anomalies.[23,24] It can be diagnosed with the imaging findings of facial canal hypoplasia on CT and small caliber of the nerve on MR (**Fig. 7**).[25]

Duplication

Duplication of the facial nerve, also called bifid facial nerve, can occur in isolation or with other ear anomalies.[23,26] Tympanic segment is the most common site, followed by the mastoid segment.[26]

Aberrant course

Abnormal course of the facial nerve can be seen in isolation but is usually seen in 3 conditions:

Table 3
Imaging techniques and suggested clinical indications with their corresponding imaging recommendations

Imaging Modality	Indications	Study Selection	Advantages	Disadvantages	
CT	• Initial evaluation after trauma • Presurgical assessment of osseous anatomy	MDCT	Widely available	Higher radiation dose compared with CBCT	HR bone algorithm with st $\leq$0.6 mm. MPR in the axial, coronal, Poschl and Stenvers planes
—	• Assessment of bone erosion in inflammation and neoplasia	CBCT	Higher resolution of small or thin structures, such as lateral wall of tympanic segment, with a lower dose	• Longer acquisition • Increased motion artifact • Requires GA in children	—
MR imaging	Nontraumatic facial palsy	Dedicated MR imaging of the temporal bones with contrast	Ability to evaluate the nerve and the brainstem in the setting of inflammation, neoplasia, or vascular lesion	• Longer acquisition • Prone to artifact	—

Abbreviations: CBCT, cone-beam CT; GA, general anesthesia; HR, high-resolution; MDCT, multidetector CT; MPR, multiplanar reconstruction; st, slice thickness.

Table 4
Sequences used for facial imaging and the corresponding abbreviations

Fluid-attenuated inversion recovery	FLAIR
Balanced steady-state free precession	b-SSFP
Fast spin echo	FSE
Constructive interference into steady state	CISS
Fast imaging employing steady-state acquisition	FIESTA
Fast imaging employing steady-state acquisition–constructive interference	FIESTA-C
Driven equilibrium radiofrequency reset pulse	DRIVE
Sampling perfection with application optimized contrasts using different flip angle evolution	SPACE
Spoiled gradient recalled acquisition in the steady state	SPGR
Volumetric interpolated breath-hold examination	VIBE
Magnetization prepared rapid gradient echo	MPRAGE
Brain volume imaging	BRAVO

- Congenital aural dysplasia: the tympanic and mastoid segments are most commonly affected.[21,23]
- Congenital middle ear and ossicular anomalies (because of common origin of the ossicles and facial nerve from second branchial arch). There is usually inferomedial displacement of the tympanic segment (**Fig. 8**A, B).[27]
- Congenital inner ear anomalies (IEAs).[28,29] The direction of abnormal displacement depends on the severity and type of underlying IEA.[29] IP-III results in superior dislocation of the labyrinthine segment, whereas cochlear aplasia/hypoplasia and common cavity anomaly usually result in anterior displacement of labyrinthine segment and inferior dislocation of the tympanic segment.

On coronal reformatted CT images, the absence of the dot beneath the lateral semicircular canal should raise suspicion of the abnormal course of the facial nerve (see **Fig. 8**A, B). Preoperative identification of such an anomalous course is crucial to surgeons and can help avoid an inadvertent iatrogenic facial nerve paralysis.[23]

Dehiscence

The facial canal dehiscence has been reported in more than half the cases histopathologically,

Table 5
Recommended magnetic resonance imaging sequences and parameters for facial neuropathy and for hemifacial spasm

Sequence	ST (mm/Gap)	FOV (cm)	Imaging Details
Limited Brain			
Sagittal T1	5/1	23–24	Entire brain
Axial FLAIR	5/1	22	Entire brain
Axial T2 brain stem	3/1	22	Include parotids
DWI	5/1	22	—
High-resolution IAC			
Axial 3D T2 b-SSFP or 3D FSE (CISS/FIESTA/DRIVE/SPACE…)	0.6	16–18	—
Precontrast and postcontrast T1WI	—	—	—
Axial 3D T1 (T1-SPACE/3D-SPGR/VIBE)	1	20–22	Preferred
Axial and coronal T1 TSE	2	16–18	≤2 mm, if 3D unavailable
Add-ons			
Postcontrast axial 3D FLAIR	1.2	22	In suspected inflammation
3D TOF MRA	—	—	For hemifacial spasm

Abbreviations: 3D, three-dimensional; DWI, diffusion-weighted imaging; FLAIR, fluid-attenuated inversion recovery; FOV, field of view; MRA, magnetic resonance angiography; TOF, time-of-flight; TSE, turbo spin echo.

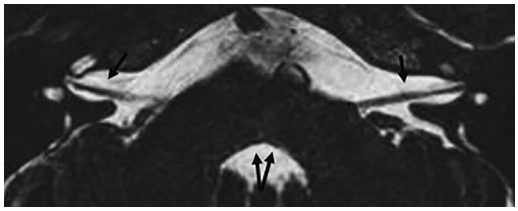

Fig. 6. Mobius sequence. Axial constructive interference into steady state (CISS) image performed on a 10-year-old boy with facial paralysis and abnormal gaze since birth, shows bilateral absence of the facial colliculi (*double arrows*) and flattening of the fourth ventricle with absence of the bilateral CN VII (*black arrows*) and CN VI. (*Courtesy of* K. K.Oguz, MD, Ankara, Turkey.)

most common in the tympanic portion, mastoid segment, and near the GG (**Fig. 8**C, D).[30] At the tympanic segment, the facial nerve may prolapse inferiorly from the dehiscence and lie in close contact with the stapes. Again, because of the embryonic association of facial nerve with other second arch derivatives, a dehiscence of the facial canal can be associated with additional middle ear or ossicular anomalies.[1,23] Although most patients with dehiscence are asymptomatic, conductive hearing loss may be the presenting symptom. In addition, a dehiscence may predispose the nerve to injury during a middle ear surgery.

Epineurial pseudocysts

An epineurial pseudocyst of the intratemporal facial nerve is a developmental lesion, located

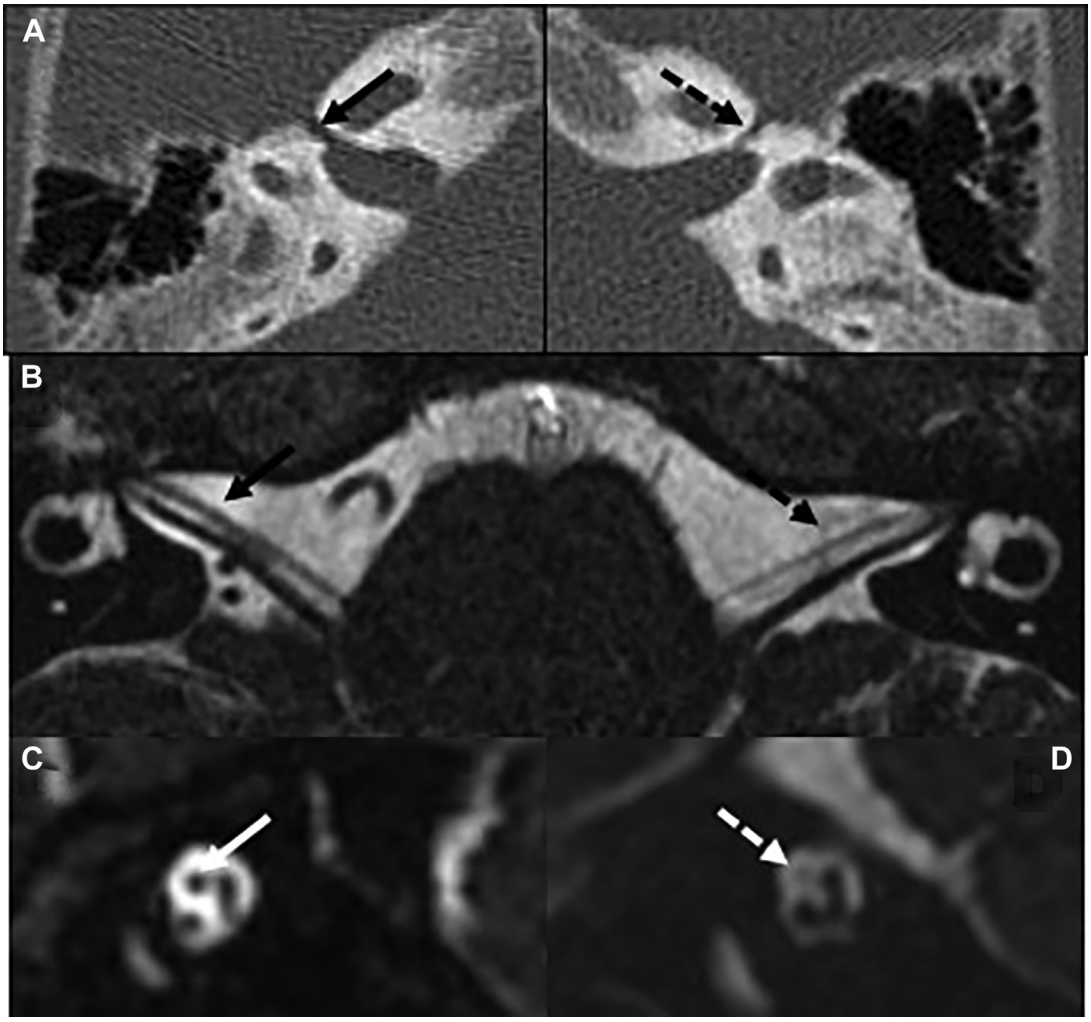

Fig. 7. Facial canal and facial nerve hypoplasia. Axial CT (*A*), axial (*B*) and bilateral sagittal oblique CISS from right (*C*) and left (*D*) IACs, showing left facial canal and facial nerve hypoplasia (*dotted arrows*) compared with the normal-sized canal and nerve (*straight arrows*) on the right.

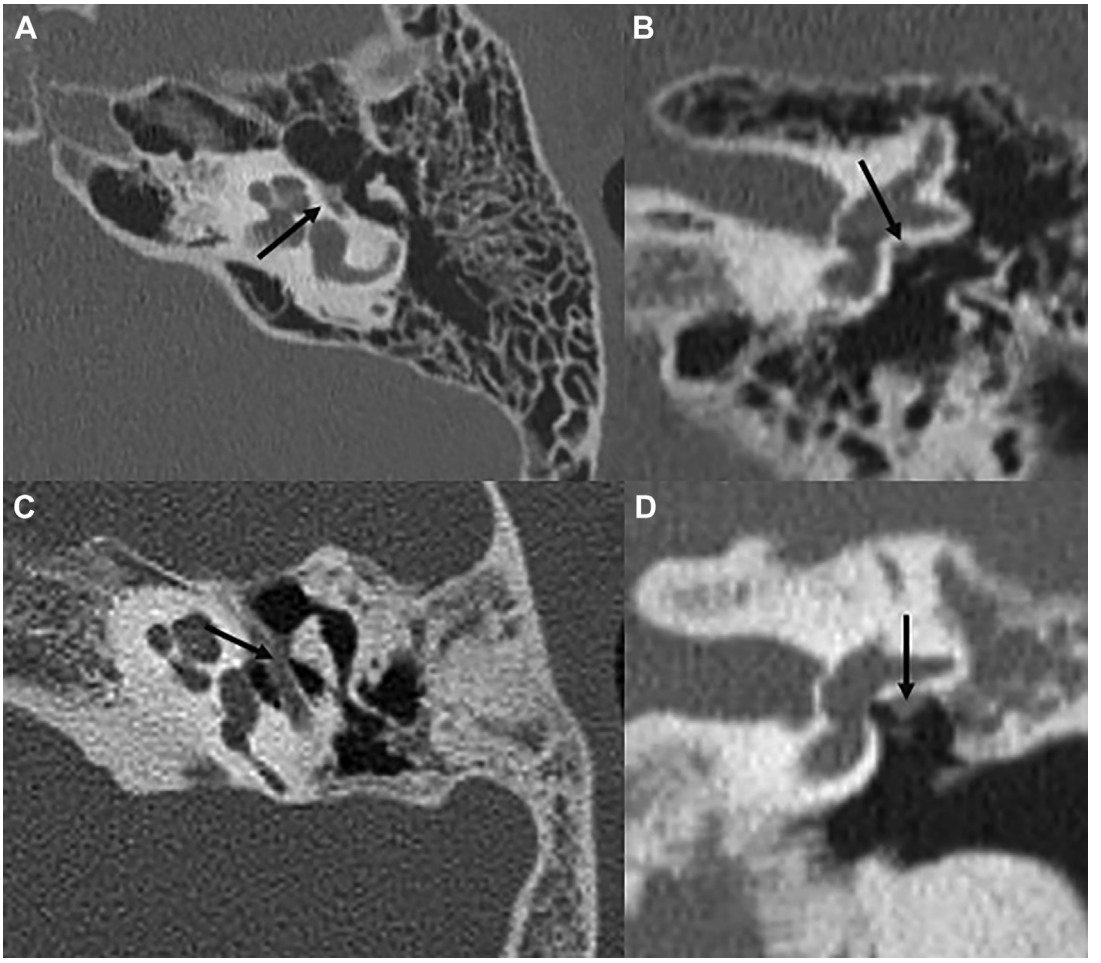

Fig. 8. Axial (*A*) and coronal (*B*) reformatted images of temporal bone CT of a 8-year-old boy showing aberrant course of the left facial nerve (*arrow*) with inferomedial displacement of the tympanic facial canal and accompanying oval window atresia (*A* and *B*). Axial (*C*) and coronal (*D*) reformatted images of temporal bone CT scans in another adult patient showing dehiscent facial canal (*arrow*).

adjacent to the mastoid segment of the facial nerve (Fig. 9). The lesions consist of dense fibroadipose tissue without a true wall and are probably an incidental imaging finding.[31]

Inflammatory/Infectious Disorders

Studies have shown that infection/inflammation accounts for more than 70% of cases presenting with facial palsy.[11]

Bell palsy

BP, also called idiopathic facial palsy, is the most common cause of facial paralysis.[32,33] It is characterized by a rapid unilateral facial nerve paresis or paralysis of unknown cause. Although a viral cause (herpes simplex virus reactivation) is suspected, the exact mechanism of BP is currently unknown.[34] The BP is thought to result from

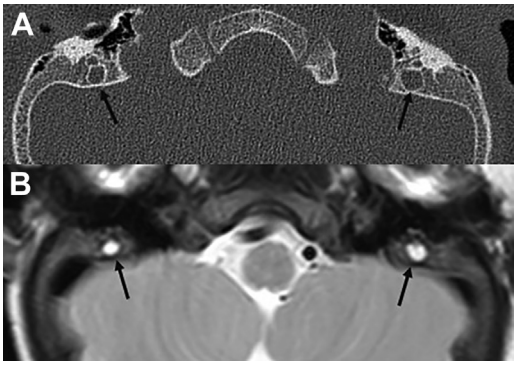

Fig. 9. Epineurial pseudocysts seen as bilateral cystic lesions (*arrows*) along the course of the mastoid facial segment on axial CT (*A*) and T2 turbo spin-echo MR image (*B*).

Box 1
Imaging indications in Bell palsy

- Slow progression of the palsy
- Spasm preceding palsy
- Second paralysis on the same side
- Paralysis of isolated branches of the facial nerve
- Paralysis associated with other CN involvement
- Worsening or persistent paralysis after 3 months

inflammatory edema of the facial nerve, with resultant compression of the vascular supply within the narrow facial canal.[34] The narrowest regions of the canal, the labyrinthine segment and especially the meatal foramen, are thus the most susceptible areas to injury from ischemia. Furthermore, those areas constitute a watershed zone between the vertebral and carotid artery systems, which makes them susceptible to ischemic injury.[11] The disease is usually self-limiting, with improving weakness in all cases, and complete recovery in up to 80% of patients at 6 months. The diagnosis of BP is one of exclusion, where history and physical examination are crucial in making the diagnosis. The latest guidelines do not consider imaging to be contributive in BP, unless the symptoms are atypical (**Box 1**).[32]

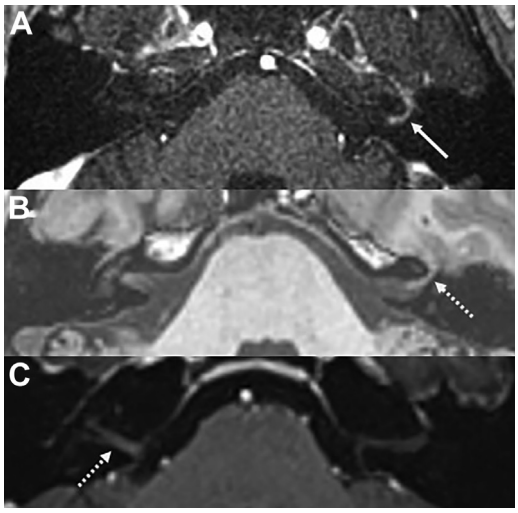

Fig. 10. BP in a 49-year-old woman presenting with left-sided facial paralysis that had started 6 weeks ago, seen as abnormal enhancement along the labyrinthine segment (*arrow*) on postcontrast 3D T1-weighted imaging (T1WI) (*A*). On different patients without any facial symptoms, increased signal along the normal nerve (*dotted arrows*) on precontrast brain volume imaging (BRAVO) sequence (*B*) and on postcontrast magnetization prepared rapid gradient echo (MPRAGE) sequence (*C*).

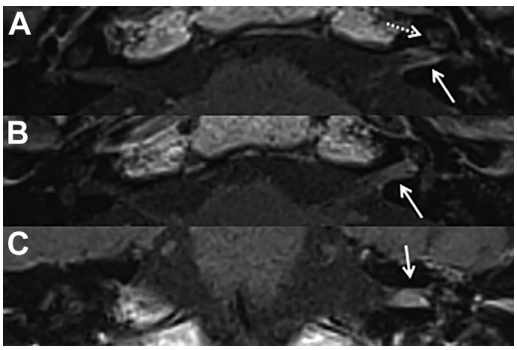

Fig. 11. Ramsey-Hunt disease. Axial (*A* and *B*) and coronal (*C*) postcontrast T1WI images of a 34-year-old woman presenting with 1-month history of left facial paralysis and vertigo. There is abnormal thickening and enhancement involving both the facial and vestibulocochlear nerves (*straight arrows*) with subtle enhancement of the cochlea (*dashed arrow*). The patient had history of vesicular rash in her pinna at the onset, confirming the diagnosis.

When imaging is needed, dedicated contrast-enhanced MR imaging of the temporal bone should be performed, covering the entire course of the facial nerve, including the parotid glands, to rule out other conditions that could present with facial paralysis.

The characteristic imaging finding of BP is linear intense contrast enhancement along the distal canalicular and labyrinthine segments of the nerve (**Fig. 10**A). Although any enhancement in the canalicular segment is considered abnormal for routine turbo spin-echo (TSE) imaging, faint focal enhancement can occasionally be seen at the IAC fundus in normal individuals, on 3D sequences such as spoiled gradient recalled acquisition in the steady state (SPGR) and fast spin-echo (FSE).[35,36] Furthermore, physiologic enhancement is usually seen in the geniculate, tympanic, and mastoid segments caused by previously described vascular plexus. In addition, compared with routine T1 spin-echo sequences, increased signal can be observed both before and after contrast and in all facial nerve segments on 3D inversion recovery–prepared fast spoiled gradient echo sequences (such as magnetization prepared rapid gradient echo [MPRAGE] and brain volume imaging [BRAVO]) (**Fig. 10**B, C).[37] Determination of pathologic facial nerve enhancement on these sequences must thus be made with caution, because the apparent enhancement may instead

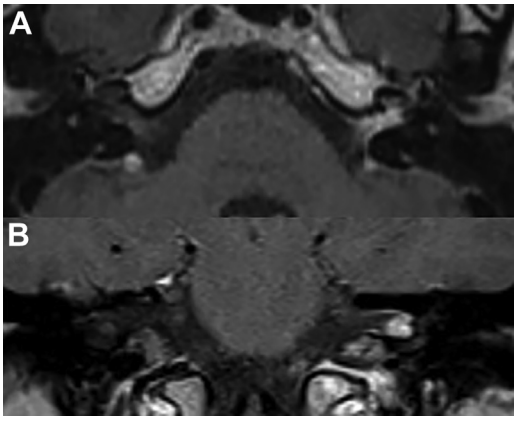

Fig. 12. Neurosarcoidosis patient with contrast enhancement at the fundus of IAC on axial (*A*) and coronal (*B*) postcontrast BRAVO.

reflect the increased visualization of the nerve on this sequence.[37] The postcontrast 3D-FLAIR images can improve the specificity and overall accuracy of MR imaging in patients with BP.[19]

Herpes zoster oticus

Herpes zoster oticus (HZO), also called Ramsay Hunt syndrome, is the second most common inflammation of the facial nerve and results from reactivation of latent varicella zoster virus in the GG.[6,11,38] It is characterized by acute facial palsy and facial pain, accompanied by CN VIII symptoms (including sensorineural hearing loss and vertigo) and also by vesicular rash in the periauricular region and external auditory canal. The characteristic imaging findings are thickening and enhancement along the involved nerves, CN VII and CN VII, a distinguishing feature from BP (**Fig. 11**).[18,39,40] The presence of associated increased signal and enhancement of the

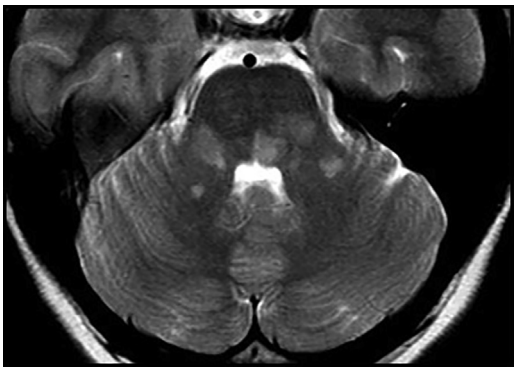

Fig. 13. Multiple sclerosis in 28-year-old woman with multiple demyelinating plaques at the brain stem, also involving the regions of the facial nerve and facial colliculus.

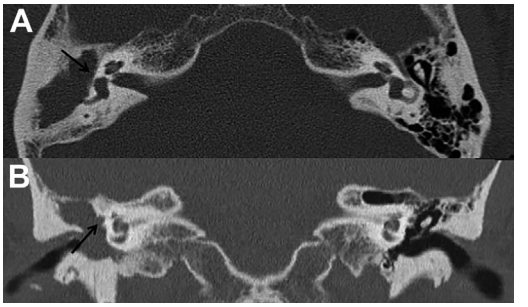

Fig. 14. Large middle ear cholesteatoma with facial canal erosion (*arrows*) on axial (*A*) and coronal (*B*) reformatted images of temporal bone CT.

labyrinth, best seen on the 3D-FLAIR sequences, was also found to be useful in distinguishing HZO from BP.[18,40]

Lyme disease

Lyme disease is caused by the infection by the spirochete *Borrelia burgdorferi* and is a common cause of facial paralysis is children. The central nervous system (CNS) involvement can cause meningitis and cranial neuropathies, facial palsy being the most common.[41] The facial paralysis can be present bilaterally in up to 25% of cases.[41] The imaging findings are nonspecific, with linear enhancement along the facial nerve.[41]

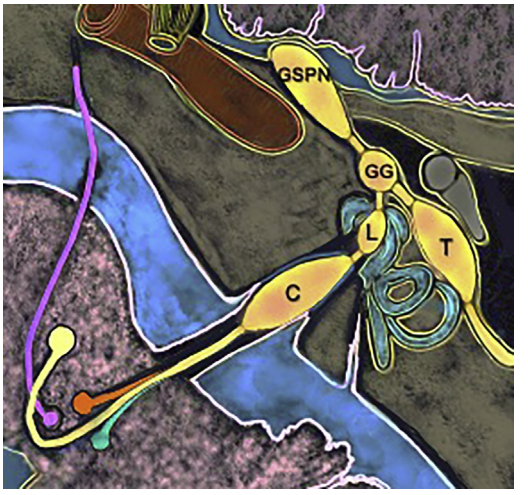

Fig. 15. The most common locations for facial schwannoma: the canalicular segment (C), the labyrinthine segment (L), the GG, the GSPN, and the tympanic segment (T). (*Adapted from* Mundada P, Purohit BS, Kumar TS, Tan TY. Imaging of facial nerve schwannomas: diagnostic pearls and potential pitfalls. *Diagnostic and interventional radiology.* 2016;22(1):40-46.)

Sarcoidosis

Sarcoidosis is a noncaseating granulomatous inflammation that can affect the CNS. Facial nerve palsy is the most commonly seen CN disorder.[42] Neurosarcoidosis manifests as leptomeningeal enhancement, typically involving the basal surfaces of the brain, and enhancing dural masses/diffuse dural thickening. The facial nerve, when involved, shows focal nodular or linear enhancement (**Fig. 12**).[42]

Other inflammatory conditions affecting the facial nerve/nucleus

Demyelinating diseases such as multiple sclerosis may involve the facial nerve nucleus, but the facial palsy is rarely isolated and is rarely the presenting symptom (**Fig. 13**).[11]

Autoimmune disorders such as chronic inflammatory demyelinating polyneuropathy or Sjögren syndrome can also present with facial nerve palsy, and enhancement along the involved nerve can be seen on postcontrast images.[43]

Secondary involvement from acute/chronic middle ear and skull base infections

Acute and chronic infections of the middle ear including cholesteatoma can result in facial palsy.[11] Facial palsy is seen in 1% of cases with cholesteatoma, and the tympanic segment of the nerve is the most frequently affected site (**Fig. 14**).[44] Similarly, facial palsy is seen in more than one-third of patients with skull base osteomyelitis and is also a sign of poor prognosis.[11,45]

Tumoral Lesions

Several benign and malignant tumors can occur along the facial nerve but the primary tumors of CN VII are rare and the nerve is more frequently affected by lesions arising in the adjacent structures (such as vestibular schwannoma) or is secondarily involved by metastatic disease and perineural tumor spread.

Facial schwannoma

Facial nerve schwannoma (FNS) is a rare benign neoplasm but it represents the most common

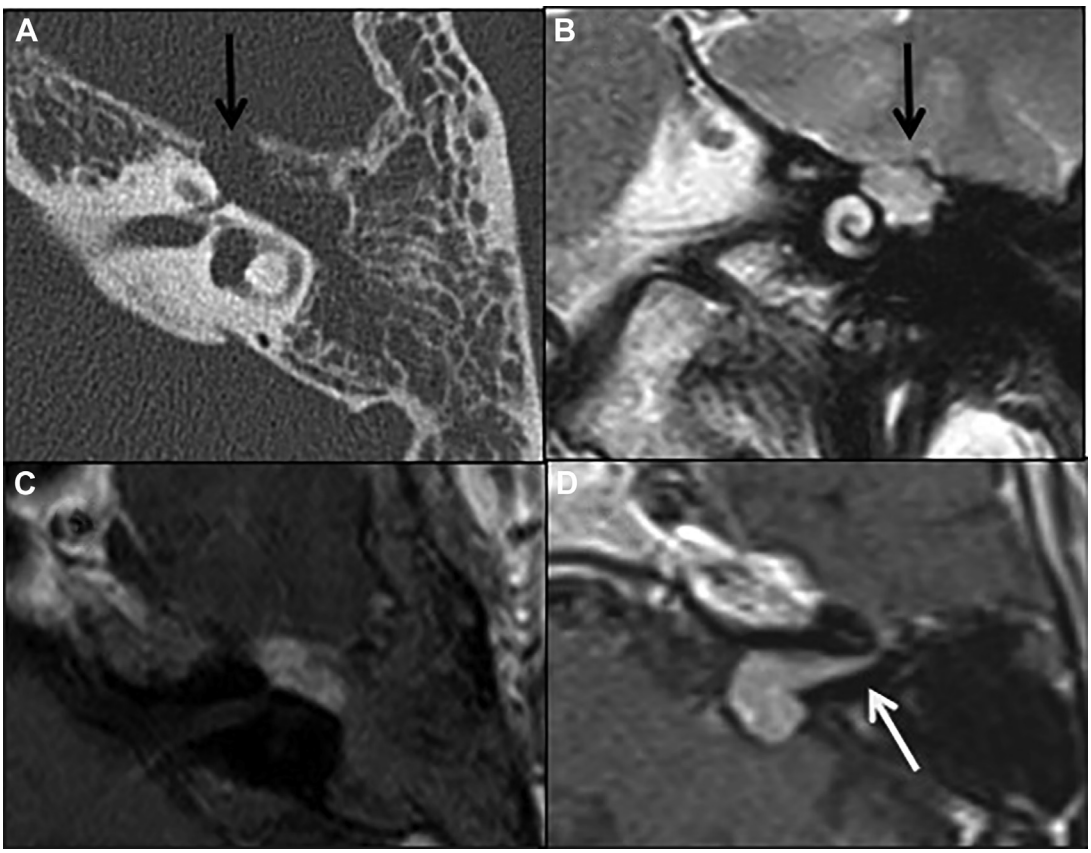

Fig. 16. Facial schwannoma (*arrow*) with widening of the GG and extension into the middle ear (*A*). The coronal T2-weighted imaging (WI) (*B*) and the axial postcontrast T1WI (*C*) show expansile, homogeneous, and avidly enhancing lesion centered at the GG. In another patient (*D*), an IAC mass extending to the facial canal (*arrow*), suggestive of a facial schwannoma.

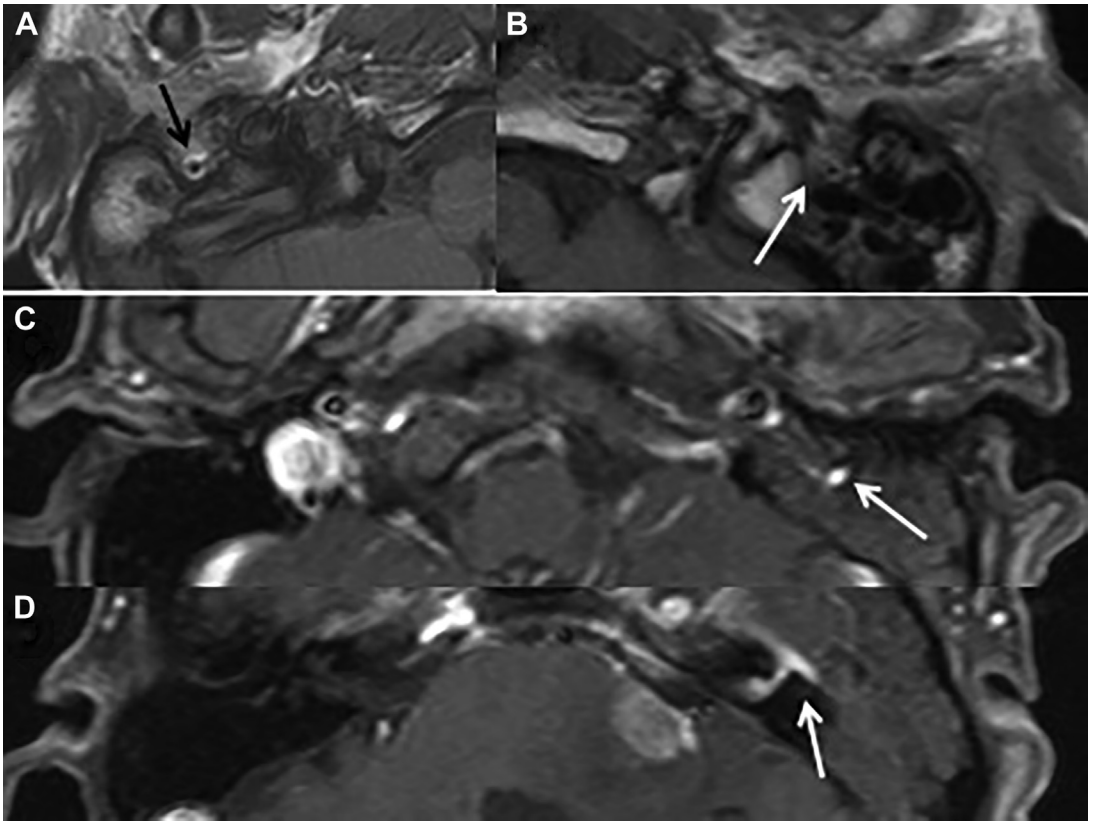

Fig. 17. Perineural tumor spread. The normal fat signal surrounding the nerve (*black arrow*) preserved on the right (*A*), decreased (*white arrow*) on the left on T1WI (*B*). Asymmetric increased enhancement (*arrow*) along the mastoid (*C*) tympanic, geniculate, labyrinthine, and cisternal segments (*D*) on the left suggestive of perineural spread.

primary neoplasm of the facial nerve. It can involve any segment, but most lesions are centered at the perigeniculate area (**Fig. 15**).[16,46] Although most cases occur sporadically, it can also be seen in patients with neurofibromatosis type 2 or after radiotherapy.[47] Facial palsy is the most frequent symptom but is not always present (because the motor nerves have thicker myelin sheaths and may be more resistant to slowly increasing compression) and the patients may present from compressive symptoms to the adjacent cochlear nerve.[2,47] FNSs are typically small fusiform tumors that grow along the path of least resistance and can involve multiple contiguous segments of the nerve.[16,46] On CT, the lesions typically cause smooth expansion of the canal, but they can protrude into the middle ear cavity (**Fig. 16**A). On MR imaging, they are usually homogeneous, T2-hyperintense, diffusely enhancing lobular lesions, although cystic degeneration may result in heterogeneous enhancement (**Fig. 16**B, C).[16] At the CPA or IAC, FNS cannot be differentiated from a vestibular schwannoma unless the tumor extends to the labyrinthine segment (**Fig. 16**D). At the GG, the lesion can enlarge superiorly and present as an extra-axial middle cranial fossa mass.

Other benign lesions affecting the facial nerve

Primary meningiomas of the facial nerve canal are very rare (they are mostly centered at the GG), and the facial nerve is mostly affected by meningiomas originating from the CPA/IAC and middle cranial fossa regions.[2,5,11]

Perineural spread of tumor

Several head and neck malignancies can involve the facial nerve as perineural tumor spread (PNTS).[48] The most common primaries are parotid gland malignancies (adenoid cystic and mucoepidermoid carcinomas), but external auditory canal squamous cell carcinoma, melanoma, and lymphoma can also spread through the nerve. The PNTS might cause gradual facial palsy but is often asymptomatic and can only be detected on

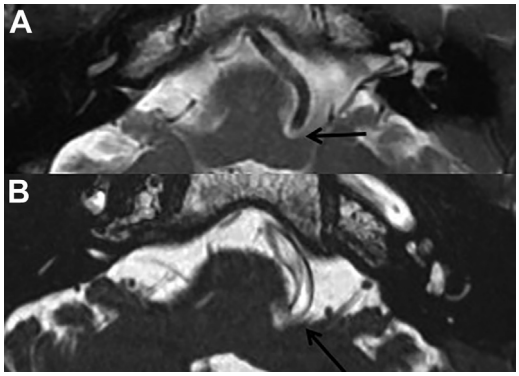

Fig. 18. In a 45-year-old male patient presenting with hemifacial spasm, the T2WI (*A*) and the FIESTA sequence (*B*) show a tortuous vertebrobasilar artery causing compression of the facial nerve at REZ (*arrows*), with indentation in the ventrolateral aspect of the brainstem.

imaging. The tumor spread typically occurs centripetally, from the extracranial nerve or greater superficial petrosal nerve toward the brain stem, but so-called skip lesions are common. Furthermore, the spread may also extend in the reverse direction from a branching point of the main nerve trunk. PNTS is seen as asymmetric and thickened enhancement along the involved nerve and can even be seen as enhancement extending all the way to the brain stem (**Fig. 17**).[49] Obliteration of the fat signal at the level of the stylomastoid foramen on T1-weighted imaging (T1WI) is an important finding suggestive of PNTS (see **Fig. 17**A, B).

Metastases to the surrounding bones as well as leptomeningeal metastases along the cisternal and canalicular segments of the nerve can also result in facial palsy. In adult patients, the most common primaries are breast and lung.[5] In addition, lymphoma and leukemia can also cause CN infiltration that usually involves multiple CNs.

Vascular Lesions

Hemifacial spasm

Hemifacial spasm is a complex symptom that consists of involuntary, painless spasms of the facial muscles, most commonly seen in middle-aged women.[9] The spasm can be secondary to the facial nerve/nucleus irritation (from tumors, demyelinating disorders, trauma, and infections) but is most frequently primary and caused by a neurovascular conflict, from a vascular structure that causes facial nerve compression.[50,51] The previously described REZ, especially the centrally myelinated axons from the RExP to TZ, are the most vulnerable for focal injury and demyelination from repeated vascular pulsations.[9] The most commonly implicated vessels are AICA, PICA, and vertebral artery (**Fig. 18**).[9,50] However, it is important to remember that numerous vessels course in the CPA and cross the facial nerve but most are not symptomatic; therefore, the radiological diagnosis or suggestion should only be used in patients who are symptomatic and who have objective distortion of the facial nerve on imaging.[51]

Venous vascular malformation of the facial nerve

Venous vascular malformation (VVM) of the facial nerve is a vascular lesion that was historically referred to as facial hemangioma or ossifying hemangioma.[52] It is most frequently seen in the GG with extension to the labyrinthine segment (usually presenting with facial nerve symptoms) or at the IAC (presenting with gradually progressive sensorineural hearing loss).[4,53] On MR, VVMs are seen as expansile, heterogenous, and avidly enhancing masses that may be difficult to differentiate from schwannomas (**Fig. 19**). However, the CT findings showing characteristic honeycomb appearance with internal bony spicules and poorly defined

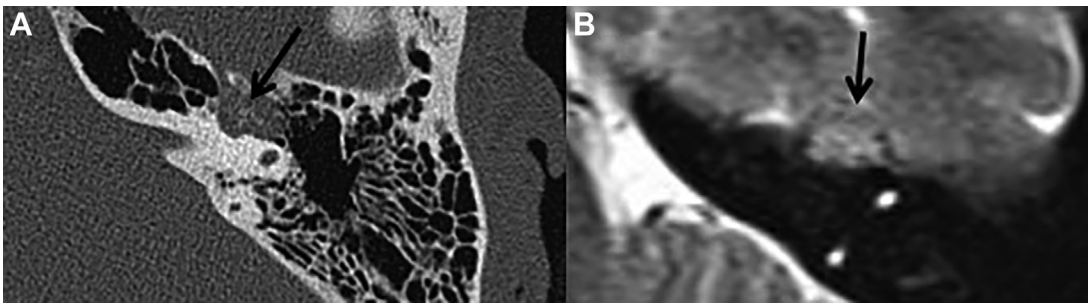

Fig. 19. VVM of the facial nerve seen as expansile mass, centered at the GG, with internal trabecular calcifications (*arrow*) on axial CT (*A*) and heterogenous appearance on T2WI (*B*).

margins allow the differential diagnosis from FNS (see **Fig. 19**).[2,11,15]

The facial nerve can also be involved in temporal trauma and can be secondarily affected by skull base tumors and tumors arising from adjacent structures, but these entities are discussed separately elsewhere in this issue.

SUMMARY

The facial nerve is a complex structure, affected by a wide spectrum of disorders. The appropriate radiological assessment of a patient presenting with facial neuropathy requires a thorough knowledge of its anatomy, of the normal imaging appearance of the nerve/canal both on CT and MR, and characteristic and atypical imaging features of different affecting disorders for an accurate differential diagnosis.

CLINICS CARE POINTS

- Abnormal course of the facial nerve can be seen in isolation but is usually seen in the setting of congenital aural dysplasia, congenital middle ear and ossicular anomalies or with congenital inner ear anomalies. It is thus crucial to assess the course of the nerve in those cases to help surgical planning and prevent iatrogenic injury.
- In routine TSE imaging any enhancement in the canalicular segment of the facial nerve is considered abnormal. However, faint enhancement can occasionally be seen at the IAC fundus in normal individuals, on 3D sequences such as SPGR and FSE. Additionally, increased signal can be observed both before and after contrast and in all facial nerve segments on 3D inversion recovery-prepared fast spoiled gradient-echo sequences (such as MPRAGE and BRAVO).
- Facial nerve might be involved by perineural tumor spread (PNTS) in several head and neck malignancies including parotid gland malignancies, external auditory canal squamous cell carcinoma and melanoma. The PNTS might cause gradual facial palsy but is often asymptomatic and can only be detected on imaging.

DISCLOSURE

The author has nothing to disclose.

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