

Lower Cranial Nerves

Theodoros Soldatos, MD, PhD^a, Kiran Batra, MD^a,
Ari M. Blitz, MD^b, Avneesh Chhabra, MD^{c,*}

KEYWORDS

- Cranial nerves • Imaging assessment • Anatomy • Pathology • Magnetic resonance neurography
- Magnetic resonance imaging

KEY POINTS

- Enhancement of the intracanalicular and/or the labyrinthine portion of the facial nerve is always abnormal.
- Vestibulocochlear nerve schwannomas typically develop as intracanalicular–cisternal masses, whereas meningiomas develop as cisternal masses.
- Simultaneous glossopharyngeal, vagal, and spinal accessory neuropathy indicates a jugular foramen lesion. In the latter setting, paraganglioma constitutes the most common etiology.
- In isolated vagal neuropathy, detailed knowledge of the clinical findings is mandatory to tailor the examination and interpret the imaging findings.
- In hypoglossal neuropathy, the most important magnetic resonance imaging (MRI) feature is unilateral signal intensity denervation changes of the tongue musculature.
- Neurosarcoidosis, subarachnoid hemorrhage, basal meningitis, and viral neuritis may involve multiple lower cranial nerves and be demonstrated on MRI as nerve thickening and contrast enhancement.

INTRODUCTION

Imaging evaluation of cranial neuropathies (CNs) is a challenging task for radiologists, requiring thorough knowledge of the anatomic, physiologic, and pathologic features of the cranial nerves, as well as detailed clinical information, which is necessary for tailoring the examinations, locating the abnormalities, and interpreting the imaging findings. Although computed tomography (CT) provides excellent depiction of the skull base foramina, the nerves themselves can only be visualized in detail on magnetic resonance imaging (MRI).¹ This review provides clinical, anatomic, and radiological information on lower CNs (VII to XII), along with high-resolution magnetic resonance images as a guide for optimal imaging technique, so as to improve the diagnosis of cranial neuropathy.

ANATOMY

CN VII: Facial Nerve

The facial nerve is a complex mixed nerve, consisting of the facial nerve proper (the larger motor component) and the nervus intermedius (the smaller sensory component). The preganglionic fibers originate from the main motor nucleus, which is situated in the midpons. These join fibers from the superior salivatory nucleus, the nucleus solitarius, and the spinal tract of CN V. The facial nerve emerges at the ventrolateral aspect of the caudal pons, crosses the cerebellopontine angle (CPA) cistern, together with the vestibulocochlear nerve, and enters the temporal bone. The intratemporal course and central connections of the facial nerve can be roughly divided into the following segments:

^a The Russell H. Morgan Department of Radiology and Radiological Science, The Johns Hopkins Hospital, 601 North Caroline Street, Baltimore, MD 21287, USA; ^b Division of Neuroradiology, The Russell H. Morgan Department of Radiology and Radiologic Science, The Johns Hopkins Hospital, Phipps B-100, 600 North Wolfe Street, Baltimore, MD 21287, USA; ^c University of Texas Southwestern, 5323 Harry Hines Blvd. Dallas, TX 75390-9178, USA
* Corresponding author.

E-mail address: avneesh.chhabra@utsouthwestern.edu

The *meatal* segment runs within the internal acoustic canal (IAC), occupying the anterosuperior portion of the latter (**Fig. 1**).

Superior to the cochlea, the *labyrinthine segment* connects with the geniculate ganglion and provides the greater superficial petrosal nerve, which participates in the innervation of the lacrimal gland and mucous membranes of the nasal cavity and palate.²

The tympanic (horizontal) segment extends from the geniculate ganglion to the horizontal semicircular canal.

At the latter location, a second genu is formed, marking the beginning of the mastoid portion, which runs inferiorly within the mastoid bone and provides a) the nerve to the stapedius muscle b) the chorda tympani nerve, which provides secretomotor innervation to the submaxillary and sublingual glands, as well as sensory innervation to the anterior two thirds of the tongue c) the auricular branch of the vagus nerve, which participates in the sensory innervation of the posterior auditory canal.

The motor component of CN VII exits the skull through the stylomastoid foramen, and provides the posterior auricular nerve, which innervates the postauricular muscles, and two small branches, which innervate the posterior belly of the digastric muscle and the stylohyoid. Subsequently, the facial nerve penetrates the parotid gland, passes lateral to the retromandibular vein, and courses superficially into the muscles of facial expression.³

CN VIII: Vestibulocochlear Nerve

The vestibulocochlear nerve is a sensory nerve, consisting of a superior vestibular, an inferior

vestibular, and a cochlear component. The fibers originate from vestibular nuclei located in the pons and medulla, and cochlear nuclei situated in the inferior cerebellar peduncles. The nerve emerges in the groove between the pons and the medulla oblongata, just posterior to the facial nerve, and courses together and parallel to the latter within the CPA cistern and the internal acoustic canal. The cochlear component runs in the anteroinferior aspect of the canal, whereas the superior and inferior vestibular components run in the posterosuperior and posteroinferior aspects, respectively (see **Fig. 1**).^{4,5} The peripheral branches of the vestibular components are distributed in the utricle, saccule and semicircular ducts, whereas the respective branches of the cochlear component end at the organ of Corti.

CN IX: Glossopharyngeal Nerve

Among the six lower CNs, the glossopharyngeal is the smallest in terms of diameter, importance, and clinical significance.⁶ The ninth nerve is a mixed nerve, which contains motor, somatosensory, visceral sensory, and parasympathetic fibers. The preganglionic fibers originate from the nucleus ambiguus, inferior salivatory nucleus, nucleus of tractus solitarius, and spinal trigeminal nucleus. The nerve exits the brain stem from the lateral aspect of the upper medulla along with cranial nerves X and XI, crosses the pontine cistern, and enters the pars nervosa of the jugular foramen (**Fig. 2**).⁷ In passing through the latter, the nerve enters the superior and the petrous ganglia, from which final peripheral branches emanate. These include:

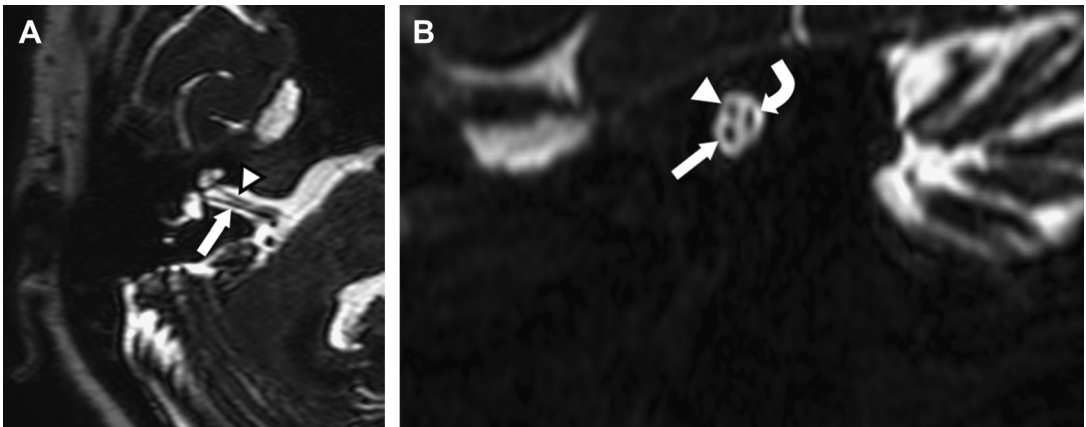


Fig. 1. Normal facial and vestibulocochlear nerves at the IAC fundus. Axial 3D DRIVE image (A) through the IAC demonstrates the facial nerve (*arrowhead*) and the vestibulocochlear nerve (*arrow*). The respective oblique sagittal image (B) shows the facial nerve anterosuperiorly (*arrowhead*), the cochlear branch of the vestibulocochlear nerve anteroinferiorly (*arrow*), and the superior and inferior vestibular branches posteriorly (*curved arrow*).

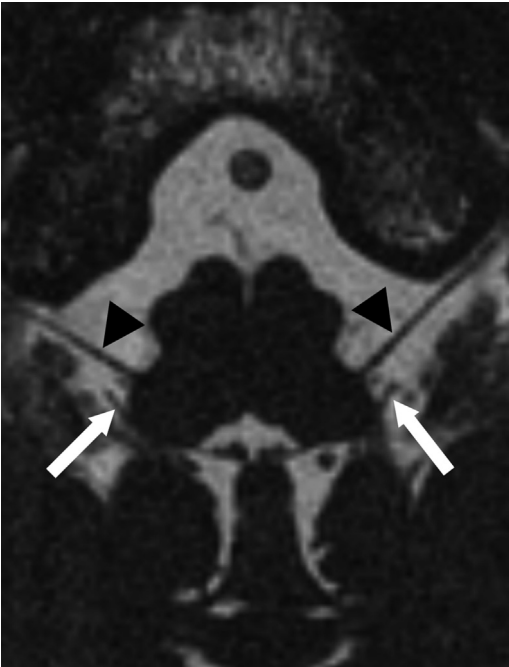


Fig. 2. Normal glossopharyngeal and vagus nerves. Axial CISS image through the brain stem demonstrates the origins of the glossopharyngeal nerves (*arrowheads*) and vagus nerves (*arrows*) from the sides of the upper medulla.

The tympanic nerve (which forms the tympanic plexus that gives off the lesser superficial petrosal nerve, a branch to join the superficial petrosal nerve and branches to the tympanic cavity)

Carotid branches (which connect with the vagus nerve and sympathetic branches)

Pharyngeal branches (which supply the muscles and mucous membrane of the pharynx)

Tonsillar branches (which supply the palatine tonsils)

Lingual branches (which supply the posterior third of the tongue and communicate with the lingual nerve)

A muscular branch (which is distributed to the stylopharyngeus)^{8,9}

CN X: Vagus Nerve

The vagus nerve contains motor, sensory, and parasympathetic nerve fibers, and features the most extensive course and distribution among all CNs, coursing through the neck and traversing in the thorax and abdomen. The preganglionic fibers emanate from the nucleus ambiguus, dorsal motor nucleus, nucleus of tractus solitarius, and spinal trigeminal nucleus. The nerve emerges

from the medulla oblongata, between the olive and the inferior cerebellar peduncle, just posterior to the glossopharyngeal nerve (see **Fig. 2**), entering the pars vascularis of the jugular foramen, where it forms the jugular and the nodose ganglia. Between the 2 latter ganglia, the vagus nerve gives off an auricular ramus, which innervates the skin of the concha of the external ear; a meningeal ramus, which runs to the dura matter of the posterior fossa; as well as a pharyngeal ramus, which forms the pharyngeal plexus with the glossopharyngeal nerve that supplies the muscles of the pharynx and soft palate (except the stylopharyngeus and tensor veli palatine muscles). Just distal to the nodose ganglion, the vagus nerve gives off the superior laryngeal nerve, which provides motor innervation to the cricothyroid muscle and sensory innervation to the larynx.¹⁰ Subsequently, CN X descends in the neck within the carotid sheath, between the common carotid artery and the internal jugular vein. At the base of the neck, it provides the superior cardiac branches and the recurrent laryngeal nerves. The right recurrent laryngeal nerve bends upward and medially behind the subclavian artery, and ascends in the ipsilateral tracheoesophageal sulcus, whereas the left branch arises to the left of the aortic arch, loops beneath the ligamentum arteriosum, and ascends in the left tracheoesophageal sulcus. The recurrent laryngeal nerve innervates all the laryngeal muscles, except the cricothyroid, which is innervated by the superior laryngeal nerve. Subsequently, the vagus nerve enters the thorax, coursing over the subclavian artery on the right side, and between the common carotid and subclavian artery on the left side, and gives off branches to the pulmonary and esophageal plexuses. After crossing through the esophageal hiatus, the nerve terminates in multiple abdominal viscera.^{11,12}

CN XI: (SPINAL) Accessory Nerve

The accessory nerve (often termed the spinal accessory nerve) is a motor nerve, composed of a small cranial part, which originates from the nucleus ambiguus and emerges from the side of the medulla oblongata, and a large spinal portion, which originates from the ventral horn of the spinal cord, between the C1 and C5 levels (**Fig. 3**). The 2 parts unite and enter the pars vascularis of the jugular foramen. The cranial part reaches the inferior vagal ganglion portion and is distributed to the striated muscles of the soft palate and larynx, whereas the spinal portion crosses the transverse process of C1 and provides innervation to the sternocleidomastoid and trapezius.^{13,14}



Fig. 3. Normal spinal accessory nerves. Axial CISS image at a slightly inferior level to Fig. 2 shows the origins of the bulbar spinal accessory nerves (arrows).

CN XII: Hypoglossal Nerve

The nucleus of the hypoglossal nerve is situated along the paramedian area of the anterior wall of the fourth ventricle in the medulla. The nerve emerges from the preolivary sulcus, runs through the hypoglossal canal, passes behind the inferior ganglion of the vagus nerve, and between the internal carotid artery and internal jugular vein (Fig. 4). After reaching the submandibular region, the hypoglossal nerve is distributed to the intrinsic muscles of the tongue (except the palatoglossus),

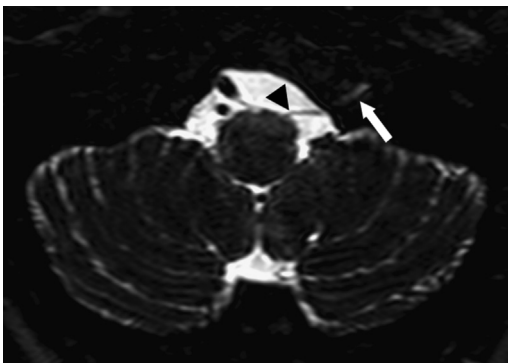


Fig. 4. Normal hypoglossal nerve. Axial 3D DRIVE image through the lower medulla demonstrates the left hypoglossal nerve (arrowhead) and entering the homonymous canal (arrow).

as well as the genioglossus, styloglossus, hyoglossus, and anterior strap muscles.³

IMAGING PROTOCOL

High-field imaging (3 T or newer 1.5 T scanners) is preferred to make use of the highest available signal-to-noise ratio and contrast-to-noise ratio, while keeping the imaging time in an acceptable range. A protocol that is commonly employed in most institutions and provides adequate high-resolution diagnostic evaluation is presented in Table 1. Thin-section imaging (1 mm) and low voxel size (0.6–1 mm for isotropic constructive interference in steady state [CISS] imaging) are essential to obtain the high-resolution evaluation of the posterior fossa CNs.

PATHOLOGIC CONDITIONS

CN VII: Facial Nerve

Facial palsy presents clinically with ipsilateral facial drop and difficulty in facial expression, pain around the jaw or behind the ear, increased sensitivity to sound, decreased ability to taste, headache, and changes in the amount of tears and saliva produced. It is crucial for clinicians to determine whether the forehead muscles are spared, which reflects pathology in the cerebral hemispheres (central facial palsy); or are affected, which implicates pathology in the facial nerve itself (peripheral facial palsy).¹⁵

After gadolinium administration, a thorough evaluation of all portions of the facial nerve is essential to detect areas of abnormal enhancement. Enhancement of the intracanalicular portion (which extends from the opening to the fundus of the IAC) and/or the labyrinthine portion (which extends from the fundus of the IAC to the facial hiatus) is always abnormal. The remaining portions of the nerve, as well as the geniculate ganglion, may normally enhance. In the case of abnormal facial nerve enhancement, the differential diagnosis includes Bell palsy, schwannoma, hemangioma, acute otitis media, lymphoma, sarcoidosis, viral neuritis, perineural tumor spread, Lyme disease, and Guillain-Barré and Ramsay-Hunt syndromes.¹⁶

In Bell palsy, there is enhancement of the intracanalicular and/or the labyrinthine portion of the ipsilateral facial nerve (Fig. 5), whereas some authors have also reported higher signal intensity ratio of the geniculate ganglion and tympanic segment on the affected side than on the normal side. The affected segment maintains linear morphology without any nodularity, and may be normal in size or slightly enlarged.^{17–19} Although the diagnosis of Bell palsy is typically clinical,

Table 1
A commonly employed protocol for the MRI evaluation of the cranial nerves

Plane	Sequence	Technique	Comment
3-plane	Scout	GRE	
Sagittal	T1 W	TSE or 3D GRE	Thin (1 mm) slices
Axial	T2 W	TSE	Fat suppression
Axial	IR	FLAIR	
Axial	DWI		
Axial	T1 W	TSE	Thin (1 mm) slices
Axial	CISS	3D	Thin (0.6 mm) slices
Axial and coronal (+GD)	T1 W	TSE	Thin (1 mm) slices
Axial (+GD)	T1 W	TSE	Brain

For all sequences except the last one, the slice coverage is through the cavernous sinus and the brain stem.

Abbreviations: CISS, constructive interference in steady state; DWI, diffusion-weighted imaging; FLAIR, fluid-attenuated inversion recovery; GD, gadolinium administration; GRE, gradient echo; T1 W, T1 weighted; T2 W, T2 weighted; TSE, turbo spin echo.

MRI is reserved for patients in whom nerve decompression is planned, when there is suspected mass lesion in nonresolving neuropathy, or when there are indeterminate results of electromyography. Imaging can be used to confirm potential swelling of the nerve proximal to the meatal foramen and to detect any associated mass lesion.²⁰

Schwannoma may develop in any portion of the facial nerve, although it has a predilection for the region of the geniculate ganglion. Typically, it presents as a well-demarcated space-occupying lesion, which is isointense to hypointense relative to gray matter on T1 weighted images and moderately hyperintense of T2 weighted images, and enhances homogeneously after gadolinium

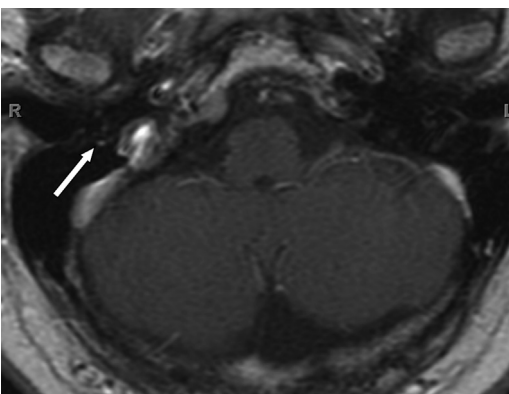


Fig. 5. Bels palsy. Contrast-enhanced axial T1 weighted image through the petrous bone demonstrates abnormal contrast enhancement of the labyrinthine portion of the right facial nerve (arrow).

administration. Larger lesions undergo internal bleeding, presenting as hyperintense zones on T1 weighted images, or cystic degeneration or necrosis appearing as hyperintense areas on T2 weighted images.²¹ CT demonstrates bony scalloping and remodeling rather than destruction.

Facial nerve venous vascular malformation (previously described as facial nerve hemangioma) also shows predilection for the geniculate ganglion. In the latter location, the lesion may be isointense to adjacent brain and only detectable on contrast-enhanced T1 weighted images, where it is expected to enhance intensely. Hemangiomas have similar signal characteristics compared with schwannomas, although in the former, the bony margins are indistinct, enabling differentiation from the latter, which feature well-defined remodeled margins. In addition, hemangiomas containing bone may feature foci of low signal intensity on MRI, and bone spicules or honeycomb morphology on CT. Associated widening of the facial nerve canal is sometimes present.^{22–24} Meningiomas infrequently arise in the geniculate ganglion, are not readily differentiated from hemangiomas on imaging studies, and are included in the differential diagnosis solely based on the aforementioned location.²⁴

In acute otitis media, there is obvious T2 hyperintensity and contrast enhancement of the tympanic segment of the facial nerve, although findings are difficult to assess because of the inflammation of the adjacent tissues. MRI is helpful in determining the degree of facial nerve involvement, as well as potential extension of the inflammation within the otic capsule and epidural and intradural spaces.²⁰

Perineural spread of parotid malignancies and squamous cell carcinoma of the parotid or face may occur along the facial nerve. MRI demonstrates enlargement and enhancement of the involved nerve portion and commonly secondary enlargement of the stylomastoid foramen. The facial nerve may be involved by neurosarcoidosis and Lyme disease, and demonstrates nerve enhancement as well as diffuse or multifocal nodular enlargement, which tends to regress after therapy.^{25,26} In Guillain-Barré syndrome, facial nerve involvement is acute, typically bilateral, and presents with enhancement.²⁷

The facial nerve is susceptible to injury in cases of temporal bone fractures. In transverse fractures, the facial nerve is injured in up to 40% of cases, whereas in longitudinal fractures, it is injured in about 10% to 20% of cases. CT is the modality of choice for assessing the integrity of the facial canal, while the role of MRI is limited in these situations.¹⁶

CN VIII: Vestibulocochlear Nerve

Dysfunction of the vestibular branch of CN VIII presents clinically with dizziness, vertigo, disequilibrium, imbalance, ataxia, nausea, and/or vomiting. When the cochlear branch is affected, manifestations include tinnitus or ear ringing, poor hearing ability, or even deafness. Combinations of the aforementioned symptoms indicate simultaneous involvement of both nerve branches.

Schwannomas of the vestibulocochlear nerve (acoustic neuromas) usually develop as combined intracanalicular–cisternal masses, and less

commonly as purely intracanalicular, extracanalicular, or intralabyrinthine lesions (Figs. 6 and 7). The lesions show the typical signal and enhancement characteristics of schwannomas, as previously described. In the IAC, intracanalicular lesions as well as segments of mixed intracanalicular–cisternal lesions demonstrate a funnel-shaped (ice cream cone) appearance with posterolateral epicenter on axial images and a short club-shaped configuration on coronal images.²⁸ In larger lesions, CT may demonstrate erosion of the temporal bone, which is limited to the boundaries of the IAC.²⁹

Vestibulocochlear meningiomas are the most common intracranial extra-axial tumors in adults. They typically involve the cisternal portion of the nerve, assume a hemispherical configuration, and are eccentric to the IAC with anteromedial epicenter, although they may cross the latter or even extend into it. Typically, these neoplasms are isointense to gray matter on T1 weighted images, hyperintense on T2 weighted images, and enhance intensely after gadolinium administration. The margin of the tumor may elongate and flatten out along the bone, producing the dural tail sign.^{30,31} On CT, associated calcifications and hyperostosis may be present.³²

CN VIII, as well as CNs IX to XI, may be stretched or displaced by posterior fossa arachnoid cysts or lipomas. Whereas lipomas have signal characteristics of fat on all imaging sequences, arachnoid cysts show angled margins and have signal characteristics of cerebrospinal fluid, do not enhance, and can be differentiated

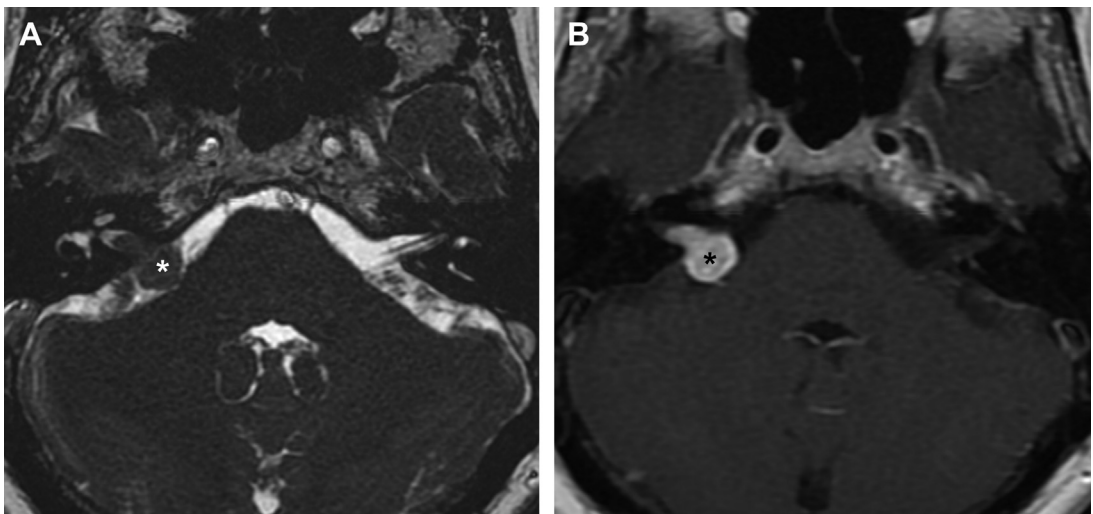


Fig. 6. Axial T2 weighted (A) and contrast-enhanced T1 weighted (B) images at the level of the cerebellopontine cisterns exhibit a round isointense, homogeneously enhancing space-occupying lesion (asterisk), extending in the right internal acoustic canal. On surgery, the lesion proved to be a vestibular schwannoma.

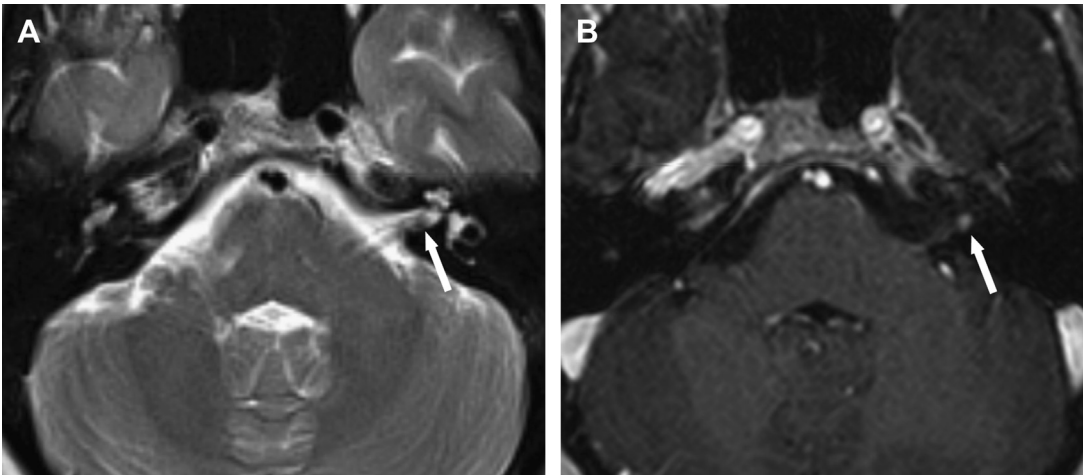


Fig. 7. Small acoustic schwannoma in a 52-year-old woman with gradual hearing loss in the left ear. Axial T2 weighted (A) and postcontrast T1 weighted (B) images at the level of the cerebellopontine cisterns demonstrate a tiny isointense enhancing lesion (arrow) within the left IAC.

from epidermoid cysts (lobulated margins) using diffusion-weighted imaging, on which the arachnoid cyst has low signal intensity, and the epidermoid cyst has high signal intensity.^{30,33}

Although previously suspected to be associated with tinnitus, vascular loops of the anterior inferior cerebellar artery extending within the IAC are considered normal anatomic variations (**Fig. 8**). However, they should be reported if detected, because they could be symptomatic. Any vascular contacts with the vestibulocochlear nerves, especially with atrophic appearance of the ipsilateral

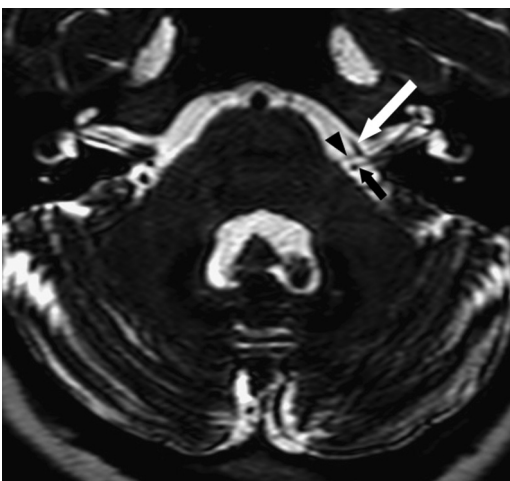


Fig. 8. Anterior inferior cerebellar artery looping within the IAC. Axial 3D DRIVE image through the IAC shows the left anterior inferior cerebellar artery (white arrow) in close anatomic relationship to the seventh (arrowhead) and eighth (black arrow), and entering the IAC.

nerves, should be reported.³⁴ Other vascular lesions that may compress the nerve include aneurysm of the anterior inferior cerebellar artery, tortuosity or dolichoectasia of the vertebrobasilar arteries, arteriovenous malformations, and dural fistulae. However, the aforementioned entities rarely cause neurogenic symptoms.³²

Congenital pathologies of the vestibulocochlear nerve include

- Aplasia, in which the nerve is absent, and the IAC is small containing only the facial nerve or no nerves at all
- Hypoplasia, in which the cochlear branch is aplastic or hypoplastic
- X-linked deafness, in which a wide neural aperture in the IAC fundus is associated with a broad communication between the cochlea and the IAC³⁰

Within the CPA and/or the IAC, CNs VII and VIII may be affected due to meningitis, postmeningitic or postoperative fibrosis, and neoplastic dural or leptomeningeal disease. Nerve thickening and enhancement are apparent on MRI, although a definite differential diagnosis cannot be established, except from cases with multifocal cerebral involvement, which indicates a neoplastic process.³⁰ Similar to the facial nerve, the vestibulocochlear nerve may be involved by neurosarcoidosis, either in isolation or as part of multifocal disease.

CN IX, X, XI: Glossopharyngeal, Vagus, and Accessory Nerves

These nerves are reviewed in the same section, because of their close anatomic, and to some

extent, functional relationship. The typical clinical scenario is complex neuropathy of CNs IX to XI, which indicates a lesion at the level of the medulla, CPA cistern, jugular foramen, or carotid space.³⁵ Intramedullary lesions, including demyelination, malignancy, motor neuron disease, syringobulbia, and infarction from occlusion of the posterior inferior cerebellar artery (PICA), can involve the nuclei of the aforementioned nerves, and present clinically as lateral medullary (Wallenberg) syndrome, which includes swallowing difficulty or dysphagia, slurred speech, ataxia, facial pain, vertigo, nystagmus, Horner syndrome, diplopia, and potentially palatal myoclonus.^{36,37}

In the CPA cistern, the nerve roots of the glossopharyngeal and vagus nerves are subject to compression by the PICA, resulting in hyperactive rhizopathy, such as glossopharyngeal neuralgia or spasmodic torticollis (**Fig. 9**). However, this compressive relationship is not always possible to confirm on imaging, and the diagnosis is of exclusion and may be confirmed at the time of explorative surgery.⁶

Upon their entrance in the jugular foramen, CNs IX to XI are subject to simultaneous injury by various entities that develop locally. Combined neuropathy of the aforementioned nerves is known as jugular foramen (Vernet) syndrome. The most common entity is paraganglioma, arising from paraganglionic tissue situated in the adventitia of the jugular vein (glomus jugulare), or in and around

the vagus nerve (glomus vagale).³⁸ Paragangliomas are generally benign and locally aggressive, but may undergo malignant degeneration in approximately 3% to 4% of cases.³⁹ On imaging, these tumors are centered at the jugular foramen or the nasopharyngeal carotid space, respectively, demonstrate ovoid or lobulated margins, and may extend in the posterior fossa or inferiorly, toward the carotid bifurcation. Unlike carotid body tumor (glomus caroticus), which splays the internal and external carotid arteries, glomus vagale displaces both vessels anteromedially.^{40,41} On MRI, paragangliomas are identified as isointense lesions that enhance avidly after gadolinium administration. Larger lesions may show a characteristic salt-and-pepper appearance on MRI, with T1 hyperintense foci representing areas of subacute hemorrhage, and T2 hypointense foci reflecting high-velocity flow voids (**Fig. 10**).⁴² High-resolution, thin-section CT images using bone windows exhibit moth-eaten permeative destructive bone changes around the jugular foramen (**Fig. 11**).^{16,35,43} The jugular foramen may also be involved by metastatic tumors (usually from prostate, breast or lung). In such cases, the contour of the foramen appears irregular on CT.⁴² Other neoplasms that may arise at the jugular foramen and cause bone destruction include meningioma, fibrous dysplasia, Paget disease, histiocytosis X, multiple myeloma, and primitive ectodermal tumor. The latter is an irregular destructive mass,

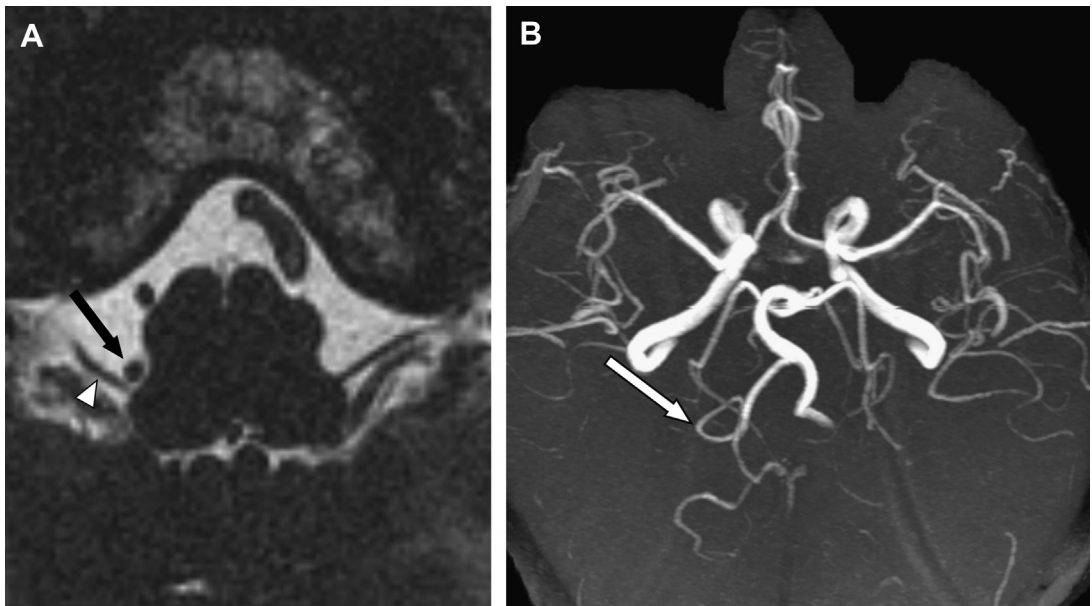


Fig. 9. Tortuous PICA compressing the spinal accessory nerve. Axial CISS image (A) through the medulla demonstrates a vessel (arrow) indenting the right glossopharyngeal nerve (arrowhead). Magnetic resonance angiography image (B) from the same case indicates the corresponding vessel is a tortuous right PICA (arrow).

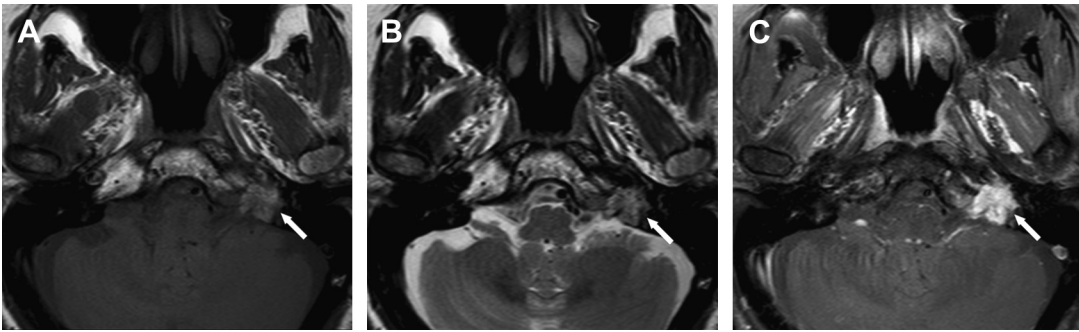


Fig. 10. Paraganglioma of the left jugular foramen. Axial T1 weighted (A), T2 weighted (B), and fat-suppressed contrast-enhanced T1 weighted (C) images display an enhancing space-occupying lesion (arrow) centered in the left jugular foramen, exhibiting a salt-and-pepper configuration.

which is isointense and slightly hyperintense to muscle on T1 and T2 weighted images, respectively. Additionally, it enhances homogeneously after contrast administration, demonstrates no tumor blush on angiography, and is surrounded by eroded bone on CT. Skull base fractures extending to the jugular foramen may also injure CNs IX to XI. Finally, the foramen may be infiltrated by extrinsic processes, which commonly originate from the temporal bone or the clivus, including cholesteatoma, epidermoid tumor, cholesterol granuloma, petrositis, abscess, mucocele, meningioma, chordoma, chondrosarcoma, chondroblastoma, osteoclastoma, fibrosarcoma, endolymphatic sac tumor, rhabdomyosarcoma, and osteomyelitis (Fig. 12).⁴² Distal to the jugular foramen, the nerves may be involved by lymphoma or extension of nasopharyngeal carcinoma.

Isolated glossopharyngeal palsy is a rare entity, which, apart from PICA compression over the nerve root zone, may be caused by intramedullary lesions, entrapment by an elongated styloid process, or an ossified stylohyoid ligament (Eagle syndrome), as well as by lesions of the

retropharyngeal or retroparotid space, such as nasopharyngeal carcinoma, adenopathy, aneurysm, abscess, trauma (eg, birth injury), and surgical procedures (eg, carotid endarterectomy).¹⁰ The disease presents as paroxysms of unilateral and severe lancinating pain in the oropharyngeal or otitic region, which is either spontaneous or elicited by actions that stimulate the region supplied by the nerve (eg, yawning, coughing, swallowing, and talking).⁴⁴

Isolated vagal neuropathy may be of peripheral or central type, corresponding to isolated impairment of the recurrent laryngeal nerve or complete vagal dysfunction, respectively. In the former case, there is injury of the recurrent laryngeal branch in the infrahyoid neck or upper thorax, with common causes including iatrogenic trauma (thyroidectomy, cervical spine, skull base, carotid or thoracic surgery, intubation), trauma (eg, motor vehicle accident), and extralaryngeal neoplasm (particularly esophageal or lung cancer). Due to its relatively medial location, the right recurrent laryngeal nerve is more susceptible to injury during thyroid or esophageal surgery. However, in up to one-third

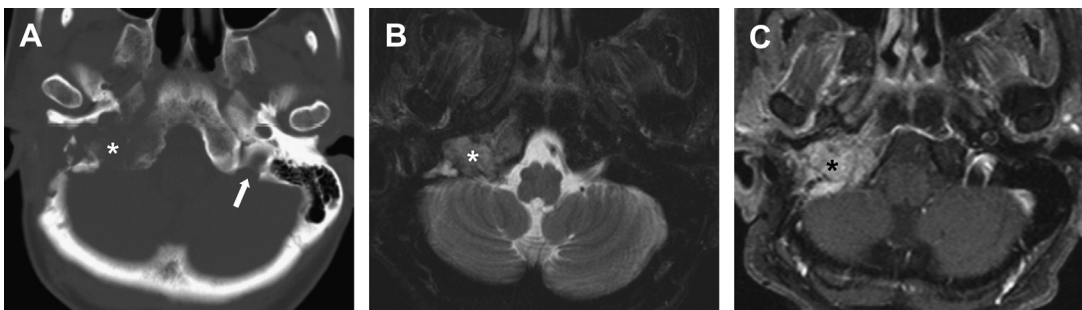


Fig. 11. Glomus tumor. CT image (A) at the level of the skull base demonstrates erosion of the right jugular foramen (asterisk), with a moth-eaten pattern. Note the normal left jugular foramen (arrow). Corresponding axial T2 weighted (B) and contrast-enhanced (C) images exhibit an isointense, avidly enhancing, mass centered within the jugular foramen (asterisk).

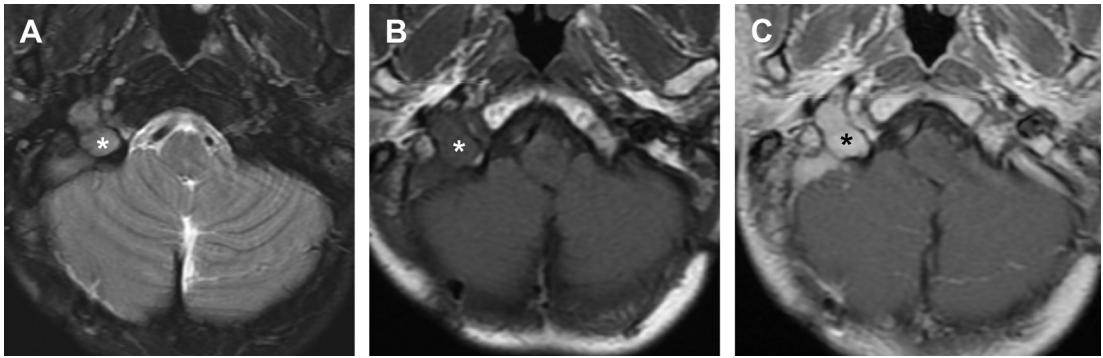


Fig. 12. Jugular foramen meningioma. Axial T2 weighted image (A) through the jugular foramina demonstrates absence of the normal flow void on the right (*asterisk*). Axial T1 weighted image (B) shows intermediate signal in this region (*asterisk*). Following contrast administration (C), an enhancing mass (*asterisk*) is evident extending inferiorly into the right carotid space.

of cases, no cause is identified, and the entity is considered idiopathic.^{10,45} The disease presents clinically with hoarseness, resulting from paralysis of all ipsilateral laryngeal muscles (except the cricothyroid). In the case of bilateral nerve damage, there is breathing difficulty and aphonia. Cross-sectional imaging, either CT or MRI, should cover the area between the skull base and the carina, and thorough evaluation of the carotid space, tracheoesophageal groove, and aortopulmonary window is mandatory to detect the causative lesion.⁴⁶ On the ipsilateral side, imaging findings suggestive of vocal cord paralysis include paramedian vocal cord position (100%), pyriform sinus and laryngeal ventricle dilatation (100%), thickening and medial deviation of the aryepiglottic fold (>75%), antero-medial deviation of the arytenoid cartilage (>45%), true vocal cord fullness (>45%), subglottic fullness, vallecula dilatation, subglottic arch flattening, posterior cricoarytenoid atrophy, and thyroarytenoid muscle atrophy.⁴⁷

In central type vagal neuropathy, the aforementioned clinical picture and imaging findings are supplemented by alterations of the parasympathetic tone in the thorax and abdomen. The injury of the vagus nerve distal to the origin of the recurrent laryngeal nerve may be caused by thoracic or abdominal neoplasms, compression by aortic aneurysm, cardiomegaly, or tuberculous sequelae.⁴⁸

Isolated spinal accessory nerve palsy may be a complication of surgery. Other causes include internal jugular vein cannulation in the posterior triangle of the neck, following carotid endarterectomy, coronary bypass surgery, and radiation therapy, as well as with shoulder blunt trauma or dislocation.⁴⁹ On MRI, signal intensity denervation changes of sternocleidomastoid and trapezius muscles are apparent. In chronic cases,

compensatory hypertrophy of the ipsilateral levator scapulae is a common finding, and should not erroneously be interpreted as a tumor.³⁵

As with all CNs, the glossopharyngeal, vagus, and spinal accessory nerves may be involved by nerve sheath tumors, and viral neuritis due to varicella zoster virus infection. In the latter case, MRI demonstrates thickening and contrast enhancement of the affected nerve(s), reflecting breakdown of the blood–brain barrier. As the clinical picture improves, nerve swelling regresses, but contrast enhancement may persist for a long period.⁵⁰

CN XII: Hypoglossal Nerve

Palsy of the hypoglossal nerve is relatively uncommon, produces distinctive clinical findings, and may be caused by injury at any point throughout its course from the medulla oblongata to the tongue.⁵¹ In supranuclear lesions, there is weakening or paralysis of the contralateral side of the tongue, although no dysfunction is usually apparent, since it is compensated for by the ipsilateral normal side. In nuclear or intranuclear lesions, there is ipsilateral tongue deviation, supplemented by muscle atrophy and fasciculation in chronic stages.⁵² After gadolinium administration, enhancement of the hypoglossal canal with minor anterior extension beneath into the nasopharyngeal region is a normal finding.⁵³

In hypoglossal nerve dysfunction, the most important MRI feature is unilateral signal intensity denervation changes of the tongue musculature, which manifest as low and high signal intensity on T1 and T2 weighted images, respectively, in the subacute phase, signifying edema, and as high signal intensity on both sequences and loss of volume in the chronic cases, representing fatty

infiltration and atrophy, respectively.⁵⁴ Once detected, the aforementioned finding should prompt for a comprehensive evaluation of skull base along the course of the nerve. Lipomas and dermoids of the tongue musculature may contain abundant amounts of fat, and caution is warranted not to misinterpret them as fatty infiltration.⁵²

The medullary portion of the 12th nerve may be affected by cerebral infarcts, gliomas and metastatic neoplasms, and less commonly, by encephalitis, multiple sclerosis and pseudobulbar palsy, amyotrophic lateral sclerosis, or poliomyelitis.^{51,55}

Skull base primary (eg, chordoma, meningioma) and secondary tumors involve the cisternal and skull base portions of the nerve. Nerve sheath tumors (schwannomas, neurofibromas) are uncommon and show typical MRI findings, whereas when located in the hypoglossal canal, they may cause expansion and bone remodeling, but no cortical rupture.^{52,56} In addition, the cisternal portion may be involved by sarcoidosis or undergo compression by aneurysm or dolichoectasia of the vertebral artery or the PICA, although, similar to the vestibulocochlear nerve, the clinical significance of this finding remains questionable. Traumatic injury to the skull base segment may be caused by occipital condyle fracture and odontoid process subluxations, and is rarely bilateral.^{51,57,58} The cisternal and skull base portions may also be damaged by subarachnoid hemorrhage or infections of the skull base (eg, osteomyelitis or basal meningitis).⁵²

The extracranial segment of CN XII may be invaded by malignancies of the nasopharynx, oropharynx, and sublingual spaces. Hypoglossal nerve palsy may also be caused by carotid artery aneurysm, ectasia or dissection, venous thrombosis, deep neck infections, odontogenic abscesses, and neck surgery (carotid endarterectomy, vascular puncture, or operations on the upper cervical spine or submandibular gland).^{51,57,58} Finally, hypoglossal nerve palsy has been reported after skull base radiation therapy.⁵⁹

SUMMARY

In the vast majority of lower CN pathologies, MRI enables accurate detection and characterization of the causative entity. Thorough knowledge of the anatomy, pathology, and radiologic appearance, as well as appropriate imaging technique and correlation with the clinical findings are mandatory for a precise diagnosis, which will help avoid surgical pitfalls and optimize management planning.

REFERENCES

1. Casselman J, Mermuys K, Delanote J, et al. MRI of the cranial nerves—more than meets the eye: technical considerations and advanced anatomy. *Neuroimaging Clin N Am* 2008;18(2):197–231.
2. Ginsberg LE, De Monte F, Gillenwater AM. Greater superficial petrosal nerve: anatomy and MR findings in perineural tumor spread. *AJNR Am J Neuroradiol* 1996;17(2):389–93.
3. Yousem DM, Grossman RI. Cranial anatomy. In: Yousem DM, Grossman RI, editors. *Neuroradiology: the requisites*. 3rd edition. Philadelphia: Mosby; 2010. p. 46.
4. Rubinstein D, Sandberg EJ, Cajade-Law AG. Anatomy of the facial and vestibulocochlear nerves in the internal auditory canal. *AJNR Am J Neuroradiol* 1996;17(6):1099–105.
5. Tian GY, Xu DC, Huang DL, et al. The topographical relationships and anastomosis of the nerves in the human internal auditory canal. *Surg Radiol Anat* 2008;30(3):243–7.
6. Soh KB. The glossopharyngeal nerve, glossopharyngeal neuralgia and the Eagle's syndrome—current concepts and management. *Singapore Med J* 1999;40(10):659–65.
7. Rubinstein D, Burton BS, Walker AL. The anatomy of the inferior petrosal sinus, glossopharyngeal nerve, vagus nerve, and accessory nerve in the jugular foramen. *AJNR Am J Neuroradiol* 1995;16(1):185–94.
8. Zhao H, Li X, Lv Q, et al. A large dumbbell glossopharyngeal schwannoma involving the vagus nerve: a case report and review of the literature. *J Med Case Rep* 2008;2:334.
9. Suzuki F, Handa J, Todo G. Intracranial glossopharyngeal neurinomas. Report of two cases with special emphasis on computed tomography and magnetic resonance imaging findings. *Surg Neurol* 1989;31(5):390–4.
10. Brazis PW, Masdeu JC, Biller J. Cranial nerves IX and X (the glossopharyngeal and vagus nerves). *Localization in clinical neurology*. 6th edition. Philadelphia: Lippincott Williams & Wilkins; 2011. p. 361–368.
11. Dionigi G, Chiang FY, Rausei S, et al. Surgical anatomy and neurophysiology of the vagus nerve (VN) for standardised intraoperative neuromonitoring (IONM) of the inferior laryngeal nerve (ILN) during thyroidectomy. *Langenbecks Arch Surg* 2010;395(7):893–9.
12. Binder D, Sonne DC, Fischbein N. Vagus nerve. *Cranial nerves: anatomy, pathology, imaging*. New York: Thieme Medical Publishers; 2010. p. 158–171.
13. Lloyd S. Accessory nerve: anatomy and surgical identification. *J Laryngol Otol* 2007;121(12):1118–25.

14. Cappiello J, Piazza C, Nicolai P. The spinal accessory nerve in head and neck surgery. *Curr Opin Otolaryngol Head Neck Surg* 2007;15(2):107–11.
15. Hazin R, Azizzadeh B, Bhatti MT. Medical and surgical management of facial nerve palsy. *Curr Opin Ophthalmol* 2009;20(6):440–50.
16. Yousem DM, Grossman RI. Temporal bone. neuro-radiology: the requisites. 3rd edition. Philadelphia: Mosby, Inc; 2010. p. 385–418.
17. Kohsyu H, Aoyagi M, Tojima H, et al. Facial nerve enhancement in Gd-MRI in patients with Bell's palsy. *Acta Otolaryngol Suppl* 1994;511:165–9.
18. Murphy TP, Teller DC. Magnetic resonance imaging of the facial nerve during Bell's palsy. *Otolaryngol Head Neck Surg* 1991;105(5):667–74.
19. Al-Noury K, Lotfy A. Normal and pathological findings for the facial nerve on magnetic resonance imaging. *Clin Radiol* 2011;66(8):701–7.
20. Kumar A, Mafee MF, Mason T. Value of imaging in disorders of the facial nerve. *Top Magn Reson Imaging* 2000;11(1):38–51.
21. Wiggins RH 3rd, Harnsberger HR, Salzman KL, et al. The many faces of facial nerve schwannoma. *AJNR Am J Neuroradiol* 2006;27(3):694–9.
22. Friedman O, Neff BA, Willcox TO, et al. Temporal bone hemangiomas involving the facial nerve. *Otol Neurotol* 2002;23(5):760–6.
23. Phillips CD, Hashikaki G, Veilon F, et al. Anatomy and development of the facial nerve. In: Swartz JD, Loevner LA, editors. *Imaging of the temporal bone*. New York: Thieme Medical Publishers; 2009. p. 444–79.
24. Larson TL, Talbot JM, Wong ML. Genuiculate ganglion meningiomas: CT and MR appearances. *AJNR Am J Neuroradiol* 1995;16(5):1144–6.
25. Oki M, Takizawa S, Ohnuki Y, et al. MRI findings of Vllth cranial nerve involvement in sarcoidosis. *Br J Radiol* 1997;70(836):859–61.
26. Saremi F, Helmy M, Farzin S, et al. MRI of cranial nerve enhancement. *AJR Am J Roentgenol* 2005;185(6):1487–97.
27. Ramsey KL, Kaseff LG. Role of magnetic resonance imaging in the diagnosis of bilateral facial paralysis. *Am J Otol* 1993;14(6):605–9.
28. Jeng CM, Huang JS, Lee WY, et al. Magnetic resonance imaging of acoustic schwannomas. *J Formos Med Assoc* 1995;94(8):487–93.
29. Feghali JG, Kantrowitz AB. Atypical invasion of the temporal bone in vestibular schwannoma. *Skull Base Surg* 1995;5(1):33–6.
30. De Foer B, Kenis C, Van Melkebeke D, et al. Pathology of the vestibulocochlear nerve. *Eur J Radiol* 2010;74(2):349–58.
31. Pickett BP, Kelly JP. Neoplasms of the ear and lateral skull base. In: Bailey BJ, Johnson JT, editors. *Head and neck surgery—otolaryngology*, vol. 6. Philadelphia: Lippincott Williams & Wilkins; 2006. p. 2003–26.
32. Enterline DS. The eighth cranial nerve. *Top Magn Reson Imaging* 1996;8(3):164–79.
33. Aribandi M, Wilson NJ. CT and MR imaging features of intracerebral epidermoid—a rare lesion. *Br J Radiol* 2008;81(963):e97–9.
34. Swartz JD. Pathology of the vestibulocochlear nerve. *Neuroimaging Clin N Am* 2008;18(2):321–46.
35. Ong CK, Chong VF. The glossopharyngeal, vagus and spinal accessory nerves. *Eur J Radiol* 2010;74(2):359–67.
36. Sacco RL, Freddo L, Bello JA, et al. Wallenberg's lateral medullary syndrome. Clinical–magnetic resonance imaging correlations. *Arch Neurol* 1993;50(6):609–14.
37. Kim JS, Lee JH, Suh DC, et al. Spectrum of lateral medullary syndrome. Correlation between clinical findings and magnetic resonance imaging in 33 subjects. *Stroke* 1994;25(7):1405–10.
38. Ramina R, Maniglia JJ, Fernandes YB, et al. Jugular foramen tumors: diagnosis and treatment. *Neurosurg Focus* 2004;17(2):E5.
39. Caldemeyer KS, Mathews VP, Azzarelli B, et al. The jugular foramen: a review of anatomy, masses, and imaging characteristics. *Radiographics* 1997;17(5):1123–39.
40. Weissman JL. Case 21: glomus vagale tumor. *Radiology* 2000;215(1):237–42.
41. Lee KY, Oh YW, Noh HJ, et al. Extraadrenal paragangliomas of the body: imaging features. *AJR Am J Roentgenol* 2006;187(2):492–504.
42. Vogl TJ, Bisdas S. Differential diagnosis of jugular foramen lesions. *Skull Base* 2009;19(1):3–16.
43. Rao AB, Koeller KK, Adair CF. From the archives of the AFIP. Paragangliomas of the head and neck: radiologic-pathologic correlation. *Armed Forces Institute of Pathology. Radiographics* 1999;19(6):1605–32.
44. Pearce JM. Glossopharyngeal neuralgia. *Eur Neurol* 2006;55(1):49–52.
45. Blau JN, Kapadia R. Idiopathic palsy of the recurrent laryngeal nerve: a transient cranial mononeuropathy. *Br Med J* 1972;4(5835):259–61.
46. Chin SC, Edelstein S, Chen CY, et al. Using CT to localize side and level of vocal cord paralysis. *AJR Am J Roentgenol* 2003;180(4):1165–70.
47. Escott EJ, Bakaya S, Bleicher AG, et al. Vocal cord lesions and paralysis. In: Lin EC, Escott EJ, Garg KD, et al, editors. *Practical differential diagnosis for CT and MRI*. New York: Thieme Medical Publishers; 2008. p. 109–10.
48. Hartl DM, Travagli JP, Lebouleux S, et al. Clinical review: current concepts in the management of unilateral recurrent laryngeal nerve paralysis after

- thyroid surgery. *J Clin Endocrinol Metab* 2005; 90(5):3084–8.
49. Brazis PW, Masdeu JC, Biller J. Cranial nerve XI (the spinal accessory nerve). Localization in clinical neurology. 6th edition. Philadelphia: Lippincott Williams & Wilkins; 2011. p. 369–76.
 50. Sniezek JC, Netterville JL, Sabri AN. Vagal paragangliomas. *Otolaryngol Clin North Am* 2001; 34(5):925–39.
 51. Thompson EO, Smoker WR. Hypoglossal nerve palsy: a segmental approach. *Radiographics* 1994;14(5):939–58.
 52. Alves P. Imaging the hypoglossal nerve. *Eur J Radiol* 2010;74(2):368–77.
 53. Voyvodic F, Whyte A, Slavotinek J. The hypoglossal canal: normal MR enhancement pattern. *AJNR Am J Neuroradiol* 1995;16(8):1707–10.
 54. Russo CP, Smoker WR, Weissman JL. MR appearance of trigeminal and hypoglossal motor denervation. *AJNR Am J Neuroradiol* 1997;18(7):1375–83.
 55. Laine FJ, Underhill T. Imaging of the lower cranial nerves. *Neuroimaging Clin N Am* 2004; 14(4):595–609.
 56. Biswas D, Marnane CN, Mal R, et al. Extracranial head and neck schwannomas—a 10-year review. *Auris Nasus Larynx* 2007;34(3):353–9.
 57. Delamont RS, Boyle RS. Traumatic hypoglossal nerve palsy. *Clin Exp Neurol* 1989;26:239–41.
 58. Freixinet J, Lorenzo F, Hernandez Gallego J, et al. Bilateral traumatic hypoglossal nerve paralysis. *Br J Oral Maxillofac Surg* 1996;34(4):309–10.
 59. Billan S, Stein M, Rawashdeh F, et al. Radiation-induced hypoglossal nerve palsy. *Isr Med Assoc J* 2007;9(2):134.