

**American College of Radiology
ACR Appropriateness Criteria®
Cranial Neuropathy**

Variant 1: Anosmia or other abnormalities of the sense of smell (olfactory nerve, CN I). Initial imaging.

Procedure	Appropriateness Category	Relative Radiation Level
MRI orbits face neck without and with IV contrast	Usually Appropriate	○
MRI head without and with IV contrast	May Be Appropriate (Disagreement)	○
MRI orbits face neck without IV contrast	May Be Appropriate	○
CT maxillofacial with IV contrast	May Be Appropriate	⊗⊗
CT maxillofacial without IV contrast	May Be Appropriate	⊗⊗
US neck	Usually Not Appropriate	○
MRA head with IV contrast	Usually Not Appropriate	○
MRA head without and with IV contrast	Usually Not Appropriate	○
MRA head without IV contrast	Usually Not Appropriate	○
MRI head with IV contrast	Usually Not Appropriate	○
MRI head without IV contrast	Usually Not Appropriate	○
MRI orbits face neck with IV contrast	Usually Not Appropriate	○
CT head with IV contrast	Usually Not Appropriate	⊗⊗⊗
CT head without and with IV contrast	Usually Not Appropriate	⊗⊗⊗
CT head without IV contrast	Usually Not Appropriate	⊗⊗⊗
CT maxillofacial without and with IV contrast	Usually Not Appropriate	⊗⊗⊗
CTA head with IV contrast	Usually Not Appropriate	⊗⊗⊗
FDG-PET/CT skull base to mid-thigh	Usually Not Appropriate	⊗⊗⊗⊗

Variant 2:

Unilateral isolated weakness of the mastication muscles, paralysis of the mastication muscles, sensory abnormalities of the face and head, facial numbness, or trigeminal neuralgia (trigeminal nerve, CN V). Initial imaging.

Procedure	Appropriateness Category	Relative Radiation Level
MRI head without and with IV contrast	Usually Appropriate	○
MRI orbits face neck without and with IV contrast	Usually Appropriate	○
MRA head with IV contrast	May Be Appropriate	○
MRA head without and with IV contrast	May Be Appropriate	○
MRA head without IV contrast	May Be Appropriate	○
MRI head without IV contrast	May Be Appropriate (Disagreement)	○
MRI orbits face neck without IV contrast	May Be Appropriate	○
CT maxillofacial with IV contrast	May Be Appropriate	☼☼
CT maxillofacial without IV contrast	May Be Appropriate	☼☼
US neck	Usually Not Appropriate	○
MRI head with IV contrast	Usually Not Appropriate	○
MRI orbits face neck with IV contrast	Usually Not Appropriate	○
CT head with IV contrast	Usually Not Appropriate	☼☼☼
CT head without and with IV contrast	Usually Not Appropriate	☼☼☼
CT head without IV contrast	Usually Not Appropriate	☼☼☼
CT maxillofacial without and with IV contrast	Usually Not Appropriate	☼☼☼
CTA head with IV contrast	Usually Not Appropriate	☼☼☼
FDG-PET/CT skull base to mid-thigh	Usually Not Appropriate	☼☼☼☼

Variant 3:**Unilateral isolated weakness of the facial expression, paralysis of the facial expression, hemifacial spasm, or Bell palsy (facial nerve, CN VII). Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
MRI head without and with IV contrast	Usually Appropriate	○
MRI orbits face neck without and with IV contrast	Usually Appropriate	○
MRA head with IV contrast	May Be Appropriate	○
MRA head without and with IV contrast	May Be Appropriate	○
MRA head without IV contrast	May Be Appropriate (Disagreement)	○
MRI head without IV contrast	May Be Appropriate	○
MRI orbits face neck without IV contrast	May Be Appropriate (Disagreement)	○
CT maxillofacial with IV contrast	May Be Appropriate	☼☼
CT maxillofacial without IV contrast	May Be Appropriate	☼☼
CT temporal bone with IV contrast	May Be Appropriate	☼☼☼
CT temporal bone without IV contrast	May Be Appropriate	☼☼☼
US neck	Usually Not Appropriate	○
MRI head with IV contrast	Usually Not Appropriate	○
MRI orbits face neck with IV contrast	Usually Not Appropriate	○
CT head with IV contrast	Usually Not Appropriate	☼☼☼
CT head without and with IV contrast	Usually Not Appropriate	☼☼☼
CT head without IV contrast	Usually Not Appropriate	☼☼☼
CT maxillofacial without and with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone without and with IV contrast	Usually Not Appropriate	☼☼☼
CTA head with IV contrast	Usually Not Appropriate	☼☼☼
FDG-PET/CT skull base to mid-thigh	Usually Not Appropriate	☼☼☼☼

Variant 4:**Multiple different middle cranial nerve palsies (CN V-VII). Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
MRI head without and with IV contrast	Usually Appropriate	○
MRI orbits face neck without and with IV contrast	Usually Appropriate	○
MRI head without IV contrast	May Be Appropriate	○
MRI orbits face neck without IV contrast	May Be Appropriate	○
CT head without IV contrast	May Be Appropriate	⊕⊕⊕
US neck	Usually Not Appropriate	○
MRA head with IV contrast	Usually Not Appropriate	○
MRA head without and with IV contrast	Usually Not Appropriate	○
MRA head without IV contrast	Usually Not Appropriate	○
MRI head with IV contrast	Usually Not Appropriate	○
MRI orbits face neck with IV contrast	Usually Not Appropriate	○
CT maxillofacial with IV contrast	Usually Not Appropriate	⊕⊕
CT maxillofacial without IV contrast	Usually Not Appropriate	⊕⊕
CT head with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT head without and with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT maxillofacial without and with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT neck with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT neck without and with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT neck without IV contrast	Usually Not Appropriate	⊕⊕⊕
CT temporal bone with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT temporal bone without and with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT temporal bone without IV contrast	Usually Not Appropriate	⊕⊕⊕
CTA head with IV contrast	Usually Not Appropriate	⊕⊕⊕
FDG-PET/CT skull base to mid-thigh	Usually Not Appropriate	⊕⊕⊕⊕

Variant 5:**Oropharyngeal neurogenic dysphagia or oropharyngeal pain (glossopharyngeal nerve, CN IX). Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
MRI head without and with IV contrast	Usually Appropriate	○
MRI orbits face neck without and with IV contrast	Usually Appropriate	○
MRI head without IV contrast	May Be Appropriate	○
MRI orbits face neck without IV contrast	May Be Appropriate	○
CT neck with IV contrast	May Be Appropriate	☼☼☼
US neck	Usually Not Appropriate	○
MRA head and neck with IV contrast	Usually Not Appropriate	○
MRA head and neck without and with IV contrast	Usually Not Appropriate	○
MRA head and neck without IV contrast	Usually Not Appropriate	○
MRI head with IV contrast	Usually Not Appropriate	○
MRI orbits face neck with IV contrast	Usually Not Appropriate	○
CT maxillofacial with IV contrast	Usually Not Appropriate	☼☼
CT maxillofacial without IV contrast	Usually Not Appropriate	☼☼
CT head with IV contrast	Usually Not Appropriate	☼☼☼
CT head without and with IV contrast	Usually Not Appropriate	☼☼☼
CT head without IV contrast	Usually Not Appropriate	☼☼☼
CT maxillofacial without and with IV contrast	Usually Not Appropriate	☼☼☼
CT neck without and with IV contrast	Usually Not Appropriate	☼☼☼
CT neck without IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone without and with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone without IV contrast	Usually Not Appropriate	☼☼☼
CTA head and neck with IV contrast	Usually Not Appropriate	☼☼☼
FDG-PET/CT skull base to mid-thigh	Usually Not Appropriate	☼☼☼☼

Variant 6:**Unilateral isolated palatal or vocal cord paralysis or both (vagal nerve, CN X). Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
MRI head without and with IV contrast	Usually Appropriate	○
MRI orbits face neck without and with IV contrast	Usually Appropriate	○
CT neck with IV contrast	Usually Appropriate	☼☼☼
MRI head without IV contrast	May Be Appropriate	○
MRI orbits face neck without IV contrast	May Be Appropriate	○
CT chest with IV contrast	May Be Appropriate	☼☼☼
CT chest without IV contrast	May Be Appropriate	☼☼☼
CT neck without IV contrast	May Be Appropriate	☼☼☼
US neck	Usually Not Appropriate	○
Radiography chest	Usually Not Appropriate	☼
MRA head and neck with IV contrast	Usually Not Appropriate	○
MRA head and neck without and with IV contrast	Usually Not Appropriate	○
MRA head and neck without IV contrast	Usually Not Appropriate	○
MRI head with IV contrast	Usually Not Appropriate	○
MRI orbits face neck with IV contrast	Usually Not Appropriate	○
CT maxillofacial with IV contrast	Usually Not Appropriate	☼☼
CT maxillofacial without IV contrast	Usually Not Appropriate	☼☼
CT chest without and with IV contrast	Usually Not Appropriate	☼☼☼
CT head with IV contrast	Usually Not Appropriate	☼☼☼
CT head without and with IV contrast	Usually Not Appropriate	☼☼☼
CT head without IV contrast	Usually Not Appropriate	☼☼☼
CT maxillofacial without and with IV contrast	Usually Not Appropriate	☼☼☼
CT neck without and with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone without and with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone without IV contrast	Usually Not Appropriate	☼☼☼
CTA head and neck with IV contrast	Usually Not Appropriate	☼☼☼
FDG-PET/CT skull base to mid-thigh	Usually Not Appropriate	☼☼☼☼

Variant 7:**Unilateral isolated weakness or paralysis of the sternocleidomastoid and trapezius muscles (accessory nerve, CN XI). Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
MRI head without and with IV contrast	Usually Appropriate	○
MRI orbits face neck without and with IV contrast	Usually Appropriate	○
CT neck with IV contrast	Usually Appropriate	⊕⊕⊕
MRI head without IV contrast	May Be Appropriate	○
MRI orbits face neck without IV contrast	May Be Appropriate	○
CT neck without IV contrast	May Be Appropriate	⊕⊕⊕
US neck	Usually Not Appropriate	○
MRA head and neck with IV contrast	Usually Not Appropriate	○
MRA head and neck without and with IV contrast	Usually Not Appropriate	○
MRA head and neck without IV contrast	Usually Not Appropriate	○
MRI head with IV contrast	Usually Not Appropriate	○
MRI orbits face neck with IV contrast	Usually Not Appropriate	○
CT maxillofacial with IV contrast	Usually Not Appropriate	⊕⊕
CT maxillofacial without IV contrast	Usually Not Appropriate	⊕⊕
CT head with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT head without and with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT head without IV contrast	Usually Not Appropriate	⊕⊕⊕
CT maxillofacial without and with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT neck without and with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT temporal bone with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT temporal bone without and with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT temporal bone without IV contrast	Usually Not Appropriate	⊕⊕⊕
CTA head and neck with IV contrast	Usually Not Appropriate	⊕⊕⊕

Variant 8:**Unilateral isolated weakness or paralysis of the tongue (hypoglossal nerve, CN XII). Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
MRI head without and with IV contrast	Usually Appropriate	○
MRI orbits face neck without and with IV contrast	Usually Appropriate	○
CT neck with IV contrast	Usually Appropriate	☼☼☼
MRA head and neck with IV contrast	May Be Appropriate	○
MRA head and neck without and with IV contrast	May Be Appropriate	○
MRA head and neck without IV contrast	May Be Appropriate	○
MRI head without IV contrast	May Be Appropriate	○
MRI orbits face neck without IV contrast	May Be Appropriate	○
CT neck without IV contrast	May Be Appropriate	☼☼☼
CTA head and neck with IV contrast	May Be Appropriate	☼☼☼
US neck	Usually Not Appropriate	○
MRI head with IV contrast	Usually Not Appropriate	○
MRI orbits face neck with IV contrast	Usually Not Appropriate	○
CT maxillofacial with IV contrast	Usually Not Appropriate	☼☼
CT maxillofacial without IV contrast	Usually Not Appropriate	☼☼
CT head with IV contrast	Usually Not Appropriate	☼☼☼
CT head without and with IV contrast	Usually Not Appropriate	☼☼☼
CT head without IV contrast	Usually Not Appropriate	☼☼☼
CT maxillofacial without and with IV contrast	Usually Not Appropriate	☼☼☼
CT neck without and with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone without and with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone without IV contrast	Usually Not Appropriate	☼☼☼
FDG-PET/CT skull base to mid-thigh	Usually Not Appropriate	☼☼☼☼

Variant 9:**Multiple different lower cranial nerve palsies or combined lower cranial nerve syndromes (CN IX-XII). Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
MRI head without and with IV contrast	Usually Appropriate	○
MRI orbits face neck without and with IV contrast	Usually Appropriate	○
CT neck with IV contrast	Usually Appropriate	⊕⊕⊕
MRA head and neck with IV contrast	May Be Appropriate	○
MRA head and neck without and with IV contrast	May Be Appropriate	○
MRA head and neck without IV contrast	May Be Appropriate	○
MRI head without IV contrast	May Be Appropriate	○
MRI orbits face neck without IV contrast	May Be Appropriate	○
CT neck without IV contrast	May Be Appropriate	⊕⊕⊕
CTA head and neck with IV contrast	May Be Appropriate	⊕⊕⊕
US neck	Usually Not Appropriate	○
MRI head with IV contrast	Usually Not Appropriate	○
MRI orbits face neck with IV contrast	Usually Not Appropriate	○
CT maxillofacial with IV contrast	Usually Not Appropriate	⊕⊕
CT maxillofacial without IV contrast	Usually Not Appropriate	⊕⊕
CT head with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT head without and with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT head without IV contrast	Usually Not Appropriate	⊕⊕⊕
CT maxillofacial without and with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT neck without and with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT temporal bone with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT temporal bone without and with IV contrast	Usually Not Appropriate	⊕⊕⊕
CT temporal bone without IV contrast	Usually Not Appropriate	⊕⊕⊕
FDG-PET/CT skull base to mid-thigh	Usually Not Appropriate	⊕⊕⊕⊕

Variant 10:**Head and neck cancer. Suspected or known perineural spread of tumor. Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
MRI head without and with IV contrast	Usually Appropriate	○
MRI orbits face neck without and with IV contrast	Usually Appropriate	○
MRI head without IV contrast	May Be Appropriate	○
MRI orbits face neck without IV contrast	May Be Appropriate	○
CT maxillofacial with IV contrast	May Be Appropriate	☼☼
CT neck with IV contrast	May Be Appropriate	☼☼☼
US neck	Usually Not Appropriate	○
MRA head and neck with IV contrast	Usually Not Appropriate	○
MRA head and neck without and with IV contrast	Usually Not Appropriate	○
MRA head and neck without IV contrast	Usually Not Appropriate	○
MRI head with IV contrast	Usually Not Appropriate	○
MRI orbits face neck with IV contrast	Usually Not Appropriate	○
CT maxillofacial without IV contrast	Usually Not Appropriate	☼☼
CT head with IV contrast	Usually Not Appropriate	☼☼☼
CT head without and with IV contrast	Usually Not Appropriate	☼☼☼
CT head without IV contrast	Usually Not Appropriate	☼☼☼
CT maxillofacial without and with IV contrast	Usually Not Appropriate	☼☼☼
CT neck without and with IV contrast	Usually Not Appropriate	☼☼☼
CT neck without IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone without and with IV contrast	Usually Not Appropriate	☼☼☼
CT temporal bone without IV contrast	Usually Not Appropriate	☼☼☼
CTA head and neck with IV contrast	Usually Not Appropriate	☼☼☼
FDG-PET/CT skull base to mid-thigh	Usually Not Appropriate	☼☼☼☼

CRANIAL NEUROPATHY

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Summary of Literature Review

Introduction/Background

The human body has 12 paired cranial nerves (CNs) that provide specialized sensory and motor innervation to the head and neck region. As a group, the CNs have both sensory and motor components similar to those of the spinal nerves. Individually the CNs may be purely sensory, purely motor, or a mixture of both sensory and motor. Functions of the CNs may be divided into three sensory and three motor categories. The sensory group includes visceral sensory, which supplies sensory input from the internal organs; general sensory, which supplies tactile, pain, temperature, and other sensations; and special sensory, which includes the special senses of smell, vision, taste, hearing, and balance. Of the three motor functions, somatic motor nerves innervate muscles that develop from the body somites; branchial motor nerves innervate muscles derived from the branchial arches; and visceral motor nerves innervate the viscera, glands, and smooth muscle [1-4].

The CNs emerge in an orderly fashion from the rostral portion of the embryologically developing neural tube, which develops to form the brain. Anatomically, the 12 pairs of CNs are designated by numbers I-XII. The CNs include the olfactory (CN I), optic (CN II), oculomotor (CN III), trochlear (CN IV), trigeminal (CN V), abducens (CN VI), facial (CN VII), vestibulocochlear (CN VIII), glossopharyngeal (CN IX), vagus (CN X), spinal accessory (CN XI), and hypoglossal (CN XII) nerves. The olfactory (CN I) and optic (CN II) nerves are actually tracts formed from the telencephalon and diencephalon, respectively, and are not considered true nerves [4,5]. The CN nuclei arise in the brainstem, largely topologically arranged between the midbrain and the rostral cervical spine (CN XI). Current knowledge of CN anatomy has been improved by modern microsurgical techniques and endoscopic dissections, allowing visualization of the CN brainstem exit zones [3,4,6-13]. Various nomenclatures for the segments of the CNs are described in the anatomical, surgical, and radiological literature [5,14,15].

The optic (CN II), oculomotor (CN III), trochlear (CN IV), and abducens (CN VI) nerves are considered functionally to be part of the visual and extraocular motor system and are discussed in the ACR Appropriateness Criteria[®] topic on “[Orbits, Vision and Visual Loss](#)” [16]. The vestibulocochlear nerve (CN VIII) is reviewed in the ACR Appropriateness Criteria[®] topic on “[Hearing Loss and/or Vertigo](#)” [17]. Therefore, this discussion will be confined to CN I, CN V, CN VII, CN IX, CN X, CN XI, and CN XII.

In approaching cranial neuropathy, several concepts should be emphasized:

1. Cranial neuropathy can result from pathology affecting the nerve fibers at any point from the CN nucleus to the end organ supplied by the nerve. Pathologic processes affecting the brain, CN nuclei, or nerve fiber tracts supplying CNs are often associated with multiple neurologic symptoms. The CNs may take long, circuitous routes to their end organs. A detailed knowledge of CN anatomy coupled with a careful neurologic examination is essential for proper clinical localization of potential lesions and for the selection of appropriate imaging protocols.

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2. Because some individual nerve fibers, such as the autonomic nerves, may travel with several different CNs from their nuclei of origin to their ultimate destinations, loss of a specific function may indicate involvement of potentially more than one CN.
3. Because of the close proximity of many CN nuclei, CN segments, exiting sites of the CNs, as well as interconnections among nerves, some lesions may result in multiple cranial neuropathies.
4. In patients with multiple cranial neuropathies, imaging is useful to exclude a potential intraparenchymal process. For detection of a potential extraparenchymal process as a cause, the addition of cerebrospinal fluid (CSF) analysis and serologic studies may be indicated [18].

Special Imaging Considerations

MRI is the standard modality for imaging the CNs. Imaging with 3.0T is preferred over 1.5T imaging because of superior signal-to-noise ratios, gradient strength, and spatial resolution, although diagnostic information can be obtained with 1.5T imaging when 3.0T imaging is not available or precluded [5]. A phased-array head coil suffices for most examinations; specialized surface coils may supplement examinations of peripherally located nerves [5,19,20].

Fundamental techniques include thin-cut T1-weighted, T2-weighted, and contrast-enhanced T1-weighted sequences. The unenhanced T1-weighted sequence remains an excellent baseline technique for anatomic evaluation because of the natural contrast provided by fat in the neck and skull base. Diffusion-weighted imaging (DWI) is useful to assess for acute infarctions, cellular tumors, or specific lesions that may affect CN function, such as epidermoids or cholesteatomas. Thin section nonecho planar DWI with decreased susceptibility artifact compared with echo planar DWI allows for increased sensitivity in detection of small cholesteatomas. The use of intravenous (IV) contrast is imperative for the evaluation of cranial neuropathy with MRI. Neck CT also requires the use of IV contrast when evaluating for lesions in the neck causing cranial neuropathy.

Thin-section imaging with high-spatial resolution is required to directly image the CNs or their course. Slice thickness should be calculated for optimal spatial resolution without introducing partial-volume effect. The primary plane of imaging varies with the CN of interest and should include orthogonal views. The CN nuclei and fascicular nerve segments (brainstem segments) are not well seen on MRI, but their location can be deduced by recognizing surrounding anatomy and are best imaged with various T2-weighted, multi-echo, and fluid-attenuated inversion-recovery (FLAIR) sequences [15]. Three-dimensional isotropic heavily T2-weighted sequences with low CSF artifact provide submillimeter high-spatial and contrast resolution to image the cisternal and dural cave segments of the CNs and can be reformatted into multiple planes [5,21-23].

Intradural and foraminal CN segments, surrounded by a vascular plexus, are best imaged with contrast-enhanced 2-D or 3-D T1-weighted images, contrast-enhanced MR angiography (MRA) or contrast-enhanced modified balanced steady-state free precession (SSFP) sequences in which the nerves appear dark while surrounded by the vascular plexus [5,15,23,24]. The peripheral or extraforaminal segments of the CNs begin as they exit the skull base where they are initially surrounded by fat and are well imaged with high-resolution axial and coronal T1-weighted and T2-weighted sequences and with SSFP sequences as they course within the face and neck [5,15]. Contrast-enhanced fat-suppressed T1-weighted techniques may emphasize abnormal nerves or lesions but can mask subtle pathology if the fat-suppression is nonuniform. Because CN examinations tend to be lengthy, strategies such as parallel imaging may improve patient compliance and image quality [15].

DWI and fiber tract imaging techniques have shown some improvement in visualization of CN fascicles and fiber tracts but are without sufficient reliability for routine clinical use [5,25,26].

In the setting of cranial neuropathy, thin-cut high-resolution CT can be complementary to MRI in characterizing the osseous integrity of the skull base and skull base foramina.

Knowledge of CN anatomy coupled with a careful neurologic examination can aid in tailoring imaging protocols, particularly if a central or peripheral CN palsy can be distinguished clinically. If the probable location of the causative lesion cannot be deduced clinically, imaging of the entire course of the relevant CN may be required because cranial neuropathy can result from pathology affecting the nerve fibers at any point along the course of the nerve and might require multiple imaging modalities. For example, complete evaluation of CN VII includes imaging of the parotid gland and face, because causative lesions can be intraparotid or cutaneous. Head and neck imaging is required to completely image the courses of CN IX, CN XI, and CN XII, which extend into the neck to innervate

end organs. Evaluation of the head, neck, and upper chest is necessary for complete evaluation of the CN X to include the recurrent laryngeal nerve. This can be accomplished by extending the neck scan into the mid thorax (aortic pulmonary window) or by adding a dedicated chest CT. Patients presenting with otalgia may require evaluation of CN V, VII, IX, and X and upper cervical nerves C2 and C3 because any of these nerves may be the source for the otalgia [27].

There is institutional variation when ordering and performing high-resolution skull base imaging. Targeted skull base imaging protocols may be similar between institutions yet can be variably referred to as either MRI brain or MRI orbits, face, and neck based on accepted institutional protocols. MRI brain or MRI orbits, face, and neck or both studies may be needed, depending on the individual's clinical need. Recognizing this, physicians ordering imaging studies should be aware of these institutional preferences when selecting the desired imaging study.

Initial Imaging Definition

Initial imaging is defined as imaging at the beginning of the care episode for the medical condition defined by the variant. More than one procedure can be considered usually appropriate in the initial imaging evaluation when:

- There are procedures that are equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient's care)

OR

- There are complementary procedures (ie, more than one procedure is ordered as a set or simultaneously where each procedure provides unique clinical information to effectively manage the patient's care).

Discussion of Procedures by Variant

Variant 1: Anosmia or other abnormalities of the sense of smell (olfactory nerve, CN I). Initial imaging.

The sense of smell is mediated by the olfactory nerve (CN I), and abnormalities thereof can be grouped into clinical categories. Quantitative disturbances include diminished or enhanced sense of smell (anosmia, hyposmia, or hyperosmia). Qualitative disturbances involve distortions of the sense of smell (dysosmia). Discrimination disturbances involve an inability to differentiate among various smells. Hallucinations or delusions in the sense of smell may also occur. The latter may be caused by temporal lobe dysfunction (see the ACR Appropriateness Criteria® topic on "[Seizures and Epilepsy](#)" [28]) or by degenerative or psychiatric disease. Taste, though not mediated by CN I, may also be affected by pathology involving the CN I as a result of disturbed olfaction. Clinical examination should focus on a thorough nasal and neurological examination. Objective testing can characterize the degree of olfaction loss.

Most patients with olfactory symptoms do not require imaging, unless history or physical examination warrants it [29]. Causative factors for olfactory impairment can be categorized into three main groups, including conduction loss from sinonasal passage obstruction, sensorineural loss from olfactory neuroepithelial damage, and dysfunction from central nervous system disorders [30,31]. Trauma, aging, upper respiratory infections, and inflammatory sinonasal disorders most commonly affect the sense of smell [30-32]. Tumors affecting the cribriform plate such as squamous cell carcinomas, meningiomas, and esthesioneuroblastoma or olfactory neuroblastomas; inflammatory lesions such as sarcoidosis and granulomatosis with polyangiitis; and congenital conditions such as cephaloceles and Kallmann syndrome can also result in impaired olfaction [32-34]. Olfactory dysfunction is also associated with neurodegenerative, cognitive, and mood disorders including Alzheimer disease, Parkinson disease, and depression [30,35-38].

CT Head

There is no relevant literature to support the use of CT head in the evaluation of the olfactory nerve.

CT Maxillofacial

CT of the paranasal sinuses and face is useful to evaluate fractures, paranasal sinus inflammatory disease, and bony anatomy that impact olfaction [29,39-41]. CT can be used to characterize sinonasal inflammatory disease. Rhinosinusitis with nasal polyposis severity, as characterized by CT, correlates with worse olfaction [31]. Although the anatomic subunits of the olfactory pathway except for the olfactory bulbs cannot be directly visualized by CT, imaging protocols should cover the major anatomic divisions of the olfactory nerve and pathway, including the olfactory epithelium, which is located in the upper nasal cavity; the olfactory neurons and bulbs, located in the cribriform plate and inferior frontal lobes; and the olfactory pathways, which travel in portions of the temporal and

frontal lobes [4]. No IV contrast is necessary for osseous evaluation including posttrauma and for uncomplicated inflammatory sinonasal disease. Contrast-enhanced CT is useful to evaluate granulomatous and neoplastic disease. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CTA Head

There is no relevant literature to support the use of CT angiography (CTA) head in the evaluation of the olfactory nerve.

FDG-PET/CT

Efforts using functional MRI, single-photon emission CT, and fluorine-18-2-fluoro-2-deoxy-D-glucose (FDG)-PET in studying olfactory dysfunction and the role of olfaction in neurodegenerative disorders remain largely investigative and are not generally used in routine evaluations [42-48].

MRA Head

There is no relevant literature to support the use of MRA head in the evaluation of the olfactory nerve.

MRI Head

MRI is the mainstay for directly imaging the olfactory apparatus and is best assessed with MRI orbits, face, and neck MRI rather than MRI head, allowing for assessment of the sinonasal components of the olfactory apparatus while simultaneously evaluating the relevant brain structures that can affect olfaction.

MRI Orbits, Face, and Neck

MRI is the mainstay for directly imaging the olfactory apparatus and sinonasal or anterior cranial fossa tumors that may impair or directly involve the olfactory apparatus. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may be useful for this clinical scenario. In a large retrospective study of patients with olfactory loss, the most common etiology was idiopathic with <1% of patients having olfactory loss attributable to a lesion identified by MRI, although selection bias may have contributed to the low diagnostic yield [29]. Recent investigations have focused on olfactory bulb volume, as measured by MRI, as an indicator of olfactory dysfunction and even a marker for such disorders as early Parkinson disease and depression [39,41,49-55]. Coronavirus disease 2019 (COVID-19)-related olfactory dysfunction occurs and has been associated with olfactory bulb abnormalities on MRI [56]. Thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section should be employed. Imaging protocols should cover the major anatomic divisions of the olfactory nerve and pathway, including the olfactory epithelium, which is located in the upper nasal cavity; the olfactory neurons and bulbs, located along the cribriform plates and inferior frontal lobes; and the olfactory pathways, which travel in portions of the temporal and frontal lobes [4].

US Neck

There is no relevant literature to support the use of ultrasound (US) neck in the evaluation of the olfactory nerve.

Variant 2: Unilateral isolated weakness of the mastication muscles, paralysis of the mastication muscles, sensory abnormalities of the face and head, facial numbness, or trigeminal neuralgia (trigeminal nerve, CN V). Initial imaging.

The trigeminal nerve (CN V) is the largest CN, providing general sensation to the face, part of the scalp, the nasal cavity, oral cavity, and teeth. It also provides branchial motor innervation to the muscles of mastication. The 4 central trigeminal nerve nuclei are located within the brainstem and include the mesencephalic nucleus, the principal sensory nucleus, the motor nucleus, and the spinal trigeminal tract and nucleus. The spinal trigeminal tract and nucleus extend from the midpons caudally into the upper cervical cord at the C2-C4 levels. The trigeminal nerve is divided into 3 main divisions, known as the ophthalmic (V1), maxillary (V2), and mandibular (V3) branches [3]. Symptoms of trigeminal neuropathy or neuropathic pain syndromes vary with the involved segment and division and may or may not include other sensory deficits (such as facial numbness) or motor deficits (such as weakness with mastication). In patients with clinical features of trigeminal neuropathy, initial imaging is performed along the entire course of the trigeminal nerve to evaluate for a causative lesion [57].

The trigeminal nerve can be affected by processes anywhere along its course from the brainstem to its peripheral branches; consequently, imaging should cover the entire course of the nerve. Brainstem lesions can affect the trigeminal nerve but rarely lead to isolated trigeminal neuropathy because of the close proximity of neural structures within the brainstem. In particular, multiple sclerosis can result in trigeminal neuralgia, necessitating imaging of the brainstem to look for demyelinating disease [58-61]. Other brainstem lesions that can affect the trigeminal nerve include infarction, hemorrhage, vascular lesions (such as compressing vascular loops, aneurysms, and

vertebrobasilar dolichoectasia), inflammatory and infectious conditions (such as meningitis, encephalitis, sarcoidosis, and demyelinating disease), and tumors (such as gliomas, lymphomas, and metastases) [59]. Tumors, vascular lesions, and inflammatory processes may also affect the cisternal, dural cave, cavernous, foraminal, and extracranial branches of the nerve as they traverse the Meckel cave, the pterygopalatine fossa, the orbit, the skull base, and the masticator space [59,62,63]. Perineural spread of tumor, discussed in Variant 10, can affect the trigeminal nerve anywhere along its course.

Trigeminal neuropathy should be distinguished from craniofacial trigeminal neuralgia, the latter typically associated with neurovascular compression of the centrally myelinated portion of the trigeminal nerve [59,63]. When secondary causes are excluded and the clinical picture is consistent with trigeminal neuralgia, MRI is useful to assess for neurovascular compression (ipsilateral to the patient's symptoms) [63].

CT Head

CT is a complementary study for evaluating the osseous integrity of the skull base and neural foramina. However, standard coverage with a CT Head is insufficient to evaluate the full extent of the trigeminal nerve divisions in the face. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CT Maxillofacial

Maxillofacial CT is a complementary study for evaluating the osseous integrity of the skull base and neural foramina, which can be accomplished without IV contrast. CT may be helpful for visualizing perineural fat planes, which can be distorted or obliterated in patients with lesions affecting the trigeminal nerve, although MRI offers improved detection of perineural tumor spread (discussed in Variant 10) compared with CT, which has a sensitivity and specificity of 88% and 89%, respectively [64-66]. CT with IV contrast offers the benefits of vascular and mucosal enhancement and may highlight enhancing lesions. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

Thin section contrast-enhanced navigation protocol CT is useful for noninvasive treatment planning in patients with known trigeminal neuralgia [67].

CTA Head

There is no relevant literature to support the use of CTA head in the initial evaluation of trigeminal neuropathy.

In patients with clinical features of trigeminal neuralgia, high-resolution combined MRI, MRA, and CTA may be used to assess for vascular loops compressing the fifth nerve. Anatomic and dedicated vascular imaging play a complementary role. Structural MRI evaluation of the nerve and its relationship to vasculature is more commonly reported in the literature, likely related to the improved soft tissue contrast of MRI compared with CT [23,68-76]. CTA has been used to characterize the relationship of arterial vasculature relative to the fifth nerve in patients with a clinical diagnosis of trigeminal neuralgia but is less commonly used than MRI and MRA because it does not provide simultaneous high-resolution imaging of the trigeminal nerve provided by MRI [77].

MRA Head

There is no relevant literature to support the use of MRA head in the initial evaluation of trigeminal neuropathy. In patients with clinical features of trigeminal neuralgia, MRA is commonly used as a complementary study to high-resolution MRI in assessing for vascular compression of the CN V with sensitivities of combined MRI with MRA ranging from 97% to 100%, specificities reported as 100%, and good to strong agreement found compared with surgical findings [69,71-73,76]. One group reported a lower sensitivity (50% at 3T and 33% at 1.5T) of combined high-resolution MRI with MRA for detection of neurovascular compression of CN V by smaller vessels such as the anterior inferior cerebellar artery [71].

MRI Head

MRI is useful for directly imaging the trigeminal nerve (CN V). Routine MRI head allows for direct visualization and excellent assessment of the brainstem and intracranial course of CN V but may not evaluate the entire extracranial course of the nerve, which can be simultaneously imaged with MRI orbits, face, and neck. Depending on institutional protocols, this may be achievable with MRI head, MRI orbits, face, and neck, or combined simultaneous use of these studies. Imaging protocols should use applicable thin-cut high-resolution techniques as described in the "Special Imaging Considerations" section through the course of CN V. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario.

In patients with clinical features of trigeminal neuralgia, 3-D heavily T2-weighted MRI sequences, MRA, and a combination of these techniques are commonly used noninvasive methods for characterizing the anatomy of vascular loops potentially compressing the fifth nerve and correlate well with surgical findings [23,68-76]. Congruence rates of imaging and intraoperative findings for neurovascular contact range from 83% to 100% [68-70,72,75,78]. Both false-positive and false-negative imaging studies occur when assessing for neurovascular contact in the setting of trigeminal neuralgia; consequently, MRI is supportive rather than diagnostic in selecting candidates for microvascular decompression and should be interpreted in the context of the site of symptoms [69,73-75,78]. Imaging evidence of vascular trigeminal nerve root compression, the degree of compression, location of contact, and nerve volume may have prognostic value [73,77,79-81]. Trigeminal nerve size measurements have been reported as smaller on the symptomatic side in trigeminal neuralgia as measured from thin-cut MRI [82-84]. Preoperative imaging is useful for surgical planning [85].

Advanced MRI techniques including diffusion tensor imaging (DTI), functional MRI, and voxel-based morphometry are being used to research the pathophysiology of trigeminal neuralgia. DTI metrics suggest microstructural tissue changes in symptomatic nerves in the setting of trigeminal neuralgia compared with asymptomatic nerves and may be useful for making treatment decisions [68,86-93]. Advanced MRI techniques have also detected structural alterations in gray matter and white matter in patients with trigeminal neuralgia [94-97].

MR neurography is an emerging technique that may be useful in characterizing the etiology of peripheral trigeminal neuropathy [98].

MRI Orbits, Face, and Neck

MRI is useful for directly imaging the trigeminal nerve (CN V). Depending on institutional protocols, this may be achievable with MRI head, MRI orbits, face, and neck, or combined simultaneous use of these studies. Imaging protocols should use applicable thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section through the course of CN V. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. In patients with trigeminal neuropathy, IV contrast should be administered. Noncontrast MRI may also be an alternate option for this clinical scenario.

In patients with clinical features of trigeminal neuralgia, 3-D heavily T2-weighted MRI sequences, MRA, and a combination of these techniques are commonly used noninvasive methods for characterizing the anatomy of vascular loops potentially compressing the cisternal segment of the fifth nerve and correlate well with surgical findings [23,68-76]. Congruence rates of imaging and intraoperative findings for neurovascular contact range from 83% to 100% [68-70,72,75,78]. Both false-positive and false-negative imaging studies occur when assessing for neurovascular contact in the setting of trigeminal neuralgia; consequently, MRI is supportive rather than diagnostic in selecting candidates for microvascular decompression and should be interpreted in the context of the site of symptoms [69,73-75,78]. Imaging evidence of vascular trigeminal nerve root compression, the degree of compression, location of contact, and nerve volume may have prognostic value [73,77,79-81]. Trigeminal nerve size measurements have been reported as smaller on the symptomatic side in trigeminal neuralgia as measured from thin-cut MRI [82-84]. Preoperative imaging is useful for surgical planning [85].

Advanced MRI techniques including DTI, functional MRI, and voxel-based morphometry are being used to research the pathophysiology of trigeminal neuralgia. DTI metrics suggest microstructural tissue changes in symptomatic nerves in the setting of trigeminal neuralgia compared with asymptomatic nerves and may be useful for making treatment decisions [68,86-93]. Advanced MRI techniques have also detected structural alterations in gray matter and white matter in patients with trigeminal neuralgia [94-97].

MR neurography is an emerging technique that may be useful in characterizing the etiology of peripheral trigeminal neuropathy [98].

FDG-PET/CT Skull Base to Mid-Thigh

There is no relevant literature to support the use of FDG-PET/CT skull base to mid-thigh in the evaluation of isolated trigeminal mononeuropathy or isolated unilateral trigeminal neuralgia.

US Neck

There is no relevant literature to support the use of US neck in the evaluation of isolated trigeminal mononeuropathy or isolated unilateral trigeminal neuralgia.

Variant 3: Unilateral isolated weakness of the facial expression, paralysis of the facial expression, hemifacial spasm, or Bell palsy (facial nerve, CN VII). Initial imaging.

The facial nerve (CN VII) is one of the most complex CNs and contains branchial motor (innervation to the muscles of facial expression), visceral motor (parasympathetic innervation to most of the glands of the head), general sensory (surface innervations to a small portion of the external ear and tympanic membrane), and special sensory (taste to the anterior two-thirds of the tongue) functions [2,99]. From its nucleus in the pons, the intraparenchymal fascicular or attached segment courses superiorly along the surface of the pons after which it turns anteriorly exiting the brainstem at the pontomedullary sulcus, which is referred to as the root exit point. Its parenchymal fascicular or attached segment courses superiorly along the surface of the pons after which it turns anteriorly, detaching from the pons at the root detachment point [5,100,101]. It then traverses the cerebellopontine angle in its cisternal segment, enters the internal auditory canal within its meatal segment, and courses through the temporal bone (which includes the labyrinthine, tympanic, mastoid segments, and geniculate ganglion). The facial nerve exits the temporal bone at the stylomastoid foramen, and the extracranial segment courses through the parotid gland.

Peripheral, nuclear, or infranuclear facial nerve paralysis presents as ipsilateral facial paralysis with involvement of the forehead (which is usually spared with a supranuclear facial nerve palsy) and can result from pathology affecting the facial nerve nucleus or any portion of the facial nerve after exiting the brainstem within its intracranial and extracranial segments. Within the pons, the facial nuclei can be affected by intra-axial conditions such as infarction, vascular malformations, tumors, and multiple sclerosis. Brainstem lesions are often accompanied by additional neurologic symptoms that can help localize the lesion clinically [102,103]. Rarely brainstem or cortical infarct can result in an isolated facial nerve palsy [102,103]. As the facial nerve exits the brainstem and courses within the cerebellopontine angle, the internal auditory canal, and through the temporal bone, it may be affected by facial and vestibular schwannomas, meningiomas, vascular lesions, inflammation, cholesteatomas, paragangliomas, epidermoid cysts, trauma, and intrinsic bone tumors. The extracranial facial nerve may be affected by inflammation, parotid tumors, conditions of the neighboring anatomic spaces, and skull base pathology including carcinomas, sarcomas, trauma, and inflammatory disease [99,102]. Perineural spread of a tumor can result in facial nerve palsy and is discussed in Variant 10 [104].

Facial nerve palsy can present with facial droop, pain around the jaw or ear, hyperacusis, tinnitus, reduced taste, and decreased lacrimation or salivation. Facial paralysis in the form of Bell palsy is the most common cause of acute peripheral facial nerve palsy, attributed to inflammation of the facial nerve, which may be idiopathic or due to herpes simplex virus. Most patients experience complete recovery of function by 6 months, which can be hastened with steroids [102]. In general, Bell palsy patients need not be imaged unless the symptoms are atypical, recurrent, or persist for 2 to 4 months [102].

Unilateral hyperactivity of the facial nerve can occur, resulting in the spasm of the facial musculature referred to as hemifacial spasm. Hemifacial spasm is most commonly the result of vascular compression of the facial nerve, usually within the centrally (oligodendrocyte) myelinated portion of the nerve [100].

CT Head

There is no relevant literature to support the use of routine CT head alone in the evaluation of unilateral isolated facial nerve palsy.

CT Temporal Bone

High-resolution thin-cut temporal bone CT provides complementary useful information to MRI by characterizing the osseous integrity of the temporal bone through which the facial nerve courses. CT imaging excels at osseous imaging and characterizes well temporal bone fractures, presurgical osseous anatomy, bony facial nerve canal involvement with inflammatory middle ear disease, facial canal foramina expansion, patterns of bone erosion, and intrinsic bone tumor matrices [2,99,105-108]. High-resolution temporal bone CT with thin sections should be obtained to evaluate the course of CN VII. Contrast may be useful in the setting of infection or when a tumor is suspected. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

High-resolution noncontrast CT of the temporal bone is useful to characterize temporal bone fractures including fractures through the facial nerve canal, but sensitivity for nondisplaced fracture has been shown to vary with fracture location and is lowest in the mastoid segment [105,108].

CT Maxillofacial

Temporal bone CT (complementary to MRI in patients with facial nerve palsy) is the preferred CT study and typically provides better spatial resolution than maxillofacial CT when assessing the fine detailed anatomy of the temporal bone and bony facial nerve canal. Maxillofacial CT provides better spatial resolution than head CT and, depending on institutional protocols, may provide sufficient spatial resolution to be complementary to MRI to characterize the osseous integrity of the temporal bone and assess the extracranial course of the facial nerve although the nerve itself is not directly imaged with CT [2,99,105-108]. Contrast may be useful when infection or a tumor is suspected. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CTA Head

There is no relevant literature to support the use of CTA head as an isolated study in the initial evaluation of acute unilateral facial nerve palsy. Rarely brainstem or cortical infarct can result in isolated facial nerve palsy, and if clinically suspected, CTA may be complementary to MRI in this clinical scenario [102,103] to characterize the vasculature.

CTA is uncommonly reported as a complementary study to thin-cut high-resolution MRI for characterizing the anatomy of vascular loops potentially compressing the centrally myelinated portion of the facial nerve [109].

FDG-PET/CT Skull Base to Mid-Thigh

There is no relevant literature to support the use of FDG-PET/CT skull base to mid-thigh in the evaluation of isolated unilateral facial nerve palsy or hemifacial spasm.

MRA Head

There is no relevant literature to support the use of MRA alone in the initial evaluation of acute unilateral facial nerve palsy. Rarely brainstem or cortical infarct can result in isolated facial nerve palsy, and if clinically suspected, MRA may be complementary to MRI in this clinical scenario [102,103] to characterize the vasculature.

MRA may be useful in the assessment of hemifacial spasm. In patients with clinical features of hemifacial spasm, MRA can be complementary to 3-D heavily T2-weighted MRI sequences for characterizing the anatomy of vascular loops potentially compressing the centrally myelinated portion of the facial nerve with sensitivity and accuracy reported as >95% and a good correlation with surgical findings [76,109,110].

MRI Head

MRI is the primary modality for evaluating the intracranial and extracranial portions of the facial nerve [20,104,111-116]. MRI head allows for direct visualization and excellent assessment of the brainstem and intracranial course of CN VII. Depending on institutional protocols, this may be achievable with MRI head, MRI orbits, face, and neck, or combined simultaneous use of these studies. Imaging protocols should use applicable thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section through the course of CN VII and its innervated structures. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario.

Volumetric and 3T imaging have been reported to provide improved visualization of the facial nerve and its surrounding perineural vascular plexus [7,19,111,113].

In general, Bell palsy patients need not be imaged unless the symptoms are atypical, recurrent, or persist for 2 to 4 months [102]. When imaging is considered, MRI is the method of choice. Variable abnormal enhancement patterns may be seen in the canalicular, labyrinthine, geniculate, tympanic, and mastoid portions of the nerve [106,117-122]. There is a lack of consensus regarding the prognostic value of MRI in Bell palsy [118-121], and MRI is most useful for excluding other causes of facial nerve palsy.

High-resolution thin-cut contrast-enhanced MRI is an especially useful method to evaluate for perineural spread of a tumor, which can affect CN VII and is discussed in Variant 10. Sensitivities for MRI detection of perineural spread of a tumor range from 73% to 100% and vary according to the nerve evaluated and timing of imaging relative to tissue sampling [65,104,123-125].

In patients with clinical features of hemifacial spasm, 3-D heavily T2-weighted MRI sequences and MRA are noninvasive methods for characterizing the anatomy of vascular loops potentially compressing the centrally myelinated portion of the facial nerve and correlate well with surgical findings [76,78,100,101,110]. Both false-positive and false-negative imaging studies occur when assessing for neurovascular contact in the setting of hemifacial spasm; consequently MRI is supportive rather than diagnostic in selecting candidates for microvascular

decompression [78,100]. In patients with failed initial microvascular decompression surgery, heavily T2-weighted MRI may be helpful to delineate the cause [101].

DTI is an advanced imaging technique that may be useful in assessing the facial nerve. Several studies suggest that DTI is accurate for the preoperative localization of the cisternal facial nerve to avoid iatrogenic injury in patients with cerebellopontine angle tumors [107,126-128]. Research indicates that DTI may have future use localizing the intraparotid facial nerve and detecting perineural spread of a tumor [129,130].

MRI Orbits, Face, and Neck

MRI is the primary modality for evaluating the intracranial and extracranial portions of the facial nerve [20,104,111-116]. Depending on institutional protocols, this may be achievable with MRI head, MRI orbits, face, and neck, or combined simultaneous use of these studies. Imaging protocols should use applicable thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section through the course of CN VII and its innervated structures. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario.

Volumetric and 3T imaging have been reported to provide improved visualization of the facial nerve and its surrounding perineural vascular plexus [7,19,111,113].

In general, Bell palsy patients do not need to be imaged unless the symptoms are atypical, recurrent, or persistent for 2 to 4 months [102]. When imaging is considered, MRI is the method of choice. Variable abnormal enhancement patterns may be seen in the canicular, labyrinthine, geniculate, tympanic, and mastoid portions of the nerve [106,117-122]. There is a lack of consensus regarding the prognostic value of MRI in Bell palsy [118-121], and MRI is most useful for excluding other causes of facial nerve palsy.

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In patients with clinical features of hemifacial spasm, 3-D heavily T2-weighted MRI sequences and MRA are noninvasive methods for characterizing the anatomy of vascular loops potentially compressing the centrally myelinated portion of the facial nerve and correlate well with surgical findings [76,78,100,101,110]. Both false-positive and false-negative imaging studies occur when assessing for neurovascular contact in the setting of hemifacial spasm; consequently MRI is supportive rather than diagnostic in selecting candidates for microvascular decompression [78,100]. In patients with failed initial microvascular decompression surgery, heavily T2-weighted MRI may be helpful to delineate the cause [101].

DTI is an advanced imaging technique that may be useful in assessing the facial nerve. Several studies suggest that DTI is accurate for the preoperative localization of the cisternal facial nerve to avoid iatrogenic injury in patients with cerebellopontine angle tumors [107,126-128]. Research indicates that DTI may have future use localizing the intraparotid facial nerve and detecting perineural spread of tumor [129,130].

US Neck

There is no relevant literature to support the use of US neck in the evaluation of isolated unilateral facial nerve palsy or hemifacial spasm.

Variant 4: Multiple different middle cranial nerve palsies (CN V-VII). Initial imaging.

Multiple CN nuclei are closely approximated within the brainstem, from which motor fibers originate and sensory fibers terminate. A small single brainstem lesion can produce severe and mixed neurologic deficits, including multiple cranial neuropathies, often times with some component of cerebellar, motor, or somatosensory deficit helping in clinically localizing the lesion. The pons connects the midbrain to the medulla. The dorsal pontine tegmentum contains white matter tracts and CN V through CN VIII nuclei, and the ventral pons contains corticospinal, corticobulbar, and corticopontine tracts [1,131]. After exiting the pons, the first and second divisions of the trigeminal nerve course along the lateral wall of the cavernous sinus, the abducens nerve courses through the cavernous sinus, and CNs VII and VIII extend through the cerebellopontine angle toward the porus acusticus. The third division of the trigeminal exits the skull base via foramen ovale.

Pontine lesions can affect CNs V, VI, VII, and/or VIII. Ischemic and hemorrhagic infarcts are the most frequent cause of acute brainstem syndromes with the pons most frequently affected [1,132]. Nonischemic lesions affecting

the brainstem include trauma, demyelinating disease, encephalitis, neoplasms, central pontine myelinolysis, neurodegenerative disorders, and syringobulbia [18,131,132]. Pontine syndromes causing variable involvement of CNs V-VIII, along with other neurologic deficits, include Millard-Gubler syndrome, Foville syndrome, locked-in syndrome, and facial colliculus syndrome [131].

The internal carotid artery with its surrounding sympathetic plexus and the abducens nerve extend through the center of the cavernous sinus, and the oculomotor, trochlear, and trigeminal nerves (1st and 2nd division) extend along the lateral wall of the cavernous sinus. Cavernous sinus lesions can result in isolated or multiple cranial neuropathies including involvement of the abducens and trigeminal nerves. Tumors, vascular lesions, infection, and inflammatory disorders can lead to cavernous sinus syndrome [18].

The cerebellopontine angle spans a craniocaudal extent that encompasses the cisternal portions of CNs V-X and is most frequently affected by benign tumors. Sensorineural hearing loss and tinnitus are the most common symptoms but can be accompanied by neuropathies of CNs V, VI, VII, IX, and X with larger tumors [18]. The combination of ipsilateral CN V and CN VII palsies raises concern for perineural spread of tumor, which is discussed in Variant 10. Additionally, leptomeningeal processes can lead to variable patterns of cranial neuropathy [18,132].

CT Head

Ischemic and hemorrhagic infarcts are the most frequent cause of acute brainstem syndromes, which can result in multiple middle CN palsies. When an acute brainstem syndrome is suspected clinically, particularly in the emergent setting, noncontrast head CT can be used as an initial screening study to assess for brainstem hemorrhage or acute infarct, although beam hardening artifact can limit sensitivity for detecting small acute brainstem infarcts by CT. To avoid delay of appropriate care, any patient with a new neurologic deficit suggesting stroke should have imaging guided by the ACR Appropriateness Criteria[®] topic on “[Cerebrovascular Disease](#)” [133]. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CT Maxillofacial

There is no relevant literature to support the use of maxillofacial CT in the initial evaluation of multiple middle CN palsies. Depending on institutional protocols used for high-resolution skull base imaging, maxillofacial CT may be complementary to an MRI exam if a skull base lesion is found to be the cause of multiple middle CN palsies and can be useful to characterize the osseous integrity of the skull base, intratumoral calcification, and skull base foramina [134]. Contrast should be administered. Noncontrast CT may also be an alternate option when used as complementary to the MRI. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CT Temporal Bone

There is no relevant literature to support the use of temporal bone CT in the initial evaluation of multiple middle CN palsies. Depending on institutional protocols used for high-resolution skull base imaging, temporal bone CT may be complementary to contrast-enhanced MRI if a skull base lesion is found to be the cause of multiple middle CN palsies and can be useful to characterize the osseous integrity of the base, intratumoral calcification, and skull base foramina [134]. Contrast should be administered. Noncontrast CT may also be an alternate option when used as complementary to the MRI. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CT Neck

There is no relevant literature to support routine use of neck CT in the initial evaluation of multiple middle cranial palsies. If perineural tumor spread (discussed in Variant 10) is suspected based on clinical features, most notably involving both CNs V and VII, neck CT is complementary to MRI for characterizing osseous changes of skull base neural foramina and may be useful to characterize perineural fat and stage the neck [64,66]. Contrast should be administered if possible. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CTA Head

There is no relevant literature to support routine use of CTA head in the initial evaluation of multiple middle cranial palsies. Ischemic and hemorrhagic infarcts are the most frequent cause of acute brainstem syndromes, which can result in multiple middle CN palsies [131,132,135]. CTA may be complementary to brain MRI or head CT to characterize the vasculature in these clinical scenarios.

FDG-PET/CT Skull Base to Mid-Thigh

There is no relevant literature to support the use FDG-PET/CT skull base to mid-thigh in the initial evaluation of multiple middle cranial palsies.

MRA Head

There is no relevant literature to support routine use of MRA head in the initial evaluation of multiple middle cranial palsies. Ischemic and hemorrhagic infarcts are the most frequent cause of acute brainstem syndromes, which can result in multiple middle CN palsies [131,132,135]. MRA may be complementary to MRI brain to characterize the vasculature in these clinical scenarios.

MRI Head

MRI is useful for investigating potential brainstem, cavernous sinus, and leptomeningeal processes leading to multiple middle CN palsies [2,83,131,134,136]. Imaging should focus on the brainstem, central skull base, and cavernous sinus. Depending on institutional protocols, this may be achievable with MRI head, MRI orbits, face, and neck, or combined simultaneous use of these studies. Imaging protocols should use applicable thin-cut high-resolution techniques described in the “Special Imaging Considerations” section of this document extending through the course of the affected CNs. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion.

DWI can be used to assess for acute brainstem infarction, cholesteatoma, and for characterizing tumor cellularity. False-negative DWI can occur in the setting of very small ischemic brainstem infarcts [137,138]. Thin-section coronal DWI or thinner axial DWI may improve sensitivity for detecting acute brainstem infarction, with nearly 25% of acute brainstem infarcts more easily seen on thin-cut coronal DWI compared with standard axial DWI in one study [138].

The combination of ipsilateral CN V and CN VII palsies raises concern for perineural spread of a tumor, which is discussed in Variant 10.

MRI Orbits, Face, and Neck

MRI is useful for investigating potential brainstem, cavernous sinus, and leptomeningeal processes leading to multiple middle CN palsies [2,83,131,134,136]. Imaging should focus on the brainstem, central skull base, and cavernous sinus. Depending on institutional protocols, this may be achievable with MRI head, MRI orbits, face, and neck, or combined simultaneous use of these studies. Imaging protocols should use applicable thin-cut high-resolution techniques described in the “Special Imaging Considerations” section of this document extending through the course of the affected CNs. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Contrast should be administered if possible. Noncontrast MRI may also be an alternate option for this clinical scenario.

DWI can be used to assess for acute brainstem infarction, cholesteatoma, and for characterizing tumor cellularity. False-negative DWI can occur in the setting of very small ischemic brainstem infarcts [137,138]. Thin-section coronal DWI or thinner axial DWI may improve sensitivity for detecting acute brainstem infarction, with nearly 25% of acute brainstem infarcts more easily seen on thin-cut coronal DWI compared with standard axial DWI in one study [138].

The combination of ipsilateral CN V and CN VII palsies raises concern for perineural spread of a tumor, which is discussed in Variant 10.

US Neck

There is no relevant literature to support the use of US neck in the initial evaluation of multiple middle cranial palsies.

Variant 5: Oropharyngeal neurogenic dysphagia or oropharyngeal pain (glossopharyngeal nerve, CN IX). Initial imaging.

The glossopharyngeal nerve (CN IX) arises in the medulla and is responsible for branchial motor innervation to the stylopharyngeus muscle, which elevates the palate, and visceral motor parasympathetic innervation to the parotid gland [2]. Visceral sensory innervation to the carotid sinus plays a role in regulating circulation and general and special sensory innervation supply sensation and taste to the posterior tongue. The nerve exits the jugular foramen in close proximity to the vagus (CN X) and the spinal accessory (CN XI) nerves [2,24]. Noniatrogenic isolated syndromes involving the glossopharyngeal nerve are very rare. Nerve root section or nerve ablation results in

variable degrees of dysphagia and ipsilateral sensory loss on the pharynx and posterior tongue [139]. Intra-parenchymal lesions can affect CN IX, including gliomas, lymphomas, metastases, vascular malformations, infarctions, demyelinating lesions, and inflammatory abnormalities. Leptomeningeal metastases, granulomatous disease, and even tortuosity or aneurysmal dilatation of vessels may affect the nerve as it enters the subarachnoid cistern. Lesions in the region of the posterior skull base and jugular foramen, such as metastases, schwannomas, paragangliomas, and meningiomas, usually also involve the other lower CNs [140,141]. Tonsillar pain syndromes and loss of a gag reflex accompanied by impaired taste and sensation along the posterior one-third of the tongue and palate may signal a glossopharyngeal nerve lesion [2,141]. Glossopharyngeal neuralgia presents as severe pain in the oropharyngeal and otic regions, classically triggered by swallowing, and is typically caused by neurovascular compression, although a minority of cases may be caused by trauma, an elongated calcified stylohyoid ligament, and neoplasms along the course of the nerve, including in the neck [142,143].

CT Head

There is no relevant literature to support the use of routine noncontrast head CT in the initial evaluation of oropharyngeal neurogenic dysphagia or oropharyngeal pain.

CT Maxillofacial

There is no relevant literature to support the use of maxillofacial CT in the initial evaluation of oropharyngeal neurogenic dysphagia or oropharyngeal pain.

CT Temporal Bone

There is no relevant literature to support the use of temporal bone CT in the initial evaluation of oropharyngeal neurogenic dysphagia or oropharyngeal pain.

CT Neck

Neck CT is complementary to MRI orbits, face, and neck in assessing patients with isolated CN IX palsy. Neck CT can delineate skull base erosion, identify deep space neck masses, intratumoral calcification, and the bony margins of the jugular foramen and nearby skull base foramina as well as image the extracranial course of the CN IX and its innervated structures in the pharynx and carotid space [144-148]. It does not image the intracranial course of CN IX or the brainstem well, which are best assessed with MRI. Imaging protocols should include thin-cut high-resolution bone windows through the posterior skull base. Contrast is strongly preferred for soft tissue characterization. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CT neck is also useful to characterize the anatomy of the stylohyoid ligament in patients with glossopharyngeal pain [142]. In cases of glossopharyngeal neuralgia, imaging should include the pharynx and larynx to exclude a mucosal neoplasm as an etiology.

CTA Head and Neck

There is no relevant literature to support the use of CTA in the initial evaluation of oropharyngeal neurogenic dysphagia or oropharyngeal pain.

FDG-PET/CT Skull Base to Mid-Thigh

There is no relevant literature to support the use of FDG-PET/CT skull base to mid-thigh in the initial evaluation of oropharyngeal neurogenic dysphagia or oropharyngeal pain.

MRA Head and Neck

There is no relevant literature to support routine use of MRA head and neck to assess vasculature in the initial evaluation of glossopharyngeal palsy, although MRA focused on the posterior skull base can be complementary to thin-cut high-resolution skull base technique, contrast-enhanced MRI orbits, face, and neck, or MRI head in patients with glossopharyngeal nerve palsy. Contrast-enhanced MRA focused on the posterior skull provides detailed imaging of the lower CNs within the jugular foramen and their relationship to the hypoglossal nerve as they exit the skull base, with CN IX reportedly well imaged in 100% of cases using contrast-enhanced high-resolution MRA [24,149].

MRA can be complementary to thin-cut high-resolution MRI sequences to assess for neurovascular compression in patients with glossopharyngeal neuralgia and has demonstrated agreement with surgically confirmed neurovascular compression in all patients in 2 small studies [143,149,150].

MRI Head

Although MRI head allows for direct visualization and excellent assessment of the brainstem and intracranial course of CN IX, it does not typically evaluate the entire extracranial course of the nerve, which can be simultaneously imaged with MRI orbits, face, and neck [2,24,143,149-151]. If institutional protocols require MRI head to assess the intracranial course of CN IX, the MRI head technique should focus on the posterior fossa and posterior skull base applying the relevant thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section of this document and can be simultaneously combined with MRI orbits, face, and neck to evaluate the extracranial course of the nerve. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario.

Thin-cut heavily T2-weighted contrast-enhanced modified balanced SSFP sequences and contrast-enhanced MRA focused on the posterior skull provide detailed imaging of the nerves within the jugular foramen and their relationship to the hypoglossal nerve as they exit the skull base, visualizing CN IX in 90% to 100% of imaged patients [24].

Various combinations of high-resolution 3-D T2-weighted imaging, MRA, and 3-D T1-weighted contrast-enhanced sequences can be used to assess for neurovascular compression [143,149-151] and have demonstrated agreement with surgical findings in all patients in two small studies [143,150].

MRI Orbits, Face, and Neck

MRI is particularly useful for directly imaging CN IX and allows for investigating possible lesions within the brainstem and along the intracranial and extracranial course of the nerve (an area not assessed on standard protocol MRI head) [2,24,143,149-151]. Depending on institutional protocols, this may be achievable with MRI orbits, face, and neck or MRI orbits, face, and neck performed simultaneously with MRI head. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario. Imaging protocols should use applicable thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section and focus on the posterior fossa, posterior skull base, and neck through the course of CN IX and its innervated structures. In cases of glossopharyngeal neuralgia, imaging should include the pharynx and larynx to exclude a mucosal neoplasm as an etiology.

Thin-cut heavily T2-weighted contrast-enhanced modified balanced SSFP sequences and contrast-enhanced MRA focused on the posterior skull provide detailed imaging of the nerves within the jugular foramen and their relationship to the hypoglossal nerve as they exit the skull base, visualizing CN IX in 90% to 100% of imaged patients [24].

Various combinations of high-resolution 3-D T2-weighted imaging, MRA, and 3-D T1-weighted contrast-enhanced sequences can be used to assess for neurovascular compression [143,149-151] and have demonstrated agreement with surgical findings in all patients in two small studies [143,150].

US Neck

There is no relevant literature to support the use of US neck in the initial evaluation of oropharyngeal neurogenic dysphagia or oropharyngeal pain.

Variant 6: Unilateral isolated palatal or vocal cord paralysis or both (vagal nerve, CN X). Initial imaging.

The vagus nerve (CN X) supplies visceral sensation to the pharynx, larynx, and viscera and general sensation to the ear. Branchial motor branches innervate muscles of the pharynx via the pharyngeal branches and the larynx via the superior and recurrent laryngeal nerves, and visceral motor branches play a predominant role in parasympathetic supply to the thorax and abdomen [2]. The vagus nerve runs the longest course in the body of any CN and is therefore vulnerable to a wide range of pathologies occurring throughout its trajectory from the posterior fossa and skull base to the neck, thorax, and abdomen. Within the neck it descends in the posterior carotid sheath. At the base of the neck, the recurrent laryngeal branch of the right vagus nerve turns upward and medial posterior to the subclavian artery, ascending in the tracheoesophageal groove. The left recurrent laryngeal nerve arises to the left of the aortic arch, turns upward in the aortopulmonary window beneath the ligamentum arteriosum, ascending in the left tracheoesophageal groove. The recurrent laryngeal nerves innervate all the laryngeal muscles, with the exception of the cricothyroid.

Isolated vagal palsy can be central or peripheral, due to complete vagal nerve dysfunction or isolated impairment of the recurrent laryngeal nerve, respectively. Lesions proximal to the pharyngeal branches cause ipsilateral palatal weakness. Because lesions anywhere along the course of the vagus nerve may potentially cause vocal cord paralysis,

the imaging protocol must visualize the full extent of the nerve from the skull base to the mid chest in this situation [2,152]. In the case of a central palsy, careful neurologic examination and patient history may help to locate the lesion to the central nervous system. Intramedullary lesions that can affect the vagus nucleus include demyelination, infarction, neoplasms, motor neuron disorders, and syringobulbia. Intracranial processes such as meningiomas, schwannomas, metastases, granulomatous disease, and infection may affect the nerve as it exits the medulla. Paragangliomas, schwannomas, meningiomas, and metastases involving the skull base may affect the vagus nerve and the neighboring glossopharyngeal nerve (CN IX) or accessory nerve (CN XI) by infiltration of fibers or by compression [2,152,153]. Direct and indirect signs of vocal cord paralysis can be seen on CT [152]. Optimal evidence-based imaging algorithms in the setting of unilateral vocal cord paralysis without an apparent cause are not established.

One of the most troubling symptoms of vagus dysfunction is vocal cord paralysis. Injury of the recurrent laryngeal branch in the neck or upper thorax can be idiopathic, due to iatrogenic injury from surgery or intubation, trauma, infection, inflammation, vascular lesions, and neoplasms arising in the neck and thorax. Moreover, thoracic causes of vocal cord paralysis, such as lung cancer, tuberculosis, and thoracic aortic aneurysm, are common [154-157].

CT Chest

Evaluation of the posterior fossa, neck, and upper chest is necessary for complete evaluation of the CN X, including the recurrent laryngeal nerve. Below the skull base, the vagus nerve is not directly imaged, although its course from the skull base to the carina can be imaged with contrast-enhanced neck CT extended through the aortopulmonary window. CT chest alone does cover the entire course of the vagus nerve, but it can be combined simultaneously and sequentially with CT neck to image the intrathoracic course of the vagus nerve. Preferably contrast should be administered. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

Although chest radiographs may detect large thoracic causes of vocal cord paralysis, chest CT is more sensitive, particularly for detecting aortopulmonary window and paratracheal lesions [158].

CT Head

There is no relevant literature to support the use of routine CT head alone in the evaluation of unilateral isolated vagal nerve palsy.

CT Maxillofacial

There is no relevant literature to support the use of maxillofacial CT in the evaluation of isolated CN X palsy. Contrast-enhanced neck CT extended through the aortopulmonary window is useful for initial evaluation of isolated CN X palsy and allows imaging of the osseous integrity of the skull base and assessment of the full extent of the recurrent laryngeal nerve.

CT Temporal Bone

There is no relevant literature to support the use of temporal bone CT in the evaluation of isolated CN X palsy.

CT Neck

Evaluation of the posterior fossa, neck, and upper chest is necessary for complete evaluation of the CN X, including the recurrent laryngeal nerve, and can be achieved well with contrast-enhanced neck CT extended through the AP window. Below the skull base, the vagus nerve is not directly visualized, although its course from the skull base to the carina can be imaged with neck CT. With its rapid scanning time, high-spatial resolution, and multiplanar capability, contrast-enhanced neck CT conveniently allows imaging of the jugular foramen, the full extracranial course of the vagus nerve, direct imaging of the larynx, and assessment of the full extent of the recurrent laryngeal nerve when extended through the aortopulmonary window. Imaging protocols should include thin-cut high-resolution bone windows through the posterior skull base. Contrast should be administered. Noncontrast neck CT offers limited evaluation of the neck and may be an alternate option for this clinical scenario. There is no relevant literature to support the use of combined pre- and postcontrast imaging. Direct and indirect signs of vocal cord paralysis can be seen on CT [152]. CT may differentiate traumatic arytenoid dislocation from neurogenic paralysis [153].

Optimal evidence-based imaging algorithms in the setting of unilateral vocal cord paralysis without an apparent cause are not established. Several studies support the use of CT for detecting a cause for unilateral vocal cord paralysis with diagnostic yields ranging from 23.5% to 47.5% [155,157]. One study found CT had a higher diagnostic yield of 40% among patients >65 years of age [131]. In patients with a diagnosis of idiopathic vocal cord

paralysis, repeat CT may be helpful to detect occult causes of paralysis [156]. Other studies report low diagnostic yields ranging from 0% to 6% [159-161].

Noninvasive virtual laryngoscopy using ultrahigh-resolution CT and cine MRI allowing the patient to perform phonation and breathing maneuvers during imaging are emerging technologies [162,163].

CTA Head and Neck

There is no relevant literature to support the use of CTA in the evaluation of unilateral isolated vagal nerve palsy.

FDG-PET/CT Skull Base to Mid-Thigh

FDG-PET/CT is not routinely used in the initial evaluation of vocal cord paralysis or vagus nerve palsy. FDG-PET/CT may be useful after initial cross-sectional imaging in patients with a known primary malignancy for both staging and therapy assessment [164]. It may yield false-positive findings if not interpreted carefully in the clinical context in patients with vocal cord palsy or following vocal fold augmentation [152,165-167].

MRA Head and Neck

There is no relevant literature to support the use of MRA in the evaluation of vasculature in unilateral isolated vagal nerve palsy.

MRI Head

Evaluation of the head, neck, and upper chest is necessary for complete evaluation of the CN X including the recurrent laryngeal nerve. For imaging of the intracranial and skull base segments of the vagus nerve, MRI is useful [24,83,143,149-151]. Although MRI head allows for direct visualization and excellent assessment of the brainstem and intracranial course of CN X, it does not evaluate the entire extracranial course of the nerve, which can be simultaneously imaged using the MRI orbits, face, and neck. If institutional protocols require MRI head to assess the intracranial course of CN X, the MRI head technique should focus on the posterior fossa and posterior skull base, applying the relevant thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section of this document and can be simultaneously combined with MRI orbits, face, and neck to evaluate the extracranial course of the nerve. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario.

Thin-cut heavily T2-weighted contrast-enhanced modified balanced SSFP sequences and contrast-enhanced MRA focused on the posterior skull provide detailed imaging of the lower nerves within the jugular foramen, including CN X, which is well visualized in 94% to 100% of imaged patients [24].

Below the skull base, the vagus nerve is not directly imaged by CT and MRI, though its course from the skull base to the carina can be imaged by either modality. Contrast-enhanced CT is typically accessible and conveniently allows complete and rapid imaging of the full extracranial course of the vagus nerve and direct imaging of the larynx and can be extended into the upper thorax to cover the entire course of the left recurrent laryngeal nerve.

MRI Orbits, Face, and Neck

Evaluation of the head, neck, and upper chest is necessary for complete evaluation of the CN X, including the recurrent laryngeal nerve. For imaging of the intracranial and skull base segments of the vagus nerve, MRI is useful [24,83,143,149-151]. Depending on institutional protocols, this may be achievable with MRI orbits, face, and neck or MRI orbits, face, and neck performed simultaneously with MRI head. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario. Imaging protocols should use applicable thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section and focus on the posterior fossa, posterior skull base, and neck through the course of CN X and its innervated structures.

Thin-cut heavily T2-weighted contrast-enhanced modified balanced SSFP sequences and contrast-enhanced MRA focused on the posterior skull provide detailed imaging of the lower nerves within the jugular foramen, including CN X, which is well visualized in 94% to 100% of imaged patients [24].

Below the skull base, the vagus nerve is not directly visible by CT and MRI, although its course from the skull base to the carina can be imaged by either modality. Contrast-enhanced CT is typically accessible and conveniently allows complete and rapid imaging of the full extracranial course of the vagus nerve and direct imaging of the larynx and can be extended into the upper thorax to cover the entire course of the left recurrent laryngeal nerve.

Radiography Chest

Chest radiography is not sufficient for the complete evaluation of thoracic causes that can contribute to vocal cord paralysis. Chest CT is more sensitive, particularly for detecting aortopulmonary window and paratracheal lesions [168]. Radiography can only reveal large lesions in the lung apex or mediastinum that may cause CN X deficits but fails to detect small clinically relevant lesions or pathology in the aortopulmonary window and the paratracheal region that can contribute to vocal cord paralysis [158,168].

US Neck

US is not able to fully image the entire course of CN X in the posterior fossa or the recurrent laryngeal nerve extending into the upper thorax, both of which can be simultaneously imaged with contrast-enhanced neck CT extended through the AP window [157,158]. US is a complementary modality that can be used to guide biopsies of lesions in the neck that contribute to a CN X palsy [158]. Transcutaneous laryngeal US is an innovative, noninvasive technique that may be suitable to directly assess vocal cord movement in select patients at high risk for iatrogenic vocal cord palsy following surgery or intubation [169-174].

Variant 7: Unilateral isolated weakness or paralysis of the sternocleidomastoid and trapezius muscles (accessory nerve, CN XI). Initial imaging.

The accessory nerve (CN XI) consists of a small cranial root originating from the nucleus ambiguus within the medulla oblongata and a large spinal root originating from the ventral horn of the spinal cord, between the C1 and C5 levels. The two components join and enter the pars vascularis of the jugular foramen [2]. The accessory nerve supplies portions of the sternocleidomastoid muscle and the upper portion of the trapezius muscle. Accessory nerve palsy is clinically manifested by weakness and atrophy of these muscles, causing decreased shoulder abduction, shoulder pain, cosmetic disfiguration, and disability. Isolated accessory nerve palsy is typically iatrogenic (due to injury from surgery or internal jugular vein cannulation) or may be due to trauma [175]. Accessory nerve palsy can also be accompanied by variable palsies of the glossopharyngeal (CN IX), vagus (CN X), and hypoglossal nerves (CN XII) in combined syndromes, particularly from lesions arising in the brainstem or jugular foramen, as discussed in Variant 9.

CT Head

There is no relevant literature to support the use of routine CT head alone in the evaluation of unilateral isolated CN XI palsy.

CT Maxillofacial

There is no relevant literature to support the use of maxillofacial CT in the evaluation of unilateral isolated CN XI palsy.

CT Temporal Bone

There is no relevant literature to support the use of temporal bone CT in the evaluation of unilateral isolated CN XI palsy.

CT Neck

Accessory nerve palsy is clinically manifested by weakness and atrophy of the sternocleidomastoid muscle and trapezius muscle and, as an isolated palsy, is typically due to iatrogenic injury. Contrast-enhanced CT neck may be useful to characterize lesions in the carotid space or posterior cervical space along the extracranial course of CN XI, as well as demonstrating atrophy of the trapezius or sternocleidomastoid muscles. MRI offers superior soft tissue contrast to demonstrate denervation changes or directly image features of neuritis and offers the benefit of being able to directly image the intracranial and high cervical portions of the nerve [2]. Imaging protocols should include thin-cut high-resolution bone windows through the posterior skull base. Noncontrast neck CT offers limited evaluation of the neck and may be an alternate option for this clinical scenario. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CTA Head and Neck

There is no relevant literature to support the use of CTA in the evaluation of unilateral isolated CN XI palsy.

MRA Head and Neck

There is no relevant literature to support the use of MRA in the evaluation of vasculature in unilateral isolated CN XI palsy.

MRI Head

MRI is useful for imaging the high cervical, intracranial, and extracranial portions of the accessory nerve as well as the brainstem [2,24,131,149]. Although MRI head allows for excellent assessment of the brainstem, intracranial, high cervical, and foraminal course of CN XI, it does not evaluate the entire extracranial course of the nerve, which can be simultaneously imaged with MRI orbits, face, and neck. If institutional protocols require complementary MRI head to assess the intracranial course of CN XI, the MRI head technique should focus on the posterior fossa and posterior skull base. The relevant thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section of this document should be utilized for optimal imaging along the course of the nerve and its innervated structures. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario.

Contrast-enhanced modified balanced SSFP sequences and MRA focused on the posterior skull provide detailed imaging of the lower CNs within the jugular foramen and as they exit the skull base [24]. Thin-cut high-resolution heavily T2-weighted imaging combined with MRA provides variable direct visualization of CN XI with the cranial segment identified in 88% of the sides and the spinal segment identified in 93% of the sides in one study [149]. Another study reported lower rates with the spinal root of CN XI visualized in 51% of subjects using contrast-enhanced modified balanced SSFP sequences [24].

MRI Orbits, Face, and Neck

MRI is useful for investigating the high cervical and intracranial portions of the accessory nerve as well as the brainstem and fully images the extracranial course of the accessory nerve innervating the sternocleidomastoid and trapezius muscles [2,24,131,149]. Depending on institutional protocols, this may be achievable with MRI orbits, face, and neck or MRI orbits, face, and neck performed simultaneously in conjunction with complementary MRI head. Imaging protocols should use applicable thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section and focus on the posterior fossa, posterior skull base, and neck through the course of CN XI and its innervated structures. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario.

Contrast-enhanced modified balanced SSFP sequences and MRA focused on the posterior skull provide detailed imaging of the lower CNs within the jugular foramen and as they exit the skull base [24]. Thin-cut high-resolution heavily T2-weighted imaging combined with MRA provides variable direct visualization of CN XI with the cranial segment identified in 88% of the sides and the spinal segment identified in 93% of the sides in one study [149]. Another study reported lower rates with the spinal root of CN XI visualized in 51% of subjects using contrast-enhanced modified balanced SSFP sequences [24].

MRI offers excellent soft tissue contrast to directly image features of neuritis or nerve sheath tumors as well as fully characterize the carotid space and posterior cervical space [2]. In patients with accessory nerve palsy, atrophy, and denervation, signal changes can be seen in the trapezius muscle on MRI [2,176].

US Neck

The accessory nerve can be directly imaged by US within the posterior cervical triangle and can provide a supportive role in the diagnosis of spinal accessory nerve injuries, although sensitivity may be user dependent [177-180].

Variant 8: Unilateral isolated weakness or paralysis of the tongue (hypoglossal nerve, CN XII). Initial imaging.

The hypoglossal nerve (CN XII) nucleus arises in the dorsal medulla. The hypoglossal rootlets traverse the premedullary cistern dorsal to the vertebral artery and then form the nerve, which enters the hypoglossal canal where it is surrounded by a venous plexus. The extracranial hypoglossal nerve travels caudally within the carotid space after which it courses anteriorly inferior to the hyoid where it distributes terminal branches providing somatic motor innervation to the intrinsic and extrinsic muscles of the tongue (except the palatoglossus muscle) and the infrahyoid strap muscles via the ansa cervicalis [2,13,181]. Hypoglossal nerve palsy typically presents clinically when a nuclear or infranuclear lesion is present and is characterized by dysarthria and deviation of the tongue to the side of the lesion upon tongue protrusion. Hypoglossal nerve palsy can occur because of injury of the nerve at any point along its course. There are a multitude of causes for hypoglossal nerve palsy including brainstem infarct, demyelinating disease, tumors, vascular lesions, trauma, inflammatory, and infectious processes. Nuclear lesions are usually accompanied by additional neurologic deficits indicative of brainstem involvement. Most hypoglossal nerve palsies are due to neoplasm involving the hypoglossal canal [2,182,183]. Within the extracranial segment, the most common causes of isolated hypoglossal nerve palsy are malignant tumors both along the course of CN XII

in the carotid space and in the sublingual space [184]. Dissection of the internal carotid artery can result in isolated acute CN XII palsy or multiple variable patterns of CN palsies including involvement of CN IX through CN XII [185-187]. Associate denervation signal alterations can be seen on MRI that vary with the duration of the palsy [2,186-188].

Hypoglossal nerve palsy can also be accompanied by variable palsies of the glossopharyngeal (CN IX), vagus (CN X), and accessory nerves (CN XI) in combined syndromes, particularly from lesions arising in the brainstem or jugular foramen, as discussed in Variant 9 [183].

CT Head

There is no relevant literature to support the use of routine CT head alone in the initial evaluation of unilateral isolated CN XII palsy.

CT Maxillofacial

There is no relevant literature to support the use of maxillofacial CT in the initial evaluation of unilateral isolated CN XII palsy.

CT Temporal Bone

There is no relevant literature to support the use of temporal bone CT in the initial evaluation of unilateral isolated CN XII palsy.

CT Neck

Evaluation of the entire course of the hypoglossal nerve is required with a hypoglossal nerve palsy. MRI is useful for evaluating hypoglossal nerve palsy, directly imaging the brainstem, the intracranial and skull base segments of the hypoglossal nerve and assessing for possible lesions along the extracranial segments of the nerve while providing improved soft tissue contrast [5,13,181,183,189]. The extracranial segment of the hypoglossal nerve within the suprahyoid neck is difficult to see with MRI and CT and inferred by knowledge of the nerve course and surrounding anatomy.

Neck CT provides complementary information to MRI, characterizing the osseous integrity of the hypoglossal canal and surrounding skull base [183]. Imaging protocols should include thin-cut high-resolution bone windows through the posterior skull base. Contrast should be administered, because tumors are the most common cause of isolated hypoglossal nerve palsy in this segment. Noncontrast CT may also be an alternate option for this clinical scenario. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

For detecting hypoglossal canal invasion by glomus tumors, CT had a sensitivity and specificity of 87.5% and 66.7%, respectively, compared with MRI which had a sensitivity of 100% and a specificity of 59% [183].

CTA Head and Neck

When dissection of the internal carotid artery is clinically suspected as a cause of acute isolated CN XII palsy, CTA may be useful to evaluate for dissection, particularly in the emergent setting. In a study comparing CTA with conventional angiography, CTA had a sensitivity of 66% when evaluating for blunt carotid vascular injury, with most false-negatives representing low grade injuries [190]. In a comparative study, multidetector CT/CTA demonstrated more features of cervical artery (internal carotid and vertebral arteries) dissection compared with MRI/MRA. There was no significant reader preference for MRI/MRA compared with CT/CTA when evaluating for internal carotid artery dissection. MRI/MRA provided additional characterization of ischemic complications [191]. In a retrospective review of the literature comparing test performance of MRI/MRA with CTA in the assessment of cervicocephalic arterial dissection with a reference standard of catheter angiography, the authors report MRI sensitivities and specificities of 50% to 79% and 67% to 99%, respectively, and CTA sensitivities and specificities of 51% to 98% and 67% to 100%, respectively (based on prospective studies) [192]. The authors concluded that the test characteristics of MRI/MRA and CTA for diagnosis of cervicocephalic arterial dissection were similar and study selection should be based on individual factors including urgency of imaging. A limitation of the literature assessing MRA and CTA for dissection is that most studies evaluate test performance in the clinical setting of traumatic dissection rather than spontaneous dissection. CTA is often used as a primary screening tool when carotid dissection is suspected in the emergent setting [190].

FDG-PET/CT Skull Base to Mid-Thigh

There is no relevant literature to support the use of FDG-PET/CT skull base to mid-thigh in the initial evaluation of unilateral isolated CN XII palsy.

MRA Head and Neck

When dissection of the internal carotid artery is suspected as a cause of acute isolated CN XII palsy, MRA may be useful to evaluate for dissection. In a retrospective review of the literature comparing test performance of MRI/MRA with CTA in the assessment of cervicocephalic arterial dissection with a reference standard of catheter angiography, the authors report MRI sensitivities and specificities of 50% to 79% and 67% to 99%, respectively, and CTA sensitivities and specificities of 51% to 98% and 67% to 100%, respectively (based on prospective studies) [192]. The authors concluded that the test characteristics of MRI/MRA and CTA for diagnosis of cervicocephalic arterial dissection were similar and study selection should be based on individual factors including urgency of imaging. In another comparative study, multidetector CT/CTA demonstrated more features of cervical artery (internal carotid and vertebral arteries) dissection compared with MRI/MRA. There was no significant reader preference for MRI/MRA compared with CT/CTA when evaluating for internal carotid artery dissection. MRI/MRA provided additional characterization of ischemic complications [191]. A limitation of the literature assessing MRA and CTA for dissection is that most studies evaluate test performance in the clinical setting of traumatic dissection rather than spontaneous dissection. CTA is often used as a primary screening tool when carotid dissection is suspected in the emergent setting [190].

MRI Head

Evaluation of the entire course of the hypoglossal nerve is required with a hypoglossal nerve palsy. MRI offers excellent soft tissue contrast and directly images the intracranial and skull base segment of CN XII [5,13,181,183,189]. MRI head does not typically evaluate the entire extracranial course of CN XII, which can be simultaneously imaged with MRI orbits, face, and neck. Depending on institutional protocols, MRI head may be complementary to MRI orbits, face, and neck to image the intracranial course of CN XII. The relevant thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section of this document should be utilized for optimal imaging along the course of the nerve and its innervated structures. Contrast should be administered because a tumor remains the most common cause of hypoglossal nerve palsy involving the intracranial, skull base, and cervical segments of the hypoglossal nerve. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario.

MRI had greater sensitivity (100%) but lower specificity (59%) for detecting hypoglossal canal invasion by glomus jugulare tumors when compared with CT (sensitivity of 87.5% and specificity of 66.7%) [183]. Denervation injury changes that occur within the tongue in the setting of hypoglossal nerve palsy vary with acuity and can be detected on MRI and CT, with MRI providing better soft tissue contrast [186-188]. Thin-cut heavily T2-weighted contrast-enhanced modified balanced SSFP sequences and contrast-enhanced MRA focused on the posterior skull provide detailed imaging of the lower nerves with 90% to 100% of imaged CN XII visible [24].

MRI Orbits, Face, and Neck

Evaluation of the entire course of the hypoglossal nerve is required with a hypoglossal nerve palsy. MRI is useful for directly imaging CN XII and allows for investigating possible lesions within the brainstem and along the intracranial, skull base, and extracranial segments of the nerve [5,13,181,183,189]. The extracranial segment of the hypoglossal nerve within the suprahyoid neck is difficult to see with MRI and CT and inferred by knowledge of the nerve course and surrounding anatomy. Depending on institutional protocols, this may be achievable with MRI orbits, face, and neck or MRI orbits, face, and neck performed simultaneously with MRI head. Imaging protocols should use applicable thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section and focus on the posterior fossa, posterior skull base, and neck through the course of CN XII and its innervated structures. Contrast should be administered if possible because a tumor remains the most common cause of hypoglossal nerve palsy. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario.

MRI had greater sensitivity (100%) but lower specificity (59%) for detecting hypoglossal canal invasion by glomus jugulare tumors when compared with CT (sensitivity of 87.5% and specificity of 66.7%) [183]. Denervation injury changes that occur within the tongue in the setting of hypoglossal nerve palsy vary with acuity and can be detected on MRI and CT, with MRI providing better soft tissue contrast [186-188]. Thin-cut heavily T2-weighted contrast-enhanced modified balanced SSFP sequences and contrast-enhanced MRA focused on the posterior skull provide detailed imaging of the lower nerves with 90% to 100% of imaged CN XII visible [24].

US Neck

There is no relevant literature to support the use of US neck in the initial evaluation of unilateral isolated CN XII palsy.

Variant 9: Multiple different lower cranial nerve palsies or combined lower cranial nerve syndromes (CN IX-XII). Initial imaging.

The medulla oblongata joins the pons to the spinal cord and is composed of a ventral portion and a dorsal tegmentum. The ventral portion contains the pyramids and olives, and the dorsal tegmentum contains CN IX through CN XII nuclei along with the white matter sensory tracts. Vascular supply to the medulla is from the anterior spinal artery, branches of the vertebral arteries, and the posterior inferior cerebellar arteries [1,131]. Outside the brainstem, CN IX through CN XI traverse the jugular foramen, and CN XII traverses the hypoglossal canal in the posterior skull base before extending caudally into the neck. Lesions affecting the medulla oblongata, the subarachnoid space, or the posterior skull base, including the jugular foramen and hypoglossal canal, can lead to multiple CN palsies affecting CN IX through CN XII in variable patterns. Patients with medullary lesions will typically have additional neurologic findings such as long tract signs, nystagmus, vertigo, ataxia, nausea, and vomiting. A thorough neurologic examination evaluating for these associated signs can localize a process to the brainstem [18,135]. Imaging protocols can then be tailored to evaluate the suspected region of anatomy affected.

Most brainstem syndromes are due to brainstem infarctions and hemorrhages [132]. Wallenberg syndrome or lateral medullary syndrome is typically due to occlusion of the posterior inferior cerebellar artery. Additional disorders that may affect the brainstem include demyelinating disease, primary brainstem tumors, metastasis, encephalitis, Arnold-Chiari malformations, and syringobulbia [18,132].

Multiple different jugular foramen syndromes are described based on variable patterns of CN palsies affecting CN IX through CN XII and include Vernet syndrome (IX, X, XI), Collet-Sicard syndrome (IX, X, XI, XII), and Villaret syndrome (IX, X, XI, XII and cervical sympathetic trunk). Lesions affecting the jugular foramen leading to jugular foramen syndromes include jugular foramen tumors (paragangliomas, schwannomas, and meningiomas), infection, leptomeningeal processes, metastasis, trauma, cholesteatoma, and vascular lesions [147,148,193].

Dissection of the internal carotid artery can result in isolated acute CN XII palsy or less commonly multiple variable patterns of CN palsies including involvement of CN IX through CN XII [185-187].

Leptomeningeal processes can lead to variable patterns of cranial neuropathy [18,132].

CT Head

There is no relevant literature to support the use of routine head CT in the initial evaluation of multiple lower CN palsies.

CT Maxillofacial

There is no relevant literature to support the use of maxillofacial CT in the initial evaluation of multiple lower CN palsies.

CT Temporal Bone

There is no relevant literature to support the use of temporal bone CT in the initial evaluation of multiple lower CN palsies.

CT Neck

Multiple different jugular foramen syndromes can result in variable patterns of CN palsies affecting CN IX through CN XII. Lesions involving the jugular foramen can extend caudally in the neck to involve the carotid space. CT is complementary to MRI in assessing jugular foramen lesions or for evaluating for carotid space lesions. Thin-cut high-resolution bone algorithm windows are useful to delineate skull base fractures, hyperostosis, skull base erosion, intratumoral calcification, and the bony margins of the jugular foramen and nearby skull base foramina [144-148]. Contrast should be administered. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CTA Head and Neck

Most brainstem syndromes are due to brainstem infarctions and hemorrhages, which are best imaged with MRI [132]. CTA may be complementary to CT or MRI to characterize the vasculature in these clinical scenarios.

When dissection of the internal carotid artery is clinically suspected as a cause of multiple lower CN palsies, CTA may be useful to evaluate for dissection, particularly in the emergent setting. In a study comparing CTA with conventional angiography, CTA had a sensitivity of 66% when evaluating for blunt carotid vascular injury, with most false-negatives representing low grade injuries [190]. In a comparative study, multidetector CT/CTA demonstrated more features of cervical artery (internal carotid and vertebral arteries) dissection compared with MRI/MRA. There was no significant reader preference for MRI/MRA compared with CT/CTA when evaluating for internal carotid artery dissection. MRI/MRA provided additional characterization of ischemic complications [191]. In a retrospective review of the literature comparing test performance of MRI/MRA with CTA in the assessment of cervicocephalic arterial dissection with a reference standard of catheter angiography, the authors report MRI sensitivities and specificities of 50% to 79% and 67% to 99%, respectively, and CTA sensitivities and specificities of 51% to 98% and 67% to 100%, respectively (based on prospective studies) [192]. The authors concluded that the test characteristics of MRI/MRA and CTA for diagnosis of cervicocephalic arterial dissection were similar and study selection should be based on individual factors including urgency of imaging. Limitations of the literature assessing MRA and CTA for dissection include most studies evaluate test performance in the clinical setting of traumatic dissection rather than spontaneous dissection. CTA is often used as a primary screening tool when carotid dissection is suspected in the emergent setting [190].

FDG-PET/CT Skull Base to Mid-Thigh

There is no relevant literature to support the use of FDG-PET/CT skull base to mid-thigh in the initial evaluation of multiple lower CN palsies.

MRA Head and Neck

Most brainstem syndromes are due to brainstem infarctions and hemorrhages [132]. MRA may be complementary to MRI to characterize the vasculature in these clinical scenarios. When dissection of the internal carotid artery is clinically suspected as a cause of multiple lower CN palsies clinically, MRA may be useful to evaluate for dissection although it is less accessible emergently compared with CTA. In a comparative study, multidetector CT/CTA demonstrated more features of cervical artery (internal carotid and vertebral arteries) dissection compared with MRI/MRA. There was no significant reader preference for MRI/MRA compared with CT/CTA when evaluating for internal carotid artery dissection. MRI/MRA provided additional characterization of ischemic complications [187]. In a retrospective review of the literature comparing test performance of MRI/MRA with CTA in the assessment of cervicocephalic arterial dissection with a reference standard of catheter angiography, the authors report MRI sensitivities and specificities of 50% to 79% and 67% to 99%, respectively, and CTA sensitivities and specificities of 51% to 98% and 67% to 100%, respectively (based on prospective studies) [188]. The authors concluded that the test characteristics of MRI/MRA and CTA for diagnosis of cervicocephalic arterial dissection were similar and study selection should be based on individual factors including urgency of imaging. Limitations of the literature assessing MRA and CTA for dissection include most studies evaluate test performance in the clinical setting of traumatic dissection rather than spontaneous dissection. CTA is often used as a primary screening tool when carotid dissection is suspected in the emergent setting [186].

MRI Head

MRI is useful for investigating for pathology involving the posterior skull base or posterior fossa, including the brainstem or leptomeningeal processes, leading to multiple lower CN palsies [2,24,83,131,134,136,149]. Although MRI head allows for direct visualization and excellent assessment of the brainstem and intracranial course of the lower CNs, it does not evaluate the entire extracranial course of the lower CNs, which can be simultaneously imaged using the MRI orbits, face, and neck. If institutional protocols require complementary MRI head to assess the intracranial course of the lower CNs, the MRI head technique should focus on the posterior fossa and posterior skull base applying the relevant thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section of this document. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario.

Thin-cut heavily T2-weighted contrast-enhanced modified balanced SSFP sequences and contrast-enhanced MRA focused on the posterior skull provide detailed imaging of the lower nerves within the jugular foramen, and their relationship to the hypoglossal nerve as they exit the skull base, with 90% to 100% of imaged CN IX, CN X, and CN XII visible [24]. There is variable direct visualization of CN XI with the cranial segment identified in 88% of the sides and the spinal segment identified in 93% of the sides in one study [149]. Another study reported lower rates with the spinal root of CN XI visualized in only 51% of subjects using heavily T2-weighted contrast-enhanced modified balanced SSFP sequences [24].

DWI can be used to assess for acute brainstem infarction, cholesteatoma, and for characterizing tumor cellularity. False-negative DWI can occur in the setting of very small ischemic brainstem infarcts [137,138]. Thinner slice axial DWI or thin-section coronal DWI may improve sensitivity for detecting small acute brainstem infarction, with nearly 25% of acute brainstem infarcts more easily seen on thin-cut coronal DWI compared with standard axial DWI in one study [138,194].

MRI Orbits, Face, and Neck

MRI is useful for investigating for pathology involving the posterior skull base or posterior fossa, including the brainstem or leptomeningeal processes, leading to multiple lower CN palsies [2,24,83,131,134,136,149]. Depending on institutional protocols, this may be achievable with MRI orbits, face, and neck or MRI orbits, face, and neck performed simultaneously with complementary MRI head. Imaging protocols should use applicable thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section and focus on the posterior fossa, posterior skull base, and neck through the course of the affected CNs. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may also be an alternate option for this clinical scenario.

Thin-cut heavily T2-weighted contrast-enhanced modified balanced SSFP sequences and contrast-enhanced MRA focused on the posterior skull provide detailed imaging of the lower nerves within the jugular foramen, and their relationship to the hypoglossal nerve as they exit the skull base, with 90% to 100% of imaged CN IX, CN X, and CN XII visible [24]. There is variable direct visualization of CN XI with the cranial segment identified in 88% of the sides and the spinal segment identified in 93% of the sides in one study [149]. Another study reported lower rates with the spinal root of CN XI visualized in only 51% of subjects using heavily T2-weighted contrast-enhanced modified balanced SSFP sequences [24].

DWI can be used to assess for acute brainstem infarction, cholesteatoma, and for characterizing tumor cellularity. False-negative DWI can occur in the setting of very small ischemic brainstem infarcts [137,138]. Thinner slice axial DWI or thin-section coronal DWI may improve sensitivity for detecting acute brainstem infarction, with nearly 25% of acute brainstem infarcts more easily seen on thin-cut coronal DWI compared with standard axial DWI in one study [138,194].

US Neck

There is no relevant literature to support the use of US neck in the initial evaluation of multiple lower CN palsies.

Variant 10: Head and neck cancer. Suspected or known perineural spread of tumor. Initial imaging.

The compact anatomy of the brainstem results in close proximity of multiple CN nuclei, and the complex anatomy of the head and neck results in close proximity and interconnection of multiple CNs, which can lead to multiple cranial neuropathies. Multiple middle and lower CN palsies are discussed in Variant 4 and 9, respectively. Perineural tumor spread can lead to isolated or multiple CN palsies and is the macroscopic spread of tumor along the course of a nerve distant from the site of the primary tumor as detected on imaging. It should be distinguished from perineural invasion, which is local invasion detected on histopathologic diagnosis at the primary tumor site [66,124]. Perineural tumor spread on imaging is associated with a worse prognosis [195]. The trigeminal (CN V) and facial (CN VII) nerves are most commonly affected by perineural tumor spread, although any nerve traveling in the vicinity of a malignancy may become involved. A multitude of tumors can result in perineural tumor spread, with squamous cell carcinoma (cutaneous and mucosal), adenoid cystic carcinoma, melanoma, lymphoma, basal cell carcinoma, and mucoepidermoid carcinoma most commonly occurring in the head and neck. Clues to perineural tumor spread include effacement of perineural and juxtaforaminal fat; asymmetric nerve enlargement or enhancement; osseous foraminal enlargement or erosion; and imaging features of denervation injury [65,66,104,123,124,196].

CT Head

There is no relevant literature to support the use of routine head CT in the initial evaluation of perineural spread of tumor.

CT Maxillofacial

There is no relevant literature to support the use of routine maxillofacial CT in the initial evaluation of perineural spread of tumor. Contrast-enhanced maxillofacial CT may be complementary to MRI and is useful for characterizing osseous changes of skull base and neural foramina; however, contrast-enhanced neck CT with thin-cut high-resolution bone algorithm windows through the skull base can provide similar information while

simultaneously staging the neck [64,66]. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CT Temporal Bone

There is no relevant literature to support the use of routine temporal bone CT in the initial evaluation of perineural spread of tumor. Temporal bone CT with IV contrast may be complementary to MRI and is useful for characterizing osseous changes of the skull base and neural foramina and may be useful to characterize perineural fat planes; however, contrast-enhanced neck CT with thin-cut high-resolution bone algorithm windows through the skull base can provide similar information while simultaneously staging the neck [64,66]. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CT Neck

MRI has better sensitivity for detection of perineural spread of tumor compared with CT and is the preferred study for initial assessment of perineural spread of tumor [65]. The trigeminal (CN V) and facial (CN VII) nerves are most commonly affected by perineural tumor spread and have branches extending into the face and neck. Neck CT may be complementary to MRI and is useful for characterizing osseous changes of the skull base and neural foramina and may be useful to characterize perineural fat planes while simultaneously staging the neck [64,66]. Contrast should be administered. There is no relevant literature to support the use of combined pre- and postcontrast imaging.

CTA Head and Neck

There is no relevant literature to support the use of CTA head and neck in the initial evaluation of perineural spread of tumor.

FDG-PET/CT Skull Base to Mid-Thigh

FDG-PET/CT is not routinely used in the initial evaluation of the perineural spread of tumor, although perineural tumor spread may be detected on metabolic imaging. FDG-PET/CT may be useful for staging and response assessment in patients with a known primary malignancy [195,197]. Contrast-enhanced PET/MR may detect perineural spread of a tumor and may be useful as an alternate to PET/CT in the evaluation of patients with head and neck cancers [164].

MRA Head and Neck

There is no relevant literature to support the use of MRA in the initial evaluation of perineural spread of tumor.

MRI Head

High-resolution thin-cut contrast-enhanced MRI is an especially useful method to evaluate for perineural spread of tumor. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Although MRI head allows for direct visualization and excellent assessment of the brainstem, intracranial, and foraminal course of CNs, it does not typically evaluate the entire extracranial course of the nerves, which can be simultaneously imaged using the MRI orbits, face, and neck. Depending on institutional protocols, MRI head may be complementary to MRI orbits, face, and neck to image the intracranial course of the relevant nerves, applying the relevant thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section of this document. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may be an alternate option for this clinical scenario.

Sensitivities for MRI detection of perineural spread of tumor range from 73% to 100% and vary according to the nerve evaluated and timing of imaging relative to tissue sampling [65,104,123-125]. MRI may underestimate microscopic perineural spread of a tumor [123-125]. Advanced imaging techniques such as tractography may be useful to detect perineural tumor spread but require further investigation [130]. Contrast-enhanced PET/MR may detect perineural spread of a tumor and may be useful as an alternate to PET/CT in the evaluation of patients with head and neck cancers [164].

MRI Orbits, Face, and Neck

High-resolution thin-cut contrast-enhanced MRI is an especially useful method to evaluate for perineural spread of a tumor. Depending on institutional protocols, this may be achievable with MRI orbits, face, and neck or MRI orbits, face, and neck performed simultaneously in conjunction with complementary MRI head. Imaging protocols should use applicable thin-cut high-resolution techniques as described in the “Special Imaging Considerations” section focused on the affected CNs. Pre- and postcontrast imaging provides the best opportunity to identify and characterize a lesion. Noncontrast MRI may be an alternate option for this clinical scenario.

Sensitivities for MRI detection of perineural spread of a tumor range from 73% to 100% and vary according to the nerve evaluated and timing of imaging relative to tissue sampling [65,104,123-125]. MRI may underestimate microscopic perineural spread of a tumor [123-125]. Advanced imaging techniques such as tractography may be useful to detect perineural tumor spread but require further investigation [130]. Contrast-enhanced PET/MR may detect perineural spread of a tumor and may be useful as an alternate to PET/CT in the evaluation of patients with head and neck cancers [164].

US Neck

There is no relevant literature to support the use of US neck in the initial evaluation of perineural spread of tumor.

Summary of Recommendations

- **Variante 1:** MRI orbits, face, and neck without and with IV contrast is usually appropriate for the initial imaging of patients with anosmia or other abnormalities of the sense of smell (olfactory nerve, CN I). The panel did not agree on recommending MRI head without and with IV contrast for this clinical scenario. Some institutions perform high-resolution imaging of CN I using MRI head without and with IV contrast, and this may be appropriate at some institutions.
- **Variante 2:** MRI head without and with IV contrast or MRI orbits, face, and neck without and with IV contrast is usually appropriate for the initial imaging of patients with unilateral isolated weakness of the mastication muscles, paralysis of the mastication muscles, sensory abnormalities of the face and head, facial numbness, or trigeminal neuralgia (trigeminal nerve, CN V). These procedures may be equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient's care) or complementary and may vary based on institutional protocols. The panel did not agree on recommending MRI head without IV contrast for this clinical scenario. There is insufficient medical literature to conclude whether or not these patients would benefit from MRI head without IV contrast.
- **Variante 3:** MRI head without and with IV contrast or MRI orbits, face, and neck without and with IV contrast is usually appropriate for the initial imaging of patients with unilateral isolated weakness of the facial expression, paralysis of the facial expression, hemifacial spasm, or Bell palsy (facial nerve, CN VII). These procedures may be equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient's care) or complementary and may vary based on institutional protocols. The panel did not agree on recommending MRI orbits, face, and neck without IV contrast for this clinical scenario. There is insufficient medical literature to conclude whether or not these patients would benefit from MRI orbits face neck without IV contrast. The panel did not agree on recommending MRA head without IV contrast, which may be complementary in the clinical setting of hemifacial spasm.
- **Variante 4:** MRI head without and with IV contrast or MRI orbits, face, and neck without and with IV contrast is usually appropriate for the initial imaging of patients with multiple different middle CN palsies (CNs V-VII). These procedures may be equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient's care) or complementary and may vary based on institutional protocols.
- **Variante 5:** MRI head without and with IV contrast or MRI orbits, face, and neck without and with IV contrast is usually appropriate for the initial imaging of patients with oropharyngeal pain (glossopharyngeal nerve, CN IX) or oropharyngeal neurogenic dysphagia. These procedures may be equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient's care) or complementary and may vary based on institutional protocols.
- **Variante 6:** MRI head without and with IV contrast or MRI orbits, face, and neck without and with IV contrast or CT neck with IV contrast is usually appropriate for the initial imaging of patients with unilateral isolated palatal or vocal cord paralysis or both (vagal nerve, CN X). These procedures may be equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient's care) or complementary and may vary based on institutional protocols and the clinical scenario.
- **Variante 7:** MRI head without and with IV contrast or MRI orbits face neck without and with IV contrast or CT neck with IV contrast is usually appropriate for the initial imaging of patients with unilateral isolated weakness or paralysis of the sternocleidomastoid and trapezius muscles (accessory nerve, CN XI). These procedures may be equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to

effectively manage the patient’s care) or complementary and may vary based on institutional protocols and the clinical scenario.

- **Variation 8:** MRI head without and with IV contrast or MRI orbits, face, and neck without and with IV contrast or CT neck with IV contrast is usually appropriate for the initial imaging of patients with unilateral isolated weakness or paralysis of the tongue (hypoglossal nerve, CN XII). These procedures may be equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient’s care) or complementary and may vary based on institutional protocols and the clinical scenario.
- **Variation 9:** MRI head without and with IV contrast or MRI orbits, face, and neck without and with IV contrast or CT neck with IV contrast is usually appropriate for the initial imaging of patients with multiple different lower CN palsies or combined lower CN syndromes (CNs IX-XII). These procedures may be equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient’s care) or complementary and may vary based on institutional protocols and the clinical scenario.
- **Variation 10:** MRI head without and with IV contrast or MRI orbits, face, and neck without and with IV contrast is usually appropriate for the initial imaging of patients with head and neck cancer and suspected or known perineural spread of a tumor. These procedures may be equivalent alternatives (ie, only one procedure will be ordered to provide the clinical information to effectively manage the patient’s care) or complementary and may vary based on institutional protocols.

Supporting Documents

The evidence table, literature search, and appendix for this topic are available at <https://acsearch.acr.org/list>. The appendix includes the strength of evidence assessment and the final rating round tabulations for each recommendation.

For additional information on the Appropriateness Criteria methodology and other supporting documents go to www.acr.org/ac.

Appropriateness Category Names and Definitions

Appropriateness Category Name	Appropriateness Rating	Appropriateness Category Definition
Usually Appropriate	7, 8, or 9	The imaging procedure or treatment is indicated in the specified clinical scenarios at a favorable risk-benefit ratio for patients.
May Be Appropriate	4, 5, or 6	The imaging procedure or treatment may be indicated in the specified clinical scenarios as an alternative to imaging procedures or treatments with a more favorable risk-benefit ratio, or the risk-benefit ratio for patients is equivocal.
May Be Appropriate (Disagreement)	5	The individual ratings are too dispersed from the panel median. The different label provides transparency regarding the panel’s recommendation. “May be appropriate” is the rating category and a rating of 5 is assigned.
Usually Not Appropriate	1, 2, or 3	The imaging procedure or treatment is unlikely to be indicated in the specified clinical scenarios, or the risk-benefit ratio for patients is likely to be unfavorable.

Relative Radiation Level Information

Potential adverse health effects associated with radiation exposure are an important factor to consider when selecting the appropriate imaging procedure. Because there is a wide range of radiation exposures associated with different diagnostic procedures, a relative radiation level (RRL) indication has been included for each imaging examination. The RRLs are based on effective dose, which is a radiation dose quantity that is used to estimate

population total radiation risk associated with an imaging procedure. Patients in the pediatric age group are at inherently higher risk from exposure, because of both organ sensitivity and longer life expectancy (relevant to the long latency that appears to accompany radiation exposure). For these reasons, the RRL dose estimate ranges for pediatric examinations are lower as compared with those specified for adults (see Table below). Additional information regarding radiation dose assessment for imaging examinations can be found in the ACR Appropriateness Criteria® [Radiation Dose Assessment Introduction](#) document [198].

Relative Radiation Level Designations		
Relative Radiation Level*	Adult Effective Dose Estimate Range	Pediatric Effective Dose Estimate Range
○	0 mSv	0 mSv
☼	<0.1 mSv	<0.03 mSv
☼☼	0.1-1 mSv	0.03-0.3 mSv
☼☼☼	1-10 mSv	0.3-3 mSv
☼☼☼☼	10-30 mSv	3-10 mSv
☼☼☼☼☼	30-100 mSv	10-30 mSv

*RRL assignments for some of the examinations cannot be made, because the actual patient doses in these procedures vary as a function of a number of factors (eg, region of the body exposed to ionizing radiation, the imaging guidance that is used). The RRLs for these examinations are designated as “Varies.”

References

1. Angeles Fernandez-Gil M, Palacios-Bote R, Leo-Barahona M, Mora-Encinas JP. Anatomy of the brainstem: a gaze into the stem of life. *Semin Ultrasound CT MR* 2010;31:196-219.
2. Soldatos T, Batra K, Blitz AM, Chhabra A. Lower cranial nerves. *Neuroimaging Clin N Am* 2014;24:35-47.
3. Joo W, Yoshioka F, Funaki T, Mizokami K, Rhoton AL, Jr. Microsurgical anatomy of the trigeminal nerve. *Clin Anat* 2014;27:61-88.
4. Lopez-Elizalde R, Campero A, Sanchez-Delgadillo T, Lemus-Rodriguez Y, Lopez-Gonzalez MI, Godinez-Rubi M. Anatomy of the olfactory nerve: A comprehensive review with cadaveric dissection. *Clin Anat* 2018;31:109-17.
5. Blitz AM, Choudhri AF, Chonka ZD, et al. Anatomic considerations, nomenclature, and advanced cross-sectional imaging techniques for visualization of the cranial nerve segments by MR imaging. *Neuroimaging Clin N Am* 2014;24:1-15.
6. Rhoton AL, Jr. Jugular foramen. *Neurosurgery* 2000;47:S267-85.
7. El-Khouly H, Fernandez-Miranda J, Rhoton AL, Jr. Blood supply of the facial nerve in the middle fossa: the petrosal artery. *Neurosurgery* 2008;62:ONS297-303; discussion ONS03-4.
8. Campero A, Campero AA, Martins C, Yasuda A, Rhoton AL, Jr. Surgical anatomy of the dural walls of the cavernous sinus. *J Clin Neurosci* 2010;17:746-50.
9. Joo W, Funaki T, Yoshioka F, Rhoton AL, Jr. Microsurgical anatomy of the infratemporal fossa. *Clin Anat* 2013;26:455-69.
10. Takemura Y, Inoue T, Morishita T, Rhoton AL, Jr. Comparison of microscopic and endoscopic approaches to the cerebellopontine angle. *World Neurosurg* 2014;82:427-41.
11. Yagmurlu K, Rhoton AL, Jr., Tanriover N, Bennett JA. Three-dimensional microsurgical anatomy and the safe entry zones of the brainstem. *Neurosurgery* 2014;10 Suppl 4:602-19; discussion 19-20.
12. Eduardo Corrales C, Mudry A, Jackler RK. Perpetuation of errors in illustrations of cranial nerve anatomy. *J Neurosurg* 2017;127:192-98.
13. Iwanaga J, Fisahn C, Alonso F, et al. Microsurgical Anatomy of the Hypoglossal and C1 Nerves: Description of a Previously Undescribed Branch to the Atlanto-Occipital Joint. *World Neurosurg* 2017;100:590-93.
14. FIPAT. *Terminologia Anatomica*. 2nd ed. Halifax, Nova Scotia, Canada: Federative International Programme for Anatomical Terminology; 2019.

15. Casselman J, Mermuys K, Delanote J, Ghekiere J, Coenegrachts K. MRI of the cranial nerves--more than meets the eye: technical considerations and advanced anatomy. *Neuroimaging Clin N Am* 2008;18:197-231, preceding x.
16. Kennedy TA, Corey AS, Policeni B, et al. ACR Appropriateness Criteria® Orbits Vision and Visual Loss. *J Am Coll Radiol* 2018;15:S116-S31.
17. Sharma A, Kirsch CFE, Aulino JM, et al. ACR Appropriateness Criteria® Hearing Loss and/or Vertigo. *J Am Coll Radiol* 2018;15:S321-S31.
18. Carroll CG, Campbell WW. Multiple cranial neuropathies. *Semin Neurol* 2009;29:53-65.
19. Chu J, Zhou Z, Hong G, et al. High-resolution MRI of the intraparotid facial nerve based on a microsurface coil and a 3D reversed fast imaging with steady-state precession DWI sequence at 3T. *AJNR Am J Neuroradiol* 2013;34:1643-8.
20. Zhao Y, Yang B. Value of Visualization of the Intraparotid Facial Nerve and Parotid Duct Using a Micro Surface Coil and Three-Dimensional Reversed Fast Imaging With Steady-State Precession and Diffusion-Weighted Imaging Sequence. *J Craniofac Surg* 2018;29:e754-e57.
21. Casselman JW, Kuhweide R, Deimling M, Ampe W, Dehaene I, Meeus L. Constructive interference in steady state-3DFT MR imaging of the inner ear and cerebellopontine angle. *AJNR Am J Neuroradiol* 1993;14:47-57.
22. Yousry I, Camelio S, Schmid UD, et al. Visualization of cranial nerves I-XII: value of 3D CISS and T2-weighted FSE sequences. *Eur Radiol* 2000;10:1061-7.
23. Blitz AM, Northcutt B, Shin J, et al. Contrast-Enhanced CISS Imaging for Evaluation of Neurovascular Compression in Trigeminal Neuralgia: Improved Correlation with Symptoms and Prediction of Surgical Outcomes. *AJNR Am J Neuroradiol* 2018;39:1724-32.
24. Linn J, Peters F, Moriggl B, Naidich TP, Bruckmann H, Yousry I. The jugular foramen: imaging strategy and detailed anatomy at 3T. *AJNR Am J Neuroradiol* 2009;30:34-41.
25. Adachi M, Kabasawa H, Kawaguchi E. Depiction of the cranial nerves within the brain stem with use of PROPELLER multishot diffusion-weighted imaging. *AJNR Am J Neuroradiol* 2008;29:911-2.
26. Wenz H, Al-Zghloul M, Hart E, Kurth S, Groden C, Forster A. Track-Density Imaging of the Human Brainstem for Anatomic Localization of Fiber Tracts and Nerve Nuclei in Vivo: Initial Experience with 3-T Magnetic Resonance Imaging. *World Neurosurg* 2016;93:286-92.
27. Chen RC, Khorsandi AS, Shatzkes DR, Holliday RA. The radiology of referred otalgia. *AJNR Am J Neuroradiol* 2009;30:1817-23.
28. Lee RK, Burns J, Ajam AA, et al. ACR Appropriateness Criteria® Seizures and Epilepsy. *J Am Coll Radiol* 2020;17:S293-S304.
29. Hoekman PK, Houlton JJ, Seiden AM. The utility of magnetic resonance imaging in the diagnostic evaluation of idiopathic olfactory loss. *Laryngoscope* 2014;124:365-8.
30. Hummel T, Landis BN, Huttenbrink KB. Smell and taste disorders. *GMS Curr Top Otorhinolaryngol Head Neck Surg* 2011;10:Doc04.
31. Alt JA, Mace JC, Buniel MC, Soler ZM, Smith TL. Predictors of olfactory dysfunction in rhinosinusitis using the brief smell identification test. *Laryngoscope* 2014;124:E259-66.
32. Patel RM, Pinto JM. Olfaction: anatomy, physiology, and disease. *Clin Anat* 2014;27:54-60.
33. Koenigkam-Santos M, Santos AC, Versiani BR, Diniz PR, Junior JE, de Castro M. Quantitative magnetic resonance imaging evaluation of the olfactory system in Kallmann syndrome: correlation with a clinical smell test. *Neuroendocrinology* 2011;94:209-17.
34. Zhang Z, Sun X, Wang C, Wang G, Zhao B. Magnetic Resonance Imaging Findings in Kallmann Syndrome: 14 Cases and Review of the Literature. *J Comput Assist Tomogr* 2016;40:39-42.
35. Kesslak JP, Cotman CW, Chui HC, et al. Olfactory tests as possible probes for detecting and monitoring Alzheimer's disease. *Neurobiol Aging* 1988;9:399-403.
36. Ponsen MM, Stoffers D, Booij J, van Eck-Smit BL, Wolters E, Berendse HW. Idiopathic hyposmia as a preclinical sign of Parkinson's disease. *Ann Neurol* 2004;56:173-81.
37. Baba T, Kikuchi A, Hirayama K, et al. Severe olfactory dysfunction is a prodromal symptom of dementia associated with Parkinson's disease: a 3 year longitudinal study. *Brain* 2012;135:161-9.
38. Jung HJ, Shin IS, Lee JE. Olfactory function in mild cognitive impairment and Alzheimer's disease: A meta-analysis. *Laryngoscope* 2019;129:362-69.
39. Hummel T, Urbig A, Huart C, Duprez T, Rombaux P. Volume of olfactory bulb and depth of olfactory sulcus in 378 consecutive patients with olfactory loss. *J Neurol* 2015;262:1046-51.

40. Saito T, Tsuzuki K, Yukitatsu Y, Sakagami M. Correlation between olfactory acuity and sinonasal radiological findings in adult patients with chronic rhinosinusitis. *Auris Nasus Larynx* 2016;43:422-8.
41. Chung MS, Choi WR, Jeong HY, Lee JH, Kim JH. MR Imaging-Based Evaluations of Olfactory Bulb Atrophy in Patients with Olfactory Dysfunction. *AJNR Am J Neuroradiol* 2018;39:532-37.
42. Wang J, Eslinger PJ, Doty RL, et al. Olfactory deficit detected by fMRI in early Alzheimer's disease. *Brain Res* 2010;1357:184-94.
43. Wang J, Sun X, Yang QX. Early Aging Effect on the Function of the Human Central Olfactory System. *J Gerontol A Biol Sci Med Sci* 2017;72:1007-14.
44. Pellegrino R, Hahner A, Bojanowski V, Hummel C, Gerber J, Hummel T. Olfactory function in patients with hyposmia compared to healthy subjects - An fMRI study. *Rhinology* 2016;54:374-81.
45. Vasavada MM, Martinez B, Wang J, et al. Central Olfactory Dysfunction in Alzheimer's Disease and Mild Cognitive Impairment: A Functional MRI Study. *J Alzheimers Dis* 2017;59:359-68.
46. Meles SK, Vadasz D, Renken RJ, et al. FDG PET, dopamine transporter SPECT, and olfaction: Combining biomarkers in REM sleep behavior disorder. *Mov Disord* 2017;32:1482-86.
47. Reichert JL, Postma EM, Smeets PAM, et al. Severity of olfactory deficits is reflected in functional brain networks-An fMRI study. *Hum Brain Mapp* 2018;39:3166-77.
48. Moon WJ, Park M, Hwang M, Kim JK. Functional MRI as an Objective Measure of Olfaction Deficit in Patients with Traumatic Anosmia. *AJNR Am J Neuroradiol* 2018;39:2320-25.
49. Negoias S, Croy I, Gerber J, et al. Reduced olfactory bulb volume and olfactory sensitivity in patients with acute major depression. *Neuroscience* 2010;169:415-21.
50. Wang J, You H, Liu JF, Ni DF, Zhang ZX, Guan J. Association of olfactory bulb volume and olfactory sulcus depth with olfactory function in patients with Parkinson disease. *AJNR Am J Neuroradiol* 2011;32:677-81.
51. Brodoehl S, Klingner C, Volk GF, Bitter T, Witte OW, Redecker C. Decreased olfactory bulb volume in idiopathic Parkinson's disease detected by 3.0-tesla magnetic resonance imaging. *Mov Disord* 2012;27:1019-25.
52. Hummel T, Haehner A, Hummel C, Croy I, Iannilli E. Lateralized differences in olfactory bulb volume relate to lateralized differences in olfactory function. *Neuroscience* 2013;237:51-5.
53. Croy I, Negoias S, Symmank A, Schellong J, Joraschky P, Hummel T. Reduced olfactory bulb volume in adults with a history of childhood maltreatment. *Chem Senses* 2013;38:679-84.
54. Negoias S, Hummel T, Symmank A, Schellong J, Joraschky P, Croy I. Olfactory bulb volume predicts therapeutic outcome in major depression disorder. *Brain Imaging Behav* 2016;10:367-72.
55. Rottstaedt F, Weidner K, Strauss T, et al. Size matters - The olfactory bulb as a marker for depression. *J Affect Disord* 2018;229:193-98.
56. Kandemirli SG, Altundag A, Yildirim D, Tekcan Sanli DE, Saatci O. Olfactory Bulb MRI and Paranasal Sinus CT Findings in Persistent COVID-19 Anosmia. *Acad Radiol* 2021;28:28-35.
57. Van Abel KM, Starkman S, O'Reilly AG, Price DL. Craniofacial pain secondary to occult head and neck tumors. *Otolaryngol Head Neck Surg* 2014;150:813-7.
58. Gass A, Kitchen N, MacManus DG, Moseley IF, Hennerici MG, Miller DH. Trigeminal neuralgia in patients with multiple sclerosis: lesion localization with magnetic resonance imaging. *Neurology* 1997;49:1142-4.
59. Borges A, Casselman J. Imaging the trigeminal nerve. *Eur J Radiol* 2010;74:323-40.
60. Lummel N, Mehrkens JH, Linn J, et al. Diffusion tensor imaging of the trigeminal nerve in patients with trigeminal neuralgia due to multiple sclerosis. *Neuroradiology* 2015;57:259-67.
61. Chen DQ, DeSouza DD, Hayes DJ, Davis KD, O'Connor P, Hodaie M. Diffusivity signatures characterize trigeminal neuralgia associated with multiple sclerosis. *Mult Scler* 2016;22:51-63.
62. Karkas A, Lamblin E, Meyer M, Gay E, Ternier J, Schmerber S. Trigeminal nerve deficit in large and compressive acoustic neuromas and its correlation with MRI findings. *Otolaryngol Head Neck Surg* 2014;151:675-80.
63. Wei Y, Zhao W, Pu C, et al. Clinical features and long-term surgical outcomes in 39 patients with tumor-related trigeminal neuralgia compared with 360 patients with idiopathic trigeminal neuralgia. *Br J Neurosurg* 2017;31:101-06.
64. Yamamoto M, Curtin HD, Suwansa-ard P, Sakai O, Sano T, Okano T. Identification of juxtaforaminal fat pads of the second division of the trigeminal pathway on MRI and CT. *AJR Am J Roentgenol* 2004;182:385-92.

65. Hanna E, Vural E, Prokopakis E, Carrau R, Snyderman C, Weissman J. The sensitivity and specificity of high-resolution imaging in evaluating perineural spread of adenoid cystic carcinoma to the skull base. *Arch Otolaryngol Head Neck Surg* 2007;133:541-5.
66. Badger D, Aygun N. Imaging of Perineural Spread in Head and Neck Cancer. *Radiol Clin North Am* 2017;55:139-49.
67. Park KJ, Kano H, Berkowitz O, et al. Computed tomography-guided gamma knife stereotactic radiosurgery for trigeminal neuralgia. *Acta Neurochir (Wien)* 2011;153:1601-9.
68. Lutz J, Linn J, Mehrkens JH, et al. Trigeminal neuralgia due to neurovascular compression: high-spatial-resolution diffusion-tensor imaging reveals microstructural neural changes. *Radiology* 2011;258:524-30.
69. Leal PR, Hermier M, Souza MA, Cristino-Filho G, Froment JC, Sindou M. Visualization of vascular compression of the trigeminal nerve with high-resolution 3T MRI: a prospective study comparing preoperative imaging analysis to surgical findings in 40 consecutive patients who underwent microvascular decompression for trigeminal neuralgia. *Neurosurgery* 2011;69:15-25; discussion 26.
70. Zhou Q, Liu ZL, Qu CC, Ni SL, Xue F, Zeng QS. Preoperative demonstration of neurovascular relationship in trigeminal neuralgia by using 3D FIESTA sequence. *Magn Reson Imaging* 2012;30:666-71.
71. Shimizu M, Imai H, Kagoshima K, Umezawa E, Shimizu T, Yoshimoto Y. Detection of compression vessels in trigeminal neuralgia by surface-rendering three-dimensional reconstruction of 1.5- and 3.0-T magnetic resonance imaging. *World Neurosurg* 2013;80:378-85.
72. Zeng Q, Zhou Q, Liu Z, Li C, Ni S, Xue F. Preoperative detection of the neurovascular relationship in trigeminal neuralgia using three-dimensional fast imaging employing steady-state acquisition (FIESTA) and magnetic resonance angiography (MRA). *J Clin Neurosci* 2013;20:107-11.
73. Maarbjerg S, Wolfram F, Gozalov A, Olesen J, Bendtsen L. Significance of neurovascular contact in classical trigeminal neuralgia. *Brain* 2015;138:311-9.
74. Yang D, Shen J, Xia X, et al. Preoperative evaluation of neurovascular relationship in trigeminal neuralgia by three-dimensional fast low angle shot (3D-FLASH) and three-dimensional constructive interference in steady-state (3D-CISS) MRI sequence. *Br J Radiol* 2018;91:20170557.
75. Jani RH, Hughes MA, Gold MS, Branstetter BF, Ligus ZE, Sekula RF, Jr. Trigeminal Nerve Compression Without Trigeminal Neuralgia: Intraoperative vs Imaging Evidence. *Neurosurgery* 2019;84:60-65.
76. Yao S, Zhang J, Zhao Y, et al. Multimodal Image-Based Virtual Reality Presurgical Simulation and Evaluation for Trigeminal Neuralgia and Hemifacial Spasm. *World Neurosurg* 2018;113:e499-e507.
77. Baliazin VA, Baliazina EV, Aksenov DP. Computed Tomography in the Diagnosis of Classical Trigeminal Neuralgia. *J Comput Assist Tomogr* 2017;41:521-27.
78. Hitchon PW, Zanaty M, Moritani T, et al. Microvascular decompression and MRI findings in trigeminal neuralgia and hemifacial spasm. A single center experience. *Clin Neurol Neurosurg* 2015;139:216-20.
79. Leal PR, Barbier C, Hermier M, Souza MA, Cristino-Filho G, Sindou M. Atrophic changes in the trigeminal nerves of patients with trigeminal neuralgia due to neurovascular compression and their association with the severity of compression and clinical outcomes. *J Neurosurg* 2014;120:1484-95.
80. Suzuki M, Yoshino N, Shimada M, et al. Trigeminal neuralgia: differences in magnetic resonance imaging characteristics of neurovascular compression between symptomatic and asymptomatic nerves. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2015;119:113-8.
81. Panczykowski DM, Frederickson AM, Hughes MA, Oskin JE, Stevens DR, Sekula RF, Jr. A Blinded, Case-Control Trial Assessing the Value of Steady State Free Precession Magnetic Resonance Imaging in the Diagnosis of Trigeminal Neuralgia. *World Neurosurg* 2016;89:427-33.
82. Erbay SH, Bhadelia RA, O'Callaghan M, et al. Nerve atrophy in severe trigeminal neuralgia: noninvasive confirmation at MR imaging--initial experience. *Radiology* 2006;238:689-92.
83. Blitz AM, Macedo LL, Chonka ZD, et al. High-resolution CISS MR imaging with and without contrast for evaluation of the upper cranial nerves: segmental anatomy and selected pathologic conditions of the cisternal through extraforaminal segments. *Neuroimaging Clin N Am* 2014;24:17-34.
84. Lee YJ, Moon HC, Tak S, Cheong C, Park YS. Atrophic Changes and Diffusion Abnormalities of Affected Trigeminal Nerves in Trigeminal Neuralgia Using 7-T MRI. *Stereotact Funct Neurosurg* 2019;97:169-75.
85. Sandell T, Ringstad GA, Eide PK. Usefulness of the endoscope in microvascular decompression for trigeminal neuralgia and MRI-based prediction of the need for endoscopy. *Acta Neurochir (Wien)* 2014;156:1901-9; discussion 09.
86. Lin W, Chen YL, Zhang QW. Vascular compression of the trigeminal nerve in asymptomatic individuals: a voxel-wise analysis of axial and radial diffusivity. *Acta Neurochir (Wien)* 2014;156:577-80.

87. Lutz J, Thon N, Stahl R, et al. Microstructural alterations in trigeminal neuralgia determined by diffusion tensor imaging are independent of symptom duration, severity, and type of neurovascular conflict. *J Neurosurg* 2016;124:823-30.
88. Lin W, Zhu WP, Chen YL, et al. Large-diameter compression arteries as a possible facilitating factor for trigeminal neuralgia: analysis of axial and radial diffusivity. *Acta Neurochir (Wien)* 2016;158:521-6.
89. Hung PS, Chen DQ, Davis KD, Zhong J, Hodaie M. Predicting pain relief: Use of pre-surgical trigeminal nerve diffusion metrics in trigeminal neuralgia. *Neuroimage Clin* 2017;15:710-18.
90. Moon HC, You ST, Baek HM, et al. 7.0Tesla MRI tractography in patients with trigeminal neuralgia. *Magn Reson Imaging* 2018;54:265-70.
91. Lee CC, Chong ST, Chen CJ, et al. The timing of stereotactic radiosurgery for medically refractory trigeminal neuralgia: the evidence from diffusion tractography images. *Acta Neurochir (Wien)* 2018;160:977-86.
92. Zhang Y, Mao Z, Cui Z, et al. Diffusion Tensor Imaging of Axonal and Myelin Changes in Classical Trigeminal Neuralgia. *World Neurosurg* 2018;112:e597-e607.
93. Tohyama S, Hung PS, Zhong J, Hodaie M. Early postsurgical diffusivity metrics for prognostication of long-term pain relief after Gamma Knife radiosurgery for trigeminal neuralgia. *J Neurosurg* 2018;131:539-48.
94. Li M, Yan J, Li S, et al. Reduced volume of gray matter in patients with trigeminal neuralgia. *Brain Imaging Behav* 2017;11:486-92.
95. Tian T, Guo L, Xu J, et al. Brain white matter plasticity and functional reorganization underlying the central pathogenesis of trigeminal neuralgia. *Sci Rep* 2016;6:36030.
96. Tsai YH, Yuan R, Patel D, et al. Altered structure and functional connection in patients with classical trigeminal neuralgia. *Hum Brain Mapp* 2018;39:609-21.
97. Liu J, Zhu J, Yuan F, Zhang X, Zhang Q. Abnormal brain white matter in patients with right trigeminal neuralgia: a diffusion tensor imaging study. *J Headache Pain* 2018;19:46.
98. Cox B, Zuniga JR, Panchal N, Cheng J, Chhabra A. Magnetic resonance neurography in the management of peripheral trigeminal neuropathy: experience in a tertiary care centre. *Eur Radiol* 2016;26:3392-400.
99. Ho ML, Juliano A, Eisenberg RL, Moonis G. Anatomy and pathology of the facial nerve. *AJR Am J Roentgenol* 2015;204:W612-9.
100. Sekula RF, Jr., Frederickson AM, Branstetter BF, et al. Thin-slice T2 MRI imaging predicts vascular pathology in hemifacial spasm: a case-control study. *Mov Disord* 2014;29:1299-303.
101. Hughes MA, Branstetter BF, Taylor CT, et al. MRI findings in patients with a history of failed prior microvascular decompression for hemifacial spasm: how to image and where to look. *AJNR Am J Neuroradiol* 2015;36:768-73.
102. Hohman MH, Hadlock TA. Etiology, diagnosis, and management of facial palsy: 2000 patients at a facial nerve center. *Laryngoscope* 2014;124:E283-93.
103. Wolf ME, Rausch HW, Eisele P, Habich S, Platten M, Alonso A. Acute Corticonuclear Tract Ischemic Stroke with Isolated Central Facial Palsy. *J Stroke Cerebrovasc Dis* 2019;28:495-98.
104. Nader ME, Ginsberg LE, Bell D, Roberts DB, Gidley PW. Evaluating Perineural Spread to the Intratemporal Facial Nerve on Magnetic Resonance Imaging. *Otolaryngol Head Neck Surg* 2019;160:1087-94.
105. Rajati M, Pezeshki Rad M, Irani S, Khorsandi MT, Motasaddi Zarandy M. Accuracy of high-resolution computed tomography in locating facial nerve injury sites in temporal bone trauma. *Eur Arch Otorhinolaryngol* 2014;271:2185-9.
106. Vianna M, Adams M, Schachern P, Lazarini PR, Paparella MM, Cureoglu S. Differences in the diameter of facial nerve and facial canal in bell's palsy--a 3-dimensional temporal bone study. *Otol Neurotol* 2014;35:514-8.
107. Li H, Wang L, Hao S, et al. Identification of the Facial Nerve in Relation to Vestibular Schwannoma Using Preoperative Diffusion Tensor Tractography and Intraoperative Tractography-Integrated Neuronavigation System. *World Neurosurg* 2017;107:669-77.
108. Chen Y, Zhang K, Xu Y, Che Y, Guan L, Li Y. Reliability of temporal bone high-resolution CT in patients with facial paralysis in temporal bone fracture. *Am J Otolaryngol* 2018;39:150-52.
109. Ohtani K, Mashiko T, Oguro K, et al. Preoperative Three-Dimensional Diagnosis of Neurovascular Relationships at the Root Exit Zones During Microvascular Decompression for Hemifacial Spasm. *World Neurosurg* 2016;92:171-78.

110. Jia JM, Guo H, Huo WJ, et al. Preoperative Evaluation of Patients with Hemifacial Spasm by Three-dimensional Time-of-Flight (3D-TOF) and Three-dimensional Constructive Interference in Steady State (3D-CISS) Sequence. *Clin Neuroradiol* 2016;26:431-38.
111. Hong HS, Yi BH, Cha JG, et al. Enhancement pattern of the normal facial nerve at 3.0 T temporal MRI. *Br J Radiol* 2010;83:118-21.
112. Lim HK, Lee JH, Hyun D, 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;33:779-83.
113. Dehkharghani S, Lubarsky M, Aiken AH, Kang J, Hudgins PA, Saindane AM. Redefining normal facial nerve enhancement: healthy subject comparison of typical enhancement patterns--unenhanced and contrast-enhanced spin-echo versus 3D inversion recovery-prepared fast spoiled gradient-echo imaging. *AJR Am J Roentgenol* 2014;202:1108-13.
114. Radhakrishnan R, Ahmed S, Tilden JC, Morales H. Comparison of normal facial nerve enhancement at 3T MRI using gadobutrol and gadopentetate dimeglumine. *Neuroradiol J* 2017;30:554-60.
115. Guenette JP, Seethamraju RT, Jayender J, Corrales CE, Lee TC. MR Imaging of the Facial Nerve through the Temporal Bone at 3T with a Noncontrast Ultrashort Echo Time Sequence. *AJNR Am J Neuroradiol* 2018;39:1903-06.
116. Guenette JP, Ben-Shlomo N, Jayender J, et al. MR Imaging of the Extracranial Facial Nerve with the CISS Sequence. *AJNR Am J Neuroradiol* 2019;40:1954-59.
117. Tien R, Dillon WP, Jackler RK. Contrast-enhanced MR imaging of the facial nerve in 11 patients with Bell's palsy. *AJNR Am J Neuroradiol* 1990;11:735-41.
118. Kress BP, Griesbeck F, Efinger K, et al. [The prognostic value of quantified MRI at an early stage of Bell's palsy]. *Rofo* 2002;174:426-32.
119. Kress B, Griesbeck F, Stippich C, Bahren W, Sartor K. Bell palsy: quantitative analysis of MR imaging data as a method of predicting outcome. *Radiology* 2004;230:504-9.
120. Song MH, Kim J, Jeon JH, et al. Clinical significance of quantitative analysis of facial nerve enhancement on MRI in Bell's palsy. *Acta Otolaryngol* 2008;128:1259-65.
121. Jun BC, Chang KH, Lee SJ, Park YS. Clinical feasibility of temporal bone magnetic resonance imaging as a prognostic tool in idiopathic acute facial palsy. *J Laryngol Otol* 2012;126:893-6.
122. Kuya J, Kuya K, Shinohara Y, et al. Usefulness of High-Resolution 3D Multi-Sequences for Peripheral Facial Palsy: Differentiation Between Bell's Palsy and Ramsay Hunt Syndrome. *Otol Neurotol* 2017;38:1523-27.
123. Nemzek WR, Hecht S, Gandour-Edwards R, Donald P, McKennan K. Perineural spread of head and neck tumors: how accurate is MR imaging? *AJNR Am J Neuroradiol* 1998;19:701-6.
124. Gandhi MR, Panizza B, Kennedy D. Detecting and defining the anatomic extent of large nerve perineural spread of malignancy: comparing "targeted" MRI with the histologic findings following surgery. *Head Neck* 2011;33:469-75.
125. Baulch J, Gandhi M, Sommerville J, Panizza B. 3T MRI evaluation of large nerve perineural spread of head and neck cancers. *J Med Imaging Radiat Oncol* 2015;59:578-85.
126. Hilly O, Chen JM, Birch J, et al. Diffusion Tensor Imaging Tractography of the Facial Nerve in Patients With Cerebellopontine Angle Tumors. *Otol Neurotol* 2016;37:388-93.
127. Borkar SA, Garg A, Mankotia DS, et al. Prediction of facial nerve position in large vestibular schwannomas using diffusion tensor imaging tractography and its intraoperative correlation. *Neurol India* 2016;64:965-70.
128. Savardekar AR, Patra DP, Thakur JD, et al. Preoperative diffusion tensor imaging-fiber tracking for facial nerve identification in vestibular schwannoma: a systematic review on its evolution and current status with a pooled data analysis of surgical concordance rates. *Neurosurg Focus* 2018;44:E5.
129. Attye A, Karkas A, Tropres I, et al. Parotid gland tumours: MR tractography to assess contact with the facial nerve. *Eur Radiol* 2016;26:2233-41.
130. Rouchy RC, Attye A, Medici M, et al. Facial nerve tractography: A new tool for the detection of perineural spread in parotid cancers. *Eur Radiol* 2018;28:3861-71.
131. Sciacca S, Lynch J, Davagnanam I, Barker R. Midbrain, Pons, and Medulla: Anatomy and Syndromes. *Radiographics* 2019;39:1110-25.
132. Keane JR. Multiple cranial nerve palsies: analysis of 979 cases. *Arch Neurol* 2005;62:1714-7.

133. American College of Radiology. ACR Appropriateness Criteria®: Cerebrovascular Disease. Available at: <https://acsearch.acr.org/docs/69478/Narrative/>. Accessed March 31, 2022.
134. Bag AK, Chapman PR. Neuroimaging: intrinsic lesions of the central skull base region. *Semin Ultrasound CT MR* 2013;34:412-35.
135. Querol-Pascual MR. Clinical approach to brainstem lesions. *Semin Ultrasound CT MR* 2010;31:220-9.
136. Yagi A, Sato N, Takahashi A, et al. Added value of contrast-enhanced CISS imaging in relation to conventional MR images for the evaluation of intracavernous cranial nerve lesions. *Neuroradiology* 2010;52:1101-9.
137. Fukuoka T, Takeda H, Dembo T, et al. Clinical review of 37 patients with medullary infarction. *J Stroke Cerebrovasc Dis* 2012;21:594-9.
138. Felfeli P, Wenz H, Al-Zghloul M, Groden C, Forster A. Combination of standard axial and thin-section coronal diffusion-weighted imaging facilitates the diagnosis of brainstem infarction. *Brain Behav* 2017;7:e00666.
139. Giorgi C, Broggi G. Surgical treatment of glossopharyngeal neuralgia and pain from cancer of the nasopharynx. A 20-year experience. *J Neurosurg* 1984;61:952-5.
140. Fayad JN, Keles B, Brackmann DE. Jugular foramen tumors: clinical characteristics and treatment outcomes. *Otol Neurotol* 2010;31:299-305.
141. Vorasubin N, Sang UH, Mafee M, Nguyen QT. Glossopharyngeal schwannomas: a 100 year review. *Laryngoscope* 2009;119:26-35.
142. Kent DT, Rath TJ, Snyderman C. Conventional and 3-Dimensional Computerized Tomography in Eagle's Syndrome, Glossopharyngeal Neuralgia, and Asymptomatic Controls. *Otolaryngol Head Neck Surg* 2015;153:41-7.
143. Zhao H, Zhang X, Zhu J, Tang YD, Li ST. Microvascular Decompression for Glossopharyngeal Neuralgia: Long-Term Follow-Up. *World Neurosurg* 2017;102:151-56.
144. Valavanis A, Schubiger O, Oguz M. High-resolution CT investigation of nonchromaffin paragangliomas of the temporal bone. *AJNR Am J Neuroradiol* 1983;4:516-9.
145. Eldevik OP, Gabrielsen TO, Jacobsen EA. Imaging findings in schwannomas of the jugular foramen. *AJNR Am J Neuroradiol* 2000;21:1139-44.
146. Macdonald AJ, Salzman KL, Harnsberger HR, Gilbert E, Shelton C. Primary jugular foramen meningioma: imaging appearance and differentiating features. *AJR Am J Roentgenol* 2004;182:373-7.
147. Vogl TJ, Bisdas S. Differential diagnosis of jugular foramen lesions. *Skull Base* 2009;19:3-16.
148. Wanna GB, Sweeney AD, Haynes DS, Carlson ML. Contemporary management of jugular paragangliomas. *Otolaryngol Clin North Am* 2015;48:331-41.
149. Linn J, Moriggl B, Schwarz F, et al. Cisternal segments of the glossopharyngeal, vagus, and accessory nerves: detailed magnetic resonance imaging-demonstrated anatomy and neurovascular relationships. *J Neurosurg* 2009;110:1026-41.
150. Gaul C, Hastreiter P, Duncker A, Naraghi R. Diagnosis and neurosurgical treatment of glossopharyngeal neuralgia: clinical findings and 3-D visualization of neurovascular compression in 19 consecutive patients. *J Headache Pain* 2011;12:527-34.
151. Jani RH, Hughes MA, Ligus ZE, Nikas A, Sekula RF. MRI Findings and Outcomes in Patients Undergoing Microvascular Decompression for Glossopharyngeal Neuralgia. *J Neuroimaging* 2018;28:477-82.
152. Paquette CM, Manos DC, Psooy BJ. Unilateral vocal cord paralysis: a review of CT findings, mediastinal causes, and the course of the recurrent laryngeal nerves. *Radiographics* 2012;32:721-40.
153. Rubin AD, Hawkshaw MJ, Moyer CA, Dean CM, Sataloff RT. Arytenoid cartilage dislocation: a 20-year experience. *J Voice* 2005;19:687-701.
154. Rosenthal LH, Benninger MS, Deeb RH. Vocal fold immobility: a longitudinal analysis of etiology over 20 years. *Laryngoscope* 2007;117:1864-70.
155. Hojjat H, Svider PF, Folbe AJ, et al. Cost-effectiveness of routine computed tomography in the evaluation of idiopathic unilateral vocal fold paralysis. *Laryngoscope* 2017;127:440-44.
156. Noel JE, Jeffery CC, Damrose E. Repeat Imaging in Idiopathic Unilateral Vocal Fold Paralysis: Is It Necessary? *Ann Otol Rhinol Laryngol* 2016;125:1010-14.
157. Bilici S, Yildiz M, Yigit O, Misir E. Imaging Modalities in the Etiologic Evaluation of Unilateral Vocal Fold Paralysis. *J Voice* 2019;33:813 e1-13 e5.
158. Kang BC, Roh JL, Lee JH, et al. Usefulness of computed tomography in the etiologic evaluation of adult unilateral vocal fold paralysis. *World J Surg* 2013;37:1236-40.

159. Badia PI, Hillel AT, Shah MD, Johns MM, 3rd, Klein AM. Computed tomography has low yield in the evaluation of idiopathic unilateral true vocal fold paresis. *Laryngoscope* 2013;123:204-7.
160. Chen DW, Young A, Donovan DT, Ongkasuwan J. Routine computed tomography in the evaluation of vocal fold movement impairment without an apparent cause. *Otolaryngol Head Neck Surg* 2015;152:308-13.
161. Paddle PM, Mansor MB, Song PC, Franco RA, Jr. Diagnostic Yield of Computed Tomography in the Evaluation of Idiopathic Vocal Fold Paresis. *Otolaryngol Head Neck Surg* 2015;153:414-9.
162. Baki MM, Menys A, Atkinson D, et al. Feasibility of vocal fold abduction and adduction assessment using cine-MRI. *Eur Radiol* 2017;27:598-606.
163. Miyamoto M, Ohara A, Arai T, et al. Three-dimensional imaging of vocalizing larynx by ultra-high-resolution computed tomography. *Eur Arch Otorhinolaryngol* 2019;276:3159-64.
164. Kuhn FP, Hullner M, Mader CE, et al. Contrast-enhanced PET/MR imaging versus contrast-enhanced PET/CT in head and neck cancer: how much MR information is needed? *J Nucl Med* 2014;55:551-8.
165. Heller MT, Meltzer CC, Fukui MB, et al. Superphysiologic FDG Uptake in the Non-Paralyzed Vocal Cord. Resolution of a False-Positive PET Result with Combined PET-CT Imaging. *Clin Positron Imaging* 2000;3:207-11.
166. Kamel EM, Goerres GW, Burger C, von Schulthess GK, Steinert HC. Recurrent laryngeal nerve palsy in patients with lung cancer: detection with PET-CT image fusion -- report of six cases. *Radiology* 2002;224:153-6.
167. Grant N, Wong RJ, Kraus DH, Schoder H, Branski RC. Positron-emission tomography enhancement after vocal fold injection medialization. *Ear Nose Throat J* 2017;96:218-24.
168. Song SW, Jun BC, Cho KJ, Lee S, Kim YJ, Park SH. CT evaluation of vocal cord paralysis due to thoracic diseases: a 10-year retrospective study. *Yonsei Med J* 2011;52:831-7.
169. Koszka C, Leichtfried FE, Wiche G. Identification and spatial arrangement of high molecular weight proteins (Mr 300 000-330 000) co-assembling with microtubules from a cultured cell line (rat glioma C6). *Eur J Cell Biol* 1985;38:149-56.
170. Woo JW, Park I, Choe JH, Kim JH, Kim JS. Comparison of ultrasound frequency in laryngeal ultrasound for vocal cord evaluation. *Surgery* 2017;161:1108-12.
171. Masood MM, Huang B, Goins A, Hackman TG. Anatomic factors affecting the use of ultrasound to predict vocal fold motion: A pilot study. *Am J Otolaryngol* 2018;39:413-17.
172. Wong KP, Au KP, Lam S, Chang YK, Lang BHH. Vocal Cord Palsies Missed by Transcutaneous Laryngeal Ultrasound (TLUSG): Do They Experience Worse Outcomes? *World J Surg* 2019;43:824-30.
173. Ruan Z, Ren R, Dong W, et al. Assessment of vocal cord movement by ultrasound in the ICU. *Intensive Care Med* 2018;44:2145-52.
174. Shah MK, Ghai B, Bhatia N, Verma RK, Panda NK. ()Comparison of transcutaneous laryngeal ultrasound with video laryngoscope for assessing the vocal cord mobility in patients undergoing thyroid surgery. *Auris Nasus Larynx* 2019;46:593-98.
175. Kim DH, Cho YJ, Tiel RL, Kline DG. Surgical outcomes of 111 spinal accessory nerve injuries. *Neurosurgery* 2003;53:1106-12; discussion 02-3.
176. Li AE, Greditzer HGt, Melisaratos DP, Wolfe SW, Feinberg JH, Sneag DB. MRI findings of spinal accessory neuropathy. *Clin Radiol* 2016;71:316-20.
177. Canella C, Demondion X, Abreu E, Marchiori E, Cotten H, Cotten A. Anatomical study of spinal accessory nerve using ultrasonography. *Eur J Radiol* 2013;82:56-61.
178. Mirjalili SA, Muirhead JC, Stringer MD. Ultrasound visualization of the spinal accessory nerve in vivo. *J Surg Res* 2012;175:e11-6.
179. Cesmebasi A, Smith J, Spinner RJ. Role of Sonography in Surgical Decision Making for Iatrogenic Spinal Accessory Nerve Injuries: A Paradigm Shift. *J Ultrasound Med* 2015;34:2305-12.
180. Shen J, Chen W, Ye X, et al. Ultrasound in the management of iatrogenic spinal accessory nerve palsy at the posterior cervical triangle area. *Muscle Nerve* 2019;59:64-69.
181. Yousry I, Moriggl B, Schmid UD, et al. Detailed anatomy of the intracranial segment of the hypoglossal nerve: neurovascular relationships and landmarks on magnetic resonance imaging sequences. *J Neurosurg* 2002;96:1113-22.
182. Keane JR. Twelfth-nerve palsy. Analysis of 100 cases. *Arch Neurol* 1996;53:561-6.
183. Gursoy M, Orru E, Blitz AM, Carey JP, Olivi A, Yousem DM. Hypoglossal canal invasion by glomus jugulare tumors: clinico-radiological correlation. *Clin Imaging* 2014;38:655-8.

184. Guarnizo A, Glikstein R, Torres C. Imaging Features of isolated hypoglossal nerve palsy. *J Neuroradiol* 2020;47:136-50.
185. Mokri B, Silbert PL, Schievink WI, Piepgras DG. Cranial nerve palsy in spontaneous dissection of the extracranial internal carotid artery. *Neurology* 1996;46:356-9.
186. Learned KO, Thaler ER, O'Malley BW, Jr., Grady MS, Loevner LA. Hypoglossal nerve palsy missed and misinterpreted: the hidden skull base. *J Comput Assist Tomogr* 2012;36:718-24.
187. Jurkiewicz MT, Stein JM, Learned KO, Nasrallah IM, Loevner LA. Hypoglossal nerve palsy due to carotid artery dissection: an uncommon presentation of a common problem. *Neuroradiol J* 2019;32:123-26.
188. Russo CP, Smoker WR, Weissman JL. MR appearance of trigeminal and hypoglossal motor denervation. *AJNR Am J Neuroradiol* 1997;18:1375-83.
189. Davagnanam I, Chavda SV. Identification of the normal jugular foramen and lower cranial nerve anatomy: contrast-enhanced 3D fast imaging employing steady-state acquisition MR imaging. *AJNR Am J Neuroradiol* 2008;29:574-6.
190. Paulus EM, Fabian TC, Savage SA, et al. Blunt cerebrovascular injury screening with 64-channel multidetector computed tomography: more slices finally cut it. *J Trauma Acute Care Surg* 2014;76:279-83; discussion 84-5.
191. Vertinsky AT, Schwartz NE, Fischbein NJ, Rosenberg J, Albers GW, Zaharchuk G. Comparison of multidetector CT angiography and MR imaging of cervical artery dissection. *AJNR Am J Neuroradiol* 2008;29:1753-60.
192. Provenzale JM, Sarikaya B. Comparison of test performance characteristics of MRI, MR angiography, and CT angiography in the diagnosis of carotid and vertebral artery dissection: a review of the medical literature. *AJR Am J Roentgenol* 2009;193:1167-74.
193. Thomas AJ, Wiggins RH, 3rd, Gurgel RK. Nonparaganglioma jugular foramen tumors. *Otolaryngol Clin North Am* 2015;48:343-59.
194. Entwisle T, Perchyonok Y, Fitt G. Thin section magnetic resonance diffusion-weighted imaging in the detection of acute infratentorial stroke. *J Med Imaging Radiat Oncol* 2016;60:616-23.
195. Derclé L, Hartl D, Rozenblum-Beddok L, et al. Diagnostic and prognostic value of 18F-FDG PET, CT, and MRI in perineural spread of head and neck malignancies. *Eur Radiol* 2018;28:1761-70.
196. Chang PC, Fischbein NJ, McCalmont TH, et al. Perineural spread of malignant melanoma of the head and neck: clinical and imaging features. *AJNR Am J Neuroradiol* 2004;25:5-11.
197. Bronstein Y, Tummala S, Rohren E. F-18 FDG PET/CT for detection of malignant involvement of peripheral nerves: case series and literature review. *Clin Nucl Med* 2011;36:96-100.
198. American College of Radiology. ACR Appropriateness Criteria® Radiation Dose Assessment Introduction. Available at: <https://www.acr.org/-/media/ACR/Files/Appropriateness-Criteria/RadiationDoseAssessmentIntro.pdf>. Accessed March 31, 2022.

The ACR Committee on Appropriateness Criteria and its expert panels have developed criteria for determining appropriate imaging examinations for diagnosis and treatment of specified medical condition(s). These criteria are intended to guide radiologists, radiation oncologists and referring physicians in making decisions regarding radiologic imaging and treatment. Generally, the complexity and severity of a patient's clinical condition should dictate the selection of appropriate imaging procedures or treatments. Only those examinations generally used for evaluation of the patient's condition are ranked. Other imaging studies necessary to evaluate other co-existent diseases or other medical consequences of this condition are not considered in this document. The availability of equipment or personnel may influence the selection of appropriate imaging procedures or treatments. Imaging techniques classified as investigational by the FDA have not been considered in developing these criteria; however, study of new equipment and applications should be encouraged. The ultimate decision regarding the appropriateness of any specific radiologic examination or treatment must be made by the referring physician and radiologist in light of all the circumstances presented in an individual examination.