Normal and pathologic radiographic anatomy of the motor innervation of the face.

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Facial motor disorders, including facial paralysis, myokymia, dyskinesia, and hemifacial spasm, are common clinical problems in which radiographic evaluation plays a crucial role. Since every segment of the motor innervation of the face from the brain to the parotid gland can now be seen radiographically, radiologists must understand the normal anatomy, the common pathologic lesions at each level, and the clinical findings that help localize the abnormality so that the most sensitive and accurate radiographic approach can be planned. Though computed tomography alone allows for visualization of every segment, other methods such as cisternography, angiography, polytomography, sialography, and magnetic resonance imaging are complementary in specific disorders.

Facial motor disorders, including facial paralysis, myokymia, dyskinesia, and hemifacial spasm, are common clinical problems in which a radiographic evaluation plays a crucial role. Though abnormalities of the motor innervation of the face need not be associated with a life-threatening illness, distortion of the face can create serious psychological problems and can significantly interfere with the unique capacity for self-expression reflected by the human countenance. An abnormality of the motor function of the face may also be the presenting symptom of progressive central or peripheral nervous system disease. With the advent of high-resolution, thin-section computed tomography (CT), the complex sinuous anatomy of the innervation of the face, from the cerebral cortex to the parotid gland, can now be routinely visualized radiographically. For simplification of discussion, the cascading pathway of the motor innervation of the face has been divided into nine descending separate anatomic levels (figs. 1 and 2).

Radiographic evaluation of the level of facial motor abnormality now approaches the accuracy of clinical localization [1-4] of the level of abnormality obtainable by a detailed neurologic examination. Since it is not possible to evaluate the innervation of the face throughout its entire course by a single survey radiographic examination, it is necessary to focus the examination on the suspected location and etiology of the abnormality. Because of this, an understanding of the clinical findings that help locate specific abnormalities and of the utility of each of the various radiographic procedures is important.

Patients with supranuclear facial nerve palsy, temporal bone fracture, malignant external otitis, atypical infranuclear facial nerve palsy, myokymia, or hemifacial spasm require localized radiographic evaluation. If surgical intervention is considered, radiographic evaluation is essential. On the other hand, patients with classic Bell palsy (unilateral facial paralysis secondary to herpes simplex infection) [5, 6], Ramsay Hunt syndrome (herpes zoster infection of the facial nerve), or facial myokymia secondary to Guillain-Barré syndrome need not have detailed radiographic workup because they are treated medically and can be diagnosed clinically.
Materials and Methods

Normal radiographs were obtained from normal patients or cadavers with no known disorder of the face. All pathologic examples were selected from patients referred for evaluation of either primary motor disorders of the face or for evaluation of a facial abnormality associated with another overriding clinical problem.

All the CT images were obtained using either a GE 8800 CT/T or Varian 360 CT scanner. The high-resolution, thin-section images of the temporal bone were made only with the GE CT/T 8800 scanner using the bone algorithm program, targeted reconstructions, and a 4000 window for review [7]. AIR [8] or metrizamide cisternography [9] and parotid sialography were used in conjunction with routine unenhanced and intravenously enhanced axial CT sections [10]. The polytomograms were made on a Philips Polytome unit. Cerebral angiography was done using a routine Seldinger transfemoral technique.

Anatomic Evaluation

The motor innervation of the face cascades through a unique sinuous path involving the brain, subarachnoid space, the longest bony canal in the body [11], the middle ear, a fatty space, the parotid gland, and the superficial facial musculature. For each of nine descending segments of the pathway innervating the face, the normal CT anatomy, the common etiologies for pathology, and the clinical findings characteristic of abnormalities of each of these levels that allow for clinical localization and targeted radiographic studies are described and explained (table 1). The description starts at the level of the brain and descends to the face.

Cerebral Hemisphere

The highest level of volitional control of the face begins in the lower part of the postcentral gyrus of the frontal lobe (fig. 3), which is seen on axial CT sections lateral to the lateral ventricles and the corpus callosum. Upper motor neuron fibers from the cerebral cortex descend medially and inferiorly through the corona radiata and the internal capsule (fig. 4) to the brainstem.

Common lesions at the cortical and corona radiata level include cortical or lacunar infarction, primary or secondary brain tumor, abscess, intra-or extraaxial hemorrhage, multiple sclerosis, and vascular malformation. Clinically, lesions rostral to the facial nucleus in the pons produce a supranuclear facial palsy, which is characterized by lower facial weakness sparing the forehead. An abnormality at these levels producing an isolated facial nerve palsy is uncommon; it is usually accompanied by other symptoms, including hemiparesis, aphasia, dysarthria, and hemisensory change.

Internal Capsule

The internal capsule is a broad band of white matter formed from the tapering corona radiata (fig. 4). The corticonuclear tracts course through the genu of the internal capsule. This is seen on routine axial CT sections of the brain at the level of the upper third ventricle as an angled, low-density strip between the head of the caudate nucleus and the thalamus medially and the basal ganglia laterally.

Common abnormalities at this level include lacunar infarction, basal ganglia hemorrhage, and multiple sclerosis. Supranuclear facial paralysis and facial dyskinesia [12] are examples of lesions that involve the adjacent basal ganglia, such as in Huntington chorea and Parkinson disease [13]. CT findings in these instances may be characteristic. An isolated supranuclear palsy is rare because of the close proximity of the other cortical bulbar and cortical spinal tracts.
TABLE 1: Anatomic, Clinical, and Radiographic Correlations

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<th>Anatomic Level</th>
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Note.—Tomographic planes are oriented in degrees above anthropologic baseline. IV = intravenous; CP = cerebellopontine; MRI = magnetic resonance imaging; PA = posteroanterior.

Fig. 3.—60-year-old man with history of supranuclear seventh cranial nerve palsy and dysarthria. Axial CT section at level of superior bodies of lateral ventricles shows contrast enhancement involving cortex due to cerebral infarction. Approximate level of postcentral gyrus (PCG) innervating face is labeled on normal side.

Fig. 4.—67-year-old man with acute dysarthria, confusion, and supranuclear seventh cranial nerve palsy. A, Internal capsule (arrows) is seen as low-density stripe between basal ganglia laterally and caudate nucleus (CN) and thalamus (T) medially. Anterior limb (AL), genu (G), and posterior limb (PL) form V pointing to foramen of Monro. B, Infarct demonstrated as low-density region is seen centered at level of internal capsule with involvement of adjacent basal ganglia (arrowhead).

Brainstem

The fibers of the internal capsule enter the cerebral peduncle to end in the seventh cranial nerve nucleus in the lowerpons (fig. 5). From the nucleus, the facial nerve fibers traverse the pons posteriorly to loop around the sixth cranial nerve nucleus forming the facial colliculus and exit at the pontomedullary junction just below the middle cerebellar peduncle.
Intraaxial abnormalities commonly seen at this level include glioma, multiple sclerosis [14], viral polyneuritis, infarct, hemorrhage, metastases, contusion, and trauma (fig. 6).

Any lesion at or distal to the facial nerve nucleus produces a peripheral facial nerve palsy characterized by flaccid paralysis affecting both the upper and lower face equally. Alternatively, lesions at this level may cause a unique dyskinesia, such as myochymia [14]. Myochymia is a constant serpentine undulating motion of the face, most often seen in patients with multiple sclerosis (best seen with magnetic resonance imaging) [15] or brainstem glioma. Because many important structures are tightly packed within the brainstem, other associated clinical abnormalities include cranial nerve palsy, ataxia, and controlateral hemiparesis, as well as abnormalities of the reticular activating system. Bilateral facial paralysis may be seen as a congenital finding in patients with Möbius syndrome [16], in which the development of the brainstem cranial nerve nuclei is abnormal.

Cerebellopontine Angle

The facial nerve traverses the cerebellopontine angle together with the roots of the eighth cranial nerve and the nervus intermedius to enter the porus acusticus. The nerves then course superiorly and laterally in close approximation to the anterior inferior cerebellar artery (fig. 7).
Acoustic neuroma, seventh cranial nerve neurollemoma (fig. 8), meningioma, exophytic brainstem tumor, meningeval carcinomatosis, petrous apex abnormalities (congenital cholesteatoma and mucocele), aneurysm, and tortuous and anomalous vessels (fig. 9) can all affect the seventh nerve at the cerebellopontine angle. Extraaxial lesions, such as chordoma, aneurysm of the basilar artery, meningioma, metastases to the clivus, and epidermoid, are unlikely to affect the facial nerve function except in late stages.

Lesions involving the facial nerve at this level may produce changes in the gustatory sensation of the anterior two-thirds of the tongue. Involvement of the nervus intermedius may also cause anatomic changes with loss of normal lacrimal function. Hemifacial spasm [17-20] may be caused by irritative abnormalities of the facial nerve in the cerebellopontine angle and is characterized by involuntary painful twitching of the face, which becomes worse during periods of anxiety. It is often a progressive disorder and can be a serious and cosmetic problem. In the past, hemifacial spasm was usually considered to be idiopathic, but recent reports implicate compression of the root fibers of the facial nerve by adjacent arterial structures.

Internal Auditory Canal

The facial nerve enters the superior anterior part of the internal auditory canal with the nervus intermedius and the eighth cranial nerve roots (fig. 10).

Acoustic neuroma is the most common lesion at this level, though facial paralysis is a rare complication, occurring in only 2% [21] of cases. A primary facial nerve neuroma can mimic an acoustic neuroma in this region [22, 23]. Other lesions that may involve the internal auditory canal include anomalous jugular bulb [24], large glomus jugulare tumor, primary cholesteatoma, transverse fracture [25, 26], metastases, petroitis, Paget disease [27], osteopetrosis, and congenital anomalies such as atresia [28] (fig. 11).

Lesions in the internal auditory canal may be difficult to distinguish from those at the cerebellopontine angle on clinical findings alone since lesions at both levels cause similar symptoms of vertigo and hearing loss.

Geniculate Ganglion

The fallopian canal (facial nerve canal within the temporal bone) begins at the geniculate ganglion where the facial nerve exits the internal auditory canal on its anterior border at the fundus. It forms a characteristic inverted V-shaped lucency on axial CT sections (fig. 12) and on coronal sections is imaged as two circular lucencies superior and lateral to the adjacent turns of the cochlea. Anteriorly, the greater superficial petrosal nerve exits along the superior margin of the petrous bone to innervate the lacrimal gland through the ciliary ganglion. Common lesions in this region include cholesteatoma, fracture, primary seventh neurona, and infection.

Clinically, lesions distal to this level do not interfere with normal lacrimal function. Destructive lesions of the geniculate ganglion may erode into the cochlea and produce neurosensory hearing loss.

Horizontal Part of the Facial Nerve Canal

The facial nerve then enters the middle ear cavity. Superiorly it is covered by dense cortical bone underlying the lateral semicircular canal, while inferiorly it is covered by only a thin bony shell. Many normal patients have dehisences of the bony wall in this segment. With high-resolution, thin-section coronal CT, this segment of the nerve can be seen as a circular soft-tissue density without a bony margin. The facial nerve canal is also close to the superior margin of the oval window.
**Mastoid Part of the Facial Nerve Canal**

From the middle ear cavity, the facial nerve turns inferiorly to descend parallel to the lateral margin of the jugular fossa (fig. 13), where it lies posterior to the external auditory canal.

Common lesions of this segment include cholesteatoma, infection, fracture [29], contusion by ossicular dislocation, surgery, glomus tumor, and congenital anomaly (such as aberrant location with associated abnormalities of the stapes and oval window) [30].

Because this segment of the facial nerve is suspended within the middle ear cavity, abnormalities associated with conductive hearing loss may occur. It is also particularly vulnerable to injury by trauma or infection.

**Subtemporal and Parotid Segments**

The subtemporal facial nerve is visible on CT as a small circular water density as it exits the stylomandibular foramen into a fatty space between the mastoid tip and styloid process [34]. Within the parotid gland the main trunk of the facial nerve rapidly divides into multiple rami to supply the peripheral facial musculature. Though not visible directly in the parotid, its position is approximately lateral to the styloid process and posterior and lateral to the retromandibular vein (fig. 14).

Common lesions involving this segment include surgical injury (parotid exploration), malignant or benign parotid tumor, metastases, infection (malignant external otitis), sarcoidosis, neurilemmona, and trauma (including forceps injury at birth) [35].

This level can be localized by the clinical findings of an infranuclear facial nerve palsy with palpable masses in the region of the parotid gland, sparing abnormalities of the autonomic nervous system and taste.
Discussion

The use of appropriate radiographic procedures directed by topographic clinical evaluation of abnormalities involving the facial musculature can result in an accurate and sensitive radiographic evaluation of the causative lesion. While CT is the mainstay in the diagnostic radiographic workup, angiography, complex motion tomography, magnetic resonance imaging, and parotid duct sialography may be necessary to aid in the diagnosis of these common problems.

Not all patients with abnormalities of motor innervation of the face, including most patients with Bell palsy and Ramsay Hunt syndrome, require complete radiographic analysis. However, patients with isolated peripheral facial palsy, supranuclear facial palsy, hemifacial spasm, trauma, myokymia, and allied neurologic symptoms do require a detailed radiographic evaluation.

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