Cranial Nerve VII: The Facial Nerve and Taste

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Definition

The motor portion, or the facial nerve proper, supplies all the facial musculature. The principal muscles are the frontalis, orbicularis oculi, buccinator, orbicularis oris, platysma, the posterior belly of the digastric, and the stapedius muscle. In nuclear or infranuclear ("peripheral") lesions, there is a partial to complete facial paralysis with smoothing of the brow, open eye, flat nasolabial fold, and drooping of the mouth ipsilateral to the lesion. Supranuclear ("central") lesions spare the brow and eyelid musculature; there is flattening of the nasolabial fold and drooping of the mouth contralateral to the lesion.

Careful and thoughtful observation is the key to discerning subtle signs of weakness of muscles supplied by the motor portion. Note especially the blink, nasolabial folds, and corners of the mouth. Asymmetry is the clue to unilateral weakness and is best perceived during conversation when the patient is unaware of being observed.

1. Blink: The eyelid on the affected side closes just a trace later than the opposite eyelid.
2. Nasolabial folds: The weak one is flatter.
3. Mouth: The affected side droops and participates manifestly less in speaking.

Ask the patient to look up or wrinkle the forehead; inspect for asymmetry. Ask him or her to close the eyes tightly. Look for incomplete closure or incomplete "burying" of the eyelashes on the affected side. Observe the nasolabial folds and mouth while the patient is concentrating on the eyes. As the orbicularis oculi contract tightly, there are milder contractions of muscles about the mouth and nose; these milder contractions are better suited to displaying slight weakness than when these muscles are tested directly.

Ask the patient to smile, show you teeth, or pull back the corners of the mouth. Look for asymmetry about the mouth.

The most subtle signs of mild facial weakness are the blink reflex and incomplete lid closure. Observe the blink reflex during conversation, or tap gently on the glabella with your index finger or reflex hammer in an attempt to bring out a mild asymmetry of blink. If you strongly suspect but are having difficulty confirming a mild facial weakness, ask the patient to lie flat on the examining table with face up. Slide the patient's head off the examining table so the head is below the body. This forces the eyelids to work against gravity. Now ask the patient to close both eyes and inspect for incomplete closure. Tap on the glabella and note asymmetry of blink.

The facial nerve participates in a number of reflexes (Dejong, 1979). Assessment of these reflexes provides valuable additional information about facial nerve function. Table 62.1 lists these reflexes, the method of eliciting them, and their clinical interpretation. Some of these reflexes are discussed in more detail in Chapter 71 (Suck, Snout, Pal- momental, and Grasp Reflexes).

Taste

The four primary tastes are bitter, sweet, sour, and salty. Screen for disorders of sweet or salty taste with salt and sugar. With the patient's eyes closed and tongue protruded, take a tongue blade and smear a small amount of salt or sugar on the lateral surface and side of the tongue. Instruct the patient to tell you the identity of the substance. Rinse the mouth thoroughly and repeat the test on the other side, using a different substance.

Basic Science

The cortical fibers of the facial nerve proper originate from the lower third of the motor strip. They course in the genu of the internal capsule and the middle third of the cerebral peduncle, supplying the seventh nucleus in the lower pons. The supranuclear innervation is bilateral to the muscles of the forehead and eyes but only contralateral to the muscles of the lower part of the face. This accounts for the sparing of the upper facial muscles with a contralateral cortical lesion. Figure 62.1 shows the anatomy of the facial nerve.

The facial nucleus participates in the corneal reflex. Corneal pain and temperature fibers go through the ophthalmic division of the fifth cranial nerve to the spinal nucleus of the fifth and thence to the ipsilateral seventh nucleus, causing the eyelid to blink. There are also central connections between the facial nucleus and the nuclei or projection systems of the second, third, fourth, sixth, and eighth cranial nerves. These connections coordinate movements among the eyelids and eyeballs and set up certain reflexes such as the blink reflex on exposure to a strong light or a loud sound. Table 62.1 lists these reflexes.

The motor fibers for the facial muscles exit from the motor nucleus, curl up and around the sixth nucleus, and descend laterally from the lower pons.
The intermediate nerve joins the motor segment at the point where it exits from the pons. The intermediate nerve is composed of contributions from three areas:

1. The superior salivary nucleus in the pons supplies secretory fibers. They go to (a) the lacrimal, nasal, and palatine glands (via the greater superficial petrosal nerve) and (b) the submandibular and sublingual salivary glands (via the chorda tympani nerve).
2. The gustatory (solitary) nucleus in the medulla supplies sensory fibers. These fibers go to taste buds on the anterior two-thirds of the tongue (via the chorda tympani nerve).
3. The dorsal part of the trigeminal tract supplies fibers that convey cutaneous sensation from the external auditory meatus and skin behind the ear (distributed with the facial nerve proper).

First-order neurons are in the geniculate ganglion. Table 62.2 summarizes the brainstem nuclei of the facial and intermediate nerves.

The facial nerve proper and intermediate nerve lie in the cerebellopontine angle with the sixth and eighth cranial nerves. The seventh, intermediate, and eighth nerves enter the internal auditory meatus. The facial and intermediate nerves then descend to the stylomastoid foramen, giving off the chorda tympani at either the stylomastoid foramen or varying distances proximal to it. The chorda tympani supplies the anterior two-thirds of the tongue and the submandibular and sublingual glands. The motor part of the facial nerve leaves the stylomastoid foramen and supplies the facial musculature. A major part of the nerve forms a plexus within the parotid gland. Table 62.3 lists the branches of the facial nerve, beginning centrally and proceeding distally.

Taste sensibility is composed of four qualities: sweet, salt, sour, and bitter. Taste receptors are located on the tongue, palate, pharynx, and larynx. Although all four qualities can be perceived throughout, there is considerable localization. The tongue, especially the tip and edges, is most sensitive to sweet and salt; the palate, to sour and bitter. The receptors are taste buds. Up to 8 are on each of the fungiform papillae on the anterior two-thirds of the tongue, and up to 100 on each of the circumvallate papillae on the posterior part of the tongue. The taste buds are barrel shaped with a pore opening. Chemoreceptive taste hairs project into the barrel from neuroepithelial sensory cells. Impulses from these cells are transmitted to the brainstem. Afferent fibers from the anterior two-thirds of the tongue travel via the lingual nerve to the chorda tympani and then as described above to the gustatory nucleus. Taste fibers from the posterior third of the tongue, the palate, and the palatoglossal arch travel via the glossopharyngeal nerve and the nodose ganglion, also ending in the gustatory nucleus. There are two ascending pathways from the gustatory nucleus. One goes to the hypothalamus. The other goes to the thalamus and
Table 62.3

The Branches of the Facial Nerve Beginning Centrally

<table>
<thead>
<tr>
<th>Branch</th>
<th>Comments</th>
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<tbody>
<tr>
<td>Greater superficial</td>
<td>Leaves just distal to geniculate ganglion, which lies in facial canal of the petrous portion of the temporal bone. Supplies lacrimal, nasal, and palatine glands.</td>
</tr>
<tr>
<td>petrosal nerve</td>
<td></td>
</tr>
<tr>
<td>Nerve to stapedius</td>
<td>Next branch to be given off, still within facial canal.</td>
</tr>
<tr>
<td>muscle</td>
<td></td>
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<tr>
<td>Chorda tympani</td>
<td>Leaves about 5 mm or less before the stylomastoid foramen. Taste to anterior one-third of tongue, submaxillary and sublingual salivary glands.</td>
</tr>
<tr>
<td>Motor branches</td>
<td>At exit from stylomastoid foramen. The posterior auricular, digastric, and stylohyoid branches are given off. In the parotid gland the nerve divides into two branches, the temporal and cervicofacial, which supply the remaining muscles.</td>
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Clinical Significance

Motor

A lesion involving the nuclear or infranuclear portion of the facial nerve will produce a peripheral facial palsy. If all motor components are involved, there is complete paralysis of all facial muscles on the involved side. The brow is smooth, the eye does not close, the nasolabial fold is flat, and that side of the mouth droops. There is no movement at all. The paradigm of this type of involvement is Bell's palsy. Idiopathic Bell's palsy may strike at any age, often after a mild viral illness. Recovery is over a period of weeks to months and is variable. The cause of the idiopathic variety is unknown. Sequelae to Bell's palsy include the following:

1. Interfacial synkinesis: When the eyes close, the mouth will twitch. This occurs when the regenerating nerve fibers do not grow back into the proper muscles. The synkinetic movements are almost always present on the involved side.
2. Because of the contractures, the face at rest may be more deeply etched on the side of the previous palsy. This can give a false impression of weakness on the opposite side.
Other causes of peripheral seventh nerve palsy include: neoplasm, trauma, middle ear infections, parotid gland surgery, granulomatous or carcinomatous meningitis, and diabetes. The disturbances of function produced by these lesions need not be complete. An important clinical point is that the clinical manifestations of these disorders are indistinguishable from idiopathic Bell's palsy.

Supranuclear involvement produces contralateral paralysis of the lower facial muscles and sparing of the upper muscles due to the bilateral supranuclear innervation of the latter. Subtle weakness is often difficult to confirm. Many, perhaps even most, normal individuals have mild lower facial asymmetries, making interpretation difficult.

Anatomic localization of lesions is made by the characteristics of the dysfunction and associated structures involved. Table 62.4 gives the location of lesions and the clinical manifestations. Refer to Figure 62.1 to see exactly where the lesions occur.

### Table 62.4

<table>
<thead>
<tr>
<th>Lesion location</th>
<th>Manifestations</th>
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<tbody>
<tr>
<td>Above the facial nucleus (supranuclear lesion)</td>
<td>Contralateral paralysis of lower facial muscles with relative preservation of upper muscles. Lesion located either in brainstem or cortex.</td>
</tr>
<tr>
<td>Pons (nuclear or fascicular lesion)</td>
<td>Ventral pontine lesion (of Millard-Gubler): ipsilateral facial monoplegia, lateral rectus palsy (VI), contralateral hemiplegia (corticospinal fibers); pontine tegmentum lesion (of Foville): ipsilateral facial monoplegia; contralateral hemiplegia (corticospinal fibers); paralysis of conjugate gaze to side of lesion (pontine paramedian reticular formation).</td>
</tr>
<tr>
<td>Cerebellopontine angle (peripheral nerve lesion, 1 in Figure 62.1)</td>
<td>Ipsilateral facial monoplegia, loss of taste to anterior two-thirds of tongue, impairment of salivary and tear secretion, hyperacusis (if VIII is not affected). Additional cranial nerves may be involved: deafness, tinnitus, vertigo (VII); sensory loss over face and absence of corneal reflex (V); ipsilateral ataxia (cerebellar peduncle).</td>
</tr>
<tr>
<td>Facial canal between internal auditory meatus and geniculate ganglion (peripheral nerve type lesion here and subsequently), 2 in Figure 62.1</td>
<td>Same as above except cranial nerves other than VII are not involved.</td>
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<tr>
<td>Facial canal between geniculate ganglion and nerve to stapedius muscle, 3 in Figure 62.1</td>
<td>Facial monoplegia; impaired salivary secretion; loss of taste; hyperacusis.</td>
</tr>
<tr>
<td>Facial canal between nerve to stapedius and leaving of chorda tympani, 4 in Figure 62.1</td>
<td>Facial monoplegia; impaired salivary secretion; loss of taste.</td>
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<tr>
<td>After branching of chorda tympani, 5 in Figure 62.1</td>
<td>Facial paralysis, distribution related to site of lesion.</td>
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### Table 62.5

**Examples of Disorders Reported to Be Associated with Gustatory Dysfunction**

**Endocrine**
- Adrenal cortical insufficiency
- Congenital adrenal hyperplasia
- Panhypopituitarism
- Cushing's syndrome
- Hypothyroidism
- Diabetes mellitus
- Turner's syndrome
- Pseudohypoparathyroidism

**Inflammatory**
- Infections:
  - Candida
  - Gingivitis
  - Herpes simplex
  - Periodontitis
  - Sialadenitis
  - Autoimmune:
    - Pemphigus
    - Sjogren's syndrome
- Local alterations of taste buds or papillae
- Chemicals, drugs
- Xerostomia

**Neurologic**
- Bell's palsy
- Familial dysautonomia
- Head trauma
- Middle ear operations with Cretinism manipulation or damage to chorda tympani
- Multiple sclerosis
- Raeder's paratrigeminal syndrome

**Nutritional**
- Cachexia
- Chronic renal failure
- Cirrhosis of the liver
- Niacin (vitamin B₆ deficiency)
- Zinc deficiency

**Psychiatric**
- Depression
- Schizophrenia

**Tumors**
- Oral cavity cancer
- Base of skull neoplasia

No proposed mechanism explains the occurrence of this phenomenon in all cases.

References