

Chapter 13: Facial Sensation and Movement and Approach to Facial Sensory and Motor Deficits: Cranial nerves 5 and 7

TRIGEMINAL NERVE (CRANIAL NERVE 5)

The principal clinically relevant functions of the trigeminal nerve are facial somatic sensation and innervation of the muscles of mastication. The trigeminal nerve also innervates one palate muscle (tensor veli palatini) and one inner ear muscle (tensor tympani).

The trigeminal nerve supplies somatic sensation to the face, the inside of the mouth (although *not* taste), the sinuses, and most of the dura mater (including all supratentorial dura and the tentorium cerebelli; the rest of the posterior fossa dura mater is supplied by cranial nerve 10). The three divisions of the trigeminal nerve and the regions for which they supply sensation are:

1. Ophthalmic division (V_1): forehead, upper lid, upper half of cornea
2. Maxillary division (V_2): cheek, lower lid, lower half of cornea, roof of mouth, upper gums/teeth/lip
3. Mandibular division (V_3): lower gums/teeth/lip, lower jaw, anterior tongue

The muscles of mastication innervated by CN 5 are:

- Masseter
- Pterygoids
- Anterior belly of the digastric (the posterior belly of the digastric is innervated by 7)
- Mylohyoid (the stylohyoid is innervated by CN 7)

See [Table 14–1](#) for a summary of the functions shared between CNs 5, 7, 9, and 10.

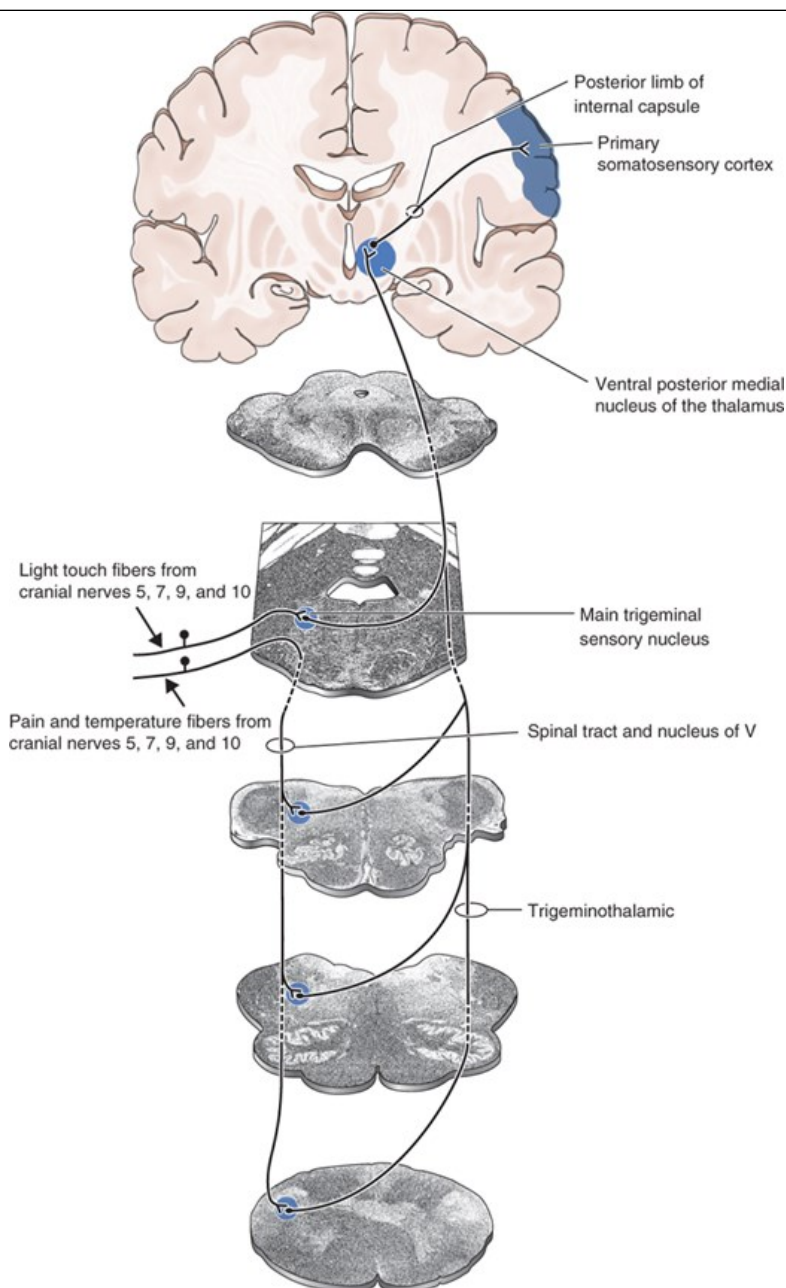
Trigeminal Pathways

Trigeminal Sensory Pathways

The facial sensory pathways of the trigeminal nerve are analogous to those for the body: All sensory information travels back to a ganglion, and pain and temperature sensation travel separately from other modalities ([Fig. 13–1](#)). The sensory ganglion of CN 5 is called the **gasserian ganglion**, which resides in **Meckel’s cave** (mnemonic: ganglion for cranial nerve **V** resides in Meckel’s cave). The gasserian ganglion receives somatosensory input from the face and transmits that information to the brainstem at the level of the pons. Distal to the gasserian ganglion, the trigeminal nerve divides into three branches: ophthalmic (V_1), maxillary (V_2), and mandibular (V_3). V_1 and V_2 pass through the cavernous sinus, whereas V_3 does **not** pass through the cavernous sinus. V_1 exits the skull through the superior orbital fissure, V_2 through the foramen rotundum, and V_3 through the foramen ovale.

FIGURE 13–1

Schematic of the trigeminal nerve pathways. The pathway for facial proprioception including the mesencephalic nucleus of CN 5 is not shown here. Reproduced with permission from Martin J: *Neuroanatomy Text and Atlas*, 4th ed. New York: McGraw-Hill Education; 2012.



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Proximal to the gasserian ganglion, the somatosensory information from CN 5 enters the brainstem at the level of the pons. Although the point of entry of CN 5 into the brainstem is at the level of the pons (as would be expected for the 5-6-7-8 schema described in Ch. 9), CN 5 has nuclei throughout the three levels of the brainstem. Light touch sensation from the face is transmitted primarily to the **main sensory nucleus of 5** (also called the **chief** or **principal sensory nucleus of 5**) at the level of entry in the pons, and the output from this nucleus crosses to join the medial lemniscus (carrying sensory information from the body), which ascends to the thalamus. Facial sensation information projects to the **ventral posterior medial (VPM) nucleus** of the thalamus, and sensation from the limbs and trunk travels to the ventral posterior lateral (VPL) nucleus of the thalamus (see Ch. 4). VPM and VPL project to the somatosensory cortex in the postcentral gyrus.

Pain and temperature sensation from the face descend to the medulla in the **spinal tract of 5** (the facial analogue of the anterolateral tract) along with its associated **spinal nucleus of 5**. The spinal nucleus and tract of 5 extend as inferiorly as the upper cervical spine. The output of the spinal nucleus of 5 crosses to join the contralateral spinothalamic tract from the body and ascends to the ventral posterior medial (VPM) nucleus of the thalamus (pain and temperature sensation from the limbs and trunk travel to the VPL nucleus of the thalamus).

Jaw proprioception information is relayed to the **mesencephalic nucleus of 5** in the midbrain.

Trigeminal Motor Pathways

The **motor nucleus of 5** is medial to the main sensory nucleus of 5 in the dorsal pons. Unlike the common symptom of facial weakness in both central and peripheral nervous system diseases (see “Upper and Lower Motor Neuron Facial Weakness” below), jaw weakness due to a lesion in the nervous system is uncommon. This is in part due to the fact that the trigeminal motor nuclei receive bilateral cortical input, so unilateral hemispheric lesions such as strokes do not typically cause unilateral jaw weakness.

Brainstem Reflexes Involving the Trigeminal Nerve

The trigeminal nerve is involved in two brainstem reflexes, the corneal reflex and the jaw jerk reflex.

Corneal reflex—The corneal reflex is elicited by stimulating the cornea, which normally leads to reflex closure of the eyes. The trigeminal nerve is the afferent limb of the reflex (sensation from the cornea is carried by V_1), and the facial nerve (CN 7) is the efferent limb responsible for closure of the eye (by contraction of the orbicularis oculi muscle). The reflex is bilateral, so stimulating one cornea normally leads to bilateral closure of both eyes.

Jaw jerk reflex—The jaw jerk reflex is elicited by asking the patient to let the jaw gently hang open, and then tapping on the upper chin gently with a reflex hammer. This reflex is mediated by the mesencephalic nucleus of 5 (afferent) and the motor nucleus of 5 (efferent). If the jaw jerk reflex is markedly brisk, this demonstrates upper motor neuron (central nervous system) pathology above the level of the spinal cord (i.e., in the brainstem or brain). The jaw jerk reflex is often tested in patients with upper motor neuron signs in the extremities to evaluate for signs of pathology superior to the spinal cord (i.e., to determine if the corticobulbar tracts are affected in addition to corticospinal tracts).

Disorders of the Trigeminal Nerve

Facial Sensory Loss

Loss of facial sensation can occur due to a lesion in:

- The trigeminal nerve in the face, skull base, or cavernous sinus
- The trigeminal pathways in the dorsolateral brainstem (e.g., loss of pain and temperature sensation in the face is part of lateral medullary syndrome along with contralateral loss of pain and temperature in the body, Horner’s syndrome, ataxia, nausea, vomiting, vertigo, dysarthria, and dysphagia)
- VPM of the thalamus
- The lateral postcentral gyrus (facial region of the somatosensory cortex)
- The subcortical white matter connecting VPM and somatosensory cortex

Isolated facial sensory loss is most likely to be due to a lesion of the trigeminal nerve, the entry zone of the nerve in the pons, or a small cortical lesion because lesions at all other sites would likely cause other deficits due to involvement of adjacent structures:

- A brainstem lesion causing facial sensory loss will often also affect other cranial nerve nuclei.
- A cavernous sinus lesion causing facial sensory loss (which will only affect V_1 and V_2 divisions since V_3 does not pass through the cavernous sinus) will often also affect CNs 3, 4, and/or 6 (see [Ch. 11](#)).
- A thalamic lesion causing facial sensory loss will often also cause sensory loss beyond the face.
- A subcortical lesion causing facial sensory loss will often also affect additional adjacent sensory fibers representing other parts of the body.

Since the face and hand have the most cortical sensory representation, a common pattern of numbness for cortical, subcortical, and small thalamic

lesions is contralateral face and hand numbness (**cheiro-oral** pattern).

Facial numbness due to an isolated trigeminal nerve lesion can be caused by:

- Malignancy: skull base tumor, cerebellopontine angle tumor, leptomeningeal metastasis
- Inflammatory disease: Sjögren's syndrome, sarcoidosis
- Spread of head and neck cancers along the trigeminal nerve (**perineural spread**), most commonly caused by squamous cell cancer of the skin on the face
- Dental pathology

Numb chin sign—Numbness of the chin (**numb chin sign**) should raise concern for metastatic malignancy affecting the mandibular (V_3) division of the trigeminal nerve in the skull base or the distal trigeminal branches in the mandible (usually the mental nerve, a branch of the inferior alveolar nerve, which is a branch of V_3). The numb chin sign may be the presenting feature of systemic malignancy or metastatic disease in a patient with known cancer. Breast cancer and lymphoma are among the most common malignancies causing the numb chin sign. When patients present with this symptom, history for other symptoms of malignancy should be elicited (e.g., weight loss, night sweats). The differential diagnosis includes dental etiologies and systemic causes of trigeminal neuropathy (e.g., Sjögren's syndrome, sarcoidosis). Evaluation should include panoramic dental x-ray, CT scan or bone scan of the jaw (to evaluate the mandible), CT scan of the head (to evaluate for a skull base lesion), and/or MRI of the brain with contrast (to evaluate the trigeminal nerve itself).

Trigeminal Neuralgia

In **trigeminal neuralgia**, brief lightening-like paroxysms of pain shoot through the face. These may be spontaneous or may be triggered by contacting the face (e.g., brushing the teeth). Trigeminal neuralgia is most commonly unilateral and most commonly affects the lower face (V_2 and/or V_3 regions). The condition may be idiopathic, or it can be caused by multiple sclerosis, a compressive vascular loop, any of the above-listed etiologies of trigeminal pathology, or it may begin after dental work (ostensibly due to irritation of the most distal branches of the trigeminal nerve).

If trigeminal neuralgia is present in a young woman and/or bilaterally, multiple sclerosis should be considered and MRI obtained. How can trigeminal neuralgia be caused by multiple sclerosis if multiple sclerosis is a central nervous system disease and the trigeminal nerves are peripheral nerves? When multiple sclerosis causes trigeminal neuralgia, the lesion is in the trigeminal entry zone in the pons, which is part of the central nervous system.

In idiopathic trigeminal neuralgia, facial sensation is generally normal. If facial sensation is diminished in a patient with trigeminal neuralgia, this is atypical and a structural lesion should be sought with neuroimaging.

Antiepileptics are used for pain control in trigeminal neuralgia, with the most supportive evidence being for carbamazepine. If neuroimaging reveals a vascular loop compressing the trigeminal nerve in a patient with trigeminal neuralgia, microvascular decompression can be considered if the patient does not respond to medications. In refractory cases of trigeminal neuralgia, surgical ablation of or radiotherapy to the gasserian ganglion may be considered.

FACIAL NERVE (CRANIAL NERVE 7)

The main clinically relevant function of the seventh cranial nerve is control of the facial musculature, although CN 7 has a number of additional functions:

- Parasympathetic:
 - Lacrimal and nasal glands
 - Submandibular and sublingual salivary glands (although the facial nerve passes through the parotid gland, it does not innervate it—CN 9 does)

- Motor (beyond facial musculature): stapedius, posterior belly of the digastric (anterior belly of the digastric is innervated by CN 5)
- Special sensory: taste to the anterior two thirds of the tongue (posterior one third is innervated by CN 9)
- Sensory: somatic sensation for the external auditory meatus (shared with CNs 9 and 10)

See Table 14–1 for a summary of functions shared between CNs 5, 7, 9, and 10.

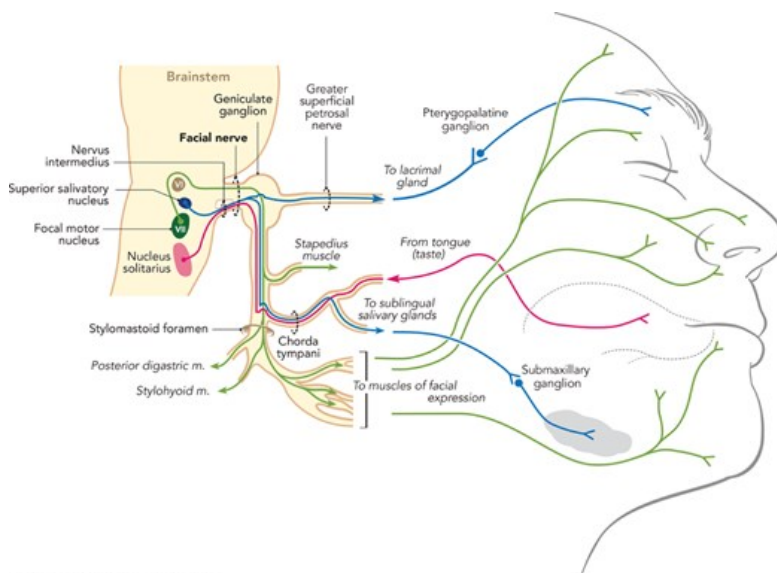
Facial Nerve Pathways

The seventh nerve nucleus is in the dorsal pons, and the nerve loops dorsally around the abducens (CN 6) nucleus before exiting anteriorly just lateral to the descending corticospinal tracts (see Fig. 9–1D). The “bump” on the dorsal pons where the seventh nerve loops around the CN 6 nucleus is called the **facial colliculus**. The nerve travels to and through the internal auditory canal with CN 8, then passes into the facial canal, finally emerging through the stylomastoid foramen to innervate the facial muscles after passing through the parotid. The nonmotor functions (sensory and visceral) are carried out by the **nervus intermedius**, which runs with the motor portion of the nerve but interacts with different brainstem nuclei related to sensory and visceral functions.

There are several important components of the facial nerve along its course in the skull before exiting the stylomastoid foramen to innervate the muscles of the face (Fig. 13–2). From proximal to distal:

FIGURE 13–2

Schematic of the facial nerve pathways. Reproduced with permission from Martin T, Corbett J: *Practical Neuroophthalmology*. New York: McGraw-Hill Education; 2013.



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- The **geniculate ganglion** is the main ganglion for all sensory and special sensory CN 7 functions (taste to anterior two thirds of the tongue, sensation around the ear).
- The **greater petrosal nerve** innervates the lacrimal and nasal glands (by way of the **sphenopalatine ganglion**, also known as the **pterygopalatine ganglion**).
- The **nerve to the stapedius** innervates the stapedius.
- The **chorda tympani** transmits taste from the anterior two thirds of the tongue and provides innervation to the salivary glands (submandibular and sublingual). Taste information travels to the **nucleus solitarius** in the medulla. (Mnemonic to recall that the nucleus solitarius is responsible for taste: **nucleus solitastiest** [although note that taste is not the sole function of the nucleus solitarius, which also receives visceral afferent

information; see [Ch. 14](#)].).

After exiting the stylomastoid foramen, subsequent branches of CN 7 are all motor: posterior auricular, digastric, stylohyoid, and the five branches to the facial muscles: temporal, zygomatic, buccal, mandibular, and cervical.

Upper and Lower Motor Neuron Pattern Facial Weakness

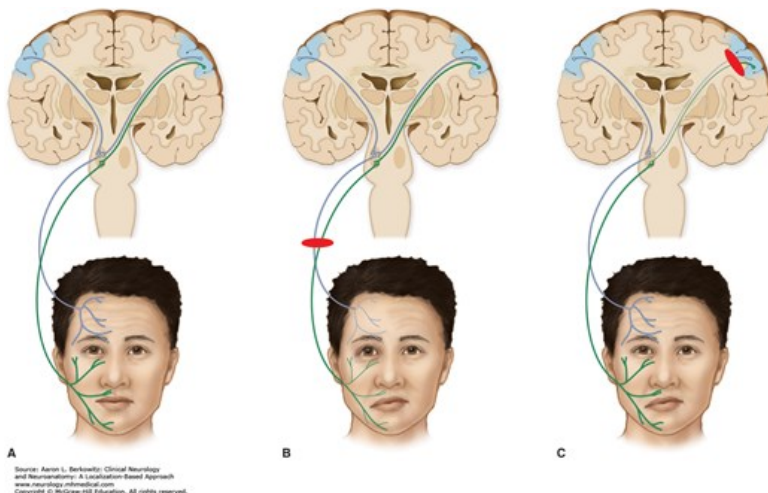
Just as the motor system for the extremities has upper motor neurons in the central nervous system and lower motor neurons in the peripheral nervous system, so too does the system for facial movements ([Fig. 13-3](#)). Facial movements are mediated by the bilateral facial nerves (CN 7), which contain the lower motor neurons in this system. Just as for the body, each half of the face is controlled by the contralateral hemisphere: The left hemisphere face region of the motor cortex communicates with the right (contralateral) CN 7 nucleus in the pons, which controls the muscles of the right side of the face; the right hemisphere face region of the motor cortex communicates with the left (contralateral) CN 7 nucleus in the pons, which controls the muscles of the left side of the face.

Episode 5: Motor Control of the Face

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FIGURE 13-3

Schematic showing upper and lower motor neuron pattern facial weakness. A: The facial nerve receives input from the contralateral motor cortex as well as an ipsilateral projection to the upper face. **B:** A lesion of the facial nerve will interrupt all input to both the upper face and lower face. **C:** A lesion of the motor cortex will cause contralateral lower facial weakness with preserved motor function of the upper face due to the ipsilateral “backup” to the upper face. (See text)



A lesion of the facial nerve will cause weakness of the whole face on that side: The patient will be unable to smile, puff the cheek, close the eye, raise the eyebrow, or wrinkle the forehead on the affected side ([Fig 13-4](#)). Early on in a seventh nerve palsy, eye closure weakness may be subtle, and noted only if the patient fails to completely “bury” the eyelashes on attempted forceful eye closure. The nasolabial fold and forehead wrinkles may be diminished or absent on the affected side.

FIGURE 13-4

Left-sided lower motor neuron pattern facial weakness (due to Bell’s palsy). A: When the patient attempts to smile, there is no movement of the left side of the face and the nasolabial fold is not visible on the left. The eye is slightly more open on the left. **B:** When the patient attempts to raise her forehead, the eyebrow elevates and the forehead wrinkles only on the right side. The flattening of the nasolabial fold on the left is again notable. **C:** On attempted gentle eye closure, the left eye does not close fully, and the sclera is seen as the eye elevates on attempted eye closure (Bell’s phenomenon). **D:** When attempting to close the eyes more tightly, the eyelashes are “buried” on the right, but still fully visible on the left due to

orbicularis oculi weakness.



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If facial weakness is due to an upper motor neuron lesion, only the lower face is affected: The patient is unable to smile on the affected side (contralateral to the brain lesion), but can still close the eye and raise the eyebrow. This is because the upper face is supplied not only by the contralateral hemisphere but also by the ipsilateral hemisphere. That is, each CN 7 contains the information from the contralateral motor cortex for the whole face and a “backup” for the upper face from the ipsilateral hemisphere. So whereas a CN 7 lesion will prevent any input—ipsilateral or contralateral—from making it to the face, an upper motor neuron lesion will only remove its input to the contralateral facial nerve, but that facial nerve will still have its ipsilateral “backup” for the upper face. Why might there be a backup for the upper face? One possibility is that it is more important to protect the eye than to preserve the ability to smile—failure of eye closure can lead to corneal abrasion and ulceration.

Upper Motor Neuron Facial Weakness

Upper motor neuron facial weakness can be caused by a lesion in the motor cortex, subcortical white matter, or in the midbrain and upper pons before the upper motor neuron pathway has made contact with the contralateral CN 7 nucleus. Therefore, a lesion in the descending motor pathways (corticospinal tract and corticobulbar tract) anywhere superior to the midpons will cause contralateral weakness in both the face and the body. Once the lesion is in the CN 7 nucleus or CN 7 fascicle in the pons, or in the facial nerve itself, facial weakness will be in a lower motor neuron pattern (upper and lower facial weakness).

Lower Motor Neuron Facial Weakness

Causes of lower motor neuron pattern facial weakness include:

- Bell's palsy, an idiopathic seventh nerve palsy (see "Bell's palsy" below).
- Infections: Lyme disease, HIV (especially at the time of seroconversion; see [Ch. 20](#)), Ramsay-Hunt syndrome.
- Inflammatory conditions: sarcoidosis, Guillain-Barré syndrome (facial weakness is common in Guillain-Barré syndrome, and is often bilateral; see [Ch. 27](#)).
- Tumor: At the cerebellopontine angle or in the internal auditory canal (e.g., vestibular schwannoma; see [Ch. 24](#)).
- Vascular: A stroke of the pons can affect the nucleus or fascicle of the seventh nerve causing a lower motor neuron pattern facial palsy in spite of the fact that the lesion is in the central nervous system (i.e., in the brainstem). When this occurs, there are often additional associated deficits, such as ipsilateral horizontal gaze palsy with dorsal lesions (due to the proximity of the CN 6 nucleus to the CN 7 nucleus) and/or contralateral weakness in the extremities with ventral lesions (if the not-yet-crossed descending corticospinal tract is affected—an example of the "crossed signs" of brainstem lesions; see [Ch. 9](#)).

All patients with facial nerve palsy should have an otoscopic examination to evaluate for vesicles in the ear that would suggest a diagnosis of **Ramsay-Hunt syndrome**, caused by varicella zoster virus (VZV) reactivation in the geniculate ganglion. Ramsay-Hunt syndrome is generally treated with steroids and antivirals.

Bell's palsy—Bell's palsy refers specifically to *idiopathic* facial nerve palsy (i.e., facial nerve palsy without an identifiable etiology). Bell's palsy is more common in diabetics and during the third trimester of pregnancy. Facial weakness usually emerges over hours, and patients may report pain behind the ear, liquids dripping from one side of the mouth when drinking, and ocular irritation from inability to close the eye. Some patients report facial numbness as a way of describing the feeling of the face being weak, although true facial numbness due to trigeminal involvement is uncommon. If assessed, taste may be impaired on one half of the tongue on the side of the facial weakness (only if the nerve is affected proximal to the chorda tympani; this does not occur in all cases). Patients often report that sounds are louder on the affected side (**hyperacusis**) due to weakness of the stapedius muscle (which normally serves to dampen loud noises). Hyperacusis can be demonstrated by passing a vibrating tuning fork between the two ears, and noting if the patient finds it to be louder on the side of the facial weakness. Facial weakness in Bell's palsy may be incomplete at presentation, and if the lower face is predominantly affected, this can mimic an upper motor neuron pattern, leading to evaluation for stroke as the cause of facial weakness.

Most patients begin to recover from Bell's palsy by 1 month and recover completely over subsequent months. A short course of oral steroids can increase the degree and speed of recovery. Some practitioners treat Bell's palsy with antivirals ([acyclovir](#)) in addition to steroids based on the hypothesis that the condition may be caused by the herpes simplex virus (HSV), but the use of [acyclovir](#) is debated.

Neuroimaging is generally unnecessary in patients with Bell's palsy unless the patient does not improve or develops additional cranial nerve palsies. In patients with Bell's palsy who undergo neuroimaging, enhancement of the affected CN 7 may be observed.

Reduced blinking and impaired eye closure in Bell's palsy (as in any seventh nerve palsy) can cause corneal inflammation (exposure keratitis), which can lead to corneal ulceration. Therefore, an important supportive measure in patients with Bell's palsy is to protect the eye with artificial tears and patching.

As the facial nerve recovers, abnormal rewiring may lead to abnormal linking of facial movements, called **synkinesis**. Examples include linking of eye and mouth movements (squinting when smiling and vice versa) and linking of lacrimation and salivation (tearing while eating, a phenomenon called "crocodile tears").

Melkersson-Rosenthal syndrome—This is a rare syndrome characterized by recurrent seventh nerve palsy and facial/lip swelling, and patients have a fissured tongue. The disorder usually begins in childhood/adolescence, and the complete triad of symptoms may not be present in all cases.