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SECTION 7: NEUROLOGIC DISORDERS

c00031

31 *Facial Nerve Palsy*

GARRETT GRIFFIN, AARON FAY, and BABAK AZIZZADEH

CHAPTER OUTLINE

s0010 Introduction

p0145 Few ailments are as physically and psychologically debilitating as facial paralysis.[1,2] Preventing corneal exposure and keratitis frequently becomes the first priority, requiring ophthalmic support early in the disease process. Thus, it is essential to understand the myriad causes of facial paralysis, diagnostic workup and potential medical and surgical interventions.

p0150 Bell palsy is the most common cause of facial neuropathy and most patients will recover with little to no sequelae. Roughly 30% of patients with facial weakness, however, *do not* have Bell palsy.[3] These patients have systemic or neoplastic disorders that, left untreated, could be life-threatening. The focused history and physical examination can be life-saving.

s0015 Historical Background

p0155 Greek and Roman scientists such as Hippocrates and Galen described facial spasm in their written works, but the earliest thorough descriptions of peripheral facial *palsy* are attributed to the Persian physicians Tabari and later Muhammad ibn Zakariya al-Razi. It was Razi who first divided facial distortion into spastic and paralytic disorders, described central and peripheral palsy and gave the first description of loss of forehead wrinkling in peripheral facial palsy.

p0160 Although facial palsy is relatively rare, it is common enough to have affected several well-known individuals, including the poet Allen Ginsburg, composer Andrew Lloyd Weber and actor George Clooney.

s0020 Fundamental Science

p0165 The facial nerve is a mixed nerve containing motor, sensory and parasympathetic fibers comprising two afferent (general sensory and special sensory taste) and two efferent (motor and parasympathetic) pathways. The path of the facial nerve is classically divided into six segments (Table 31.1; Fig. 31.1 and Table 31.2 further illustrate the different components of this complex cranial nerve (CN).) It is important to remember that the upper motor neuron tracts destined for the upper face project to the bilateral facial motor nuclei, preserving bilateral upper facial movement (including eyelid blinking) in unilateral cerebrovascular accidents.[4]

p0170 The facial nerve motor nucleus is located in the caudal aspect of the ventrolateral pons. Efferent motor fibers take a circuitous path anteromedially to loop around the abducens nucleus and then enter the internal auditory canal with the cochlear and superior and inferior vestibular nerves. The mnemonic '7 UP over COKE' reminds one that in the distal internal auditory canal, the CN VII is superior to the cochlear nerve. Motor fibers then take a sharp turn posterolaterally as they pass through the geniculate ganglion (ganglion for taste and sensory fibers) and enter the middle ear space. In the middle ear, the motor nerve passes just superior to the oval window and stapes footplate. The motor nerve then makes its 'second genu' as it leaves the middle ear to enter the mastoid bone. As the motor branch exits the stylomastoid foramen to enter the body of the parotid gland, fibers innervate the auricular, stylohyoid, posterior digastric and occipitalis muscles. In the face, the nerve arborizes extensively. Classic descriptions depict the nerve dividing into an upper and lower division at the *pes*

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t0010 **Table 31.1** Facial Nerve Segments

Name	Length	Extent	Key Components
Intracranial	23 mm	Brainstem to internal auditory canal (IAC)	Contains fibers from the two afferent and two efferent nerve pathways.
Meatal	8–10 mm	Fundus of IAC to meatal foramen	In the IAC, the facial nerve runs superior to the cochlear nerve and anterior to the superior vestibular nerve. Meatal foramen is the narrowest portion of the Fallopian canal and is felt to be the site at which nerve edema may cause Bell palsy.
Labyrinthine	3–5 mm	Meatal foramen to geniculate ganglion	Gives off the greater superficial petrosal nerve (preganglionic parasympathetic fibers causing lacrimation and salivation). The geniculate ganglion contains the cell bodies of afferent general and special sensory fibers.
Tympanic	8–11 mm	Geniculate ganglion to second genu	The nerve passes through the middle ear space superior to the oval window. Site of most injuries to the facial nerve related to temporal bone fractures.
Mastoid (vertical)	10–14 mm	Second genu to stylomastoid foramen	Gives off branches to the stapedius muscle and chorda tympani nerve (afferent taste fibers from anterior two-thirds of tongue).
Extratemporal segment	NA	Stylomastoid foramen to facial muscles	Facial nerve innervates the stylohyoid, posterior digastric and intrinsic auricular muscles in addition to the muscles of facial expression.

t0015 **Table 31.2** Components of the Facial Nerve

Type	Component	Function
Efferent	Branchial motor	To supply the stapedius, stylohyoid, posterior digastric and muscles of facial expression, including the buccinators, platysma and occipitalis muscles.
	Visceral motor (general visceral efferent)	Preganglionic parasympathetic fibers passing through the greater superficial petrosal nerve. Stimulates the lacrimal, submandibular and sublingual glands. Also glands within the mucous membrane of the nose and hard and soft palates.
Afferent	General sensory (general somatic afferent)	Sensory fibers from the skin of portions of the auricle and external auditory canal. These fibers pass through the nervus intermedius.
	Special sensory	Taste from the anterior two-thirds of the tongue.

anserinus (Latin for 'goose foot'), and then into five major branches at the anterior edge of the parotid gland: temporal, zygomatic, buccal, marginal and cervical. Table 31.3 presents some surgical pearls and landmarks for these main branches. More recent cadaver dissections demonstrate significant variability in branching pattern with eight to 14 large branches in most individuals.[5] The facial nerve is deep to the superficial muscular aponeurotic system (SMAS) and all of the muscles of facial expression except for the mentalis, buccinator and levator anguli oris.[6]

p0175 Efferent parasympathetic fibers originate in the superior salivary nucleus in the pontine tegmentum. These fibers traverse the cerebellopontine angle completely separate from the motor root, within the *nervus intermedius* (which also contains afferent taste fibers), before joining the motor fibers at the geniculate ganglion. Some efferent parasympathetic fibers exit almost immediately as the greater superficial petrosal nerve (GSPN). The GSPN is joined by sympathetic fibers jumping off the internal carotid artery as the deep petrosal nerve to form the vidian nerve. The vidian nerve enters the pterygopalatine ganglion and preganglionic parasympathetic nerves destined for the lacrimal gland synapse. Postganglionic parasympathetic fibers then leave the pterygopalatine fossa, joining the infraorbital and then lacrimal nerves to reach the lacrimal gland. Efferent parasympathetic fibers destined for the submandibular and sublingual salivary glands pass through the geniculate ganglion and leave the main motor nerve in the mastoid segment as the chorda tympani nerve. These fibers synapse in the submandibular ganglion.[7]

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p0180 Afferent taste fibers from the anterior two-thirds of the tongue travel with the lingual nerve before joining the chorda tympani nerve, with cell bodies in the geniculate ganglion and a final target within the nucleus solitarius. Touch sensory fibers from the auricle and ear canal are the second afferent pathway. These fibers also have cell bodies in the geniculate ganglion and pass through the nervus intermedius before terminating in the spinal tract of the trigeminal nerve.[4] This is one of many ways that the facial and trigeminal nerves are closely aligned.

EMBRYOLOGY

s0025

p0185 During the third week of gestation, the facioacoustic primordium appears and differentiates into CNs VII and VIII. By the fifth week of gestation, the nervus intermedius, geniculate ganglion, chorda tympani and greater superficial petrosal nerves are visible. The branchial arches are each associated with a CN, and the facial nerve is the nerve associated with the second branchial arch derivatives, which include all of the targets described above, as well as the stapes, styloid process, stylohyoid ligament and portions of the hyoid bone. By the sixth week of gestation, the first and second branchial arches give rise to small condensations of mesoderm termed the *hillocks of His*. These fuse to form the auricle around the 12th week of gestation.

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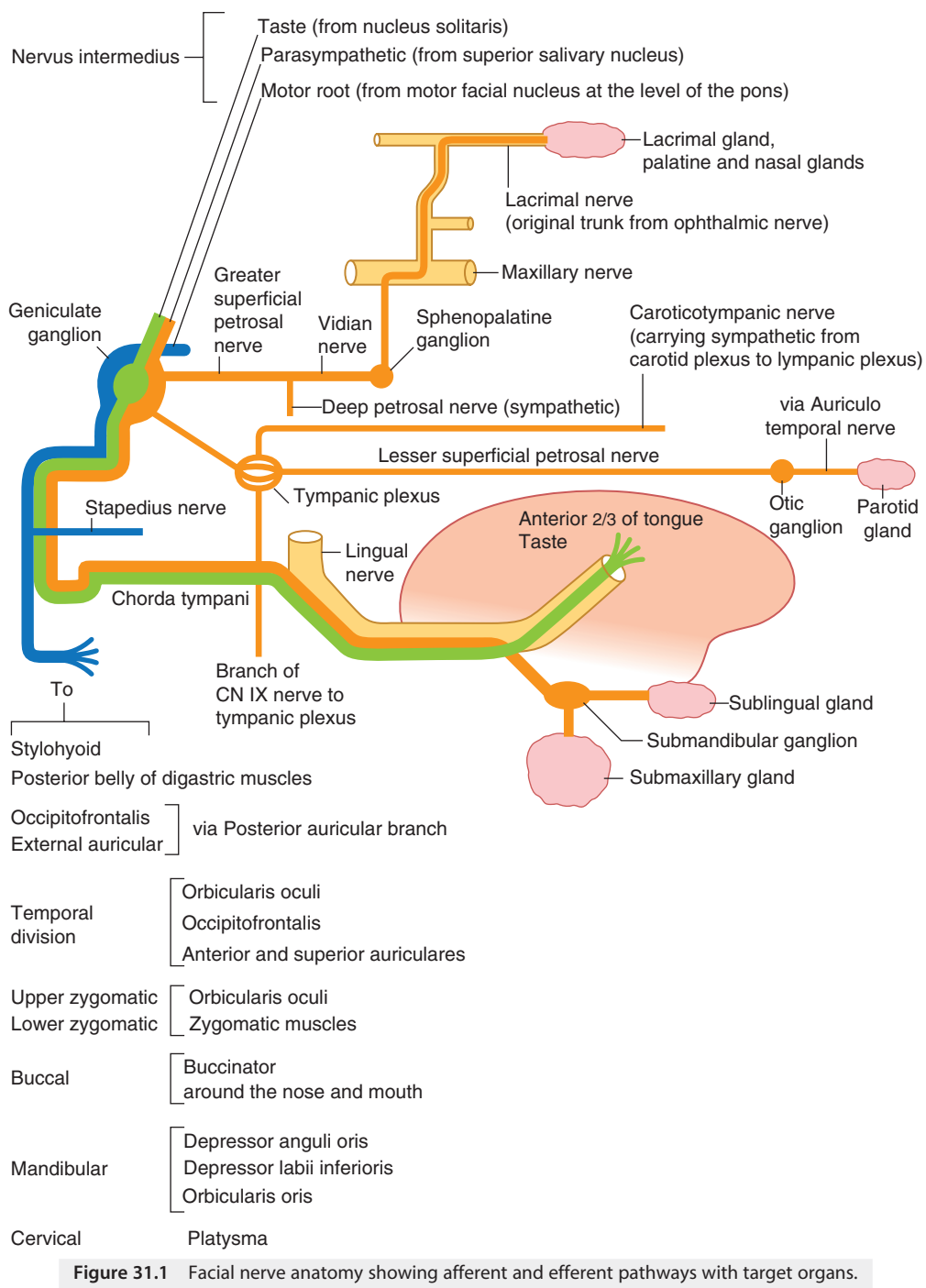

f0010 **Figure 31.1** Facial nerve anatomy showing afferent and efferent pathways with target organs.

Malformations of the pinna suggest a developmental anomaly within the second branchial arch and should alert the physician to possible abnormalities in the structure and/or function of the facial nerve.[8]

p0190 The parotid gland begins as an outpouching of oral ectoderm during the sixth week of gestation. The facial nerve grows anteriorly as the parotid gland grows posteriorly, eventually encapsulating much of the facial nerve. The parotid gland is the last major salivary gland to encapsulate, which is why it contains lymph nodes. The facial nerve divides the parotid gland into 'superficial' and 'deep' lobe; however, these two regions are not distinct entities.[9] There may be lymph nodes in both the superficial and deep lobes. This fact must be kept in mind when considering regional lymphadenectomy for advanced cutaneous malignancies.[10]

Epidemiology s0030

p0195 It is difficult to estimate the relative frequency of facial palsy etiologies. Most data is kept at large academic facial nerve centers that and probably do not represent a general population of patients. The most common etiologies of facial paresis among surgical patients at the University of Pittsburgh Facial Nerve Center between 1963 and 1996 were

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t0020 11

Table 31.3 ••

Branch	Surgical Pearls/Landmarks
Frontal (temporal)	Innervates the frontalis muscle Typically 2–4 branches cross the zygomatic arch between roughly 2 cm anterior to the root of the helix and 2 cm posterior to the junction of the arch and the body of the zygoma. The nerve is quite deep over the zygomatic arch, beneath the parotidomasseteric fascia The nerve enters the deep aspect of the temporoparietal fascia starting 15 mm superior to the arch and 15 posterior to the lateral orbital rim.
Zygomatic	Innervates the orbicularis oculi. Relatively well-protected under the orbicularis oculi and zygomatic major muscles.
Buccal	Innervates the zygomaticus major making it the main driver of smile. Innervates orbicularis oris. There are typically several large buccal branches, often with contributions from the upper and lower divisions of the facial nerve. Quite exposed between the anterior edge of the parotid gland and the zygomatic major muscle. Branches can be seen directly on the surface of the masseteric muscle, deep to the parotidomasseteric fascia. Some research suggests that terminal branches innervate the 'medial canthal' segment of the orbicularis muscle, which is most important for lower eyelid tone and blink. Terminal branches pass inferior to the orbit to innervate the glabellar musculature (corrugator, procerus).
Marginal mandibular	Innervates the lower lip depressors. Easiest branch to injure due to its long course. Usually passes within 1 cm of the angle of the mandible but has been found several centimeters inferior to the mandible lateral to the facial artery. Always above the inferior border of the mandible anterior to where the facial artery crosses the mandible.
Cervical	Innervates the platysma. Relatively unimportant. Injury can cause smile asymmetry as a result of loss of lip depressor function of the platysma.

acoustic neuroma surgery (25%), Bell palsy (17%), parotid and skin neoplasms (8%), varicella zoster (6%), trauma (6%) and genetic or congenital causes (6%).[11] The Massachusetts Eye and Ear Infirmary Facial Nerve Center recently reported a series of 2000 patients presenting with facial palsy, not necessarily requiring procedural intervention. The most common etiologies were Bell palsy (38%), acoustic neuroma surgery (10%), cancer (7%), iatrogenic injuries (7%) varicella zoster (7%), congenital palsy (5%), and Lyme disease (4%).[12] About 60% of patients in both series were female. Because most patients with Bell palsy will recover to normal facial function, they are underrepresented in series from facial nerve centers. It is thought that roughly 70% of cases of facial paresis are caused by Bell palsy.

p0200 Nearly 10% of patients with Bell palsy have had a prior episode.[13] Findings of studies are mixed as to whether the prognosis is worse for a second attack. Recurrent ipsilateral facial palsy necessitates a careful investigation. In one study, 20% of patients with a second bout of paresis on the same side were found to have a tumor along the course of the facial nerve.[14]

s0035 Classification of Peripheral Nerve Injury

p0205 Understanding the fundamentals of peripheral nerve injury provides a useful framework for thinking about how much recovery to expect and is critical to the correct implementation and interpretation of electrophysiologic (EP) facial nerve studies (see below). Seddon initially classified peripheral nerve injury into three types: (1) neuropraxia, (2) axonotemesis and (3) neurotemesis.[15] Sunderland expanded on Seddon's work by further subclassifying the neurotemesis category, which resulted in five distinct degrees of nerve injury.[16] Table 31.4 displays the theoretical characteristics of each type of nerve injury along with a range of possible outcomes from electromyography (EMG) and electroneurography (ENOG).

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p0210 Each nerve fiber consists of an axon that contains cytoplasm, aptly named *axoplasm*. The axoplasm conducts electrical impulses along the axon. The axon is surrounded by a myelin sheath made of Schwann cells (in peripheral nerves) and other connective tissues. This sheath is often referred to in the literature as the *endoneurium*, *neural tubule* or *endoneural tube.* Several axonal sheaths are bundled and held together by connective tissues (called the *perineurium*) to form the funiculus or fascicle. Finally, several fascicles are held together by areolar connective tissue, which, in the facial nerve, becomes more compressed outside the temporal bone, where it is called the *epineurium*. The epineurium, perineurium and endoneurium can be viewed as three 'protective layers' of connective tissue that serve to shelter the axon (Fig. 31.2).[17]

p0215 Sunderland's five degrees of nerve injury are based on the extent of damage to various functional anatomic components and layers of the nerve.

- u0145 *First-degree injury (neuropraxia):* First-degree injuries are the result of a conduction block that typically occurs after nerve compression or ischemia. There may be localized damage to the myelin sheath but there is no axonal degeneration; axonoplasmic continuity remains intact distal to the lesion. Therefore, neuropraxic injuries continue to conduct a neural impulse if an electrical stimulus is delivered distal to the site of lesion. This is the basic premise on which ENOG testing is based. Recovery from this type of injury is spontaneous and complete.

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Table 31.4 ••

Category	Degree of Injury				
Seddon's classification[5]	Neurotemesis	Axonotemesis	Neuropraxia		
Sunderland's classification[6]	First degree Conduction block	Second degree Axonal continuity	Third degree Endoneural tubule	Fourth degree Funiculus	Fifth degree Complete nerve trunk
Recovery[6]	Complete <2 weeks	Complete or mild	Residual deficit	Some spontaneous recovery that is rarely useful	If untreated, rare, and residual deficit, if treated
Wallerian degeneration[5,6,12,15]	Does not occur	6–21 days, often >14 days	6–21 days often <14 days		100% by 3–5 days
ENOG[7-17,19-21]	Normal	There is a >50% chance of an incomplete recovery when the ENOG response is reduced by ≥90%			100% denervation by 3–5 days*
EMG (resting)[20-22,25, 29,30]	Absent patholo gic spontaneous activity	Pathologic spontaneous activity is present after 14–21 days, which heralds a high probability (80 %+) of an incomplete recovery			
EMG (volitional)[20-22,25,29-30]	Volitional responses can be intact, reduced or absent. Early intact volitional responses at multiple sites or Improvement in the firing pattern suggest a good outcome. Absent (or minimal) volitional predicts a poor recovery.				No volitional activity*

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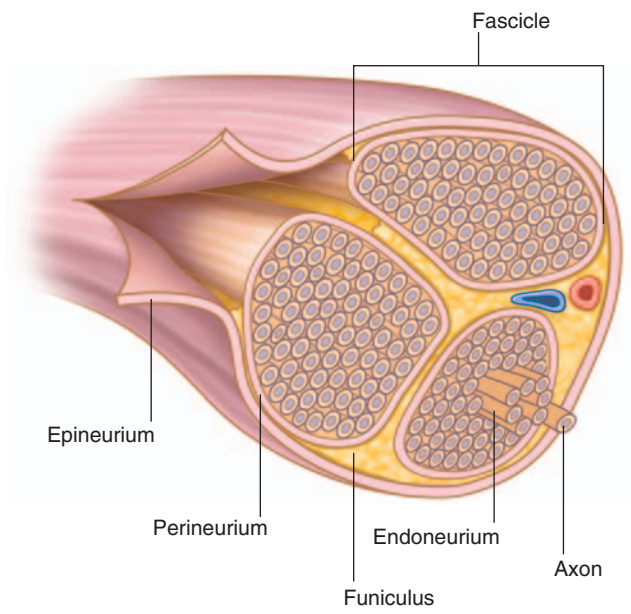
EMG, Electromyography; ENOG, electroneuronography.



f0015

Figure 31.2 Peripheral nerve ultrastructure.

u0150 ■ *Second-degree injury (axonotemesis):* In this degree of injury, complete interruption of the axoplasm contained within the axon is seen. The endoneural tubule, however, is preserved. Anterograde axonal degeneration (Wallerian degeneration) occurs, resulting in the peripheral end organ being isolated from its corresponding neuron. This loss of nerve supply to the end organ is referred to as *denervation.* Because the endoneurium remains intact, the axon can regenerate toward its original target through the intact tubule. This leads to a better prognosis in terms of recovering motor function following the injury. *Preservation of the endoneurium prevents synkinesis.*

u0155 ■ *Third-degree injury (neurotemesis):* Third-degree injury involves damage to the endoneural tube and its contents. Retrograde disturbances are more significant because regeneration can now occur across disrupted endoneural tubules. Axons may reach functionally related end organs or they may enter totally foreign endoneurial tubes. This results in internal disorganization because some axons do not regenerate to their original end organs. The resulting abnormal healing creates a distorted and less efficient firing pattern, with the clinical manifestations of synkinesis and/or contracture. *Synkinesis* is the involuntary contraction of an erroneously reinnervated muscle during a contraction of the normally innervated muscle.[17] For example, a patient's eye may blink when she attempts to smile. Synkinesis can also manifest as increased baseline tone (contracture). Overall recovery is longer and usually incomplete in third-degree injuries.

u0160 ■ *Fourth-degree injury (neurotemesis):* Fourth-degree injuries involve damage to the funiculus and its contents. The entire funiculus is involved and all bundles are breached. Funicular bundles become so disorganized that they are no longer distinguishable from the surrounding connective tissues of the epineurium. Large numbers of regenerating axons escape and infiltrate foreign tubes. Some spontaneous recovery can occur, but it is of little functional value.

u0165 ■ *Fifth-degree injury (neurotemesis, complete nerve transection):* A fifth-degree injury involves transection of the entire nerve trunk. The majority of axons do not reach their designated funiculi or endoneural tubule because of the separation of the nerve ends and scarring. Recovery will not occur without surgical intervention, and complete restoration of function is impossible, even if the nerve ends are repaired.

Clinical Features s0040

HISTORY s0045

p0245 One of the most important aspects of the clinical evaluation is to try to diagnose the cause of facial palsy. Many patients with facial paresis are told they have 'Bell palsy' by their physicians when, in fact, their presentation is *not* consistent with Bell palsy. *Bell palsy should progress from normal facial function to maximum facial weakness in less than 72 hours.*

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Viral causes of facial paresis involve the entire hemiface, including the forehead. Varicella zoster can present identically to Bell palsy with delayed onset or no onset of a vesicular skin eruption. Lyme disease also presents identically to Bell palsy with occasional bilateral facial paralysis. Thus, all patients presenting with rapid-onset peripheral facial weakness in Lyme disease–endemic areas should be considered for this diagnosis as well.

p0250 Gradual-onset, recurrent or fluctuating facial paralysis is *not* Bell palsy and should raise suspicion of a neoplasm along the course of the facial nerve. The parotid bed and neck should be carefully palpated for asymmetry or masses that could indicate a neoplasm. These patients require imaging and should be referred to an otolaryngologist for further evaluation.

s0050 PHYSICAL EXAMINATION

p0255 Physical examination of the patient with facial paralysis should follow the regional approached proscribed in the Sunnybrook and House-Brackman grading scales. Each 'zone' should be evaluated for resting, dynamic and synkinetic findings. Patients may present in the acute phase, shortly after paralysis or much later in the course when function has started to return.

p0260 Bell palsy involves all branches of the facial nerve. Weakness in only part of the face is not consistent with a viral etiology and suggests pathology involving specific distal branches of the nerve. The finding of involvement in just one part of the face is a particular cause for concern with regard to perineural invasion by facial cutaneous squamous cell carcinoma and should prompt a careful history and physical examination for any prior or current skin cancers. Facial weakness caused by perineural invasion can appear months to years after removal of the primary skin malignancy.

s0055 Forehead

p0265 The eyebrow and forehead are important for facial expression and impact a person's perceived age. Forehead movement and creases will be intact in central facial paralysis but absent in complete peripheral paralysis. In children and young adults, forehead paralysis will not usually result in brow ptosis, whereas in older adults forehead paralysis is associated with severe brow ptosis. Patients with synkinesis will often exhibit subtle brow elevation with smile and eye closure that may be amenable to botulinum toxin injections.

s0060 Eyelids

p0270 Evaluation of the periocular region should follow a system based on anatomic zones and the three conditions that distinguish the phases of facial nerve recovery. In this chapter, [3] brow evaluation is discussed separately (see above).

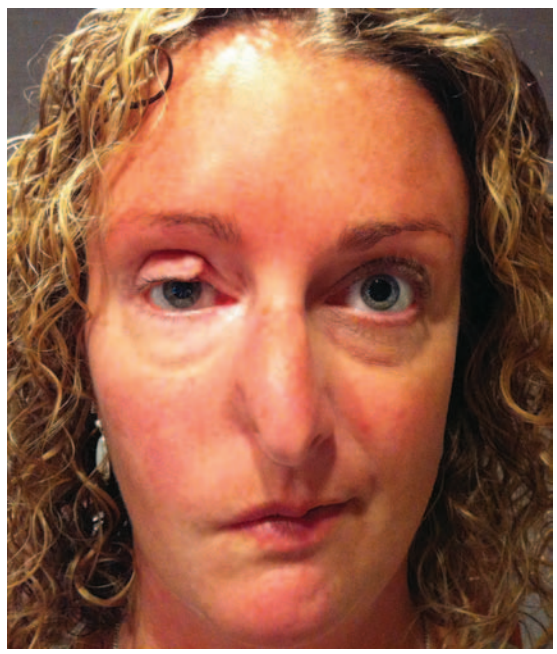
p0275 Upper eyelids are primarily responsible for ocular surface protection but also provide a seal against liquids, keep light out and move the tears across the ocular surface to the lacrimal puncta. Evaluation should note resting position as well as gentle and forced closure. To assess synkinesis, the lids should be observed after asking the patient to smile. Lagophthalmos should be measured with the patient in the upright and supine positions. Many patients will demonstrate 2 to 3 mm of upright lagophthalmos but 8 to 19 mm in the supine position. This discrepancy may be greatest in patients who have had a gold or platinum weight implanted. The eyelashes should be assessed for lash ptosis, particularly if surgery is anticipated. Meibomian gland function should be assessed.

p0280 In the early phase, the ocular surface is at high risk for exposure keratopathy or frank corneal ulceration. The cornea and conjunctiva should be observed closely under the slit lamp, with both fluorescein and lissamine green staining. In the late phase, an increased tear lake may be seen as a result of synkinetic stimulation of the lacrimal gland, especially when eating (gustatory lacrimation).

p0285 The lower eyelids provide protection to the inferior one-fourth of cornea. They may be well positioned immediately after a paralytic insult but tend to become ectropic and [4] retracted. The lid must be assessed for horizontal laxity as well as anterior lamellar shortening, which may progress rapidly in the first few months of paralysis. They should be assessed at rest and with forced closure. Typically, the lower lid rises in the supine position, masking the inferior corneal exposure. Synkinesis manifests with horizontal shortening of the palpebral fissure and lower lid elevation, particularly with talking, smiling or chewing.

Midface s0065

p0290 The midface contains all of the lip elevators. Patients with complete facial paralysis are unable to smile on that side of the face, which makes social interaction more challenging. Buccinator weakness causes difficulty chewing on that side, as the food bolus cannot be held between the teeth, and flapping or 'puffing out' of the cheek during speech. An underappreciated consequence of facial palsy is nasal obstruction, caused by deviation of the nose toward the intact side as well as loss of muscular support around the affected nostril and nasal sidewall (Fig. 31.3).



f0020 **Figure 31.3** Nasal collapse.

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p0295 It is common for patients with synkinesis to have decreased elevation of the corner of the mouth with smile because of synkinetic firing of the lip depressors (platysma, depressor anguli oris), risorius and buccinator. The most severe form of this is a 'frozen smile', where the upward and downward force on the oral commissure is roughly equal and there is minimal movement of the mouth with attempted smile. Synkinetic contracture of the lip elevators also contributes to poor smile excursion and can cause mild to severe midface pain.

s0070 Lower Face

p0300 Lower facial paralysis is relatively well tolerated compared with paralysis of the periocular and midface regions. The most bothersome result is lack of lower lip depression making the smile asymmetric. In older individuals, platysma paralysis will make the appearance of the neck asymmetric as well.

p0305 Lower facial synkinesis can impact the ability to generate a smile, as discussed above. The mentalis muscle is commonly involved in synkinesis manifested as unsightly chin dimpling and/or lower lip elevation during smile and eye closure.

s0075 FACIAL NERVE FUNCTION GRADING SCALES

p0310 Clinical facial nerve grading scales provide a convenient language that providers can use to convey what they see on physical examination. They are also useful in outcomes research. The House-Brackmann[18] and Sunnybrook scales (Fig. 31.4) are the two most commonly used physician-graded facial nerve grading scales in use today. They were developed in different ways by different types of clinicians, and for different purposes. House was a neuro-otologist interested in measuring *what happens to facial function after different kinds of injury to the main trunk of the facial nerve.* The original scale has since been updated to include a regional assessment of facial function.[19] In contrast, the Sunnybrook system[20] was developed by physical therapists and physicians interested in accurately measuring clinical change in facial function *after an intervention meant to improve that function.* As such, the Sunnybrook system is a more continuous scale that collects more finite information. Both the House-Brackmann and Sunnybrook scales are valid and reliable tools that are best used in their separate, intended ways.

s0080 Investigations

s0085 ELECTROPHYSIOLOGIC TESTING

p0315 EP testing is a valuable, objective tool to quantify facial nerve function. Electroneurography (ENOG) and electromyography (EMG) are the two tests that are most useful today.

s0090 Electroneuronography

p0320 The ENOG examination was originally proposed and popularized by Esslen[21-23] and Fisch[23,24] in the late 1970s. The goal of ENOG is to measure the amount of neural degradation that has occurred distal to the site of nerve injury by measuring the muscle response to electrical stimulation of the main trunk of the facial nerve. ENOG measures the compound muscle action potential (CMAP) of the facial muscles following this electrical stimulus. The electrical stimulus is delivered distal to the site of nerve injury. The amount of denervation is represented by comparison of the CMAP from the affected side of the face to the nonaffected side. In effect, ENOG differentiates first-degree 'conduction block' injuries (neuropraxia) from those that have developed Wallerian degeneration (second to fifth degree).

p0325 The CMAP represents the sum of the action potentials of all axons within a nerve. Normally, an electrical stimulus depolarizes all of the nerve fibers at roughly the same time, causing the distal motor units to fire synchronously.[15] These action potentials would then be summated to create a CMAP much larger than the individual action potentials from single axons. Fig. 31.5, A, portrays several near-simultaneous motor unit action potentials being summated into a compound response. If very few axons are stimulable, the compound action potential is smaller than expected (see Fig. 31.5, B).

p0330 Patients with facial neuromas or those recovering from a facial nerve injury often have fibers in different states of recovery.[25] This means that the action potentials may not occur at the same time and will not summate and can, in fact, cancel each other creating a low CMAP (see Fig. 31.5, C). This can sometimes result in discrepancies between the ENOG and EMG responses. The implications for interpreting these discrepant results are discussed in the 'Interpretation of EMG' section below.

s0095 p0335 **Interpretation of Electroneuronography.** ENOG's primary utility is in determining the long-term prognosis of facial function. Additionally, some authors have tried to use ENOG to determine which patients might benefit from surgical intervention such as decompression of the facial nerve.[25] Patients who demonstrate facial paresis in the setting of a normal ENOG study (ie, no signs of Wallerian degeneration) are likely to have a conduction block (first-degree injury) and will tend to have a satisfactory recovery.[26]

Electromyography s0100

p0340 EMG is the recording of motor unit action potentials (MUAPs). MUAPs are the spikes in electrical activity generated when a motor unit fires. A motor unit consists of a motor neuron and the corresponding muscle fibers innervated by the neuron. Each motor unit consists of the neuron and its axon, which has *multiple* synaptic junctions that are affiliated with corresponding muscle fibers. These synaptic junctions are called *myoneural junctions.* Each myoneural junction and muscle fiber generates a small electrical potential when activated. The synchronized discharge arising from all of the axon's myoneural junction potentials combine to form the larger MUAP.

p0345 EMG differs from ENOG most importantly in that EMG relies on the patient's nervous system to generate an impulse proximal to the site of injury and to pass this stimulus through the facial nerve to the target muscle fibers, which is measured. ENOG is an artificially generated electrical impulse applied to the facial nerve distal to the site of injury. Q

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Level	Characteristics
1. Normal	Normal function in all areas of the facial nerve
2. Mild dysfunction	Clinical observation Slight muscular weakness observed on examination **There may be disordered movements** At rest, the face appears symmetrical and with tones **Movements:** Forehead: moderate to good function Eyes: total closure at the slightest effort Mouth: mild asymmetry
3. Moderate dysfunction	Clinical observation Clear difference between the two hemifaces but not total asymmetry Nonserious disordered movements may be observed Contractura of the facial musclesor spasm in hemifaces **Movements:** Forehead: moderate mobility Eyes: total closure with effort Mouth: weakness of the muscles at maximum effort
4. Moderately severe dysfunction	Clinical observation Clear weakness and/or almost total asymmetry At rest, normal symmetry and maintenance of muscle tone **Movements:** Forehead: no movement Eyes: incomplete closure Mouth: asymmetry at maximum effort
5. Severe dysfunction	Clinical observation Hardly any mobility observed Asymmetry at rest **Movements:** Forehead: no movement Eyes: incomplete closure Mouth: hardly any mobility
6. Total palsy	No movement in any part of the facial nerve

f0025 A

Figure 31.4 A, House-Brackmann facial nerve grading scale. Physical examination findings in bold are considered key differentiators between the grades. (**A,** from House JW, Brackmann DE. Facial nerve grading system. Otolaryngol Head Neck Surg 1985 ;93:146–47.)

s0105 p0350 **Performing EMG.** Recordings are made from various mimetic muscles that are innervated by the facial nerve. Typically, five muscle sites are tested corresponding to the five main facial nerve distributions (frontal, zygomatic, buccal, marginal mandibular and cervical). Electrical activity at each test location is recorded both at rest and during attempted volitional movement. Granger demonstrated that patients with voluntary EMG potentials in four to five muscles 72 hours after paralysis onset has a 91% chance of a favorable outcome.[27] Abnormal resting potentials are a sign of neural degeneration and appear as early as 7 days[28] but more frequently after 10 to 14 days postinjury.[29,30]

Q

s0110 p0355 **Interpretation of EMG.** Several authors have recommended using the presence or absence of pathologic spontaneous activity (fibrillation potentials and/or positive sharp waves) with a goal of separating out those patients who will recover spontaneously from those patients who will require further intervention.[31-33] Under this system a first-degree injury is defined as decreased or absent EMG activity without pathologic spontaneous activity after 10 to 14 days. Inversely, the presence of pathologic spontaneous activity along with decreased or absent volitional activity is categorized as a second or higher degree of injury and thus predicts a poor outcome. It is important to understand that this classification system is not valid until after 10 to

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Resting symmetry	Symmetry of voluntary movement						Symmetry of voluntary movement				
Compared to normal side **Eye (choose one only)** normal 0 narrow 1 wide 1 eyelid surgery 1 **Cheek (naso-lablal fild)** normal 0 absent 2 less pronounced 1 more pronounced 1 **Mouth** normal 0 corner dropped 1 corner pulled up/out 1	**Degree of muscle excursion compared to normal side**						**Rate the degree of involuntary muscle contraction associated with each expression**				
	Standard expressions	Unable to initiate movement/ no movement	Initiates slight movement	Initiated movement with mild excursion	Movement almost complete	Movement complete	**None:** No synkinesis or mass movement	**Mild:** Slight synkinesis	**Moderate:** Obvious but not disfiguring synkinesis	**Severe:** Disfiguring synkinesis/ Gross mass movementof several muscles	
	Forehead wrinkle (FRO)	1	2	3	4	5 ☐	0	1	2	3	☐
	Gentle eye closure (OCS)	1	2	3	4	5 ☐	0	1	2	3	☐
	Open mouth smile (ZYG/RIS)	1	2	3	4	5 ☐	0	1	2	3	☐
	Snarl (LLA/LLS)	1	2	3	4	5 ☐	0	1	2	3	☐
Resting symmetry score **Total** ☐ **Total X 5** ☐	Lip Pucker (OOS/OO1)	1	2	3	4	5 ☐	0	1	2	3	☐
		Gross asymmetry	Severe asymmetry	Moderate asymmetry	Mild asymmetry	Normal symmetry					
						Total ☐					
	Voluntary movement score: **Total X 4** ☐						**Synkinesis score:** **Total** ☐				
Patient's name _____ Dx _____ Date	**Vol mov't score** ☐ – **Resting symmetry score** ☐ – **Synk score** ☐ = **Composite score** ☐										

B

Figure 31.4, cont'd **B,** Sunnybrook facial grading scale. (**B,** from Ross BG, Fradet G, Nedzelski JM. Development of a sensitive clinical facial grading system. Otolaryngol Head Neck Surg 1996;114:380–6)

14 days when the abnormal spontaneous responses will appear.

s0115 **Prognostic Value of EMG.** Volitional EMG responses at
p0360 more than four of the five recording sites is very predictive of a good recovery (House-Brackmann I or II). Several studies have clearly shown the utility of EMG to prognosticate following facial nerve injury.[31,33] The high positive and negative predictive values of EMG studies performed 2 weeks or later after the onset of injury suggests that EMG may be the study of choice for patients who present to the clinic several weeks after the onset of their injury, as well as for patients who are undergoing long term monitoring.

p0365 EMG and ENOG findings associated with good and bad outcomes are summarized in Table 31.5.

Table 31.5 Good Versus Bad Outcome Indicators t0030

Good Outcome Indicators	Bad Outcome Indicators
Normal ENOG after 14–21 days	Weakness at time of trauma
Late onset of Wallerian Degeneration	Early onset Wallerian Degeneration
Normal resting potentials on EMG after 14–21 days	Progressive decay of ENOG
Early volitional motor units <4 days	Abnormal spontaneous activity on EMG after 14–21 days
Improving volitional EMG	Delayed recovery > 3 months (Bell palsy) functional & EMG
Volitional EMG recorded at 2 + sites (4–5 better)	No Volitional EMG or Volitional EMG only at 1 recording site

EMG, Electromyography; ENOG, electroneuronography.

s0120 Timing Considerations for ENOG and EMG

p0370 When performing ENOG and EMG, it is important to consider how much time has elapsed since the onset of the injury. Table 31.6 suggests a timeline for EMG and ENOG evaluations. Key issues related to timing of the evaluation include the following:

1. ENOG should never be performed earlier than 3 days following injury because abnormal ENOG responses rely on the onset of Wallerian degeneration, which requires a minimum of 72 hours to occur. o0170
2. Early signs of denervation on ENOG are a poor prognostic sign, as they herald a more severe nerve injury. o0175

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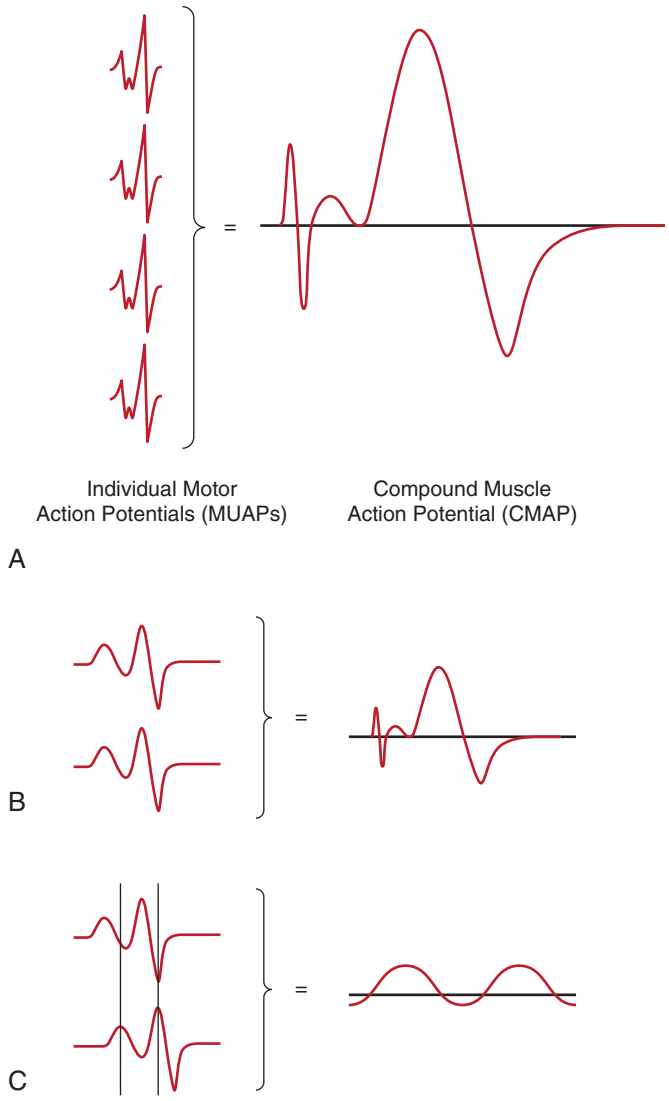

f0030

Figure 31.5 **A,** Example of how individual motor unit action potentials (MUAPs) summate to yield the compound muscle action potential (CMAP). **B,** Example of how smaller MUAPs create a smaller CMAP. **C,** Example of how asynchronous MUAPs yield a smaller and different CMAP.

o0180 3. EMG can be performed at any time after the injury. Early responses on voluntary EMG, especially at multiple locations, are a positive indicator for recovery and rule out complete nerve transection.

o0185 4. EMG's prognostic value improves when testing can be deferred until 10 to 14 days after the onset of injury. This will allow signs of abnormal healing or abnormal resting potentials to occur. However, when facial nerve decompression is being considered, delaying treatment may compromise recovery.

s0125 Pathogenesis

p0395 The most common cause of facial palsy is Bell palsy, more appropriately termed *acute idiopathic facial palsy*. The term *Bell palsy* should be used only after other causes have been carefully considered. The mnemonic VINDICATES (Vascular, Infectious, Neoplastic, Degenerative, Iatrogenic, Congenital, Autoimmune, Traumatic, Environmental, pSychosocial) can help with the differential diagnosis (Table 31.7).

Table 31.6 Timing Considerations for ENOG and EMG t0035

Onset	If face is immediately weak at time of trauma, this is concerning for severe injury. Later onset is more favorable than early onset.
0–3 days	ENOG will always be normal (unless injury is distal to point of stimulation).
	Presence of any volitional activity on EMG can rule out a complete nerve transection; intact responses at multiple sites are a positive sign, abnormal (unfavorable) spontaneous activity cannot be ruled out at this time.
3–5 days	Evidence of Wallerian degeneration (via ENOG) in this early stage after injury is concerning for a fifth-degree injury (complete transection).
6–14 days	Evidence of onset of Wallerian degeneration (via ENOG) in this time frame is suggestive of a grade 3 to 4 injury. The cutoff for surgical decompression in Bell palsy is 12 to 14 days.
14–21 days	Evidence of later onset Wallerian degeneration on ENOG suggests type 2 injury. EMG can be evaluated for presence of abnormal spontaneous activity (which suggests a second-degree or worse injury and an unfavorable outcome).
8–12 months	EMG can be used to monitor for improved volitional responses and to help determine the patient's candidacy for a dynamic facial reanimation procedure.

EMG, Electromyography; ENOG, electroneuronography.

Table 31.7 Differential Diagnosis of Facial Paralysis (VINDICATES) t0040

Type of Process	Differential Diagnosis
Vascular	Stroke (spares upper facial movement) Carotid artery aneurysm
Infectious/ Inflammatory	Viral: VZV, HSV-1, HIV Bacterial: Lyme, acute otitis media, syphilis Fungal: systemic dissemination/meningitis
Neoplastic	Benign and malignant skull base tumors Facial nerve tumors: facial schwannoma Parotid cancers, large skin malignancies
Degenerative	Guillain-Barré, cholesteatoma
Iatrogenic/ Idiopathic	Surgery Birth trauma
Congenital	Facial nerve agenesis, congenital unilateral lower lip palsy Syndromes: hemifacial microsomia Birth trauma
Autoimmune	Sarcoidosis, multiple sclerosis
Trauma	Temporal bone fracture, deep facial laceration
Environmental	Thalidomide exposure during pregnancy
p**S**ychosocial	Very difficult to malinger

HIV, Human immunodeficiency virus: HSV, herpes simplex virus; VZV, varicella zoster virus.

BELL PALSY s0130

Bell palsy accounts for approximately 70% of cases of facial palsy.[3] Historically, this was considered a diagnosis of p0400

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exclusion, but now more emphasis is placed on history and physical examination findings specific for the diagnosis. A recent consensus statement by the American Academy of Otolaryngology defined Bell palsy as acute, unilateral facial nerve paresis or paralysis with onset over less than 72 hours and without another identifiable cause.[34] Patients with Bell palsy go from normal facial movement to *peak facial weakness* in less than 72 hours. They will often report going to bed normal and waking up with significant paralysis. Gradual onset or fluctuating facial weakness is *not* Bell palsy and is often a sign of more ominous causes.

s0135 ## Incidence

p0405 The annual incidence of Bell palsy is 11 to 53 per 100,000, and varies in the population studied.[34] Most studies suggest a lower but not insignificant incidence in children.[35,36] Both sides of the face are affected equally. Gender distribution is equal, although some studies have reported that it is slightly more common in females. Pregnancy is considered a risk factor, with an incidence roughly three times greater than expected compared with age-matched nonpregnant women.[37] Roughly two-thirds of patients will present with complete paralysis.[38] Concurrent bilateral paralysis occurs in 0.3% of patients, and 9% of patients have a history of previous paralysis. There is a family history in 8% of patients.[39]

s0140 ## Etiology

p0410 The cause of Bell palsy is still unknown. The leading hypotheses have been ischemic neuropathy, autoimmune reactions and viral infection. Evidence supports a viral cause, although it is unclear whether the ultimate cause of the palsy is a direct effect of the virus on the nerve or secondary to an ischemic neuropathy. Mumps, rubella, human immunodeficiency virus, herpes simplex virus and Epstein-Barr virus have all been implicated in facial paralysis. It is not uncommon for patients with Bell palsy to have other cranial nerve abnormalities, most commonly of the trigeminal, glossopharyngeal and vagal nerves.[40] As discussed above, the facial nerve has multiple important functions beyond facial movement and can be affected in Bell Palsy, leading to hyperacusis, dysgeusia and decreased tearing.

p0415 Current evidence suggests that herpes simplex virus (HSV) or varicella zoster virus (VZV) reactivation is the cause. Murakami et al.[13] used polymerase chain reaction (PCR) and Southern blot analysis to look for viral DNA in the postauricular muscle and endoneurial fluid from patients diagnosed with Bell palsy and Ramsay Hunt syndrome. In patients with Bell palsy, HSV DNA was recovered from 11 of 14 patients and VZV from none. Conversely, VZV DNA was recovered from all nine patients with Ramsay Hunt syndrome, whereas none had HSV DNA. Control patients did not have DNA from either virus.[13]

p0420 There likely exists a third clinical entity called *zoster sine herpete* (ZSH), and those affected by it include patients affected by VZV, with acute-onset facial paralysis and no skin vesicles. Lee et al. defined patients with ZSH as those with acute facial paralysis and deep muscular or superficial prickling pain. When they compared anti-HSV and anti-VZV immunoglobulin A (IgA) and IgM levels between patients and controls, they found that anti-HSV IgA and IgM were present in all subjects but that anti-VZV levels four times the normal were present in the ZSH and Ramsay Hunt groups but not in the Bell palsy or control groups. Furthermore, when patients with ZSH were randomized to receive treatment with prednisone or prednisone plus antivirals, the patients receiving antiviral medication recovered better.[41]

Pathophysiology and Pathology s0145

p0425 Bell palsy is the most common acute mononeuropathy, yet viruses affect many CNs. The primary difference between the facial nerve and the other CNs is its long bony canal. Fisch measured the diameter of the facial nerve canal throughout its course and found the narrowest point to be 0.68 mm at the junction of the internal auditory canal and labyrinthine segment. Fisch termed this area the *meatal foramen* and hypothesized that swelling of the nerve within this bony confine could impede axoplasmic flow and nerve conduction.[42] EP tests have confirmed this location as the site of the lesion in patients with Bell palsy and herpes zoster oticus (Ramsay Hunt syndrome).[23]

p0430 Multiple studies looking at autopsy specimens of the facial nerve in patients who had died shortly after the onset of apparent Bell palsy have identified degeneration of axons and myelin sheaths with phagocytosis, an osteoclastic reaction with bone resorption around the geniculate ganglion and sometimes engorgement of venules with fresh hemorrhage. These findings are consistent with Wallerian degeneration and typically were found starting in the labyrinthine portion of the nerve, just distal to the meatal foramen.[43-45] Histologic specimens of nerve obtained during facial nerve decompression surgery corroborated these findings.[46]

Management s0150

p0435 When history and physical examination are consistent with Bell palsy, no additional testing is needed.[33] Gradual onset or incomplete facial nerve involvement suggest other diagnoses. Idiopathic facial paralysis with no recovery after 6 months requires thorough re-evaluation, including imaging studies.

p0440 Two different randomized placebo-controlled trials have looked at the impact of steroids and antivirals on recovery in Bell palsy. Both studies identified a statistically significant improved chance of a good recovery (House-Brackmann I or II) if a high-dose steroid taper was initiated within 72 hours of the onset of facial weakness. Neither study showed any additional benefit to prescribing antiviral medications.[47,48] However, many authors' recommend that patients with Bell palsy be treated with both corticosteroids and a zostercidal dose of antivirals to make sure that patients with ZSH are treated maximally. One suggested protocol includes prednisone 1 mg/kg up to 60 mg/day for 5 days with a 5-day taper-off, along with valacyclovir 1000 mg three times daily for 7 days.

p0445 There is some evidence that surgical decompression of the facial nerve can improve outcomes in patients with Bell palsy who develop greater than 90% degeneration, as seen on ENOG, and have no voluntary motor unit potentials on EMG within 2 weeks of onset of complete paralysis.[25] Complete decompression of the intratemporal facial nerve is a major skull base surgery and is typically only performed at large academic centers.

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s0155

RAMSAY HUNT SYNDROME (HERPES ZOSTER OTICUS)

p0450 Ramsay Hunt syndrome is the second most common cause of acute facial paralysis, presenting as acute facial neuropathy in combination with a vesicular eruption of the auricle, ear canal, face or upper aerodigestive tract mucosa. It is essentially shingles of the facial nerve distribution which sometimes extends to other nerves, including the vestibulocochlear (hearing loss and vertigo), trigeminal (pain) and glossopharyngeal distributions. Compared with Bell palsy, patients with Ramsay Hunt syndrome have more severe symptoms with greater pain, more profound facial paralysis and less functional recovery.[49] The facial neuropathy develops before the vesicular eruption in up to 10% of patients, and patients with ZSH never develop skin changes despite rising antibody titers against VZV.[50]

p0455 The mainstay of treatment for VZV infections, including cephalic zoster and Ramsay Hunt syndrome, is systemic corticosteroids.[51] The early institution of steroids has been shown to decrease pain and vertigo acutely while also decreasing the incidence of postherpetic neuralgia.[52] Antiviral agents targeting herpes viruses, including acyclovir, valacyclovir and famcyclovir, are recommended to treat Ramsay Hunt syndrome as well. These drugs are preferentially taken up by VZV-infected cells, making the drug nontoxic to uninfected cells. These drugs have no significant side effects and are relatively low cost as well. A typical protocol recommends valacyclovir 1000 mg orally three times daily for 7 days.

s0160

LYME DISEASE

p0460 Lyme disease is caused by the bacteria *Borrelia burgdorferi* and is transmitted from the bite of black-legged ticks (*Ixodes scapularis*) commonly known as *deer ticks*. Deer and mice serve as reservoirs for this bacterial species. In 2013, over 95% of cases were reported from 14 states in the Northeast, mid-Atlantic, and Upper Midwest regions of the United States; thus, it is considered a regional disease according to the Centers for Disease Control and Prevention (CDC). Patients should be questioned about travel to these regions of the United States. In endemic areas, up to half of the cases of facial paralysis in children are caused by Lyme disease.[53]

p0465 Lyme disease is thought to occur in stages in a manner similar to syphilis. The most common primary sign is *erythema migrans,* an erythematous ringlike rash with central clearing that slowly expands (Fig. 31.6). The pathognomonic cutaneous lesion may grow to 5 cm in diameter at the site of the tick bite. The central clearing sometimes has an erythematous center, forming a bull's eye appearance, and can be bluish instead of flesh colored. The rash can resemble ringworm (*tinea corporis*) infection and other dermatologic conditions.[54] Fatigue and lymphadenopathy are common findings. Up to 10% of patients do not develop a recognizable rash. The second stage occurs weeks to months after the bite, with neurologic abnormalities that can include meningitis and neuropathy. The third stage can manifest months to years later with chronic arthritis, subtle mental disorders, or even carditis and heart block.[55]

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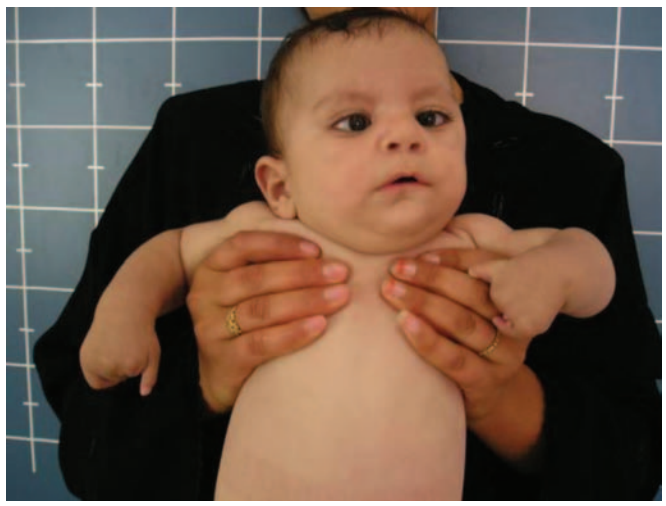

f0035 **Figure 31.6** Mobius syndrome demonstrating mild bilateral esotropia and blank expression.

p0470 Cranial neuropathies, especially of the seventh nerve, are one of the most common neurologic manifestations of Lyme disease. The facial paresis can be bilateral in up to one-third of cases and is often part of a cranial polyneuropathy. Cerebrospinal fluid (CSF) may be normal in patients with facial paralysis. Some patients will develop facial weakness before the rash or in the absence of skin lesions.[56]

p0475 Diagnostic testing is difficult as serologies may be negative early in the disease course. Enzyme-linked immunosorbent assay (ELISA) for IgM and IgG is recommended early, and positive or equivocal results are confirmed with Western blot testing. If a symptomatic patient is ELISA negative, ELISA should be repeated in 30 days. Even this two-stage testing approach is only 40% to 50% sensitive early in the disease course. Consultation with a rheumatologist and consultation of the CDC website are recommended.[55]

p0480 Treatment for Lyme disease depends on disease severity. For erythema migrans, Lyme carditis and facial paresis without meningitis, treatment is with doxycycline, amoxicillin or cefuroxime for 14 days. If arthritis is present, treatment with oral antibiotics should be continued for 28 days. Treatment for meningitis or radiculopathy requires ceftriaxone 2 mg intravenously daily for 14 days. Pregnant women and young children should not be given doxycycline.

s0165

MELKERSSON-ROSENTHAL SYNDROME

p0485 Melkersson-Rosenthal syndrome (MRS) is a rare neuromucocutaneous granulomatous disorder of unknown etiology, characterized by the clinical triad of facial palsy, lingua plicata (fissured tongue) and orofacial edema. Onset usually occurs in childhood or adolescence. Orofacial edema is the pathognomonic feature, with facial palsy and lingua plicata each being present in roughly half of the patients. Thus, the complete triad is only present in 25% of patients. The orofacial edema typically involves the lips and the buccal area, but the tongue and the gingiva can be affected as well. The facial edema can extend to the supraorbital region. Neoplastic, infectious and autoimmune causes of the edema must be investigated. The lips become chapped, fissured

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and discolored and over time may become permanently deformed.

p0490 The onset of the facial nerve paralysis is identical to that of Bell palsy. Many patients will experience recurrent and/or bilateral attacks of facial palsy. Diagnosis is based on the clinical history and mucosal or skin biopsy showing noncaseating epithelioid cell granulomas.[57] No randomized trials have evaluated the efficacy of steroids. The recovery of facial movement after multiple attacks can gradually worsen, although there is some evidence that complete facial nerve decompression can eliminate future episodes of facial paresis and improve facial nerve outcomes.[14]

p0495 In one recent meta-analysis, Crohn disease was diagnosed in 40% of adolescents with orofacial granulomatosis. The two diseases have essentially identical histopathologic changes. The orofacial edema preceded the diagnosis of Crohn disease in most patients.[58] Patients with recurrent orofacial edema should be questioned about perianal and gastrointestinal symptoms and referred to the appropriate specialist for further evaluation.

s0170 BILATERAL PARALYSIS

p0500 Bilateral facial palsy is even more devastating than the unilateral form. Both eyes may require taping or patching to prevent corneal exposure, rendering the patient functionally blind. Oral competence is significantly affected, and facial nonverbal communication is entirely lost.

p0505 Metachronous bilateral facial paralysis resulting from Bell palsy is possible but rare and should raise the suspicion of systemic disease. Most commonly associated with bilateral facial paralysis are Guillain-Barré, multiple idiopathic cranial neuropathy, brainstem encephalitis, benign intracranial hypertension, syphilis, leukemia, sarcoidosis, Lyme disease, parapontine neoplasm and bacterial meningitis. Workup may entail lumbar puncture, MRI of the brain, VDRL (Venereal Disease Reference Laboratory) test for syphilis, neurologic referral and other laboratory testing based on the history and physical examination.

p0510 Guillain-Barré is a progressive ascending motor paralysis after viral or *Campylobacter* infection, which usually affects the lower limbs but can affect the upper body and face as well. Diagnosis is based on the clinical picture and the presence of 'albuminocytologic dissociation' – an elevated protein and normal or low white blood cell count in CSF. The etiology is thought to be the stimulation of antiganglioside antibodies that attack the nervous system by the preceding infection. First-line treatment is intravenous immunoglobulin. Some patients require mechanical ventilation during the acute phase of the disease until their nervous system recovers enough to provide adequate ventilation.[59]

s0175 TRAUMATIC FACIAL PARALYSIS

p0515 Temporal bone fractures, gunshot wounds, and penetrating soft tissue injuries represent 5% or less of facial paralysis cases. Patients who have suffered trauma should be medically stabilized before considering any type of operative intervention. Facial lacerations lateral to the lateral canthus extending into the parotid tissue with any deficit in facial movement should be explored as soon as possible to identify cut nerve endings for primary repair or nerve grafting. Injuries medial to the lateral canthus typically involve facial nerve fibers so small that they are difficult to identify and cause little functional deficit. Temporal bone fractures can affect facial nerve function in several ways, including direct penetration of the nerve, traction on the nerve contusion and edema. If a patient with a nonpenetrating injury has complete facial paralysis immediately after the injury, electrophysiologic EP testing should be performed. Fisch has stated that if the ENOG shows greater than 90% degeneration by 6 days after injury, then facial nerve exploration, decompression and possible repair should be performed.[26] High-resolution temporal bone CT can help identify the area of likely injury and serves as a road map for decompression surgery. Patients who initially have some facial nerve function but who progress to complete paralysis are typically affected by nerve edema and will recover well with conservative measures only.

IATROGENIC INJURY s0180

p0520 Injury to the facial nerve is possible during lateral skull base, otologic, temporomandibular joint, maxillofacial, head and neck oncologic, and facial cosmetic surgery, including facelift. Almost all parotid surgeries require identification and dissection of the facial nerve, and many patients undergoing parotidectomy will have temporary weakness of the marginal mandibular nerve for up to 3 months after surgery as a result of praxis.

p0525 Accidental facial nerve transection that is recognized at the time of surgery should be repaired at that time with 9-0 interrupted permanent sutures through the epineurium. This is best done using a two-headed operating microscope with an assistant. Enough sutures should be placed so that no fascicles are visible bulging through the epineurial repair.

p0530 If a patient awakens from surgery with an unexpected facial weakness and it is possible the facial nerve was accidentally injured, 6 hours should be given for any local anesthetic to wear off. If the facial weakness persists beyond 6 hours and is in the zygomatic or buccal distribution, it is best to take the patient back to surgery as soon as possible to look for and repair the injured branch of the nerve. After 72 hours, Wallerian degeneration will have occurred and a nerve stimulator will no longer be able to stimulate the branch distal to the injury, which can make it harder to identify and repair the nerve. Unrecognized injuries to the frontal, marginal and cervical branches are less devastating, and a decision can be made with the patient whether or not to try to repair the nerve.

SARCOIDOSIS s0185

p0535 Sarcoidosis is another chronic, noncaseating granulomatous disease that causes thoracic adenopathy, arthralgias, hepatic dysfunction and elevated serum calcium levels. Heerfordt syndrome (uveoparotid fever) is a rare variant of sarcoidosis that causes parotitis, uveitis, mild fever and cranial nerve paralysis, most commonly of the facial nerve (50%). The facial paresis can follow the parotitis by days to weeks, and is thought to result from direct inflammation of the nerve (in contrast to compression from the parotitis).[60]

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Sarcoidosis can be diagnosed clinically, but definitive diagnosis requires tissue biopsy. The angiotensin converting enzyme level will be elevated in about two-thirds of cases. The mainstay of treatment for facial paralysis associated with sarcoidosis is a high-dose steroid taper (1 mg/kg/day for 10 days with a taper). The facial palsy has an excellent prognosis if treated with steroids shortly after onset.

p0540 Mycophenolate mofetil, cyclosporine or infliximab may be necessary in patients whose symptoms are refractory to steroids. Involvement of a rheumatologist is recommended to manage these medications and the other manifestations of sarcoidosis.

s0190 OTITIS MEDIA

p0545 Acute suppurative otitis media can cause facial paralysis in children and adults. These patients typically have a dehiscent facial nerve in the middle ear space. Treatment requires wide myringotomy and intravenous antibiotics. Some authors recommend steroids to reduce inflammation of the nerve. Facial palsy is more commonly associated with chronic otitis media and cholesteatoma. The cause of the paralysis in cholesteatoma can be inflammation or pressure, but inflammation likely plays a greater role. With proper management of the chronic otitis media, the facial palsy typically resolves without sequelae.

s0195 BAROTRAUMA

p0550 There are several reports of facial palsy associated with changes in barometric pressure. These patients usually notice the onset of palsy during scuba diving, when flying or driving at altitude, or after nose blowing. The palsy completely resolves when the ambient pressures return to normal. The pathophysiology appears to be elevated middle ear pressures transmitted directly to the facial nerve through dehiscence in the Fallopian canal. Pressure-equalization tubes should prevent further episodes.

s0200 IDIOPATHIC INTRACRANIAL HYPERTENSION

p0555 Idiopathic intracranial hypertension (IIH; also pseudotumor cerebri, benign intracranial hypertension) is a disorder of increased intracranial pressure in the absence of a mass lesion. It is most common in obese women 20 to 40 years of age. Typical presentation includes morning headaches, but symptoms can also include pulsatile tinnitus, nausea, vomiting and loss of vision. The most common associated cranial neuropathies affect the abducens (diplopia) and facial nerves. IIH is one cause of bilateral facial paralysis. Diagnosis requires MRI to eliminate structural lesions, and spinal tap to determine intracranial pressure. Acetazolamide is used to decrease CSF production, although serial spinal tap, ventriculoperitoneal shunting or optic nerve sheath fenestration may be required. When the pressure is normalized, the facial paresis resolves.[61]

s0205 FACIAL PARALYSIS IN CHILDREN

p0560 The incidence of congenital facial paralysis is estimated at 2 per 1000 live births.[62] Some authors divide facial paralysis in infants into congenital (caused by diseases acquired in utero or related to the birth process) and developmental causes (genetic basis). The differential diagnosis is summarized in Table 31.8.

t0045 **Table 31.8** Congenital Versus Developmental Facial Palsy

Congenital	Developmental
Birth trauma	Congenital unilateral lower lip palsy
Intracranial hemorrhage	Mobius syndrome (bilateral CN VI and VII)
Syphilis	Craniofacial macrosomia
Poliomyelitis	Oculoauriculovertebral dysplasia
Infectious mononucleosis (Epstein-Barr virus)	Poland syndrome
Varicella	Teratogens including thalidomide
Bell palsy	CHARGE syndrome (*CHD7* abnormalities)
Acute otitis media	Hereditary developmental facial paralysis
Rubella	

CHARGE, Coloboma, Heart defect, Atresia choanae, Retarded growth and development, Genital hypoplasia, Ear anomalies/deafness; CN, cranial nerve.

p0565 Almost 80% of cases of facial paralysis in infants are related to birth trauma, half as a result of forceps delivery and half to vaginal or Cesarian delivery.[63] Infants with facial nerve injury caused by birth trauma will often have ipsilateral facial ecchymoses and/or hemotympanum. They rarely have any other cranial nerve abnormalities. Multiple cranial nerve abnormalities or abnormalities on the newborn brainstem audiometry hearing test suggest a developmental etiology.

p0570 Congenital unilateral lower lip palsy is the mildest form of facial dysfunction. It is caused by a brainstem lesion and is associated with isolated deficient lip depressor function and otherwise normal facial movement.[64]

p0575 Mobius syndrome is a defect of the rhomboencephalon and its motor nuclei. The syndrome classically refers to the phenotype of bilateral abducens and facial nerve palsies, but this term is used to cover a broad range of abnormalities, including isolated unilateral facial paralysis up to and including the glossopharyngeal, vagus, hypoglossal and extraocular motor nerves.[65] Mobius syndrome is often recognized in young children on observation of difficulty feeding, absence of facial expression when crying, and the need to turn the entire head to look laterally (Fig. 31.7). Eye care is important. These patients often have limb and chest wall abnormalities as well.

p0580 Children and young adults can also develop Bell palsy, although likely at a lower rate compared with adults. Young patients with Bell palsy typically have excellent recovery, and some studies have suggested that recovery is the same with or without steroids. Because short-course corticosteroids present low risk and may improve recovery, treatment is recommended (1 mg/kg of prednisone per day for 5 days followed by a 5-day taper).

Management s0210

p0585 Treatment planning depends on etiology, severity, distribution, timing, duration of paralysis and the patient's age and goals. A logical method of management can be found in temporal and geographic approaches.

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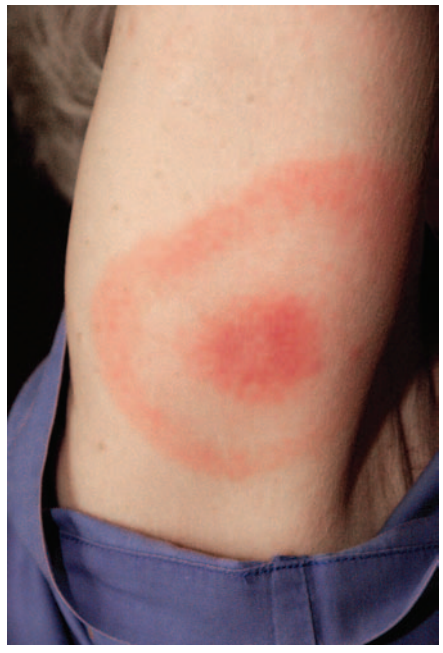


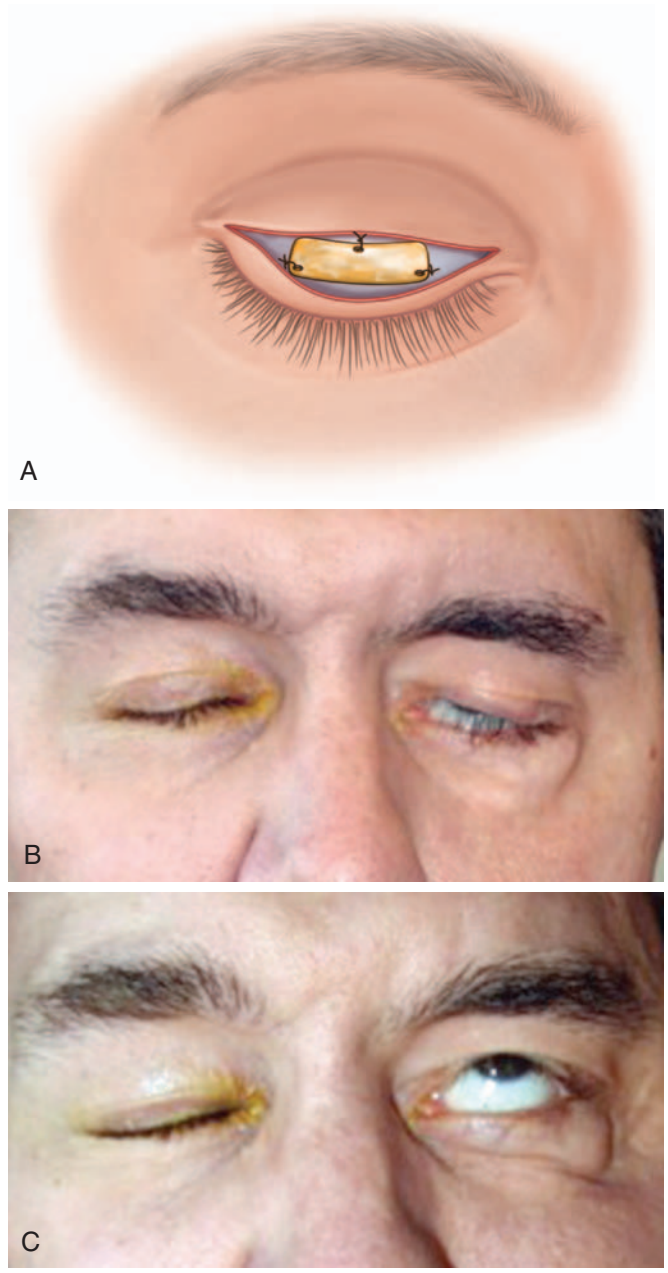
f0040 **Figure 31.7** The classic bulls-eye rash of erythema migrans. The central erythema is not always present.

p0590 Acute management related to specific etiologies (eg, Ramsay Hunt syndrome) has been reviewed earlier in this chapter.

s0215 **Short-Term Support.** All patients with facial paralysis, p0595 whether temporary or permanent, require short-term support. As in clinical evaluation, short-term management can be approached by considering four anatomic zones.

s0220 FOREHEAD. In the acute and subacute settings, forehead p0600 and eyebrow ptosis can help protect the ocular surface. The loss of frontalis function eliminates a secondary source of eyelid elevation, and the gravitational effect on the supraorbital soft tissues can help keep the eyelid closed. Therefore, brow ptosis should not be corrected permanently in the immediate aftermath of facial nerve dysfunction. Furthermore, brow reconstruction should be planned in the context of eyelid and ocular surface evaluation to preserve all possibilities for eyelid management. Reversible brow suspension using tape or prolene sutures can be safely offered in the subacute setting.

s0225 EYELIDS AND OCULAR SURFACE. The periocular region can p0605 be managed systematically, according to four regional subzones (brow, upper eyelid, ocular surface, lower eyelid). In this chapter, brow ptosis is addressed separately. The paralytic upper eyelid, notable for paralytic retraction, can be managed in the short term by applying a bead of ointment (Lacrilube or Genteal Gel) and gently squeezing the lids closed or by taping the lid closed before bed. Provided the patient has adequate contralateral vision, the eyelid can be taped closed with a narrow strip of tape or broadly covered with Tegaderm. Weight-loading with gold or platinum implants is a more invasive but reversible intervention that should be considered in high-risk patients even if the paralysis is expected to resolve in several months (Fig. 31.8). High-risk patients include those with moderate or severe dry eye syndrome, and those who are not reliable to care for the eye. Eyelid weight-loading increases the risk of nocturnal lagophthalmos and may require eyelid taping or ointment despite the implant. Massry has used a technique in which preaponeurotic fat is draped over the implant to provide additional camouflage, although this is a more invasive procedure.[66] Goldberg et al. have described hyaluronic acid filler as a temporary upper lid weight that can be augmented over time or dissolved with hyaluronidase, if necessary.[67]



f0045 **Figure 31.8** Eyelid weight-loading. **A,** Artist's representation of a single-piece weight, either gold or platinum, implanted in the pretarsal plane. Some authors describe pre- or postaponeurotic implantation. **B,** Left upper eyelid weight produces protective but incomplete closure in this patient in the upright position. **C,** Counterproductive retraction and corneal exposure in the recumbent position.

p0610 Short-term ocular surface solutions include viscous artificial tears (Refresh, Systane), which can be applied several times per day, and ointments can be applied at night.

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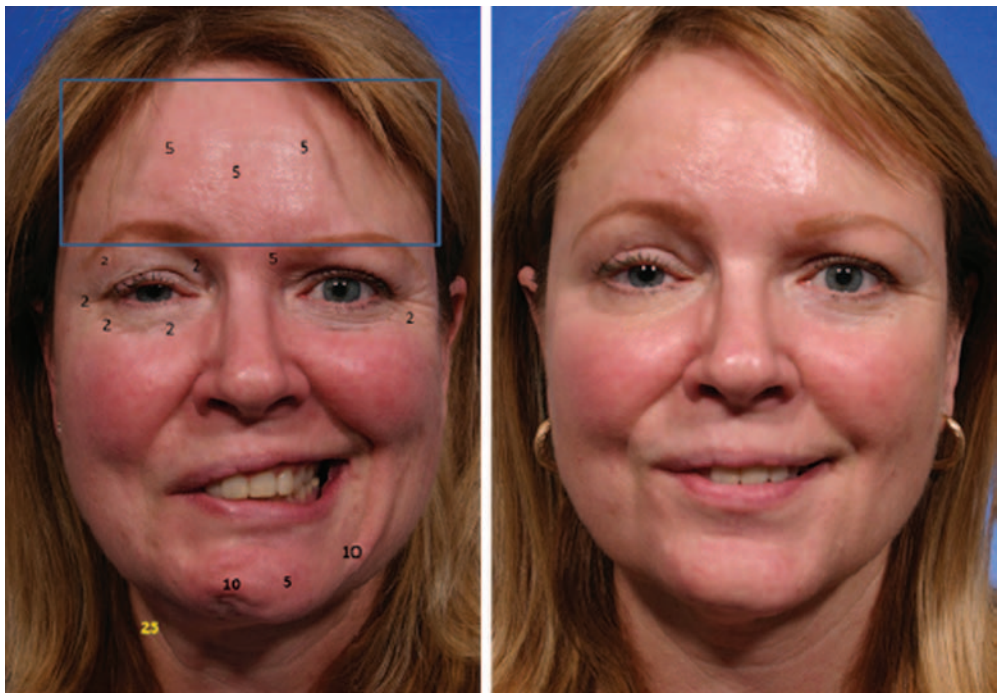

f0050

Figure 31.9 **A,** This woman developed significant periocular asymmetry following Bell palsy. Botox to the lateral canthal region did not elevate the brow adequately. **B,** Three months following left unilateral endoscopic brow lift and left upper blepharoplasty with 3 mm resection of orbicularis. With permission from ?

10

Silicone or collagen punctal plugs can be used to prolong survival of the natural tear film as well as tear supplements, but punctal destruction should be avoided in this uncertain situation. Patients often experience excess tearing even as the ocular surface dries because of poor tear movement that results from eyelid paralysis. Contact lenses may be useful in some cases but can also increase the risk of corneal ulceration. Patients with bandage contact lenses are often given a single drop per day of fluoroquinolone solution (ofloxacin, moxifloxacin) prophylactically. Moisture chambers can be applied directly to the skin or can be fitted to special eyeglasses that seal the area around the orbital rim. Similarly, Tegaderm can be placed over the eye after dressing the cornea with ointment, and in some cases, Saran wrap can provide equal protection without burdening the skin with adhesive. The ocular surface should be examined at monthly intervals during the early stages of paralysis.

5

p0615 Tarsorrhaphy can be performed using temporary, reversible or irreversible techniques. Suture tarsorrhaphy may be the best alternative for ocular surface protection in the acute setting, particularly if patients are hospitalized. Suture tarsorrhaphy is easily removed and provides ongoing access for ocular examination or topical medications. 4-0 or 5-0 nylon or prolene can be tied in a bow or in a permanent knot. Reversible surgical tarsorrhaphy is reasonable if the paralysis is expected to last several weeks or months. The anterior lamella is preserved, whereas the tarsal edges are fused, allowing surgical repair of the lid margins at a later date. Tarsorrhaphies diminish visual field and are often not cosmetically ideal, two features that limit their application. Permanent lateral and medial tarsorrhaphies also paradoxically inhibit eyelid movement and may worsen central corneal exposure.

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p0620 Lower eyelid weakness is sometimes less obvious early in the course of facial paralysis. Ectropion and retraction both contribute to epiphora and also contribute to ocular surface injury by exposing the inferior cornea and conjunctiva. Both ectropion and retraction can be managed temporarily by using thin strips of tape placed obliquely from the lateral lower lid to the temple region above the lateral canthal tendon. Irreversible reconstructive procedures should be delayed until the expected degree of functional recovery has been achieved. Temporary elevation of the lower lid is possible by using injectable hyaluronic acid fillers, whereas autologous fat or dermis are difficult to reverse and should be considered permanent.[68,69]

s0230 MIDFACE AND LOWER FACE. In the immediate aftermath of facial nerve injury, the midface and lower face areas do not p0625 require intervention. In cases where facial nerve recovery is possible but uncertain (intact but injured facial nerve during vestibular schwannoma resection), it is reasonable to consider reversible techniques such as a facial static sling with dermal allograft as well as suture suspension of the external nasal valve (Fig. 31.9). The contralateral lip depressor can be weakened with botox to improve facial symmetry; however, bilateral lower lip depressor weakness commonly causes issues with oral competence.

Long-Term Reconstruction

s0235

s0240 FOREHEAD. When no further return of function is antici- p0630 pated, permanent surgical solutions can be undertaken. Forehead lifting is enticing because it provides immediate and obvious improvement in appearance, but it should be planned in concert with eyelid reconstruction to avoid iatrogenic lagophthalmos. When indicated, direct, midforehead, pretrichial and coronal techniques are all useful.

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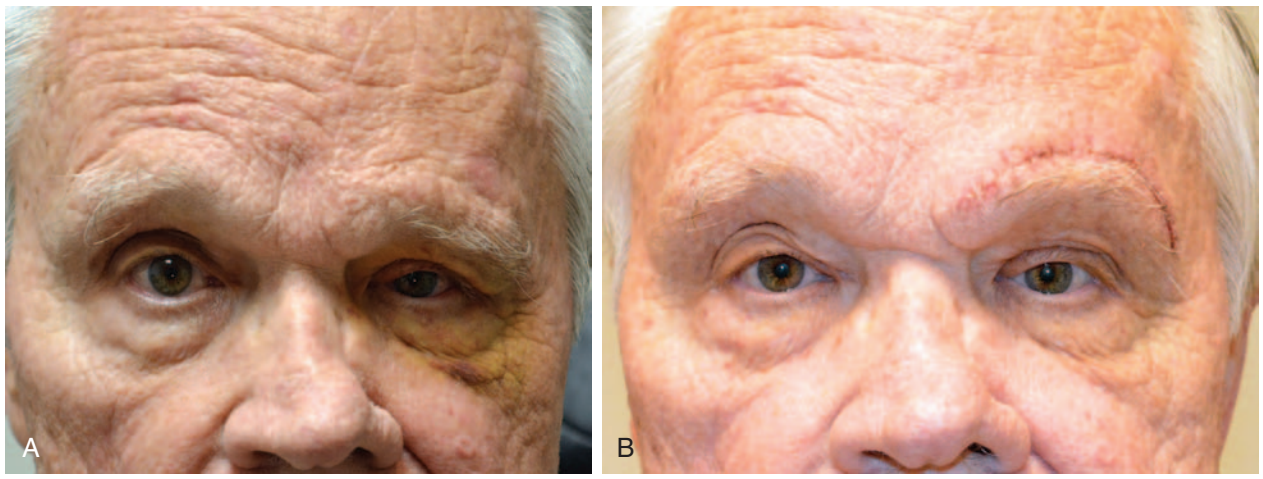

Figure 31.10 Paralytic brow ptosis. **A,** Before surgical elevation. **B,** After surgical elevation.

t0050

Table 31.9 Indications and Contraindications for Different Forehead and Brow-Lifting Techniques

Procedure	Indications and Advantages	Contraindications and Disadvantages
Coronal forehead lift	Treats all aspects of agng forehead and brow	Limited use in men Elevates hairline Vertically lengthens upper third of face Elongated scar Possible prolonged hypesthesia of scalp Less fine-tuning of brow position
Pretrichial forehead lift	High hairline No vertical forehead lengthening Preserves hairline Treats all aspects of aging forehead and brow	Possible visible (exposed) scar Possible prolonged hypesthesia of scalp
Mid-forehead lift	Prominent horizontal forehead creases Preserves hairline Improved fine-tuning of brow position Corrects brow asymmetry	Possible visible (exposed) scar Avoid in oily, thick skin
Direct brow lift	Accurate brow elevation Preserves forehead/scalp sensation Patients with abundant or thick brow hair preferred Immediate scar camouflage (with hair) Corrects brow asymmetry	Possible visible (exposed) scar Treats brows only
Temporal lift	Ideal and immediate scar camouflage (with hair) Improves lateral hooding	Not useful for mid-forehead glabellar creases No effect on medial aspect of brow
Browpexy	Performed through upper blepharoplasty or small superior edge of eyebrow incision Indicated for mild brow ptosis	Possible prolonged eyelid edema Possible brow asymmetry Possible unsatisfactory results
Endoscopic brow lift	Less invasive with small incisions Excellent scar camouflage High hairline No vertical forehead lengthening Preserves hairline Treats most aspects of aging forehead and brow	Less fine-tuning of brow position Possible contour irregularities in scalp as a result of paramedian bony fixation

The endoscopic approach does not typically provide the degree of elevation sought in paralysis cases but is useful in patients who want no visible scarring (Fig. 31.10). The relative advantages and disadvantages of these techniques are summarized in Table 31.9.

s0245 p0635 **EYELIDS.** Permanent upper eyelid paralysis can be managed in several ways. Weight-loading is most common, as described above. The platinum chain is better concealed because it contours to the tarsus but is considerably more expensive than the other weights and a bit more challenging to implant. Platinum is more dense than the 24-karat gold used in upper lid weights, allowing platinum weights to be thinner and hence more cosmetic. There is some evidence that platinum weights have a lower extrusion rate as well.[70] Implant size can be determined in the office by using external 'stick on' weights with the patient in the upright position. Patients undergoing eyelid weight loading should be forewarned about the increased risk of nocturnal lagophthalmos.

6

An alternative to eyelid weight loading is eyelid spring loading. The Levine palpebral spring has been used successfully in over 1000 eyes since the technique was refined in p0640

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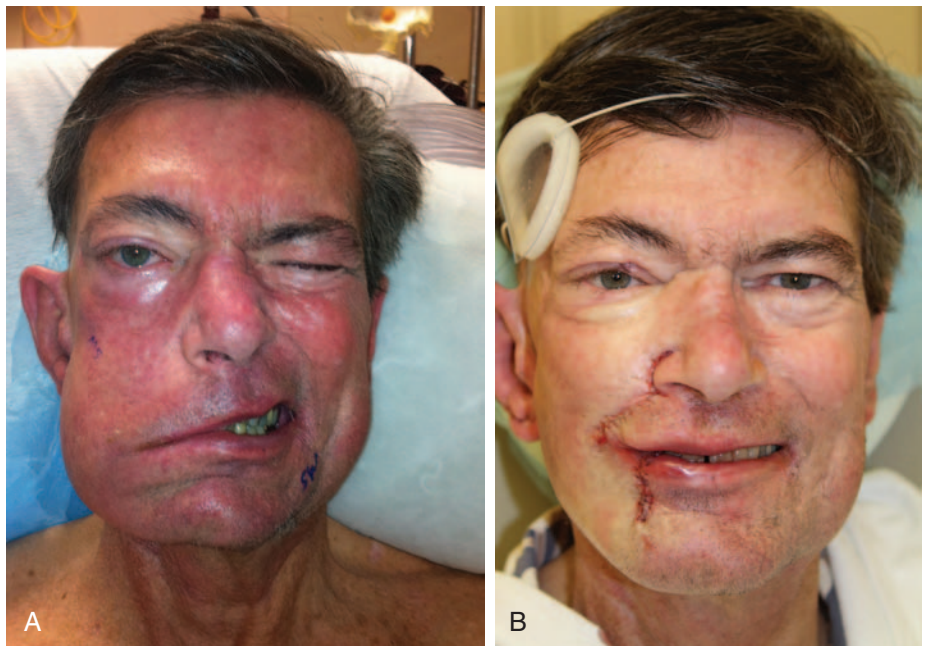

f0060

Figure 31.11 Middle-aged gentleman before and immediately after right facial static sling, lower lip wedge resection and nasal valve reconstruction.

1990. The surgery involves a custom-fitted, nickel wire implant that is created by the surgeon in the office or operating room. The single coil helical spring works much like a safety pin. One arm is secured to tarsus, and the other is fitted into the superior orbit, thus forcing the eyelid downward. In contrast to eyelid weights that require only 15 to 20 minutes of operating time, a Levine palpebral spring requires a 2½- to 3-hour operation under monitored sedation. Revisions are common.

p0645 Although silicone cerclage is not presently in use, synthetic materials are under investigation as replacements for the paralyzed orbicularis muscle.

p0650 Patients with a longstanding history of partially recovered facial paralysis may develop otherwise unexplained unilateral ptosis. They typically have moderate to normal eyelid excursion. They typically respond well to levator shortening procedures or conjunctival Müllerectomy.

p0655 Lower eyelid reconstruction requires elevation and inversion. The mainstay of lower eyelid shortening is the tarsal strip procedure. Medial shortening is exceedingly more complex mostly because of the presence of the lacrimal drainage system. An intact inferior canaliculus will reliably be kinked and damaged by simple tightening of the medial canthal tendon inferior ramus, a reasonable sacrifice in some cases. Such patients may later require conjunctivorhinostomy with a Jones tube. Simple medial tarsorrhaphy has been used with some success and minimal cosmetic deformity.

p0660 Although horizontal laxity can be corrected with shortening surgery, paralytic retraction may require recession of the lower lid retractors with an interspacer graft. Suitable materials include cartilage, donor sclera, dermis, porcine intestine and numerous other materials.

p0665 Some patients with longstanding facial paralysis develop anterior lamellar deficiency. In these patients, anterior lamellar augmentation with skin grafting may be required in concert with the other procedures listed above (Fig. 31.11).

Q

p0670 Midface lifts have historically been popular in treating lower eyelid retraction, although the durability of midface elevation in this setting is unclear. Volumetric replacement with injectable fillers or autologous fat grafting shows promise in supplanting surgical midface elevation.[69,70]

s0250 p0675 **MIDFACE AND LOWER FACE.** One of the most important long-term objectives is restoration of facial expression and smiling. For patients with complete unilateral flaccid paralysis, there are five types of operations to address mid- and lower facial paralysis.[11]

1. o0190 *Static procedures:* Static procedures attempt neither to repair the damaged nerve, nor to achieve facial movement. The aptly named 'static sling' is a common surgery in which the paralyzed cheek is suspended in a more symmetric, elevated position using dermal allograft or tensor fascia lata. Other static techniques include lysis of the lip depressors on the intact side to improve facial symmetry (Fig. 31.12). Eyebrow elevation and lower eyelid tightening are also static techniques.
2. o0195 *Nerve grafts:* When a portion of the facial nerve is lost to trauma or surgery and both cut ends of the nerve can be identified, the 'gap' can be filled with a piece from a different nerve, as is common after excision of parotid gland malignancies that invade the facial nerve. When the main trunk of the facial nerve is grafted, the best recovery possible is House-Brackmann III/VI (considerable synkinesis).
3. o0200 *Nerve substitutions:* If the facial nerve is damaged or removed in its intracranial portion, a nerve graft is generally not possible. For example, vestibular schwannomas and other cerebellopontine angle tumors are a common cause of facial paralysis because these masses grow adjacent to the facial nerve. In this location, the

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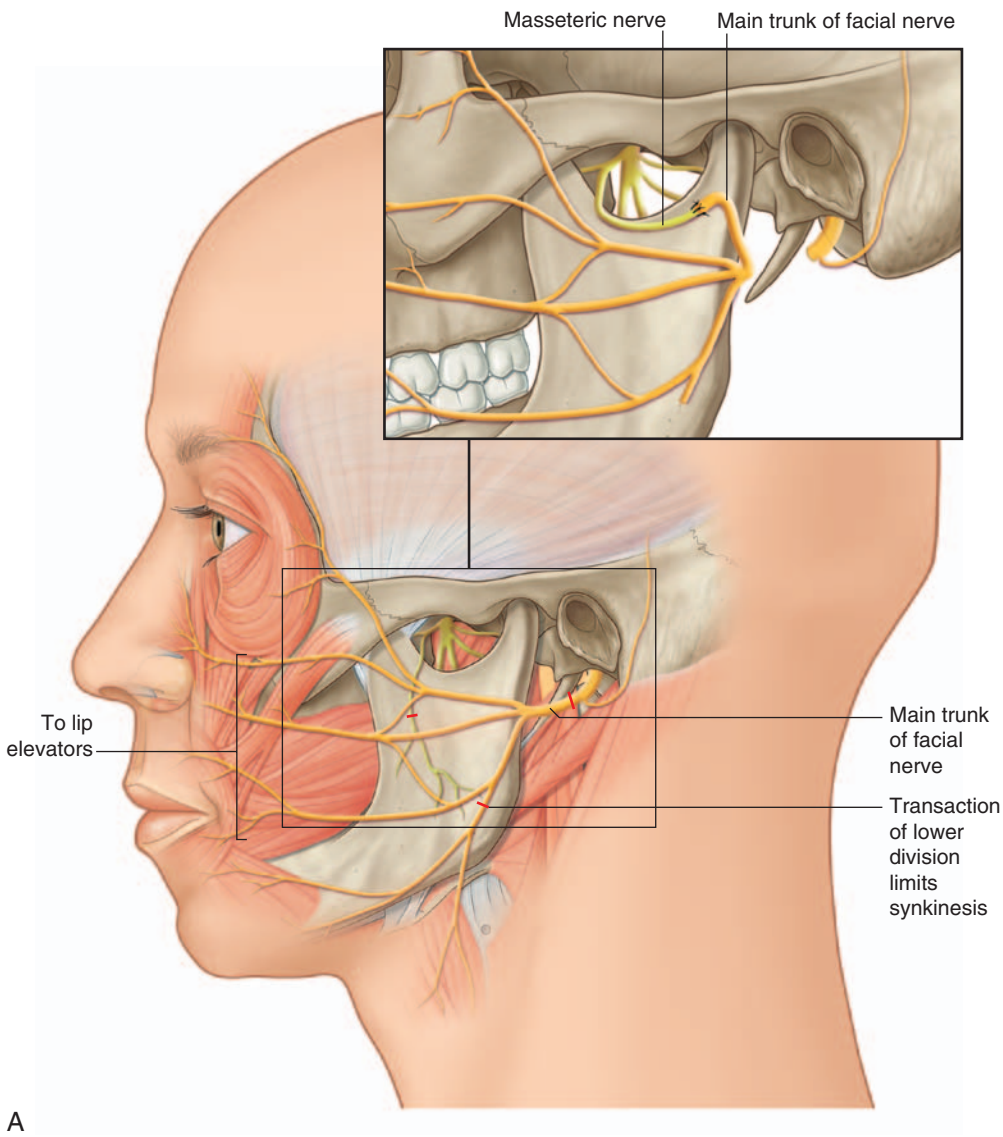

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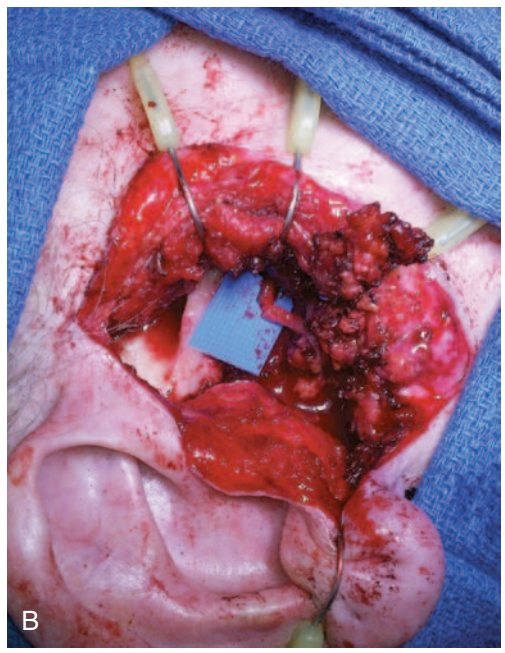

Figure 31.12 **A,** Diagram showing how the masseteric nerve is cut in the infratemporal fossa and sewn to the distal cut edge of the facial nerve. **B,** Intraoperative view of the masseteric and facial nerves being sutured together. The zygomatic arch is visible just anterior to the pinna.

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f0070

Figure 31.13 **A,** A 60-year-old woman with complete flaccid facial paralysis at rest. **B,** After right upper lid gold weight, closed canthoplasty and retractor recession in combination with masseteric-facial nerve transfer. **C,** With attempted smile before surgery. **D,** With biting down to achieve a more symmetric smile after surgery.

nerve stump is frequently too attenuated to graft. Instead, a different motor nerve in the head and neck can be sutured to the distal end of the injured facial nerve. The axons within the donor motor nerve will regenerate and eventually innervate the facial muscles. However, muscle activation requires volitional effort by the patient. Classically, the hypoglossal nerve was the most common donor nerve, but recently the masseteric nerve has been popularized as the donor nerve of choice (Figs. 31.13 and 31.14).[71] Advantages of the masseteric nerve are that it can be used end to end with minimal donor deficit and with a low incidence of mass movement. To smile, these patients must try to bite down. Patients with nerve substitutions are never able to achieve a truly spontaneous emotional smile with laughter, which is only possible when movement is controlled by the facial motor nucleus.

The contralateral facial nerve has also been used as the donor nerve in a procedure called *cross-face nerve grafting*. Typically, a sural nerve graft measuring 10 to 12 cm is tunneled across the upper lip to connect the two nerves (Fig. 31.15). Nerves in younger patients seem to have a better potential for regeneration, making this technique most appropriate in children. When successful, this

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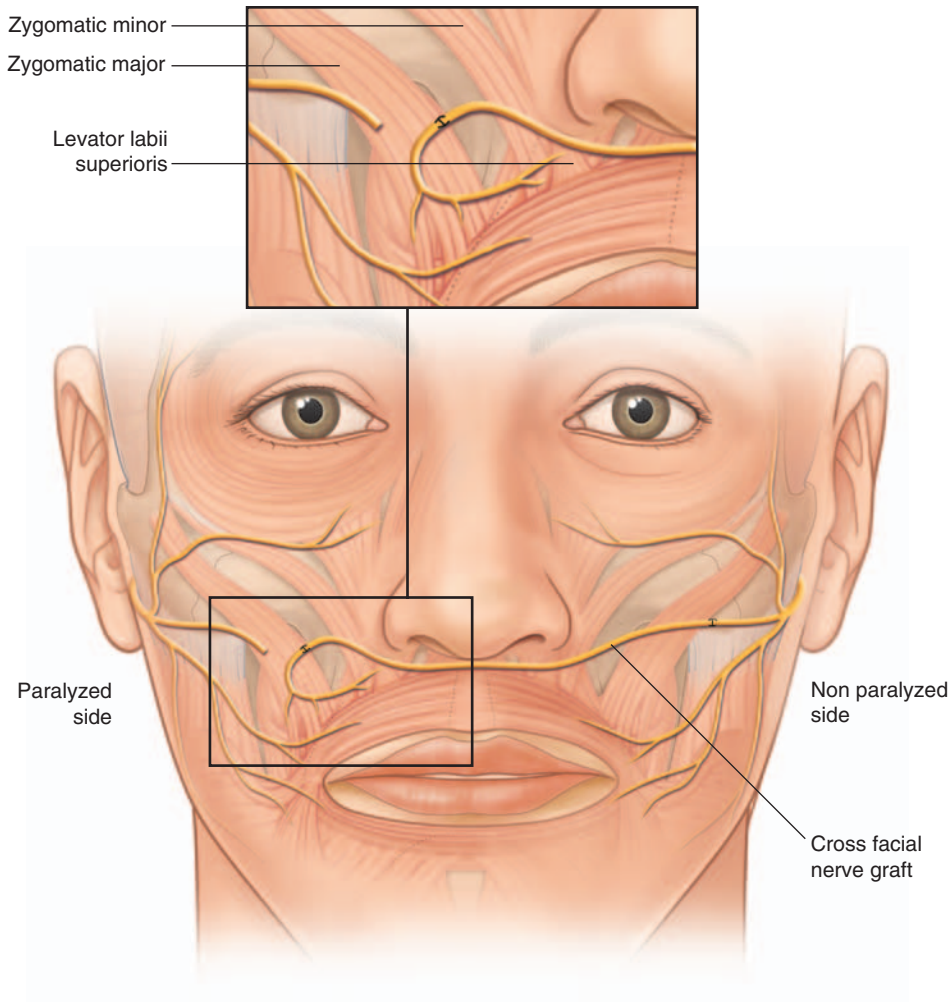

f0075

Figure 31.14 Diagram of cross-facial nerve grafting.

technique does provide a complete spontaneous emotional smile because the contralateral facial nucleus is driving the facial musculature on the affected side.

o0205 4. *Muscle substitutions (temporalis tendon transfer):* When the muscles that move the face are denervated for 2 years or longer, they atrophy and are no longer functional, even if provided with nerve input. In these instances, the surgeon must bring a new muscle into the face. The most common and modern example of this technique is called *temporalis tendon transfer*. In this surgery, the temporalis tendon is detached from the mandible and sewn to the modiolus and nasolabial fold. A tensor fascia lata spacer graft is often necessary to take tension off of the transfer (Fig. 31.16). When the patient wants to generate a smile, he or she bites down and the temporalis muscle shortens, generating an appropriate superolateral vector on the corner of the mouth. A similar technique can also be performed using a free muscle transfer, typically the gracilis muscle from the leg. The motor nerve to the transferred muscle is anastamosed to the masseteric nerve. Thus, when the patient bites down, the transplanted gracilis muscle is stimulated to create a smile.

o0210 5. *Cross-facial nerve graft and free muscle transfer:* The only way to restore a completely emotional and spontaneous smile is to connect facial musculature to the motor nucleus of the facial nerve. If a patient desires a spontaneous smile but the native facial muscles have atrophied, it is possible to perform a two-stage surgery called cross-facial nerve grafting with free muscle transfer (Fig. 31.17). This process takes 2 to 3 years and has been shown to have a 20% failure rate in most large studies. Like all cross-face nerve grafting techniques, outcomes are best in younger patients.

SYNKINESIS

s0255

p0710 Involuntary ipsilateral spasm of facial muscles occurs in a considerable subset of patients who have recovered some movement after an injury to the main trunk of the facial nerve. Most commonly, the orbicularis oculi is stimulated synchronously with the smile. Patients describe midfacial tightness, poor smile excursion and asymmetry. Botulinum toxin is generally effective (Fig. 31.18). Improved smile 14 excursion is the most difficult to achieve; some patients with a 'frozen smile' require transoral injection of the buccinators and risorius. Surgical myectomy (platysma) or neurectomy (marginal nerve) can produce more long-lasting results in the lower face. Many who develop contracture of the midfacial SMAS will benefit from physical therapy, Q

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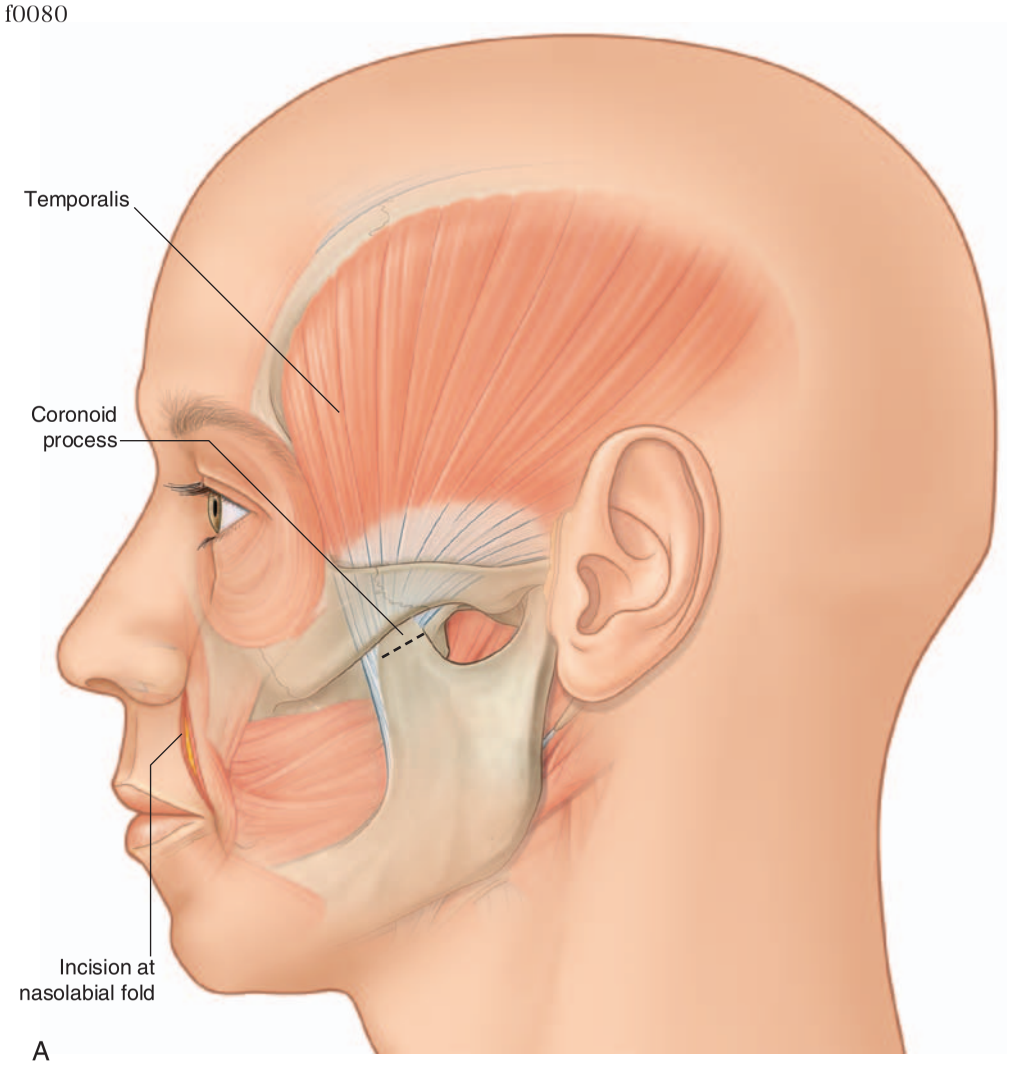

Figure 31.15 **A,** Through an incision in the nasolabial fold, the coronoid process is divided from the mandible with the temporalis tendon still attached. **B,** The cut coronoid or the tendon itself are sutured to the nasolabial fold with or without a fascial extension graft.

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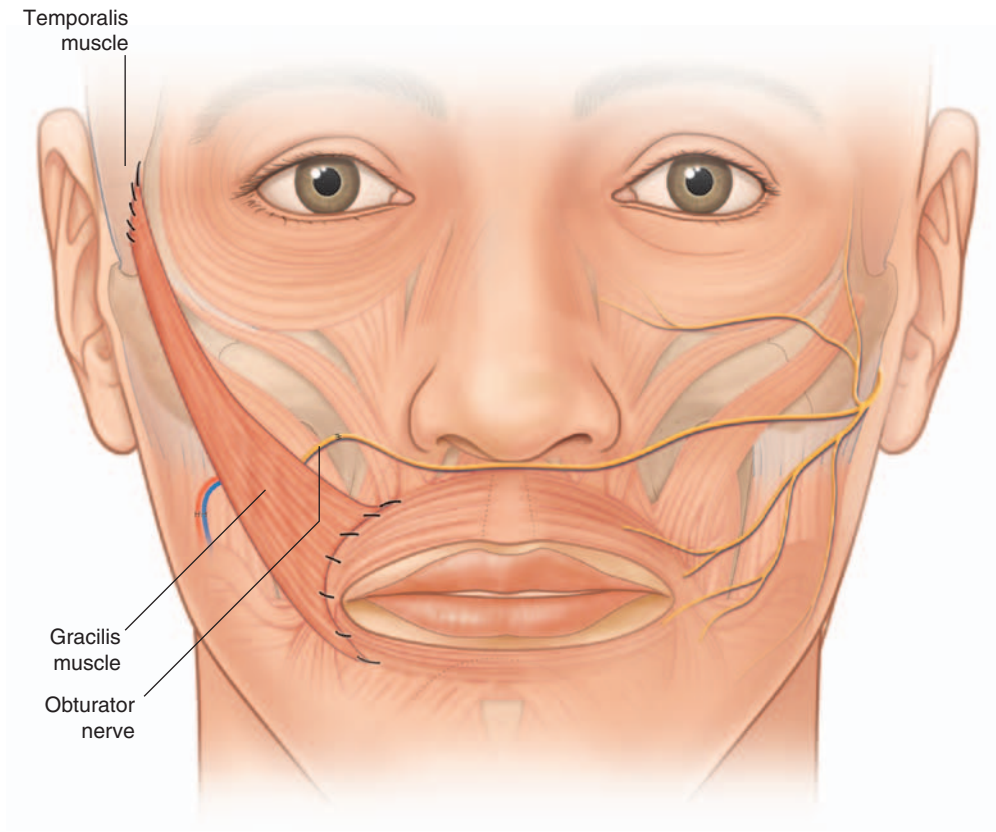

f0085

Figure 31.16 Two-stage free muscle transfer with cross-facial nerve graft. In the first surgery, a sural nerve graft is sutured to a cut buccal branch on the intact side and tunneled across the upper lip. After approximately 6 months, axons will have grown across the nerve graft. At this point, the second-stage free gracilis muscle transfer can take place. The gracilis muscle is transferred with its obturator nerve and its feeding artery and vein. The obturator nerve is sutured to the cross-facial nerve graft to provide stimulation for smile from the facial nucleus on the intact side. The gracilis vessels are sutured to the facial artery and facial vein to provide blood flow.

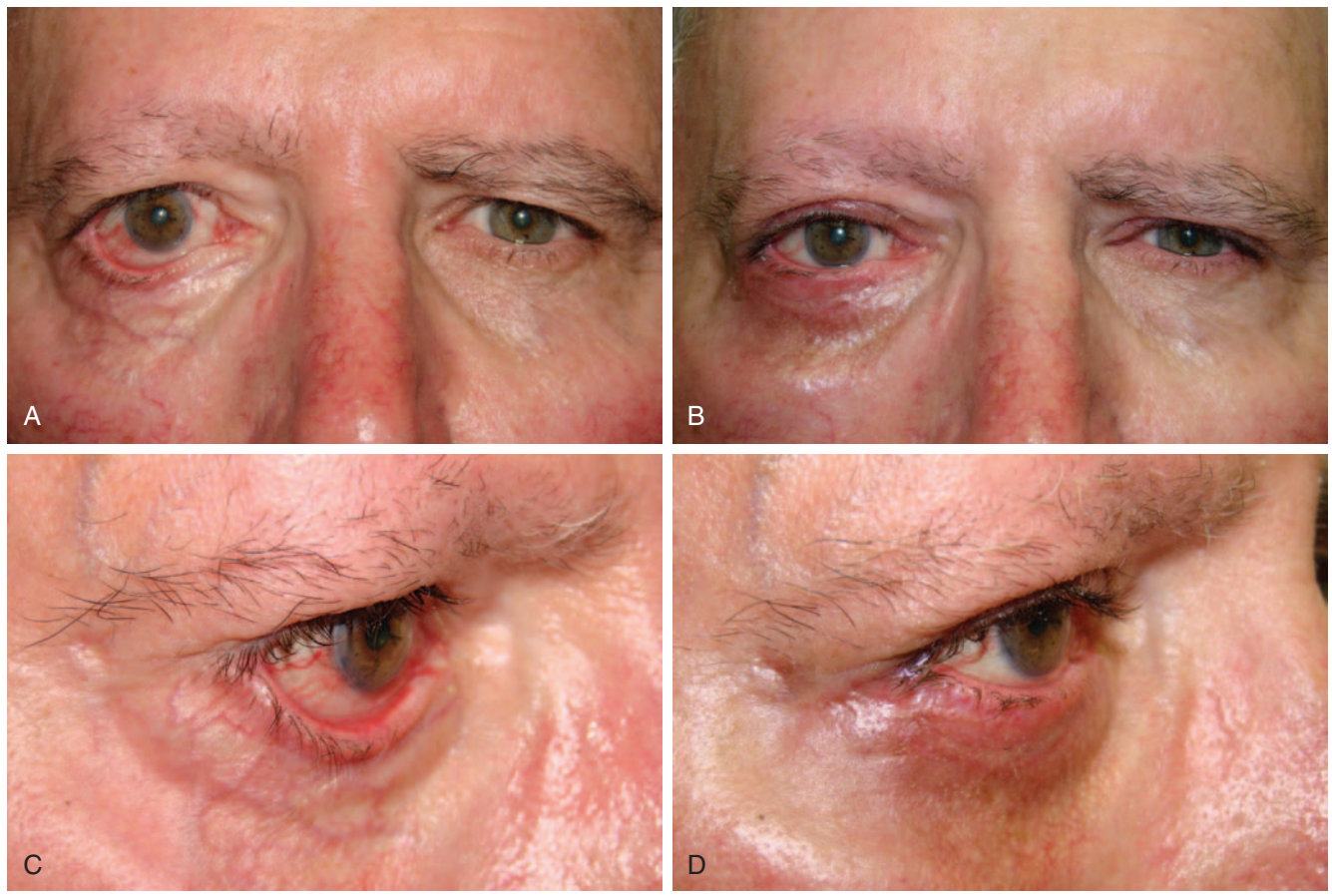

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Figure 31.17 Patient with longstanding right facial synkinesis and contracture with decreased facial tightness and improved resting facial symmetry after botulinum toxin injections.

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allowing the zygomaticus major to relax to a more normal location on the length-tension curve. Neuromuscular retraining can achieve improved facial control but requires buy-in from the patient and the daily practice.

p0715 Selective neurolysis has garnered renewed interest in recent years. With the patient under general anesthesia, the facial nerve branches to the synkinetic muscles are dissected out, and loops of nerve are passed through stab incisions in the skin. The preauricular incision is then closed, and the patient is awakened. The branches that have been passed through the skin are then cut one by one with the patient awake until the synkinesis resolves. The major risk of this technique is cutting too many branches resulting in paralysis of the targeted muscle(s).

p0720 Injectable fillers are another useful tool in patients with synkinesis. Frequently, these patients will have a deeper nasolabial fold on the affected side, with a more prominent malar eminence, as a result of midface contraction. Fillers can be injected to efface the deeper nasolabial fold on the affected side and to volumize the cheek on the unaffected side, thereby improving facial symmetry.

s0260 Future Directions

p0725 The recent progress made with cochlear and retinal implantation has stimulated interest in possible implantation of an electrode for restoring blink and facial movement. Most early work in this area was performed by ophthalmologists and otolaryngologists. In 1986, Rothstein and Berlinger definitively showed in a rabbit facial nerve injury model that contraction of the orbicularis oculi and zygomaticus muscles could be detected via EMG and used to stimulate their denervated counterparts on the opposite side of the face.[72] That same year, Otto et al. demonstrated the same in a canine model. He would later show that eye-blink could be rehabilitated continuously for 6 weeks in rabbits.[73]

p0730 Recent technological advances permit the realistic development of a permanent electrical prosthesis for facial paralysis. The envisioned implant would electrically pace several denervated facial muscles using EMG impulses from the paired muscles on the unaffected side. Because many patients with facial paralysis develop self-esteem issues and even true psychiatric difficulties preventing optimal work productivity, an effective facial implant would be worth the significant financial cost.

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