Chapter 122 – Facial Nerve Decompression

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Facial paralysis causes significant functional and aesthetic defects that often lead to great psychosocial distress. The potential causes of acute facial paralysis are numerous and listed in Table 122-1. This chapter focuses on management of the most common causes of facial paralysis that are amenable to surgical decompression, including Bell's palsy, facial paralysis associated with acute and chronic otitis media, and facial paralysis resulting from surgical trauma. Facial paralysis associated with temporal bone trauma is discussed in Chapter 128.

The goal of management in patients with facial paralysis of any etiology is to maximize functional recovery and minimize cosmetic deformity. In an effort to improve our understanding of neural injury and recovery, Sunderland introduced a histopathophysiologic classification system for nerve injuries that remains widely used today.[1] Injuries that induce only a conduction block within the nerve (neuropraxia, first–degree injury) do not disrupt axoplasmic continuity, and neural discharges can still be conducted if an electrically evoked stimulus is presented distal to the conduction block. The gross nerve structure remains intact. Second–degree injury involves axonal disruption (axonotmesis), without disruption of surrounding Schwann cell and neural connective tissue integrity. Full recovery usually occurs with the first two degrees of injury. Third- and fourth–degree injuries involve damage to the endoneurium and perineurium, respectively. The most severe forms of injury are characterized by complete neural tube disruption (neurotmesis, fifth–degree injury). In second- to fifth–degree injuries, the facial nerve undergoes distal wallerian degeneration. As a result, these nerves are not able to propagate electrically generated evoked potentials distal to the site of injury.

During recovery, if the axon regenerates through an intact neural tubule, complete return of motor function occurs without synkinesis. However, any violation of the neural support structures (endoneurium, perineurium, and epineurium) will result in misdirection of the regenerating neural fibers and cause synkinesis and incomplete motor recovery. When complete paralysis is due to either anatomic discontinuity or irreversible neural degeneration, the facial nerve requires repair or decompression for the most optimal functional and aesthetic results. Nonetheless, with completely severed nerves, some residual weakness and synkinesis are to be expected, even with the best surgical outcomes.

Table 122-1 -- DIFFERENTIAL DIAGNOSIS OF FACIAL PARALYSIS

Birth
Traumatic vaginal delivery
Myotonic dystrophy
Möbius' syndrome (facial diplegia associated with other cranial nerve deficits)
Traumatic
Cortical injuries
Basilar skull fractures
Brain stem injuries
Penetrating injury to the middle ear
Facial injuries
Barotrauma
Neurologic
Opercular syndrome (cortical lesion in the facial motor area)
Millard-Gubler syndrome (abducens palsy with contralateral hemiplegia because of a lesion in the base of the pons involving the corticospinal tract)
Infectious
Malignant otitis externa
Acute or chronic otitis media
Cholesteatoma
Meningitis Parotitis
Chickenpox

Herpes zoster oticus (Ramsay Hunt syndrome)
Encephalitis
Poliomyelitis (type I)
Mumps
Mononucleosis
Leprosy
Human immunodeficiency virus and acquired immunodeficiency syndrome
Influenza
Coxsackievirus
Malaria
Syphilis
Tuberculosis
Botulism
Mucormycosis
Lyme disease

Genetic and Metabolic
Diabetes mellitus
Hyperthyroidism
Pregnancy
Alcoholic neuropathy
Bulbopontine paralysis
Oculopharyngeal muscular dystrophy

Neoplastic
Facial nerve neuroma
Facial nerve hemangioma
Vestibular schwannoma
Glomus jugulare tumor
Meningioma von Recklinghausen's disease
Cholesterol granuloma
Carcinoma (invasive or metastatic from the breast, kidney, lung, stomach, larynx, prostate, or thyroid)

Toxic
Thalidomide (Miehlke's syndrome: involvement of cranial nerves VI and VII with atretic external ears)
Tetanus
Diphtheria
Carbon monoxide
Lead intoxication

Iatrogenic
Mandibular block anesthesia
Antitetanus serum
Vaccine treatment of rabies
Otologic, skull base, and parotid surgery
Embolization

Idiopathic
Bell's palsy
Melkersson-Rosenthal syndrome (recurrent facial palsy, furrowed tongue, fasciolabial edema)
Hereditary hypertrophic neuropathy (Charcot-Marie-Tooth disease, Dejerine-Sottas disease)
Autoimmune syndromes of temporal arteritis, periarteritis nodosa, and other vasculitides
Guillain-Barré syndrome (ascending paralysis)
Multiple sclerosis
Myasthenia gravis
Sarcoidosis (Heerfordt's syndrome, uveoparotid fever)
Wegener's granulomatosis
Eosinophilic granuloma
Amyloidosis
Hyperostoses (e.g., Paget's disease, osteopetrosis)

Kawasaki disease (infantile acute febrile mucocutaneous lymph node syndrome)
Vascular
Benign intracranial hypertension
Intratemporal aneurysm of the internal carotid artery

Data modified from May M: Differential diagnosis by history, physical findings and laboratory results. In May M (ed): The Facial Nerve. New York, Thieme-Stratton, 1986.

PATIENT SELECTION

Electrical Testing

Electrical testing constitutes the primary diagnostic modality for surgical decision making in patients with facial paralysis. Surgical management of a patient with facial paralysis is based on the premise that decompression or repair of the injured nerve will lead to better long–term functional outcomes than is the case with spontaneous recovery in a conservatively managed patient. Several electrical tests are available to evaluate the status of the facial nerve, estimate the severity of nerve injury, and prognosticate spontaneous recovery. Because most injuries to the facial nerve affect the intratemporal portion of the nerve, which is not usually readily accessible for evaluation, assessment of injury is based on measuring downfield potentials in the degenerating distal nerve. The extent and rate of progression of wallerian degeneration after injury are used as relative indicators of the severity of neural injury. Rapid wallerian degeneration is associated with neurotmesis, whereas nerves that degenerate more slowly are more likely to exhibit axonotmesis. The two most reliable and objective electrical tests for facial nerve assessment are electroneuronography (ENoG) and facial muscle electromyography (EMG). Because complete or nearly complete recovery is to be expected with incomplete facial paralysis, electrical testing is of value only in assessing a patient with complete facial paralysis.

ENoG measures facial motor activity in response to a suprathreshold electrical stimulus applied to the facial nerve distal to the site of injury. Compound muscle action potentials are measured with surface electrodes placed in the nasolabial fold. The amplitudes of compound muscle action potentials elicited on the normal and involved sides are compared. In the first 3 days after the onset of complete facial paralysis, the distal facial nerve will continue to stimulate normally until wallerian degeneration occurs. ENoG testing is therefore delayed until at least 3 days after the onset of acute facial paralysis and continued every second or third day until day 14. Reduction of the ENoG response relative to the unaffected side correlates with the degree of facial muscle denervation, which in turn reflects the extent of neural degeneration on the paralyzed side. Because severe neural degeneration is associated with poor functional recovery, surgical decompression is offered when ENoG testing indicates greater than a 90% decrease in function on the affected side versus the normal side.

EMG with needle electrodes placed within the facial musculature measures spontaneous and voluntary electrical activity in the facial muscles. It is useful for assessing the presence and extent of muscle denervation and reinnervation. EMG testing is a necessary adjunct to the interpretation of ENoG results if surgical decompression is being considered. Deblocking of the regenerating nerve fibers results in asynchronous firing of electrical potentials and therefore the absence of measurable compound action potentials on ENoG testing with skin surface electrodes. Because voluntary motor unit action potentials measured on needle EMG testing do not require synchronous electrical activity, they may be detected early in the recovery of facial nerve function and indicate a favorable prognosis. In addition, polyphasic action potentials on voluntary facial muscle contraction indicate muscle reinnervation and may precede clinical signs of recovery by 6 to 12 weeks. Spontaneous fibrillation potentials detected 2 to 3 weeks after injury indicate significant muscle denervation and poor recovery.

Bell's Palsy

Bell's palsy, by far the most common cause of acute facial paralysis, accounts for approximately 70% of cases.[2] There is enough evidence to support the concept that the facial paralysis associated with Bell's palsy is the result of an inflammatory response to herpes simplex virus type 1 that induces edema and vascular compromise, which results in functional impairment.[3,4] This entrapment neuropathy is believed to occur in the labyrinthine segment of the facial nerve, where the fallopian canal is narrowest in diameter.

Unilateral facial paralysis affecting all branches of the nerve begins suddenly over the course of 24 to 48 hours and may progress to complete paralysis within 3 to 7 days. Symptoms may be preceded by otalgia on the affected side. With the exception of facial dysfunction and, rarely, an erythematous chorda tympani nerve, no other abnormalities are typically detected on clinical examination of these patients. Hearing and balance are not affected. Progressive loss of facial function beyond 2 weeks, absence of recovery by 4 months, fluctuating function, ipsilateral recurrence, and the presence of facial twitching should alert the physician to the possible presence of an underlying tumor and prompt early imaging of the facial nerve. Bell's palsy may recur in up to 15% of patients,

although it more commonly occurs on the contralateral side.

Sixty–five percent to 85% of these patients regain good facial function with medical treatment alone consisting of systemic steroids with or without antiviral therapy. The prognosis for satisfactory recovery of women in whom Bell's palsy with complete facial paralysis develops during pregnancy is significantly worse.[5] Facial movement usually begins in these patients approximately 3 weeks after onset of the paralysis. Permanent residual weakness or secondary abnormalities, such as synkinesis or facial spasm, occur in 15% to 35% of patients. In patients with electrical tests indicating severe neural degeneration, surgical decompression may offer improved recovery of facial function. In a multicenter prospective clinical trial involving individuals with Bell's palsy who had 90% or greater degeneration on ENoG testing within the first 14 days of onset of complete paralysis and absence of motor unit potentials on voluntary EMG testing, surgical decompression of the facial nerve at the meatal foramen, labyrinthine segment, and geniculate ganglion resulted in a 91% chance of good outcome 7 months after paralysis as opposed to a 42% chance of good recovery in patients with the same ENoG and EMG parameters who were treated with steroids only.[6] The results of this study suggest that surgical decompression of the labyrinthine facial nerve may be beneficial for a select group of patients with Bell's palsy who meet electrical test criteria for unfavorable recovery. However, some controversy remains regarding the benefit of surgical decompression for these patients.[7]

All patients seen within the first 2 weeks after the onset of acute facial paralysis should be treated with systemic steroid therapy (prednisone, 1 mg/kg/day for 10 days with an additional 5-day taper). Additionally, a 10-day course of antiviral therapy may be prescribed (acyclovir, 800 mg five times a day, famciclovir, 500 mg three times a day, or valacyclovir, 1 g two times a day).[8,9] Antiviral therapy should preferably be commenced within 72 hours of the onset of symptoms.[10,11] With the exception of protective eye care, no additional treatment is necessary for patients with incomplete paralysis. Those who progress to complete paralysis should undergo electrical testing beginning 3 days after complete loss of clinical function. If ENoG testing indicates greater than 90% degeneration within the first 2 weeks after the onset of paralysis, the patient has a 50% chance of residual facial weakness and synkinesis. Surgical decompression may be offered to these patients if additional EMG testing fails to reveal any evidence of neural regeneration.

Herpes Zoster Oticus

Herpes zoster oticus (Ramsay Hunt syndrome) is a syndrome of acute peripheral facial paralysis associated with otalgia and varicelliform lesions of the auricle and external auditory canal. It is distinguished from Bell's palsy by the characteristic viral eruptions, a higher incidence of associated cochlear and vestibular dysfunction, and a poorer prognosis for recovery of facial function. Primary treatment of herpes zoster oticus is antiviral agents such as acyclovir or famciclovir and high–dose systemic corticosteroids. Unlike Bell's palsy, there is no literature to support surgical decompression of the facial nerve in these patients.

Acute Facial Paralysis Resulting from Surgical Trauma

Traumatic injuries to the facial nerve from temporal bone fractures, penetrating trauma, and iatrogenic injury represent the second most common cause of facial paralysis. The facial nerve is at risk for injury during surgery on the parotid gland, temporal bone, and cerebellopontine angle. Complications from parotid gland or temporomandibular joint surgery can result in injuries that affect the facial nerve from the stylomastoid foramen to the facial musculature. In the cerebellopontine angle, facial nerve disruption generally results during removal of an acoustic neuroma. The lack of a tough epineural sheath and anatomic distortion by the tumor make the nerve particularly prone to injury in this site.[12]

Within the temporal bone, iatrogenic injuries to the facial nerve occur most commonly during middle ear and mastoid surgery. The incidence of unexpected injury to the facial nerve as a result of an operative procedure is highest during surgery for chronic otitis media with or without cholesteatoma, and such injury occurs in up to 2% to 4% of cases. In surgery for chronic ear conditions, the facial nerve is most vulnerable to injury at three sites: the tympanic segment, the second genu, and the mastoid segment. The facial nerve is especially vulnerable in the tympanic segment because the fallopian canal is naturally dehiscent at this site in up to 30% of individuals.[13] Furthermore, this site is frequently involved with granulation tissue or cholesteatoma, which can cause erosion of the fallopian canal. If the fallopian canal is dehiscent or eroded, dissection of diseased mucosa or cholesteatoma from the nerve sheath can injure the nerve. The tympanic segment of the facial nerve is also at risk for injury caused by drilling, especially during removal of the posterior canal wall in a canal wall down mastoidectomy. The vertical portion of the facial nerve can be injured during drilling of the mastoid when the posterior canal wall is taken down, when the facial recess is opened, or when the retrofacial air cell tracts are opened. The key to avoiding iatrogenic injuries in temporal bone surgery is thorough knowledge of facial nerve anatomy within the temporal bone.[14]

Iatrogenic facial nerve trauma is best prevented by acquiring a thorough understanding of the course of the nerve through the temporal bone and by clearly identifying landmarks within the middle ear and mastoid that relate

closely to the nerve. Injuries to the facial nerve detected at the time of surgery should be addressed immediately whenever possible. Surgical decompression of the fallopian canal 1 cm proximal and distal to the site of an injury is usually adequate if the nerve is intact but contused. The epineurium may be incised if the nerve is significantly edematous or contused. By contrast, if more than 50% of the nerve has been transected, repair by primary neurorrhaphy or interposition grafting is warranted. If facial nerve paralysis is unexpected and noted immediately in the recovery room, any packing in the ear should be removed, and the patient should be observed for 2 hours to allow any effect of the lidocaine used during the procedure to dissipate. Operative trauma to the facial nerve must be suspected if the facial paralysis persists. Neural integrity can also be confirmed by the presence of voluntary motor unit action potentials on needle EMG testing in the immediate postoperative period. Surgery to explore and repair the facial nerve should be arranged within 24 to 48 hours. Delayed facial paralysis may also occur after mastoid surgery and is generally a result of neural edema and secondary entrapment neuropathy after mild manipulation of the facial nerve in a dehiscent segment. Packing should be removed from the mastoid and ear canal, and serial ENoG testing should be performed to detect any significant neural degeneration. All patients with facial paralysis resulting from surgical trauma should be treated with high-dose systemic corticosteroids (prednisone, 1 mg/kg/day for 10 days with an additional 5-day taper).

Acute Facial Paralysis Associated with Acute and Chronic Otitis Media

Facial paralysis associated with acute suppurative otitis media is typical in children or young adults who have clinical signs and symptoms of a middle ear abscess. The facial paralysis usually progresses rapidly over a period of 2 to 3 days and is associated with the acute onset of otalgia and otorrhea. The pathophysiology appears to be related to the presence of natural bony dehiscences in the fallopian canal along the tympanic segment of the facial nerve that allow inflammatory products to produce inflammation and edema of the nerve.

Management of facial paralysis associated with acute otitis media should be aggressive. Myringotomy is performed immediately to drain the purulent exudate and to obtain material for culture and sensitivity testing. A ventilation tube is placed to maintain aeration of the middle ear. Intravenous broad–spectrum antibiotics are begun empirically and may be modified when the results of the culture and sensitivity testing are available. Topical antibiotic otic drops are started, and daily aspiration of the middle ear is performed. Corticosteroids (prednisone, 1 mg/kg/day) are prescribed for 10 days. If computed tomography (CT) of the temporal bone reveals coalescent mastoiditis or intracranial extension of the infection, a cortical mastoidectomy should be performed. Patients with intracranial complications should be managed in consultation with the neurosurgical service. The prognosis for recovery of function in those with facial paralysis secondary to acute otitis media is good without surgical decompression.[15]

In chronic otitis media, facial nerve paralysis is most commonly associated with cholesteatoma or chronic inflammatory granulation tissue involving the tympanic and vertical segments of the facial nerve.[16] As with acute otitis media, facial nerve dysfunction may be caused by inflammation, edema, and subsequent entrapment neuropathy. Alternatively, extraneural and intraneural compression may also result from an enlarging cholesteatoma or abscess. Chronic otitis media complicated by facial paralysis is usually managed surgically. Removal of cholesteatoma, with or without surgical decompression of the affected facial nerve segment, intravenous antibiotics, and corticosteroid therapy usually result in favorable functional recovery. Patients with chronic suppurative otitis media without cholesteatoma appear to have a better functional outcome than do those with cholesteatoma.[17] The prognosis for recovery of facial function in these patients is related to the time of intervention.[18]

Acute Facial Paralysis Associated with Tumors

The most common neoplasms causing facial paralysis are malignant tumors of the parotid gland. Facial nerve neuroma, acoustic neuroma, and primary brain tumors are less common causes of facial paralysis. Surgical management is dictated by tumor pathology and is described in detail in other chapters of this textbook.

PREOPERATIVE PLANNING

Electrical tests, including ENoG and EMG, are used to determine surgical candidacy when no clinical function is observed. General guidelines for offering surgical decompression include 10% or less muscle function on the affected side versus the normal side as determined by ENoG performed between days 3 and 14 after the onset of complete facial paralysis, along with the absence of motor unit action potentials on EMG testing.

High-resolution axial and coronal CT scanning of the temporal bone using a bone algorithm should be performed if surgery is planned. This is of particular value in patients with facial paralysis resulting from otitis media and trauma. CT enables the surgeon to localize the site of pathology preoperatively. Magnetic resonance imaging will demonstrate enhancement of the labyrinthine facial nerve in all cases of Bell's palsy but is not routinely performed before surgical decompression unless there is clinical suspicion of underlying neoplastic disease.

Audiometric testing should be performed in all cases of facial nerve paralysis to detect any associated hearing loss, which may aid in the diagnosis, especially of any injury to the cochlea, and help dictate the surgical approach. It also provides a baseline for monitoring recovery in the postoperative period.

Determination of which regions of the facial nerve require exploration and the approach to be used is based primarily on the cause of the facial paralysis and the expected site of injury. In Bell's palsy, the labyrinthine segment and perigeniculate region are decompressed via a middle fossa approach. In facial paralysis caused by acute or chronic otitis media, the mastoid and tympanic segments are explored, depending on the site and extent of disease. Canal wall down mastoidectomy may be required to properly exteriorize a cholesteatoma involving the facial nerve. Surgery for facial paralysis secondary to intraoperative injury is directed at the site of surgical injury.

Lubrication and protection of the affected eye should be instituted at the time of initial diagnosis and continued until adequate eye closure is achieved. An ophthalmologic opinion should be obtained if exposure keratitis is present.

SURGICAL APPROACHES

Selection of the surgical approach is determined by the location of the facial nerve injury and hearing status in the affected ear (Fig. 122-1).

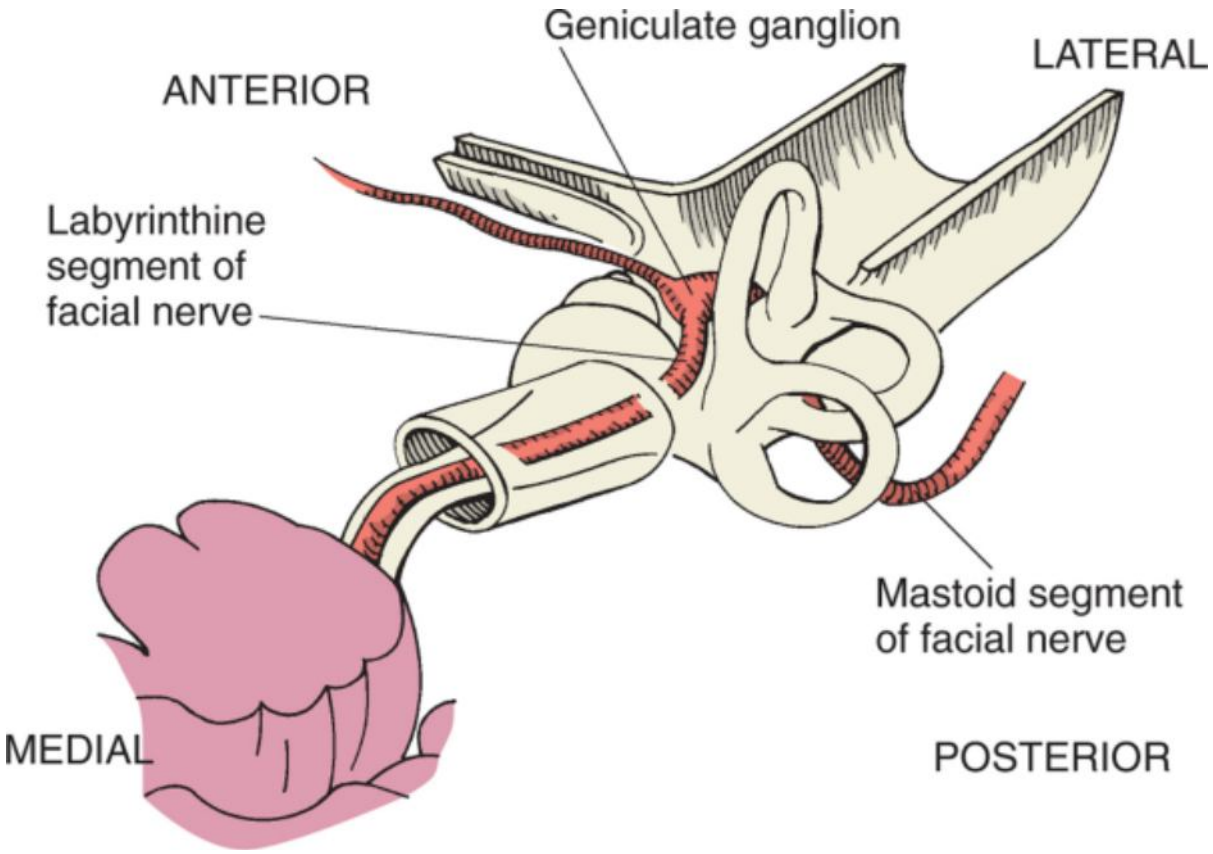

Figure 122-1 Anatomic course of the facial nerve.

Transmastoid Approach

This approach is used when facial nerve injury is limited to the tympanic and mastoid segments of the intratemporal facial nerve. The mastoid cortex is exposed through a standard postauricular incision and soft tissue dissection as described in Chapter 115. A complete mastoidectomy is performed until the mastoid antrum is entered medially. The lateral semicircular canal is identified and bone dissection is continued anterosuperiorly to expose the body of the incus in the epitympanum. Great care should be taken to avoid inadvertently drilling on the ossicular chain and violating the tegmen when drilling in the epitympanum.

The mastoid segment of the facial nerve is identified by two important surgical landmarks: the lateral semicircular canal superiorly and the digastric ridge inferiorly (Fig. 122-2). The lateral semicircular canal is the most important landmark for the facial nerve because it defines both the anteroposterior and the mediolateral location of the facial nerve at the second genu. As the digastric ridge is followed anteriorly, the underlying fascia spirals to form the sheath of the facial nerve at the stylomastoid foramen. Thus, the course of the mastoid segment of the facial nerve

can be predicted by visualizing a line connecting the lateral semicircular canal and the anterior aspect of the digastric ridge.

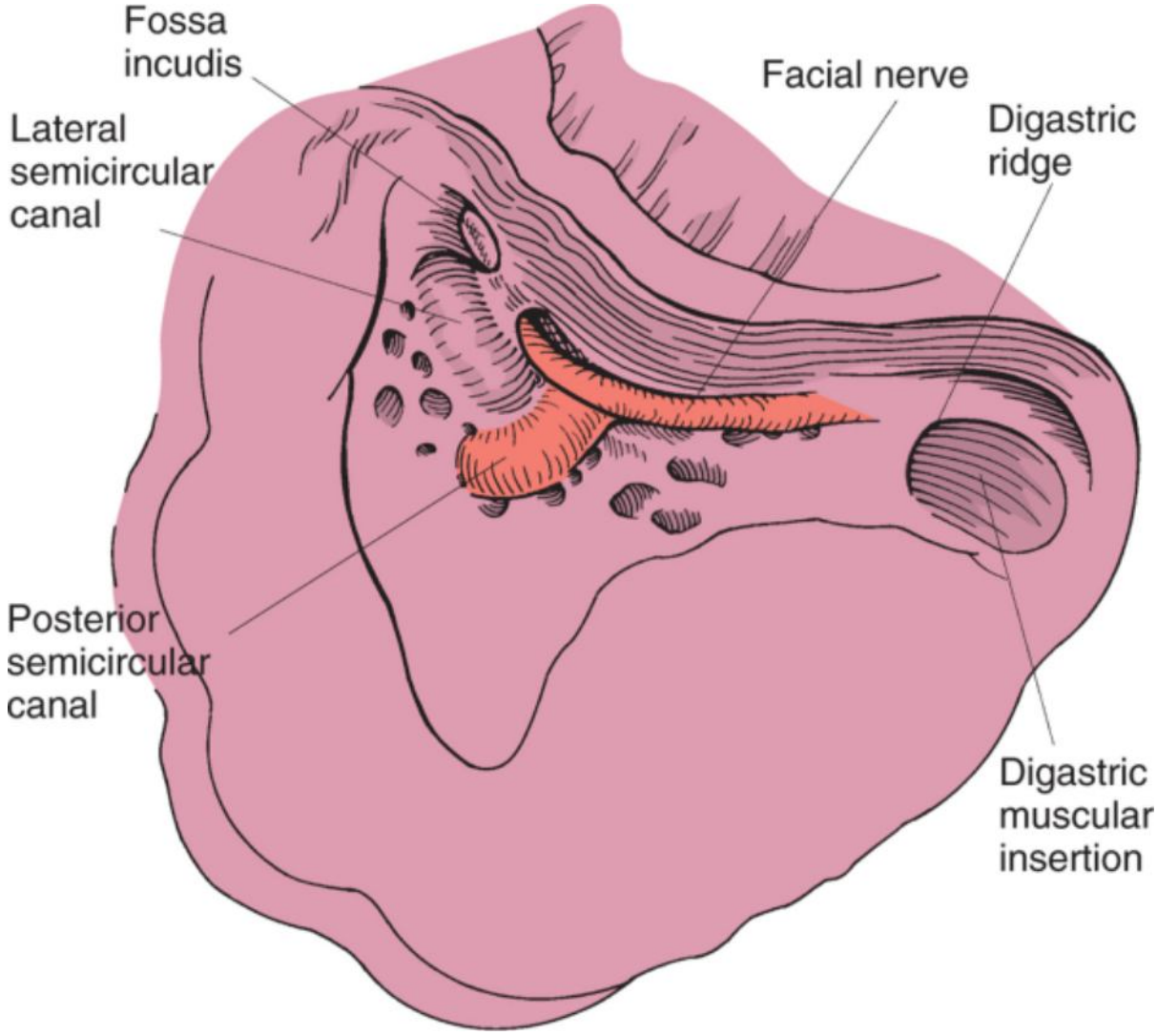

Figure 122-2 Surgical landmarks for the mastoid segment of the facial nerve.

Once the course of the mastoid segment of the facial nerve is identified, the facial recess is opened with 1- to 3-mm diamond burrs (Fig. 122-3). Drilling should be carried out parallel to the course of the nerve with the use of copious irrigation to minimize the risk of thermal injury to the nerve. A narrow bridge of bone is preserved along the inferior border of the fossa incudis to protect the incus. Bone is removed over the lateral surface of the facial nerve until a thin shell of bone remains. Inferior and lateral exposure of the facial recess is limited by the chorda tympani nerve. Anterior and slightly medial to the facial nerve, additional bone removal is often necessary to maximize exposure within the facial recess.

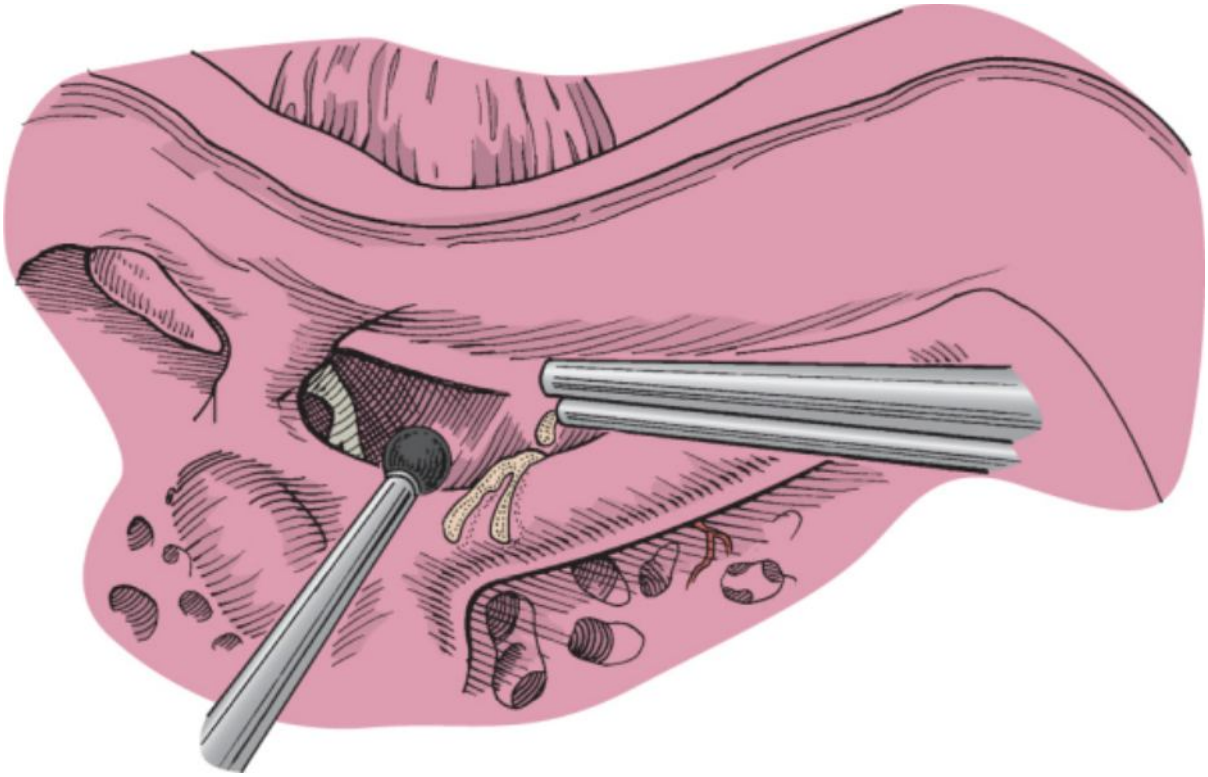

Figure 122-3 The facial recess is opened.

At this point, exposure of the facial nerve is sufficient to allow decompression of the mastoid segment, second genu, and distal tympanic segment. Decompression begins by exposing the thick periosteum of the stylomastoid foramen with a 2- to 2.5-mm diamond burr while leaving the thick fibrous sheath around the facial nerve intact in this location. The circumference of the facial nerve should be exposed for 180 degrees along its posterior and superior surface, between the lateral semicircular canal and the stylomastoid foramen (Fig. 122-4).

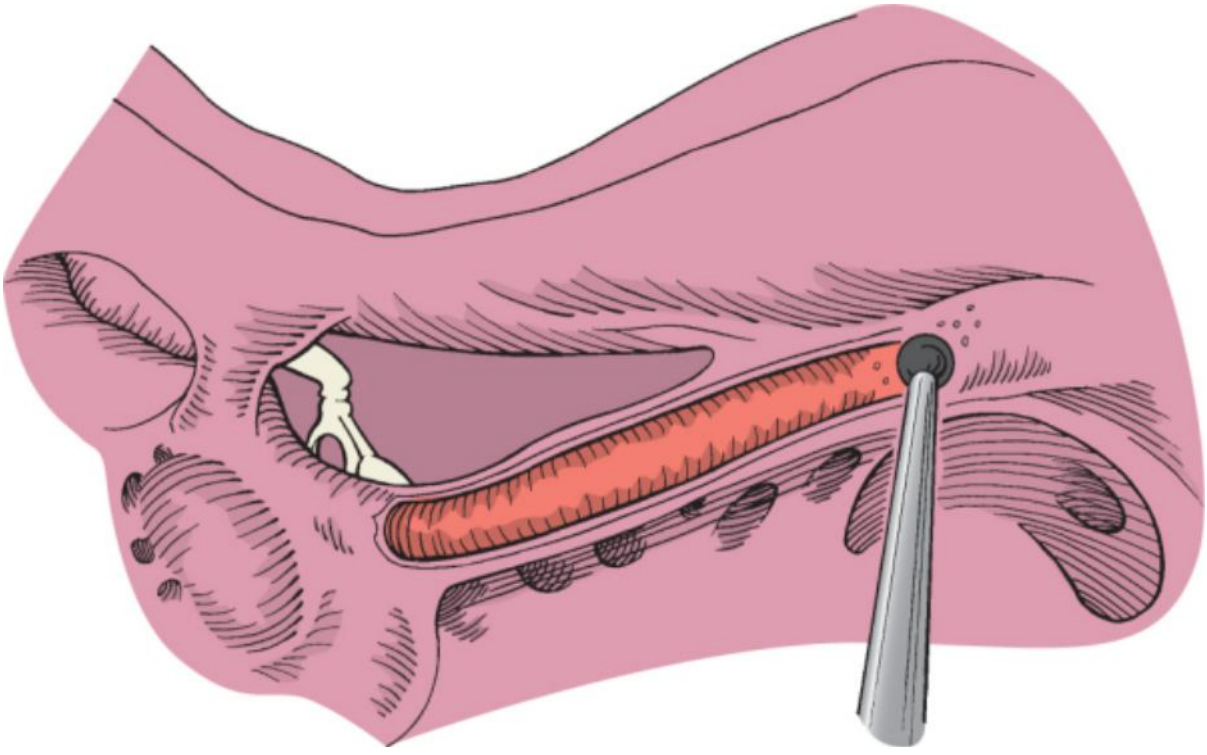

Figure 122-4 The mastoid segment of the facial nerve is decompressed.

A small diamond drill burr is then used to decompress the second genu and distal tympanic segment of the facial

nerve by rotating the diamond burr from the posterior surface of the nerve to the lateral and anterolateral surface of the nerve. This maneuver, called *barber poling,* prevents fenestration of the lateral semicircular canal (Fig. 122-5).

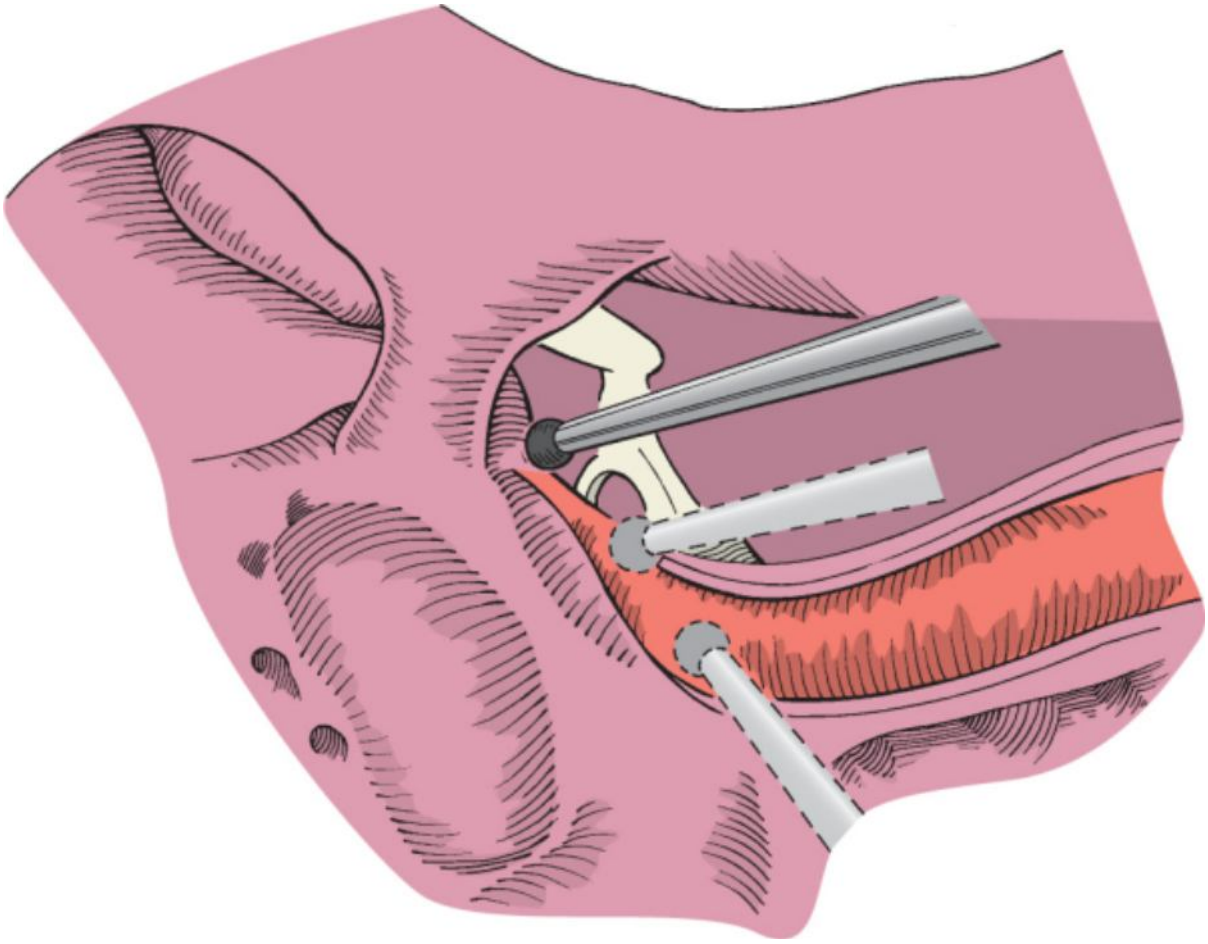

Figure 122-5 The second genu and distal tympanic segment of the facial nerve are decompressed.

Depending on the position of the incus, the distal tympanic segment and second genu of the facial nerve may be decompressed with a microdrill and microcurette while leaving the incus intact in some individuals (Fig. 122-6). If the relative proximity of the incus to the fallopian canal does not allow safe dissection of the nerve, the incus should be removed for exposure and replaced at the completion of the procedure.

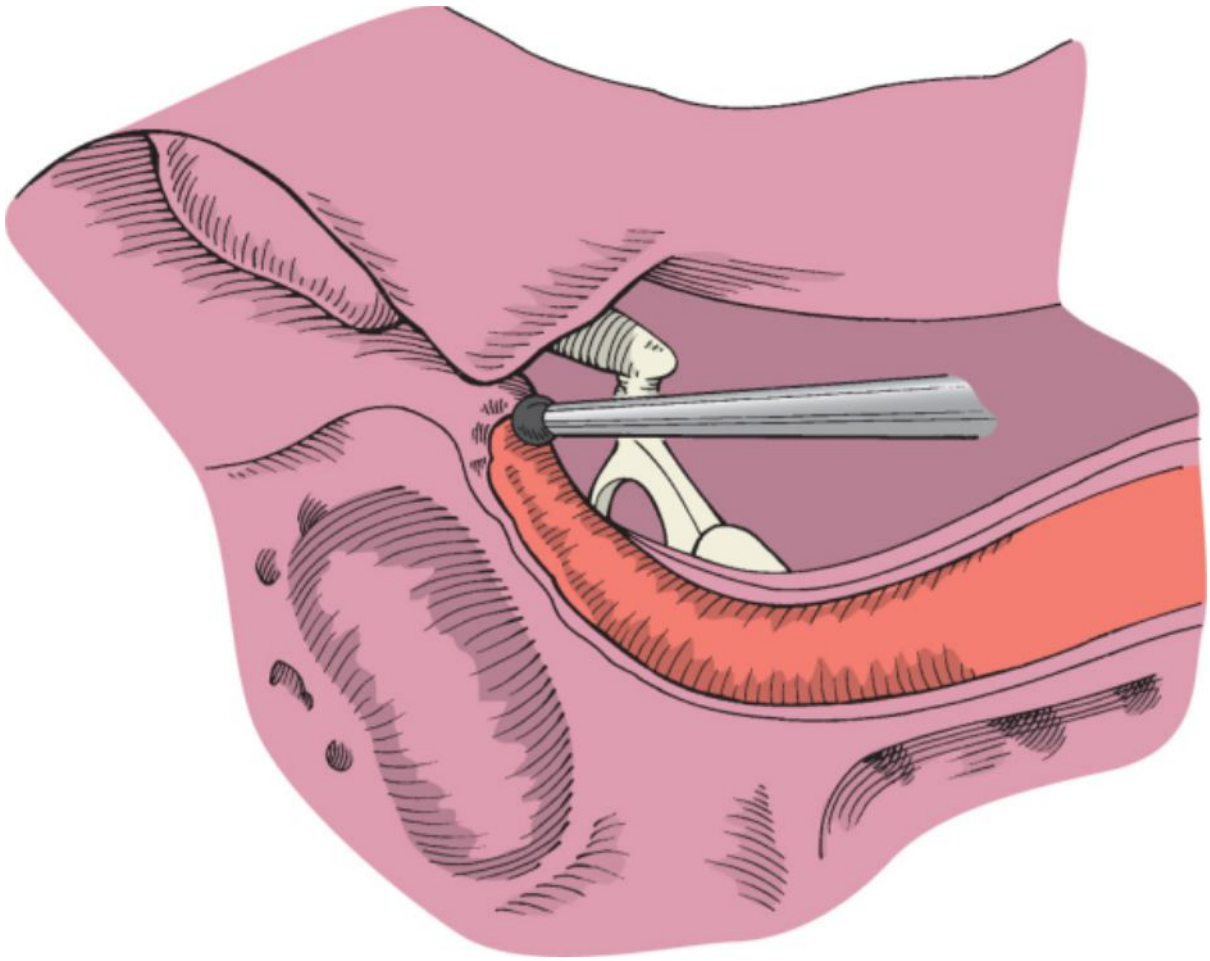

Figure 122-6 The proximal tympanic segment is decompressed with the incus in place if space permits.

The ability to expose the perigeniculate region via the transmastoid approach depends on the position of the middle fossa plate within the epitympanum and requires removal of the incus and occasionally the head of the malleus. The incudostapedial joint is separated through the facial recess, and the incus is rotated posteriorly out through the epitympanum by placing a 90-degree hook under the incus body. The tympanic facial nerve lies superior to the oval window and then courses between the cochleariform process inferiorly and the cog superiorly before diving medially toward the geniculate ganglion. The proximal tympanic segment is decompressed up to and beyond the cochleariform process with a microdrill and microcurette. The junction of the facial nerve and geniculate ganglion is reached with further anterior and medial dissection under the head of the malleus (Fig. 122-7).

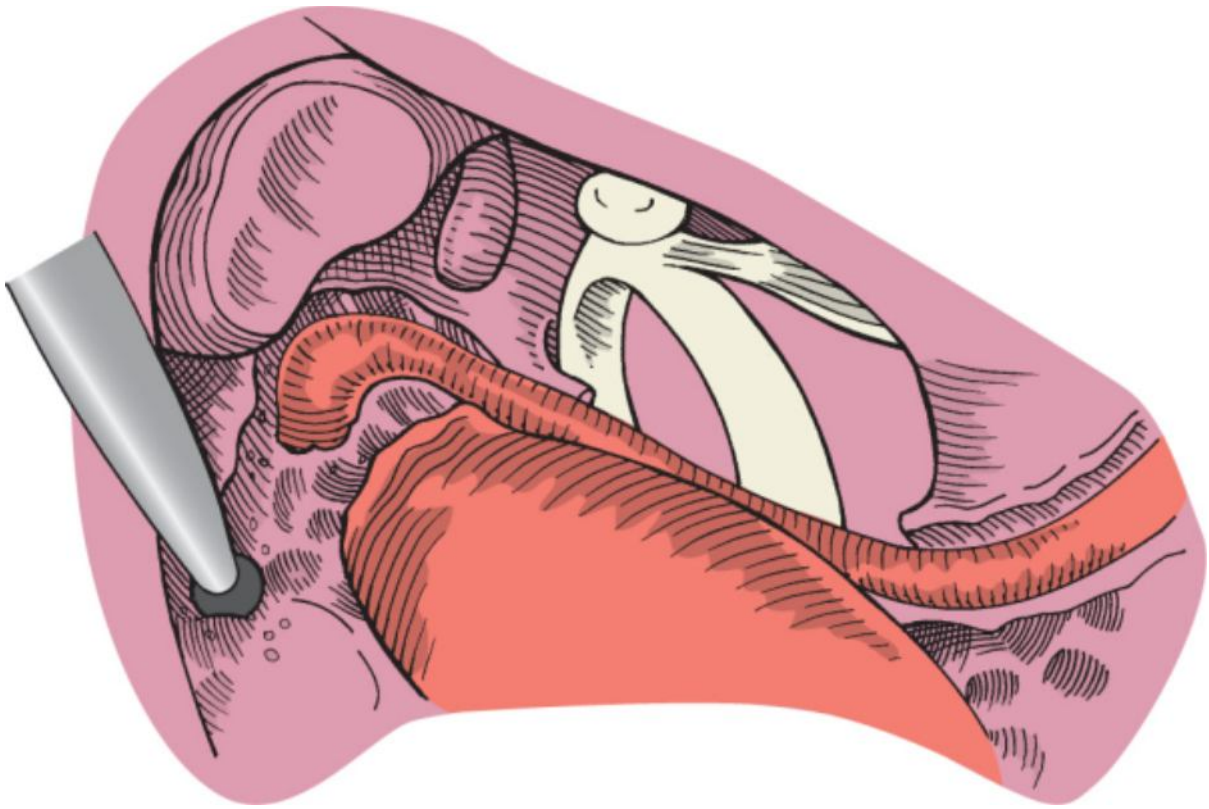

Figure 122-7 The perigeniculate and distal labyrinthine segments of the facial nerve are decompressed.

The facial nerve turns abruptly posterior, medial, and inferior at the geniculate ganglion as it enters the labyrinthine segment. The ampulla of the superior canal blocks further exposure of the labyrinthine facial nerve through the transmastoid approach. Complete exposure of the labyrinthine segment of the facial nerve requires a middle fossa approach (if hearing is present) or a translabyrinthine approach (if hearing is absent).

Once the fallopian canal in the tympanic and mastoid segments has been exposed, any residual impinging bony spicule is removed. The nerve sheath is opened at the site of injury and for a short distance proximal and distal to the site of injury to assess the severity of injury to the fascicles. If the fascicles are intact, the decompression procedure is complete. If more than 50% of the nerve fascicles have been violated or the nerve is completely transected, primary neurorrhaphy or cable grafting is indicated.

The removed incus is replaced at the end of the procedure and supported in place with saline-soaked Gelfoam pledgets. If removal of the malleus head was necessary for surgical exposure, the incus may be sculpted and interposed between the stapes capitulum and malleus handle. The postauricular wound is closed in layers with absorbable suture, and a mastoid dressing is applied to the operated ear for 24 hours. Postoperative hospitalization for transmastoid decompression alone is not necessary.

Translabyrinthine Approach

This approach is used to decompress the facial nerve proximal to the geniculate ganglion in a nonhearing ear. The surgical technique is identical to that used for translabyrinthine removal of acoustic neuromas and is detailed in Chapter 124.

Middle Fossa Approach

The primary advantage of the middle fossa approach is that it provides optimal exposure of the perigeniculate and labyrinthine facial nerve while preserving residual hearing in the operated ear. This is the approach used for surgical decompression of carefully selected patients with Bell's palsy and temporal bone fracture. It may also be used in combination with a transmastoid approach if complete intratemporal facial nerve decompression is necessary.

The patient is positioned supine with the head turned so that the operated ear is facing up. Intraoperative facial nerve monitoring is established at the beginning of the procedure. Paralytic agents are not used after induction of general anesthesia. Although the facial nerve is paretic, direct electrical stimulation of the intratemporal facial nerve during surgery may sometimes identify the site of conduction block. Brain stem auditory evoked responses are

also monitored routinely because hearing preservation is desired. Perioperative medications administered include broad–spectrum antibiotics with good cerebrospinal fluid (CSF) penetration (ceftriaxone), furosemide (Lasix), 20 mg mannitol, 0.5 gm/kg, and dexamethasone, 10 mg. Bacitracin (50,000 units/L of saline solution) is used in the irrigation fluid.

An incision is made in the preauricular crease, starting at the level of the lower border of the zygoma and extending superiorly above and behind the auricle to form a reverse question mark (Fig. 122-8). Anterior and posterior skin flaps lateral to the temporalis fascia are elevated to expose the temporalis muscle. An anteroinferiorly based temporalis muscle flap is created by incising the muscle along linea temporalis with Bovie electrocautery. A 4- × 5-cm craniotomy centered two thirds anterior and one third posterior to the external auditory canal and inferiorly based at the root of the zygoma is created with a 4-mm cutting burr (Fig. 122-9). The bone flap is carefully elevated from underlying dura with a dural elevator, removed, and soaked in bacitracin solution until the end of the procedure. Any bleeding from the dura may be controlled with bipolar electrocautery. The inferior border of the craniotomy is then lowered to the level of the middle fossa floor. Dura is carefully elevated off the middle fossa floor in a posterior-to-anterior direction to expose the anatomy of the floor of the middle fossa (Fig. 122-10). The limits of exposure are the middle meningeal artery anteriorly, the sulcus of the superior petrosal sinus medially, and the arcuate eminence overlying the dome of the superior semicircular canal posteriorly. Dura over the greater superior petrosal nerve (GSPN) tends to be densely adherent to the nerve. Great care is needed in dissection at this point because of the possibility of injuring a dehiscent geniculate ganglion. The dura is elevated in a posterior–to-anterior direction to minimize injury to the GSPN and geniculate ganglion (Fig. 122-11). Once exposure of the floor of the middle fossa is complete, the tongue of the middle fossa self-retaining retractor is secured at the sulcus of the superior petrosal sinus to retract the temporal lobe (Fig. 122-12).

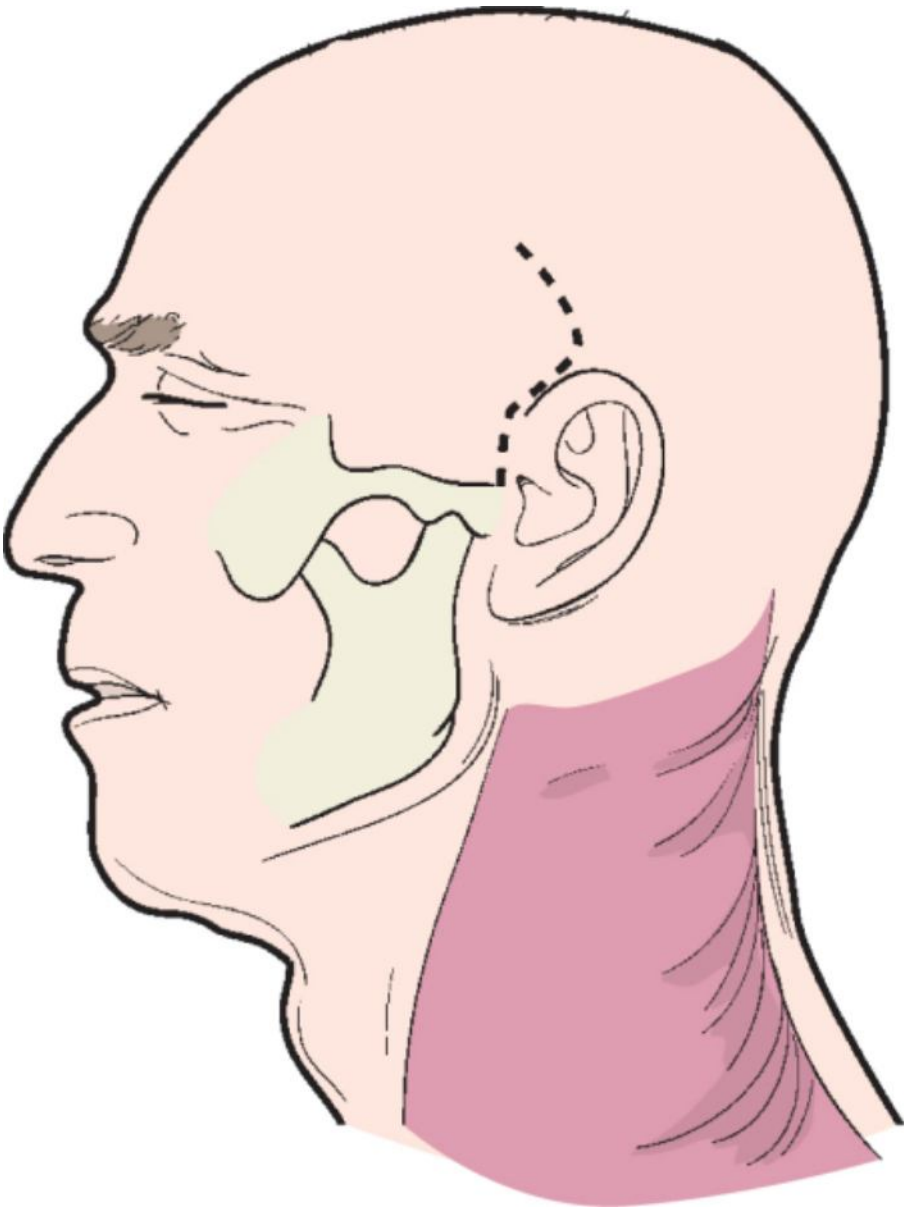

Figure 122-8 Skin incision for the middle fossa approach.

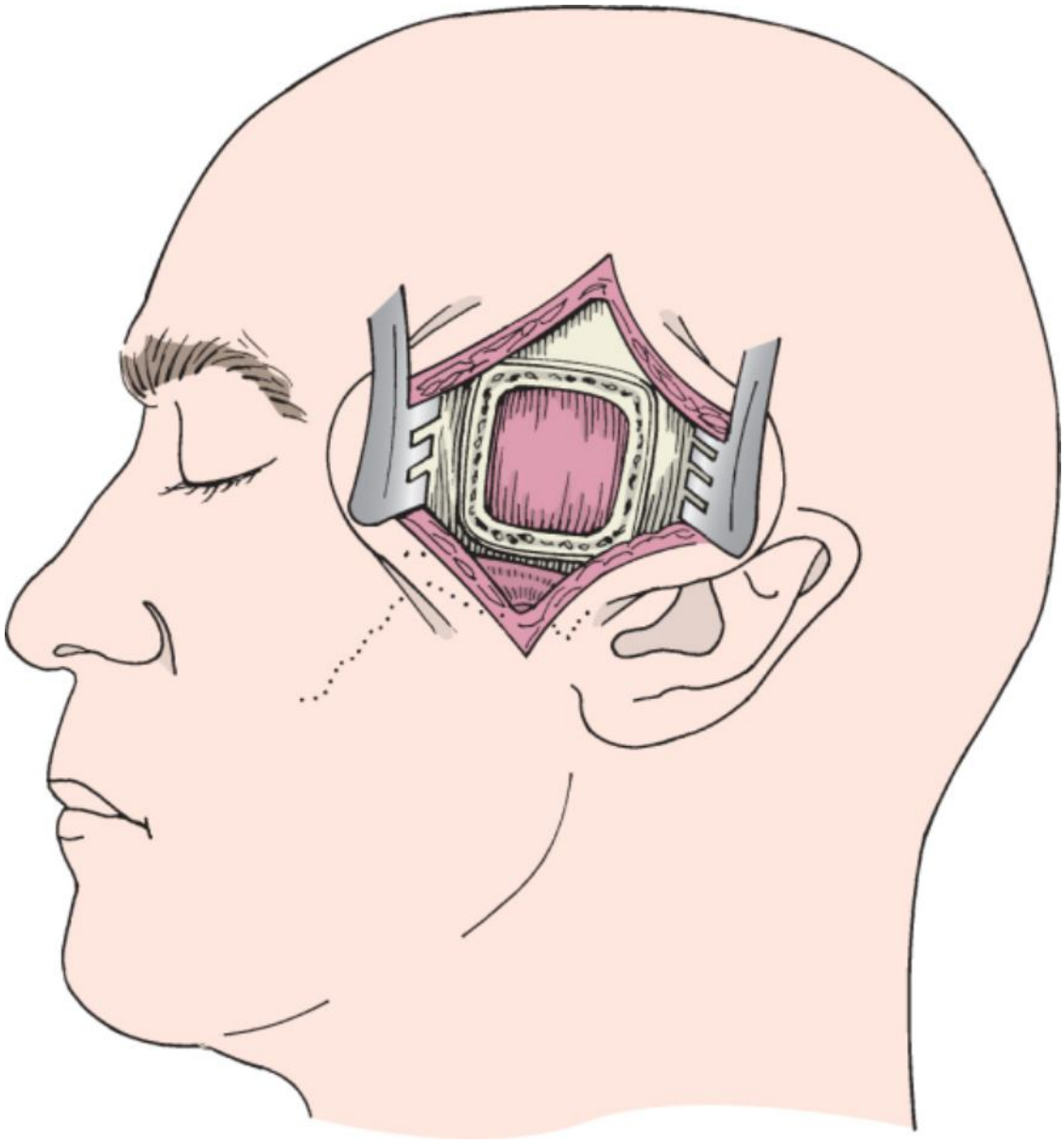

Figure 122-9 Temporal craniotomy used in the middle fossa approach.

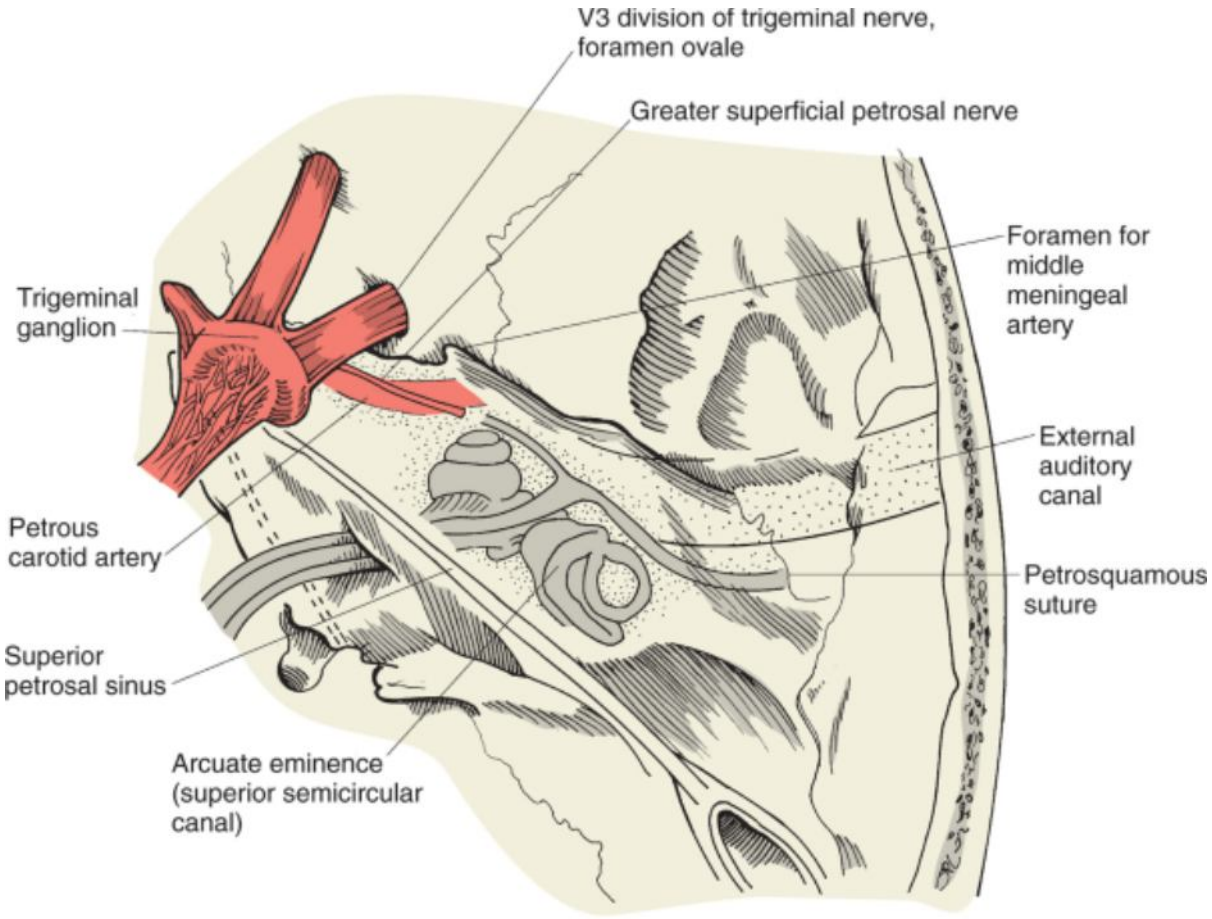

Figure 122-10 Anatomy of the floor of the middle fossa.

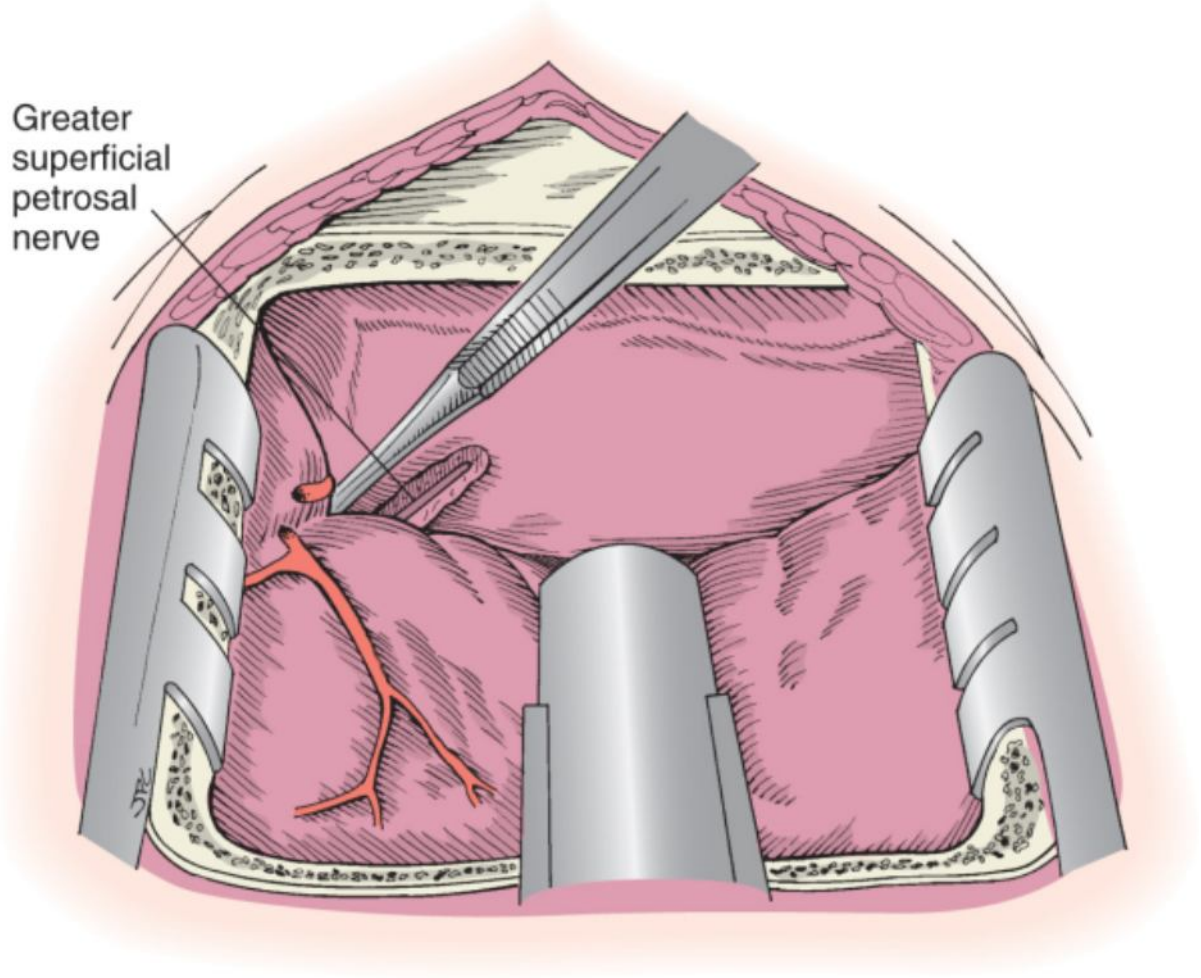

Figure 122-11 The greater superficial petrosal nerve is identified and preserved.

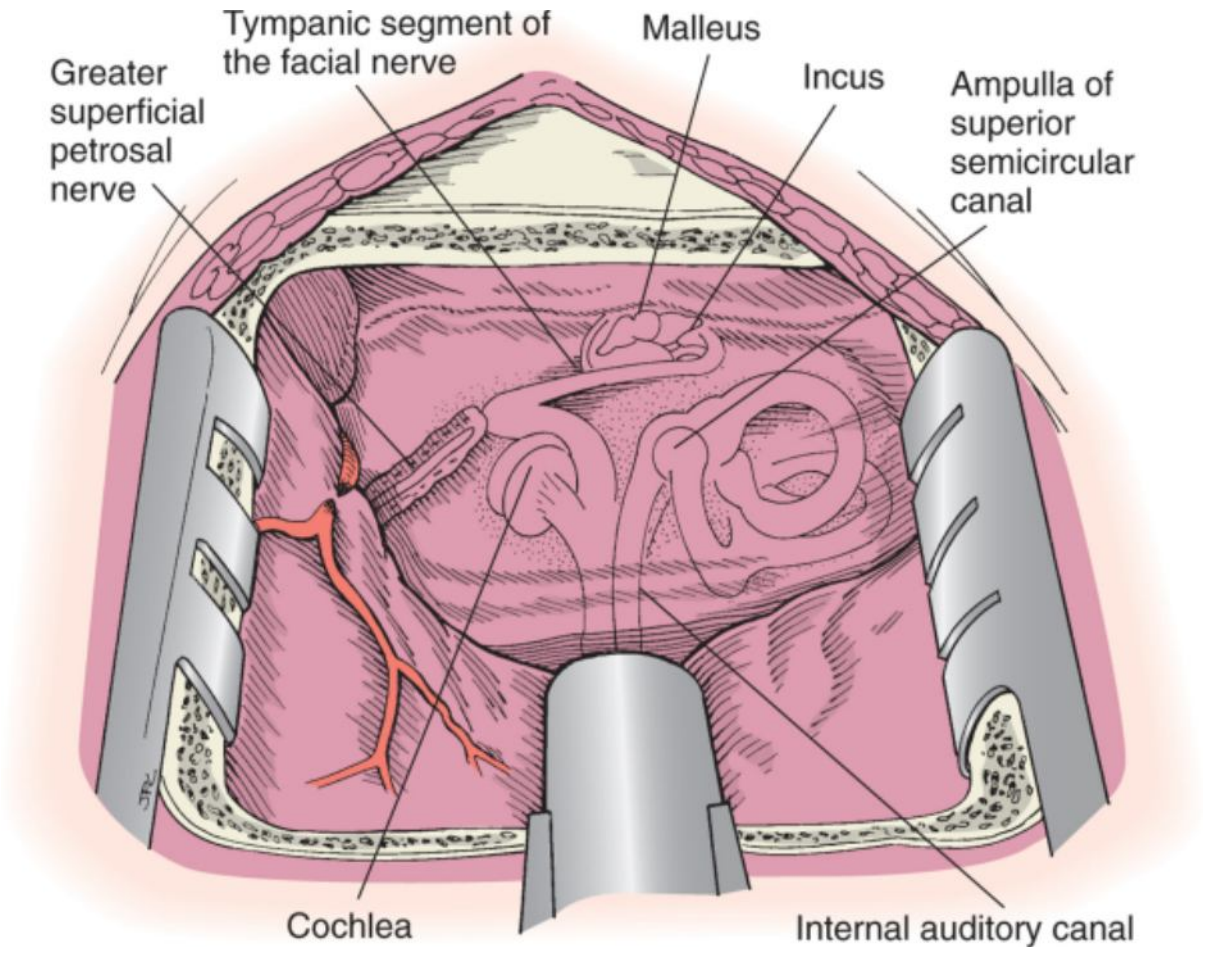

Figure 122-12 The floor of the middle fossa is exposed, and the self-retaining middle fossa retractor is secured.

The GSPN is used as the primary anatomic landmark for locating the facial nerve along the middle fossa floor. The arcuate eminence overlying the dome of the superior semicircular canal is located, and the positions of the cochlea, facial nerve, internal auditory canal (IAC), and middle ear ossicles are estimated. The location of the IAC is determined by bisecting the angle formed by the GSPN and arcuate eminence (Fig. 122-13). The roof of the middle ear is opened to identify the head of the malleus and the cochleariform process. The perigeniculate region is exposed by following the GSPN posteriorly and the tympanic segment of the facial nerve anteriorly into the geniculate ganglion with a 1-mm diamond burr (Fig. 122-14).

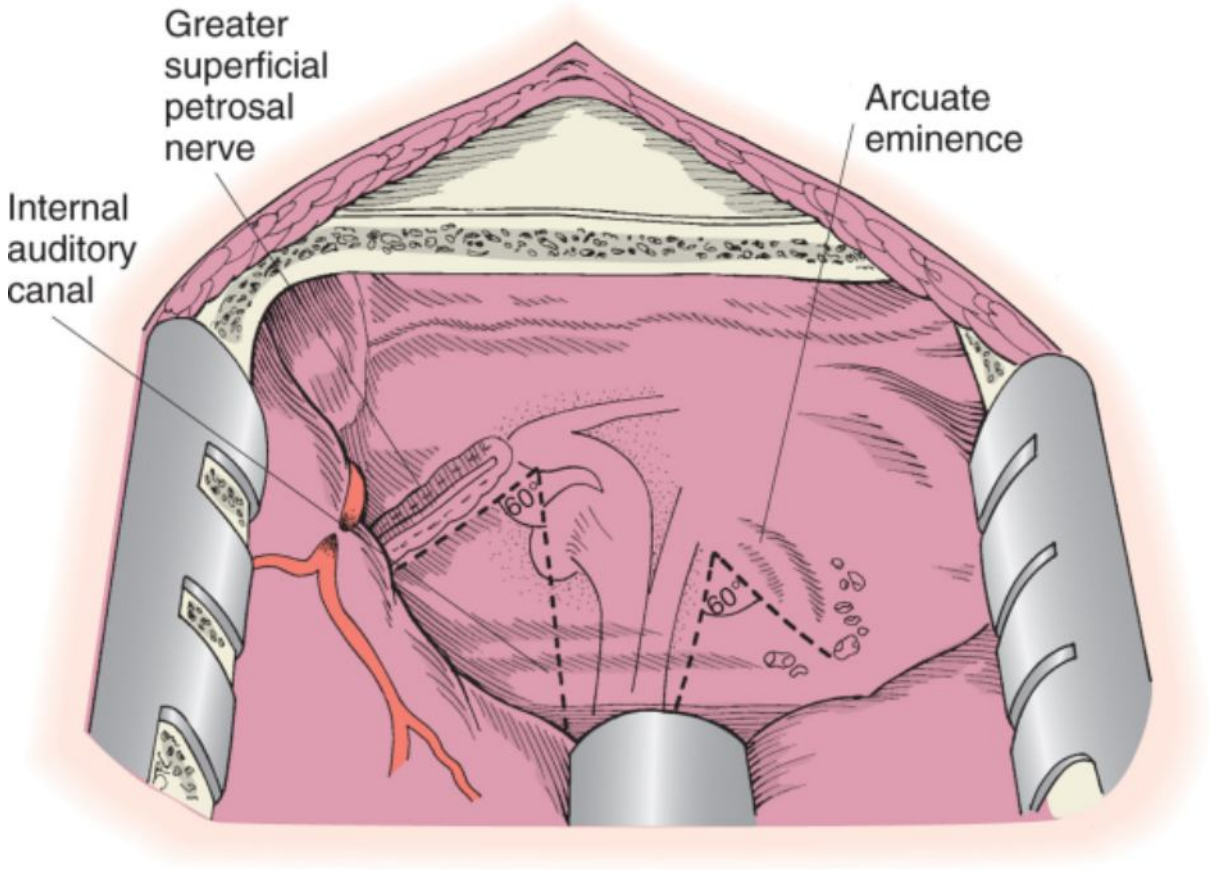

Figure 122-13 The position of the internal auditory canal is identified by using the anatomic relation of the greater superficial petrosal nerve and the arcuate eminence.

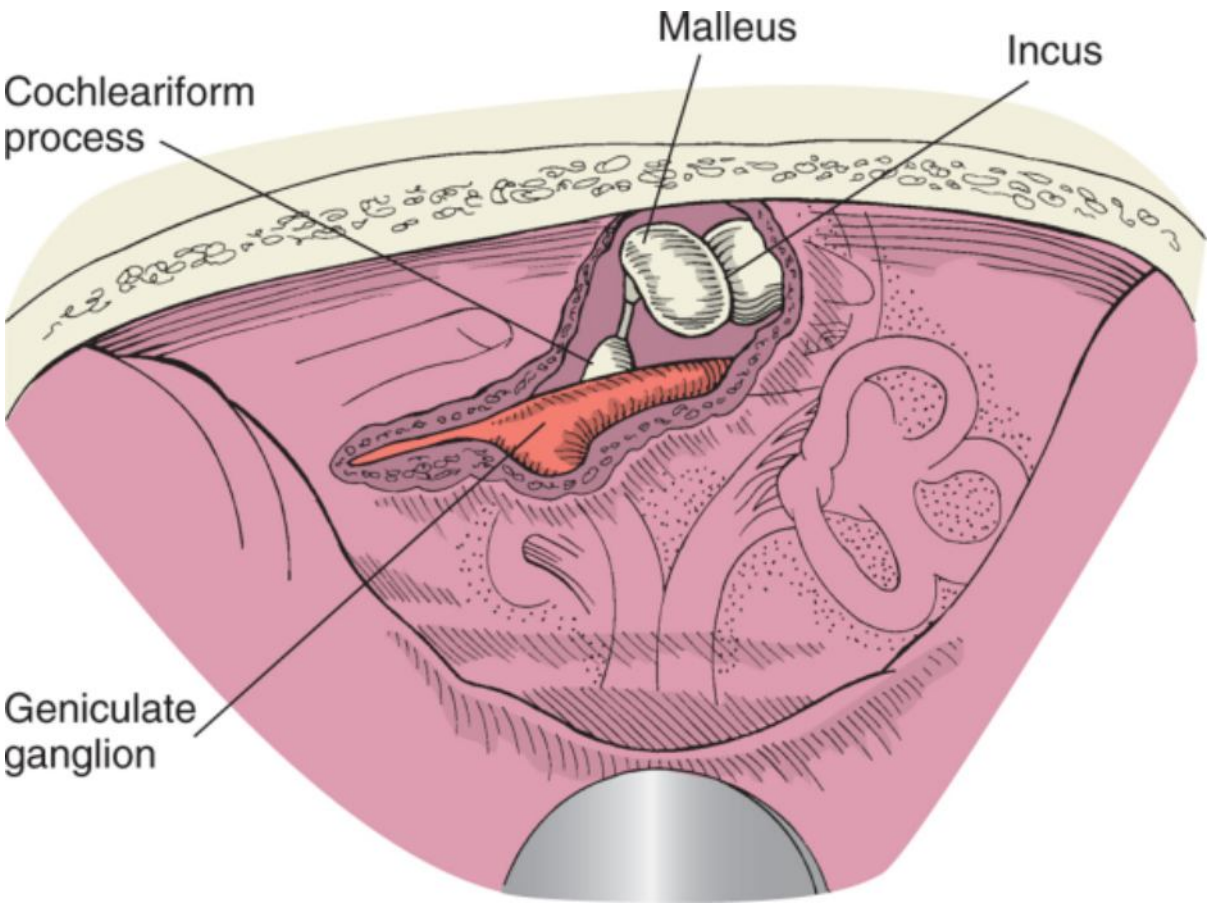

Figure 122-14 The tegmen tympani is unroofed to identify the malleus, incus, and cochleariform process. The geniculate ganglion is exposed. The perigeniculate and labyrinthine segments of the facial nerve are identified.

Bone over the labyrinthine facial nerve is then carefully removed by following the nerve from the geniculate ganglion posteriorly and medially into the IAC (Fig. 122-15). The labyrinthine facial nerve lies immediately lateral to the upper basal turn of the cochlea. The diameter of the fallopian canal in the labyrinthine segment is less than 1 mm, and it runs between the upper basal turn of the cochlea and the ampulla of the superior semicircular canal. The distance between the upper basal turn of the cochlea and the ampulla of the superior semicircular canal is 5.5 ± 1.0 mm (Fig. 122-16). A 1-mm diamond burr is used to uncover the superior surface of the labyrinthine segment. To prevent inadvertent fenestration of the cochlea, the area immediately anterior to the labyrinthine segment is closely observed for the blue line of the upper basal turn of the cochlea. Similarly, the area posterior to the labyrinthine segment is observed for the blue line of the ampulla of the superior semicircular canal. If the cochlea is inadvertently fenestrated, suctioning should be avoided in the area and the bone defect immediately plugged with bone wax. Bone over the tegmen tympani is removed to complete bony decompression of the perigeniculate facial nerve. Once bony exposure of the facial nerve has been achieved, the dura of the IAC and any dural thickening surrounding the meatal foramen are incised. The periosteum and perineurium of the facial nerve are split to a point just distal to the geniculate ganglion (Fig. 122-17).

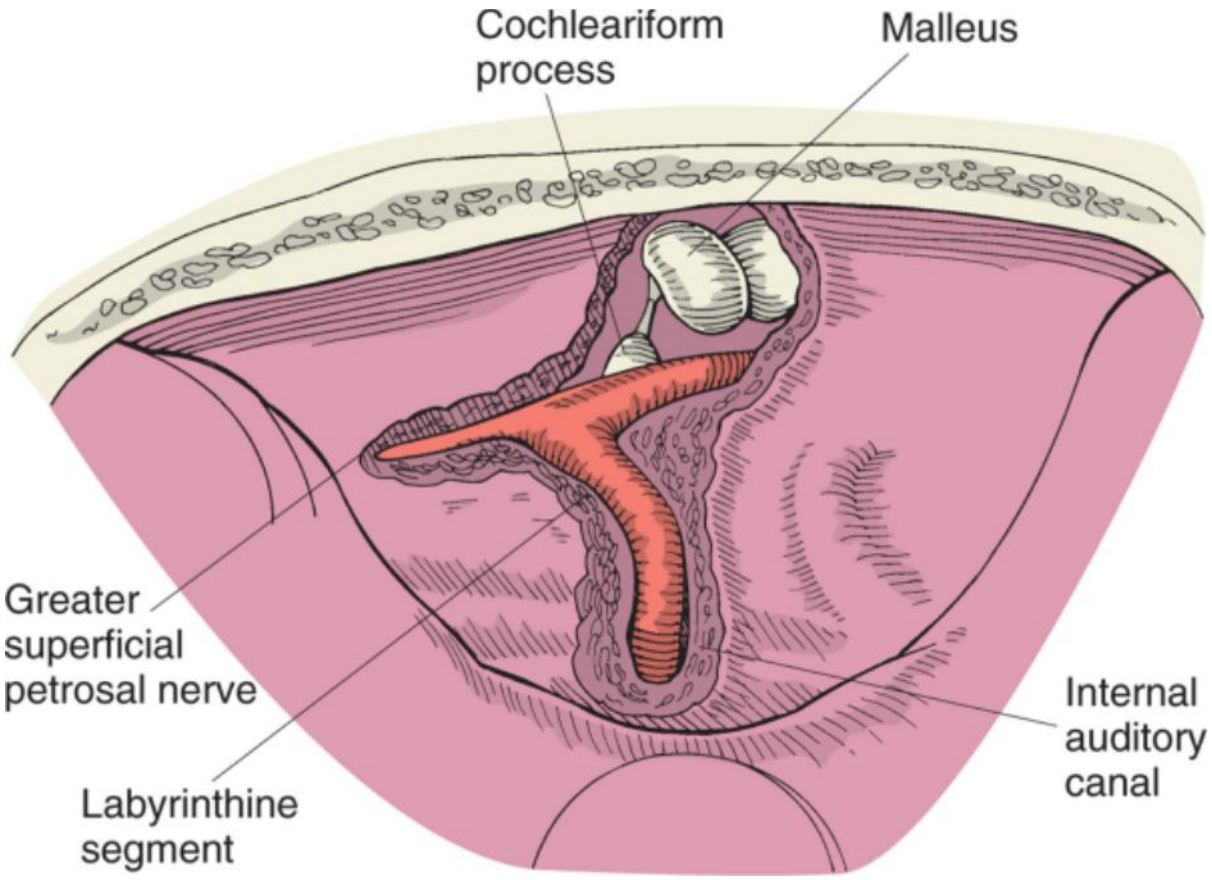

Figure 122-15 The labyrinthine segment of the facial nerve is exposed.

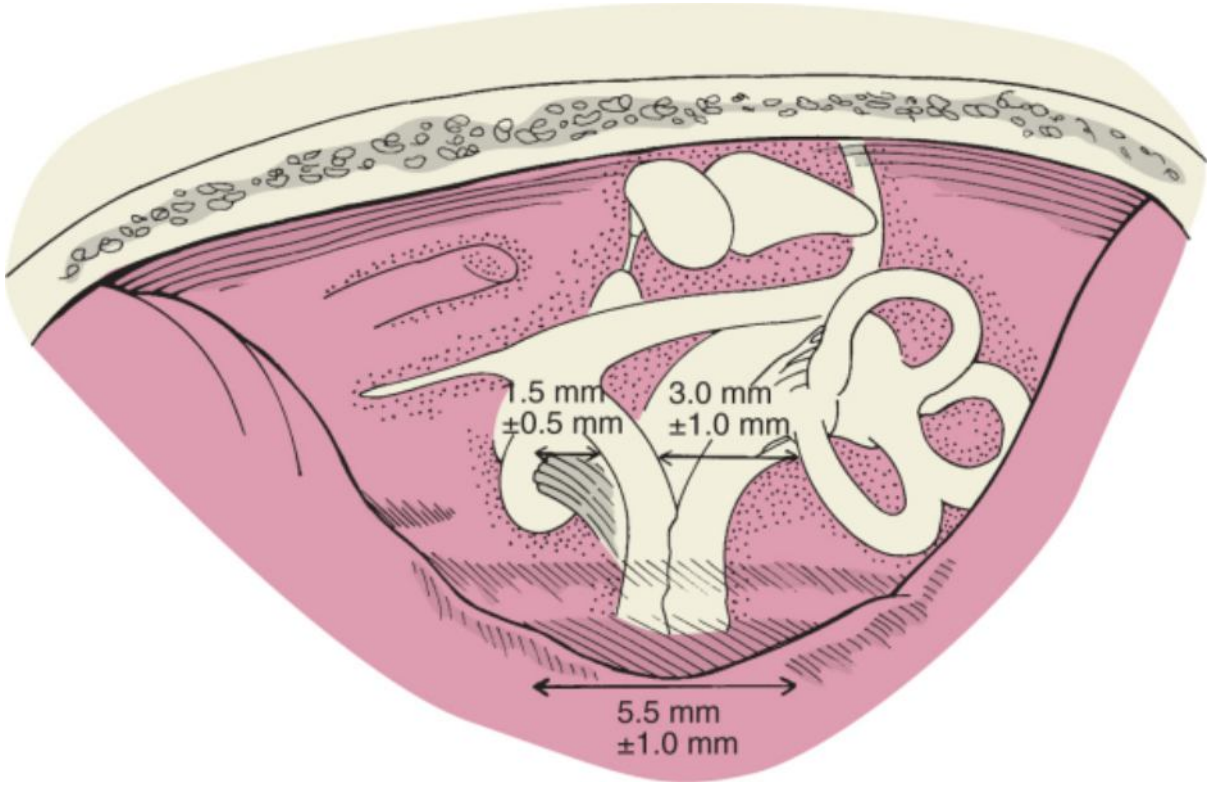

Figure 122-16 Average anatomic measurements between the labyrinthine segment of the facial nerve, the upper basal turn of the cochlea, and the ampulla of the superior semicircular canal.

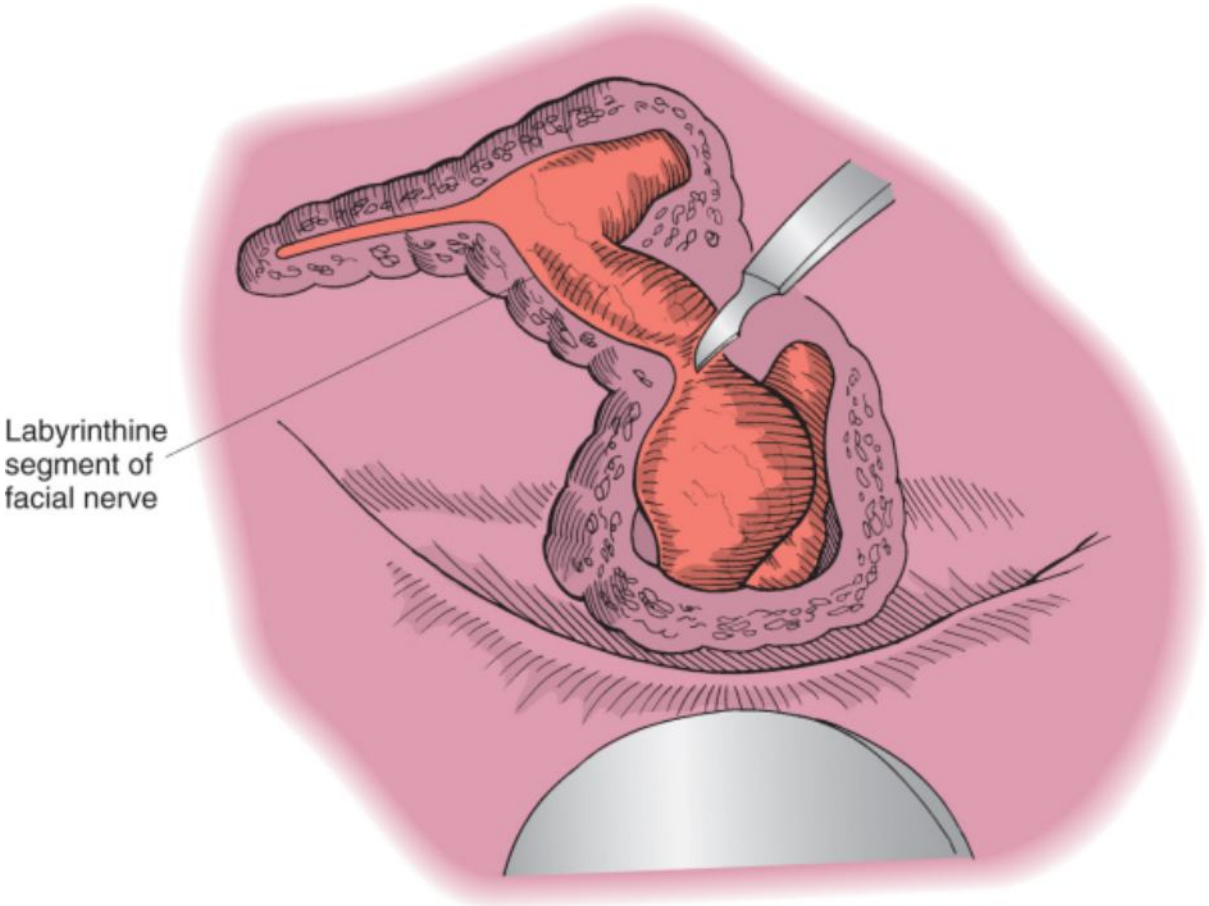

Figure 122-17 The dura of the internal auditory canal and the periosteum and perineurium of the facial nerve are incised.

Before closure, any open mastoid air cells are occluded with bone wax and the resultant epitympanic defect covered with temporalis fascia. The roof of the IAC is sealed with a small abdominal fat graft. A free bone graft is harvested from the temporal craniotomy flap for placement over the fascia to provide additional structural support and prevent herniation of dura/brain into the middle ear. The middle fossa retractor is released and removed to allow the temporal lobe to expand back over the middle fossa floor. The remainder of the craniotomy flap is replaced and secured by reapproximating the overlying temporalis muscle. The skin flap is then reapproximated in two layers without the use of any drain. A mastoid dressing is placed over the operated ear for 3 days postoperatively, and the patient is hospitalized for 4 to 5 days. The patient is monitored postoperatively in routine manner for intracranial surgery, with attention paid to CSF leakage from the wound or nose. Placement of a lumbar drain usually resolves this problem.

Facial Nerve Repair

Restoration of anatomic continuity of the facial nerve without tension is the primary goal of facial nerve repair. In general, the earlier that repair of the injured facial nerve takes place, the better the functional outcome. In patients with neural transection from surgery or trauma, immediate exploration and primary repair are advocated. If not possible, delayed exploration and repair should be performed as soon as medically feasible (within 30 days) for the best functional outcome.[9] The transected ends of the facial nerve should preferably be tagged to facilitate identification of the nerve endings at the time of secondary surgery.

Three anastomotic techniques have been reported in the literature. Epineural nerve repair is the most common anastomosis technique. It involves placing nonabsorbable suture through the epineurium on each side of the anastomosis and reapproximating the ends. The potential disadvantage is that it fails to approximate the individual nerve fascicles. Perineural repair consists of suturing the individual perineurium of each fascicle. The epineurium is sutured as a second layer. The advantage of this technique is that better contact of individual nerve fascicles may be achieved. Fascicular and interfascicular nerve repair involves individually approximating the fascicles. Natural fiber, fibrinogen adhesives, or fine sutures (10-0 or 11-0 nylon) are used to coapt the individual fascicles. To date, no definitive studies have documented the superiority of tubulization or enclosing the anastomosis to prevent tissue invasion and abnormal axon growth.[9] Accurate surgical approximation remains the primary goal. It is important to remember that the working portion of the nerve is the endoneural surface and not the nerve ends. Therefore, epineurium needs to be trimmed back to uncover the endoneural surface for good axonal volume match at the anastomotic sites.

In general, partial or complete transaction of the facial nerve should be repaired by primary neurorrhaphy or cable grafting when the proximal and distal ends of the facial nerve are accessible.

Primary Repair

Primary repair is the ideal surgical option whenever feasible because there will be only one anastomosis and minimal size incongruency of the reapproximated nerve endings. For this reason, some surgeons prefer nerve mobilization and primary anastomosis over other techniques.

Intratemporal rerouting of the distal nerve segment involves a translabyrinthine exposure, sectioning of the GSPN and chorda tympani branches, and complete skeletonization of the facial nerve within the fallopian canal to gain another 1.5 cm in length for tension-free anastomosis. The disadvantage of this approach is that the blood supply to the distal nerve segment may be disrupted and lead to further neural injury. The cut endings of the nerve are freshened by sharply trimming the endings back to normal–appearing nerve tissue. Any excess surrounding soft tissue is trimmed to allow proper identification of the nerve sheath. In delayed repairs, fibrous scarring or traumatic neuromas at the proximal end of the cut nerve should be excised before repair. Suture anastomosis of the nerve is generally unnecessary within the temporal bone. When outside the fallopian canal, one to two 9-0 monofilament sutures are used to reapproximate the epineural layer. At the cerebellopontine angle, the lack of a resilient epineural layer may necessitate a single through–and-through suture to secure the cut ends of the nerve together. The suture is passed through the distal end first to avoid placing tension and traction on the proximal end, which may lead to tearing of the proximal end. After suture repair, the anastomosis may be further reinforced with fibrinogen. Use of nerve tubes and conduits is unnecessary.

Cable Grafting

Cable or interposition grafting involves placing a free nerve graft between the proximal and distal segments of the facial nerve. This procedure provides a conduit for growth of axons to the facial musculature. It is indicated when tension-free primary anastomosis cannot be performed. Disadvantages of this technique are the requirement for two anastomoses and decreased axon availability.

The great auricular nerve is the most widely used donor nerve in the head and neck for most short–segment facial nerve defects because of its close proximity to most surgical fields, excellent size match, and limited donor site

morbidity. Its limitations are its relatively short length (up to 8 cm), restricted branching pattern, and potential for involvement by malignant disease. The great auricular nerve is located midway between the mastoid tip and clavicle along the posterior border of the sternocleidomastoid muscle.

The sural nerve is the second most popular donor nerve for facial nerve reconstruction. It provides excellent size match, is available in lengths of up to 40 cm for grafting, and has an extensive arborization pattern. It is an ideal choice for near–total facial nerve reconstruction after oncologic procedures, as well as for cross–facial grafting. Harvesting of the sural nerve requires a second operative site. The sural nerve is located between the lateral malleolus and Achilles tendon, deep or posterior to the saphenous vein.

Details on the technique of harvesting great auricular and sural nerve grafts are provided in Chapter 121. Neural anastomosis is accomplished in a fashion similar to primary anastomosis. Cable grafts laid within the fallopian canal may be supported with Gelfoam alone without suture approximation (Fig. 122-18).

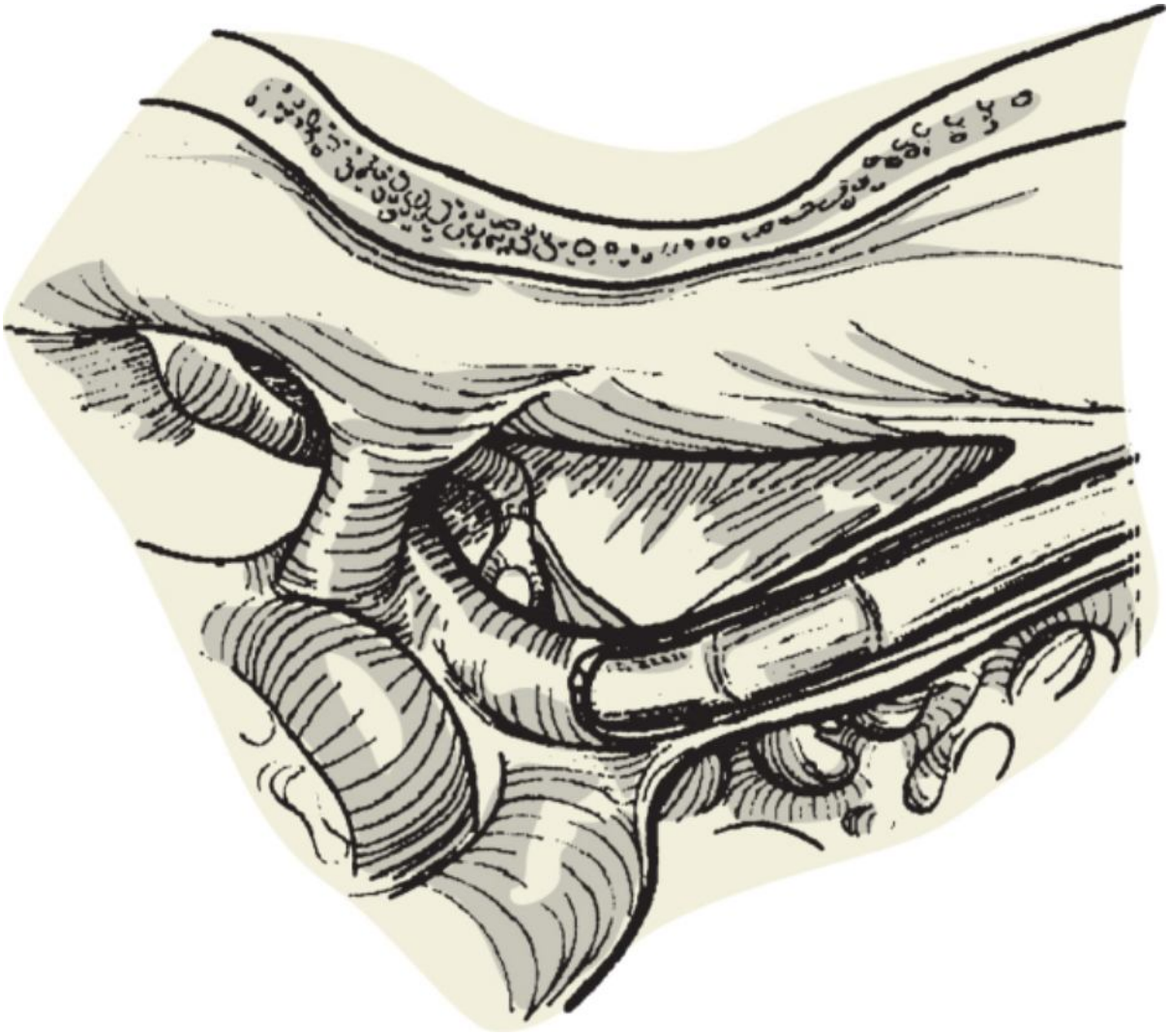

Figure 122-18 Interposition nerve graft laid in the mastoid segment of the fallopian canal.

POSTOPERATIVE MANAGEMENT

Patients undergoing transmastoid exploration of the facial nerve are discharged home on the same day. If a craniotomy is performed, the patient will require hospitalization for several days for close observation. Perioperative antibiotics are routinely administered for all craniotomy cases. The patient is instructed about eye protection, and this is reinforced until good eye closure is restored. Static reanimation of the eye with an upper eyelid gold weight or palpebral spring is recommended at the time of facial nerve repair or grafting in patients requiring neurorrhaphy, because recovery of facial function is not anticipated for 6 to 12 months. Reanimation is also recommended for patients with facial paralysis who lack a normal Bell phenomenon and those with a decreased or absent corneal reflex in the affected eye.

COMPLICATIONS

Potential complications of transmastoid facial nerve decompression include further surgical trauma to the facial nerve, hearing loss (either conductive or sensorineural), vertigo, CSF leak, and wound infection. All drilling along the facial nerve should be performed with a diamond burr and copious irrigation to prevent thermal injury. The inner ear is most at risk at the lateral semicircular canal during decompression of the mastoid segment and second genu of the nerve. The ampullated ends of the superior and lateral canals are at risk during exposure of the perigeniculate region from the transmastoid approach. It is important to recognize inadvertent fenestration of the inner ear as soon as it occurs and to seal the fenestration with bone wax. Sensorineural hearing loss can also occur from inadvertently drilling on the ossicular chain. This injury is prevented by disarticulating and removing the incus if there is not enough room to safely decompress the tympanic segment without touching the incus. Care should be taken to avoid trauma to the middle fossa dura to prevent CSF leakage. To minimize trauma to the dura, it is important to use the properly sized burr, watch all edges of the burr while drilling, and switch to a diamond burr when in tight areas or when the dura has been exposed. If the dura is violated, repair with fascia, muscle, bone, or any combination of these tissues is necessary. Techniques for repair of dural and tegmen defects are described in Chapter 127.

Potential intraoperative and postoperative complications of the middle fossa approach include sensorineural hearing loss, vertigo, edema of the temporal lobe or contusion, subdural hematoma, CSF leak, and meningitis. Sensorineural hearing loss and vertigo result from inadvertent fenestration of the cochlea and vestibular labyrinth during exposure of the labyrinthine facial nerve and IAC. Should fenestration occur, the site should be immediately occluded with bone wax. Temporal lobe edema or contusion (or both) and subdural hematoma may occur as a result of direct injury during craniotomy or temporal lobe retraction. These complications are avoided by taking steps to reduce intracranial pressure at the start of surgery (e.g., reducing Pco_2; administering intravenous mannitol, furosemide, and dexamethasone). If the temporal lobe is tight, a small incision in the dura can be made to release CSF before retracting the temporal lobe. If injury to the temporal lobe is suspected, a CT scan of the head should be performed postoperatively to assess the degree of edema, and a neurosurgeon should be consulted.

Although functional outcome does not seem to be related to the length of the nerve graft, nerve grafts placed distal to the meatal foramen appear to do better. Timing of repair after injury is also a critical factor in determining recovery. The best functional recovery after nerve repair or grafting still results in mild to moderate facial weakness with synkinesis. Facial movement may not be clinically evident until at least 6 months after surgery and may continue to improve for 2 years or more after neurorrhaphy. Therefore, adjuvant facial reanimation procedures (see Chapter 88) may be indicated if poor oral and ocular sphincter control is bothersome.

PEARLS

- Incomplete facial paralysis portends a favorable prognosis and should be managed conservatively.
- ENoG and EMG testing should be used to identify patients who would potentially benefit from surgical decompression of the facial nerve.
- Thorough understanding of temporal bone anatomy and the intratemporal course of the facial nerve is critical in preventing iatrogenic trauma to the nerve.
- Tension-free anastomosis is critical for the best functional results after neurorrhaphy.
- Static facial reanimation procedures should be considered after facial nerve repair if significant oral or ocular sphincter deficiencies are encountered.

PITFALLS

- ENoG results alone may underestimate the extent of recovery in a regenerating nerve and should therefore never be used independently to determine candidacy for surgical decompression.
- Temporal lobe retraction is poorly tolerated in elderly individuals and results in an unacceptably high risk of intracranial complications.
- Inadvertent drilling on the incus or stapes during decompression of the distal tympanic facial nerve will result in postoperative sensorineural hearing loss.
- Thermal injury to the facial nerve may occur if inadequate irrigation is used while decompressing the fallopian canal with a diamond drill burr.
- Mobilization of the facial nerve from the fallopian canal to gain nerve length for primary anastomosis entails a moderate risk of further nerve devascularization and trauma.