**Pediatric Facial Plastic and Reconstructive Surgery**

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# Chapter 21: Facial Nerve Abnormalities

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Facial nerve abnormalities in children represent a broad spectrum of pathologies, including numerous congenital and acquired etiologies. However, the principles of managing facial paralysis in children are essentially the same as those for adults. What follows is a review of the various facial nerve abnormalities encountered by otolaryngologists. In addition, patient selection adn the surgical procedures used in managing specific facial nerve disorders are presented.

## Facial Nerve Abnormalities

### Congenital

### Möbius Syndrome (Congenital Facial Diplegia)

Möbius syndrome is a rare congenital disorder that usually includes bilateral seventh nerve paralysis and unilateral or bilateral sixth nerve paralysis. Since the disorder was described, multiple authors have studied families withthe syndrome (for review see McKusick). It is considered to have an autosomal dominant inheritance pattern with variable expressivity. The inheritance pattern is thought to be no higher than 1 in 50 in families in whom myopathies or other extremity anomalies such as clubfoor, arthogryposis, or digital anomalies are not present.

The etiology of Möbius syndrome is unclear. Neuropathologic studies have noted that the nuclei of cranial nerves VI, VII, and XII are abnormal, with lesser abnormalities being found in the nuclei of cranial nerves III and XI. Other authors have reported that the facial nerves are smaller or absent at autopsy. Alternatively, primary failure of facial muscle development has been proposed as the etiology by Pitner et al, who also noted normal facial nuclei in their postmortem study.

The clinical observation of congenital extraocular muscle paralysis and facial paralysis is the typical presentation of this disorder. No mass lesions will be found on magnetic resonance imaging (MRI). Ophthalmologic consultation and management is mandatory. Reinnervation procedures such as cross-face grafts or hypoglossal-facial nerve anastomosis yield poor results, either due to the paucity of motor end plates or the atrophic seventh nerves. Significant improvements of resting tone and voluntary animation can result from temporalis muscle transposition, which brings in a new neuomuscular system.

### Hemifacial Microsomia

The term *hemifacial microsomia* refers to patients with unilateral microtia, macrostomia, and mandibular hypoplasia. Goldenhar's syndrome (oculoauriculovertebral

dysplasia) is considered to be a variant of this complex and is characterized by vertebral anomalies and epibulbar dermoids. Although approximately 25% of patients with hemifacial microsomia have facial nerve weakness. one patient with Goldenhar's syndrome has been reported to have had aplasia of the facial nerve.

### Osteopetrosis

Osteopetrosis is a generalized dysplasia of bone that may have an autosomal dominant or recessive inheritance pattern. The recessive form is more rapidly progressive and causes hepatosplenomegaly and severe neural atrophy secondary to bony overgrowth at neural foramina. Optic atrophy, facial paralysis, sensorineural hearing loss, and mental retardation are common in the recessive form, and death usually occurs by the second decade. However, in these severe cases of osteopetrosis, which were previously fatal, bone marrow transplantation has been reported to be of value.

The dominant form causes progressive enlargements of the cranium and mandible and clubbing of the long bones. Increased bone density is seen radiographically. Progressive optic atrophy, trigeminal hypoesthesia, recurrent facial paralysis, and sensorineural hearing loss are common. Complete decompression of the intratemporal facial nerve should be performed in patients with recurrent facial paralysis and radiographic evidence of osteopetrosis.

## Acquired

### Trauma

Approximately 90% of all congenital peripheral facial nerve paralysis improves spontaneously and most can be attributed to difficult deliveries, cephalopelvic disproportion, high forceps delivery, or intrauterine trauma. These types of congenital facial paralysis are often unilateral and partial, especially involving the lower division of the facial nerve. Since these etiologies involve extratemporal compression, surgical exploration or bony decompression are not indicated.

Blunt trauma resulting in temporal bone fracture is an unusual cause of facial paralysis in children. The lower incidence of skull and temporal bone fracture in children is due to adults sustaining head injuries more frequently and the increased skull compliance in children. These fractures are best evaluated with high-resolution temporal bone computed tomography (CT) scans.

Temporal and parietal blows to the head may occur anywhere along a coronal arc from the vertex to the cranial base. When the vector of force is directed toward the base, it classically passes toward the external auditory canal, deflects off the otic capsule, and extends anteromedially along the anterior edge of the petrous bone to the foramen lacerum and foramen ovale. The resulting fracture is described as a longitudinal temporal bone fracture. This is the most common type of temporal bone fracture (approximately 90%) and is also the most common type of fracture associated with facial nerve injury. The geniculate ganglion region of facial nerve is most frequently injured. The indications for facial nerve decompression and exploration are the same as those discussed in detail under the Bell's palsy section of this chapter.

Frontal and particularly occipital blows to the head tend to result in transverse fractures of the temporal bone. More severe head injury is usually required to cause these fractures. Since they often extend through the internal auditory canal or across the otic capsule, hearing loss and vertigo commonly result. Although only 10% to 20% of temporal bone fractures are transverse in orientation, they cause facial nerve injury in approximately 50% of patients. The anatomic region of the facial nerve most commonly injured is the labyrinthine segment.

Penetrating injuries to the extratemporal facial nerve should be explored urgently in order to facilitate identification of the transected distal branches using a facial nerve stimulator. If primary repair is not possible, the principles of facial nerve repair using cable grafts described later in this chapter should be followed. In infected wounds, urgent exploration and tagging of identified distal branches should precede control of the infection and granulation. Subsequent repair usually requires the use of cable grafts.

The risk of otologic surgical injury of the facial nerve is particularly high in children with congenital ear malformations. The discussion of specific malformations and the relative risk to the facial nerve is beyond the scope of this chapter. An additional group at higher risk for injury to the facial nerve are newborns who are undergoing mastoid surgery since the mastoid tip has not become pneumoatized and the facial nerve exits the stylomastoid foramen laterally. In these children a semihorizontal, curvilinear incision should be used and as is the case with all otologic surgery, a facial nerve monitoring system should be used.

## Infection

### Bacterial

Facial paralysis as a complication of otitis media has become a rare disease in children due to the ready access to medical care and antibiotics. Takahashi et al reviewed their series of over 1.600 patients with facial paralysis and found that only 11 of these patients were younger than 20 years old and had facial paralysis due to otitis media (0.69%). They described the facial paralysis in this group of patients as having a slower progression and less complete paralysis than that seen in Bell's palsy. Temporal bone CT should be performed in all patients in order to eliminate the diagnosis of coalescent mastoiditis. Intravenous antibiotics in combination with myringotomy and tympanostomy tube placement remain our initial management algorithm for bacterial otitis media complicated by facial paralysis. Bacterial cultures should always be obtained at the time of myringotomy, and antibiotic selection should be tailored to the culture results.

Facial paralysis complicating mastoiditis or cholesteatoma is also rare. In Sheehy's series of over 180 children undergoing surgery for cholesteatoma, only one patient (0.6%) had facial nerve weakness. The surgical management of these patients includes mastoidectomy, excision of the cholesteatoma, and appropriate antibiotic therapy.

Infection with the spirochete *Borrelia burgdorferi* (Lyme disease) can result in facial paralysis. This tick-borne infection is endemic to the Northeast, and is named for the town of Lyme, Connecticut. Widespread infections have been reported from the West Coast, Midwest, and East Coast as well as throughout Europe and Australia. As is the case with

other spirochete infections, the clinical manifestations of Lyme disease are protean. Facial diplegia has been reported in Lyme disease and should be considered in children presenting with facial paralysis. Serologic diagnosis should be followed by antibiotic therapy. Tetracycline is considered to be the agent of choice; however, erythromycin and penicillin have been successfully used.

## Viral

Herpes zoster oticus (Ramsay Hunt syndrome) is the etiology of facial paralysis in 3% to 12% of adults and approximately 5% of children reported in various series. It is characterized by severe pain and small, clear vesicles in the external ear canal and pinna, and rapidly progressive facial paralysis. Cranial polyneuropathy is frequently seen, particularly in the vestibulocochlear nerve with resulting auditory and vestibular dysfunction. Involvement of cranial nerves V, IX, X, XI, and XII is much less common. Although surgical managament using the middle cranial fossa approach for facial nerve decompression in herpes zoster oticus has been recommended, we currently do not advocate this specific surgical therapy since the facial nerve is more diffusely involved in this disorder than in Bell's palsy. In adults we routinely use intravenous acyclovir in the management of acute herpes zoster oticus; however, due to the mechanism of action of this drug, ie, DNA incorporation of acyclovir triphosphate and subsequent termination of DNA synthesis, its use in children is controversial. If a child is immunocompromised and has herpes zoster oticus, particularly with cranial polyneuropathies, we would, however, administer this medication in consultation with out pediatric colleagues. Other viral infections such as primary chicken pox, mononucleosis, mumps, and poliomyelitis can result in facial paralysis that may or may not resolve spontaneously. For these specific viral infections, immunization when available, is the most effective preventative measure, and supportive care is required during the active infection. Facial reanimation procedures are sometimes required after adequate follow-up suggests that spontaneous recovery will not occur.

## Benign or Malignant Neoplasms

Tumor involvemengt of the facial nerve should be considered in children with facial paralysis if one or more of the following clinical features are present: facial paralysis that progresses slowly over 3 weeks, recurrent facial paralysis involving the same side, facial weakness associated with twitching, long-standing facial paralysis (greater than 6 months), facial paralysis associated with other cranial nerve deficits and/or deficits referable to the brain stem, or evidence of malignancy elsewhere in the body. Gadolinium-enhanced MRI is extremely useful in imaging solid tumors involving the facial nerve, and high-resolution CT scans are useful in identifying bony erosion of the fallopian canal. In May's series of 280 patients ranging in age from newborn to 18 years, he reported 15 patients (5.4%) with tumors causing facial paralysis. This group of benign and malignant tumors included the following diagnoses and number of patients: leukemia (3), schwannoma (3), meningioma (2), capillary hemangioma (1), granular cell myoblastoma (1), brain stem glioma (1), mesenchymal mass (1), congenital cholesteatoma (1), sphenoid giant cell tumor (1), and neurilemmoma (1). Rhabdomyosarcoma is one of the most common neoplasms in children, accounting for 10% to 15% of all childhood neoplasms, and therefore should be considered in children presenting with unilateral facial paralysis. If the underlying disorder requires surgical resection, the resulting gap in the facial nerve should be reconstructed at the time of resection with a greater

auricular nerve or sural nerve graft, as described later in this chapter. If the management of the disorder involves chemotherapy rather than surgical intervention, facial reanimation procedures as described by May are indicated if the child has persistent facial nerve dysfunction.

## Miscellaneous Disorders

### Guillain-Barré Syndrome

The onset of simultaneous bilateral facial paralysis suggests Guillain-Barré syndrome, sarcoidosis, sickle-cell disease, or some other systemic disorder. Guillain-Barré syndrome is a relatively common neurologic disorder affecting children and is an acute inflammatory polyradiculoneuropathy that progresses to varying degrees of paralysis. The etiology remains unknown; however, autoimmune or viral mechanisms have been considered. Classical histopathologic features of the syndrome include a lymphocytic cellular infiltration of peripheral nerves and destruction of myelin. The facial paralysis seen in these children is typically bilateral in nature and often resolves spontaneously after a prolonged course of paralysis. Although there is no role for surgical decompression of the facial nerve in this disorder, reanimation is only considered late in the course of the disease.

### Melkersson-Rosenthal Syndrome

Merkersson-Rosenthal syndrome is a neuromucocutaneous disease with a classic triad of recurrent facial (labial) edema and recurrent facial paralysis associated with a fissured tongue. Patients with Melkersson-Rosenthal syndrome may not present with the complete triad, and although facial paralysis is the best-known neurologic symptom, it is not mandatory for the diagnosis. Headache, granular cheilitis, trigeminal neuralgiform attacks, dysphagia, laryngospasm, and a variety of cranial nerve and cervical autonomic dysfunction may also occur. The patient with Melkersson-Rosenthal syndrome may present at any age with any variety of classic and associated features that may wax and wane. Approximately one-third of the patients have recurrent facial paralysis as part of their syndrome. The underlying etiologic factor has been thought to be a neurotropic edema causing compression and paralysis of the facial nerve as it passes through the fallopian canal. Since the anatomically most constricted area of the fallopian canal is the meatal foramen and because most prior reports observed recurrence after transmastoid decompression, Graham and Kemink elected to decompress the proximal segment in addition to the mastoid segment of the facial nerve in all such cases by performing a combined transmastoid and midle cranial fossa facial nerve decompression and neurolysis of the nerve sheath. The preliminary data presented by Graham and Kemink suggest that edematous involvement of the facial nerve in recurrent facial paralysis does occur intratemporally and that the recurrent paralysis can be prevented by transmastoid and middle cranial fossa total facial nerve decompression with neurolysis of the facial nerve sheath. Recurrent paralysis over a prolonged period of time usually results in increasing residual dysfunction. If evidence of residual paresis exists, facial nerve decompression of the labyrinthine segment and geniculate ganglion through a middle cranial fossa exposure is recommended at the time of the next episode of paralysis.

## Bell's Palsy

As is the case in adults, Bell's palsy is the most common diagnosis in cases of facial paralysis in children. Spontaneous recovery of normal function of the facial nerve has been reported to be 90% in children, as compared to 71% in the adult population. However, the incidence of Bell's palsy has been reported to be greater in adults (for review see Jenkins et al). It must be noted that Bell's palsy is a diagnosis of exclusion and represents loss of facial nerve function without a proven etiology.

Gadolinium-enhanced MRI has been advocated as a diagnostic tool in assessing facial paralysis. Gadolinium enhancement of the normal facial nerve does not occur. Therefore, enhancement of this structure would be due to increased extracellular fluid from edema, inflammation, or neoplasm. Our observations with gadolinium-enhanced MRI, as well as those of others, are supportive of Fisch's hypothesis of axoplasmic damming at the meatal segment with subsequent edema and nerve conduction impairment. However, a recent study demonstrated that there was no prognostic significance of gadolinium enhancement of the facial nerve on MRI in patients with Bell's palsy. Therefore, gadolinium-enhanced MRI is not indicated in every child with facial paralysis. In patients suspected of having a tumor from clinical or electrodiagnostic data, gadolinium-enhanced MRI along with a high-resolution CT of the internal auditory canal, fallopian canal, skull base, and parotid should be performed.

In addition to the MRI evidence of axoplasmic damming at the meatal foramen, Jackson et al published histologic data that also support this entrapment hypothesis of Fisch. In their case report, they presented the pathologic observation that there was a sharp demarcation between sclerotic nerve proximal to and necrotic nerve distal to the meatal foramen within the fallopian canal. A morphometric temporal bone comparative study of the fallopian canal at the meatal foramen in children and adults was published by Eicher et al. They found that the nerve diameter to canal diameter ratio at the meatal foramen was statistically significantly smaller in children. These findings that the facial nerve is not as tightly contained at the meatal foramen in children may explain the higher rate of spontaneous recovery in children with Bell's palsy.

Electroneurography (ENoG) provides a quantitative assessment of facial nerve function and allows a relative comparison between the normal and affected sides. Electroneurography is widely used in adults, and inour experience, as well as that of others, ENoG is equally useful in the evaluation of facial paralysis in children. Our criteria for surgical decompression include ENoG degeneration greater than 90% relative to the unaffected side and the operation within 14 days of onset. The figure illustrates the outcome of a child who was followed for 10 days with serial ENoGs that showed progressive degeneration, until she reached 100% degeneration preoperatively. Her complete facial paralysis resolved by 2 months postoperatively when her facial nerve function was assessed as a House-Brackmann grade 1.

## Surgical Anatomy of the Facial Nerve

Detailed knowledge and familiarity with the complex course of the facial nerve and its anatomic relationship to other vital structures are essential to the surgeon who plans to operate in this area. The facial nerve (cranial nerve (CN) VII) exits the brainstem at the pontomedullary junction approximately 1.5 mm anterior to the vestibulocochlear nerve (CN

VIII). The facial nerve is smaller in diameter (approximately 1.8 mmm) than the oval CN VIII (approximately 3 mm in the largest diameter). A third smaller nerve, the nervus intermedius, emerges between CN VII and CN VIII and eventually becomes incorporated within the sheath of CN VII. After leaving the brain stem, CN VII follows a rostrolateral course through the cerebellopontine cistern for 15 to 17 mm, entering the porus of the internal auditory canal (IAC) of the temporal bone. Other important structures in the cerebellopontine cistern include the anterior inferior cerebellar artery (AICA) and the veins of the middle cerebellar peduncle. The AICA passes near or between CN VII and CN VIII; the veins are more variable in position and number. On enteringthe IAC the facial nerve occupies the anterosuperior quadrant of this channel for 8 to 10 mm. Then it enters the fallopian canal at the fundus of the IAC. The IAC is anterior to the plane of the superior semicircular canal. Superiorly, the bone overlying the IAC is within a 60° angle, whose vertex is the superior semicircular canal ampulla. At the entrance of the fallopian canal (meatal foramen) CN VII narrows to its smallest diameter, 0.61 to 0.68 mm. Only the pia and arachnoid membranes form a sheath around the nerve at this point, since the dural investment terminates at the fundus of the IAC. Many authors believe that the small diameter of the meatal foramen is an important factor contributing to the etiology of facial paralysis in certain diseases such as Bell's palsy and Ramsay Hunt syndrome.

The intratemporal course of the facial nerve has three distinct anatomic regions: the labyrinthine, tympanic, and mastoid segments. The labyrinthine segment is shortest (approximately 4 mm), extending from the meatal foramen to the geniculate ganglion. This segment travels anterior, superior, and lateral, forming an anterior medial angle of 120° with the IAC portion. The basal turn of the cochlea is closely related to the fallopian canal and lies anteroinferior to the labyrinthine segment of the facial nerve. At the lateral end of the labyrinthine segment the geniculate ganglion is found and the nerve makes an abrupt posterior change in direction, forming an acute angle of approximately 75°. Anterior to the geniculate ganglion the greater superficial petrosal nerve exists the temporal bone through the hiatus of the facial canal. The hiatus of the facial canal is quite variable in its distance from the geniculate ganglion. The hiatus of the facial canal also contains the vascular supply to the geniculate ganglion region. The tympanic, or horizontal, segment of the nerve is approximately 11 mm long, running between the lateral semicircular canal (LSC) superiorly and the stapes inferiorly, forming the superior margin of the fossa ovalis. Between the tympanic and mastoid segments, the nerve gently curves inferiorly for about 2 to 3 mm. The mastoid, or vertical, segment is the longest intratemporal portion of the nerve, measuring approximately 13 mm. As the nerve exits the stylomastoid foramen at the anterior margin of the digastric groove, an adherent fibrous sheath of dense vascularized connective tissue surrounds it. The stylomastoid artery and veins are within this dense sheath.

## General Principles in Facial Nerve Surgery

Whenever the facial nerve is to be surgically exposed, several technical points must be observed. First, a system for monitoring facial nerve function during the operation should be employed. One of the simplest monitoring methods is visual observation during critical stages of the operation. Needle electromyography can also be used if the equipment is available. No matter which monitoring technique is used, it is necessary that the side of the face in which the nerve is to be exposed be draped in a manner that allows visual observation.

The forehead, eye, mouth, and chin should be visible. The endotracheal tube should be secured to the opposite side without placing tape on the side of the mouth to be observed. Towels drape the posterior, superior, and inferior margins; a fourth towel is placed along the anterior profile. An abdominal clear, noniodinated plastic adhesive drape is placed over the face and operative area. An observer is thus able to see the entire face during the procedure and determine if any of the muscles move in response to surgical manipulation or electrical stimulation of the nerve. The scrub nurse or circulating nurse has been found to be the best observer if the surgeon asks for observation during critical periods of the procedure. Electromyographic needle electrodes can be placed in the orbicularis oculi and oris muscles and attached to electromyographic equipment for auditory feedback of neural activity throughout the surgical procedure.

Instrumentation is crucial to a successful exposure of the facial nerve. The largest diamond burr that the operative site can safely accommodate should be used when the surgeon is near the fallopian canal. Cutting burrs have a tendency to catch and jump unexpectedly and can cause severe injury to the nerve. Continuous suction-irrigation keeps the burrs clean and also dissipates heat, which can induce neural damage.

The final layer of bone over the nerve should be removed by blunt elevators specally designed for this purpose, such as the Fisch raspatory. These instruments are thin but strong enough to remove a thin layer of bone. Stapes curettes are usually too large and can cause compression injury to the nerve. If a neurolysis is to be performed, disposable microblades are available (Beaver no 59-10). Sharp dissection is less traumatic than blunt elevation when the nerve must be lifted out of the fallopian canal. The medial surface of the nerve usually adheres to the bone and contains a rich vascular supply. Cauterization near the nerve should be performed only with bipolar electrocautery and insulated microforceps.

## Middle Cranial Fossa (Transtemporal) Approach: IAC Porus to Tympanic Segment

The middle cranial fossa exposure is used to expose the IAC and labyrinthine segments of the facial nerve when preserving existing auditory function is desirable. The geniculate ganglion and tympanic portions of the nerve can also be decompressed from this approach.

### Technique

The patient is placed supine on the operating table with the head turned so that the involved temporal bone is upward. The hair is shaved 6 to 8 cm above and anterior to the ear and 2 cm posterior to it. The surgeon is seated at the head of the table with the instrument nurse at the anterior side of the patient's head. A 6x8 cm posteriorly based trapdoor incision is marked in the hairline above the ear. If exposure of the mastoid is necessary, the inferior limb of the incision can be carried postauricularly. The skin flap is elevated to expose the temporalis muscle and fascia. A 4x4 cm temporalis fascia graft is harvested for use during closure of the IAC dural defect. An anteriorly based trapdoor incision is used to elevate the temporalis muscle and periosteum. Staggering the levels of the muscle and skin incisions provides for a double-layer, watertight closure at the completion of the procedure.

The temporal root of the zygoma is exposed during elevation of the temporalis muscle. This landmark represents the level of the floor of the middle fossa. Dural fishhooks are placed in the skin and temporalis muscle flaps for retraction. A 3x5 cm bone flap for facial nerve decompression, or 4x5 cm bone flap for tumor excisions, centered above the temporal root of the zygoma, is fashioned with a medium cutting burr. It is important to keep the anterior and posterior margins of the craniotomy parallel to facilitate placement of the self-retaining retractor.

Branches of the middle meningeal artery are occasionally embedded within the inner table of the skull; therefore, elevation of the bone flap must be performed in a controlled manner. Bipolar coagulation and bone wax may be necessary to control bleeding. Elevation of the dura from the floor of the middle fossa can be one of the most difficult steps. Blunt dissection and magnification greatly facilitate dural elevation. The dura is elevated from the posterior to anterior direction to prevent accidental injury to an exposed geniculate ganglion and greater superficial petrosal nerve. Bipolar coagulation is used to cauterize dural reflections within the petrosquamous suture before transection with a scissors.

The elevation proceeds until the petrous ridge is identified medially and the arcuate eminence, meatal plane, and greater superficial petrosal nerve are exposed anteriorly. No attempt is made to identify the middle meningeal artery and accompanying troublesome bleeding veins. The tip of a self-retaining (House-Urban) retractor is placed at the petrous ridge anterior to the arcuate eminence and medial to the meatal plane. A medium diamond burr and a suction-irrigation apparatus are used to identify the blue line of the superior semicircular canal (SSC). A preoperative Stenvers' projection radiograph helps to determine the level of the SSC in relation to the floor of the middle fossa and the degree of pneumatization above the SSC.

Drilling begins posterior to the arcuate eminence over the mastoid air cells until the dense yellow bone of the otic capsule is identified. Otic capsule bone is slowly removed until the blue outline of the SSC is seen. The IAC is located by removing bone with a 60° angle anterior to the blue line of the SSC, and with the vertex based at the SSC ampulla. This dissection is continued until approximately 180° of the IAC are exposed for facial nerve decompressions, or 270° of the IAC are exposed for schwannomas. Because of the close proximity of the SSC and the basal turn of the cochlea, only approximately 120° of the circumference of the IAC can be safely removed in its lateral 5 mm or so. The facial nerve occupies the anterosuperior portion of the IAC. Laterally the vertical crest (Bill's bar) marks the division between the superior vestibular nerve and the meatal foramen containing the facial nerve.

The entrance to the fallopian canal is the narrowest, most delicate portion of the facial nerve and consequently the most challenging portion of the dissection. At the meatal foramen the facial nerve turns anterior and slightly superior. The basal turn of the cochlea can be within 1 mm inferiorly, and the ampulla of the SSC can be directly posterior to the nerve. The labyrinthine segment is followed to the geniculate ganglion. If the facial nerve needs to be exposed distal to the geniculate ganglion (eg, as with facial neuromas or with some traumatic injuries to the facial nerve) the tegmen tympani is removed with care to avoid injury to the head of the malleus and incus. The tympanic segment is easily seen to turn

abruptly posterior; it is followed to where it coursed inferior to the lateral semicircular canal (LSC). It is advisable to leave a thin shell of bone covering the nerve until its entire course is identified. The final layer of bone is removed by small blunt elevators. The nerve is tightly confined within the labyrinthine segment of the fallopian canal; larger curettes should be avoided to prevent compression injury. If the nerve is to be decompressed, a neurolysis is the final step. A disposable microscalpel (Beaver no 59-10) is used to slit the periosteum and epineural sheath.

Alternative methods to locate the facial nerve may be necessary, especially in traumatic cases. The greater superficial petrosal nerve can be traced posteriorly to the geniculate ganglion, or the tegmen tympani may be fractured and the tympanic segment visible through the fracture. The tympanic segment is then used to locate the geniculate ganglion and labyrinthine segments.

At the end of the procedure a free temporalis muscle graft is placed within the IAC and a corner piece of the bone flap is fashioned to cover the defects in the tegmen tympani and IAC. This prevents herniation of the temporal lobe into the middle ear or IAC. The temporalis fascia previously harvested is placed over the free bone graft to help seal the dural defect at the IAC. The squamosal craniotomy bone flap is replaced, and the temporalis muscle is closed with interrupted absorbable sutures. The skin is closed in layers with particular care in closing the galea. No drain is placed. A mastoid-type pressure dressing is applied.

## Advantages and Uses

The middle cranial fossa route is the only method that can be used to expose the entire IAC and labyrinthine segment with preservation of hearing. This, in combination with the retrolabyrinthine and transmastoid approaches, enables visualization of the entire course of the facial nerve and still preserves function of the inner ear. The middle cranial fossa technique is the most commonly used for decompression of the facial nerve in Bell's palsy, herpes zoster oticus, and longitudinal temporal bone fractures. However, as described earlier in this chapter, this approach may be useful in the management of patients with schwannoma of CN VII or CN VIII, as well as with patients with Melkersson-Rosenthal syndrome.

## Postoperative Care - Complications and Their Treatment

The anatomy of the floor of the middle cranial fossa is quite variable and presents some difficulty in identification of landmarks. The Stenvers' projection radiograph provides important anatomic information regarding the degree of pneumatization above the superior semicircular canal, and should be performed in all cases to minimize the risk of surgical injury to the SSC. In addition, the surgeon must have a precise knowledge of three-dimensional anatomy of the temporal bone. Many hours in a temporal bone dissection laboratory are required to attain the delicate microsurgical skills that are necessary for this type of surgery.

Conductive and sensorineural hearing losses can both result from middle cranial fossa facial nerve decompression. Conductive hearing loss can be secondary to temporal lobe herniation or ossicular disruption during dissection in the attic. A free bone graft, as already described, prevents temporal lobe herniation. Sensorineural hearing loss can result from direct

injury to the inner ear by the drill exposing the cochlea or semicircular canals or from translational injury by the drill striking an ossicle. Should the SSC be entered during the surgical dissection, the fenestration should be immediately occluded with bone wax. Injury to the internal auditory vessels within the IAC can also result in loss of inner ear function. Loss of vestibular function can occur by the same mechanisms.

Postoperative intracranial complications including meningitis, temporal lobe edema, and epidural hematoma formation are possible. Perioperative antibiotics administered over 48 hr are recommended. Fluid restriction and Decadron are used for the first 3 days postoperatively in order to minimize temporal lobe edema following intraoperative retraction. In addition, our longer craniotomy flap decreases the amount of temporal lobe retraction required for complete exposure of the internal auditory canal and fallopian canal. With adequate intraoperative hemostasis using the bipolar cautery, Oxycel, and dural tacking sutures, we have never had a clinically significant postoperative epidural hematoma develop.

Leakage of cerebrospinal fluid (CSF) must be avoided in order to prevent meningitis. All exposed mastoid air cells must be obstructed with bone wax. A temporalis muscle free graft is placed into the superior aspect of the IAC to separate the posterior fossa from the extradural floor of the middle cranial fossa. Temporalis fascia is then used to provide a second layer of closure between the posterior fossa and the extradural middle fossa. Meticulous care must be taken to assure that there are no dural dehiscences overlying the temporal lobe through which CSF may drain. If these are identified, a temporalis fascia or muscle patch must be used to repair the dural tears to prevent CSF leaks. After a three-layer watertight closure of the temporalis muscle, galea, and scalp, a mastoid-type dressing is applied daily for 5 days postoperatively. Should CSF leakage persist, a temporary lumbar fluid drain is placed and the patient is kept on bed rest. If the CSF leakage does not resolve within 7 days after placement of the lumbar drain, reexploration of the surgical field is indicated to identify and seal the area of CSF egress.

Uncontrolled bleeding or injury to the AICA poses the most serious complication during the operation. The middle cranial fossa approach does not provide adequate access to the entire cerebellopontine angle. The AICA and accompanying veins can loop into the IAC. Control of bleeding of these vessels may require a suboccipital exposure. Injury to the AICA results in brain stem and cerebellar infarction of a variable degree, depending on its size and the area of its terminal arterial supply.

## Nerve Repair

Whenever the continuity of the facial nerve has been disrupted by traumatic injury, iatrogenic injury, or tumor invasion, every effort should be made to restore its continuity. In some instances an end-to-end reapproximation can be accomplished, but if any tension occurs at the anastomotic site, an interposition nerve graft has a better chance of providing facial movement. All nerve repair techniques produce synkinesis, but sphincteric function of the mouth and eye are usually restored. Newer microsuture techniques and instrumentation should be employed to enhance return of function.

In general the injured ends of the nerve should be freshened at a 45° angle. Experimental evidence has shown that cutting the nerve at this angle exposes more neural

tubules and improves regrowth of the nerve. In addition, a fresh razor blade induces less crush to the nerve than a scalpel blade or scissors does. We have found that the perineurium of CN VII does not hold 10-0 sutures, and attempting to suture it increases trauma to the neural tubules. Removing a portion of the epineurium before suturing prevents connective tissue growth at the anastomotic site. If the epineurium is cleaned from the end of the nerve for approximately only 0.5 mm, sutures can still be placed in the epineurium for reapproximating the nerve segments. Three or four sutures of 10-0 nylon are placed with jeweler's forceps or longer instruments (19 cm microforceps) for anastomosis in the cerebellopontine angle. At the brain stem two or three sutures are placed.

When an interposition graft is required, the greater auricular nerve and sural nerve are the preferred graft donor sites. The greater auricular nerve is readily available near the operative field if it is not involved in resection of a neoplasm, and has approximately the same diameter as that of the facial nerve. It is easily located midway, perpendicular to a line drawn between the mastoid tip and the angle of the mandible. If a graft of greater than 8-10 cm is required, the sural nerve should be used. The sural nerve has another advantage, in that the peripheral portion of the nerve has many branches that can be used to reconstruct the branching pattern of the facial nerve. There is little discomfort from removing the sural nerve, since it provides only a small area of sensation to the lateral lower leg and foot. The sural nerve is found immediately posterior to the lateral malleolus, along the saphenous vein. The nerve graft should be 10% to 20% larger in diameter than the facial nerve and long enough to ensure a tension-free anastomosis.