

Section 14 – FACIAL NERVE

Chapter 121 – Tumors of the Facial Nerve

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Primary tumors of the facial nerve are relatively rare. Bell's palsy is the most common disorder that primarily affects the facial nerve. Facial paralysis frequently results from other pathologic processes affecting the posterior fossa, temporal bone, parotid gland, and infratemporal fossa. Secondary involvement of the facial nerve can occur as a result of trauma or erosive tumor such as cholesteatoma, primary squamous cell carcinoma, glomus tumors, acoustic neuroma, meningioma, malignant neoplasms of the parotid gland, and tumors metastatic to the temporal bone.

The most common primary tumors of the facial nerve are neuromas and hemangiomas. Other less common primary tumors of the facial nerve include neurofibroma, granular cell tumor, meningioma, and primary glomus tumors. Malignant epithelioid cranial nerve sheath tumor involving the facial and trigeminal nerves has also been described,^[1] but most malignant tumors of the facial nerve are the result of metastasis or perineural invasion. Cancer of the parotid gland, such as adenoid cystic carcinoma and mucoepidermoid carcinoma, is known to invade the facial nerve at the skull base.^[2] Breast, lung, and renal cancers can also metastasize to the facial nerve, most often in the internal auditory canal (IAC).^[3] Although primary tumors of the facial nerve are relatively rare, the admonition of Sir Terence Cawthorne, "All that palsies is not Bell's," must be considered when slowly progressive facial paresis develops.^[4] This chapter focuses on facial nerve neuroma and hemangioma.

The nomenclature for neurogenic tumors of the facial nerve includes the terms *neurinoma*, *schwannoma*, and *neurilemmoma*, although *neuroma* is used in this chapter to describe this tumor. Neuromas arise from Schwann cells, the myelin-producing cells peripheral to the axon, and are usually located eccentrically and compressing the nerve. The histopathology may follow one of two patterns. The first is that of a dense array of cells in a regular pattern (Antoni A), and the second demonstrates areas of loose stroma and vacuolation (Antoni B) (Fig. 121-1). Often there is a mixture of both patterns, although neither suggests any prognostic implications. This is in contrast to neurofibromas, which arise from endoneurial connective tissue. These tumors are more typically associated with neurofibromatosis 1, or von Recklinghausen's disease.

Neuroma of the facial nerve is considered to be a slowly growing tumor. It appears grossly as a diffuse bulge of the facial nerve but characteristically involves multiple segments along its course. A neuroma may arise anywhere along the course of the facial nerve from the root entry zone in the cerebellopontine angle (CPA) to the extratemporal trunk of the nerve within the parotid gland. Figure 121-2 combines the results of two publications reviewing 287 cases of facial nerve neuroma.^[5,6] The tympanic segment is most commonly involved, followed by the geniculate/labyrinthine and vertical segments.

It is reported that up to 50% of patients with primary neuroma do not present with signs or symptoms of facial nerve dysfunction.^[7] The structures of the IAC, otic capsule, and middle ear are subject to encroachment and erosion by an expanding tumor of the facial nerve. Common initial symptoms, other than facial nerve paresis, include hearing loss, tinnitus, and vertigo or dysequilibrium.

The existence of neuroma of the facial nerve isolated to the porus of the IAC and CPA has been questioned. Published reports of such tumors have provided insight into radiologic findings that preoperatively may differentiate an acoustic neuroma from a facial nerve neuroma. Acoustic neuromas are usually centered on the IAC. Demonstrating the bulk of tumor to be eccentric to the axis of the IAC raises suspicion of facial nerve neuroma.^[7] Facial nerve neuroma limited to the IAC or fallopian canal may not demonstrate bony expansion on computed tomography (CT). Contrast-enhanced magnetic resonance imaging (MRI) is more sensitive in revealing tumors of the facial nerve isolated to the IAC.

Hemangioma is considered to be a vascular hamartoma composed of blood vessels. The blood supply to the facial nerve originates from three separate sources, thus creating a vascular arcade. The intracranial and intrameatal segments of the facial nerve are vascularized by the internal auditory artery, which originates from the anterior inferior cerebellar artery. The geniculate ganglion area is supplied by the petrosal artery, which follows the course of the greater superficial petrosal nerve. The petrosal artery is a branch of the middle meningeal artery. The third

blood supply to the vertical and horizontal facial nerve segments is from the stylomastoid artery. This source has its origin from the posterior auricular or occipital arteries. These vessels create a prominent watershed vascular plexus over the geniculate ganglion area.

Depending on the size of the vessel involved, hemangiomas of the facial nerve are categorized as *capillary* or *cavernous* hemangioma. Similar to neuromas, this histologic subclassification does not have prognostic implications. Hemangiomas of the facial nerve predominantly occur at the geniculate ganglion and the IAC. They rarely originate more distally along the horizontal or vertical segments of the facial nerve. Hemangiomas usually arise eccentrically from the nerve and initiate remodeling of the surrounding trabeculated bone, on which they create spicules and centers of ossification. Because of their extrinsic origin, it was believed that hemangiomas could be dissected from the underlying nerve more easily than is the case with schwannomas. However, recent publications suggest that hemangiomas of the geniculate ganglion often infiltrate the facial nerve, thus requiring sacrifice of the nerve with cable graft repair for complete resection.^[8]

The initial symptoms of hemangioma of the facial nerve are dependent on the location. In contrast to neuroma, hemangiomas of the facial nerve typically manifest signs of facial nerve paresis or paralysis unless they are isolated to the IAC. In this location, they frequently cause progressive sensorineural hearing loss with reduced word discrimination, similar to an acoustic neuroma. In contrast, hemangiomas of the geniculate ganglion predominantly produce slowly progressive facial paralysis occurring over a period of months to years. Facial nerve hemangioma is also an uncommon cause of atypical hemifacial spasm.^[9,10] The degree of facial paresis seen with hemangioma is considered to be more severe than that occurring with a neuroma of the same size. The gross appearance of hemangioma of the facial nerve is similar to that of a red sponge. Small tumors may cause severe symptoms and, until recently, defied easy detection.

Radiographic imaging plays an important role in the differential diagnosis of these tumors. Similar to facial neuroma, identification of hemangioma of the facial nerve requires high-resolution bone-windowed CT and MRI focused along the course of the facial nerve. Recent reports suggest that gadolinium-enhanced MRI has greater sensitivity than CT in detecting and differentiating hemangiomas from other tumors of the geniculate ganglion area.^[11] The CT findings may demonstrate irregular, indistinct bone margins containing bone spicules within the tumor. This honeycomb appearance has been termed *ossifying hemangioma*.^[12] It must be emphasized that the patient's history and diagnostic imaging studies provide the critical information necessary to make a presumptive diagnosis of primary neoplasm of the facial nerve.

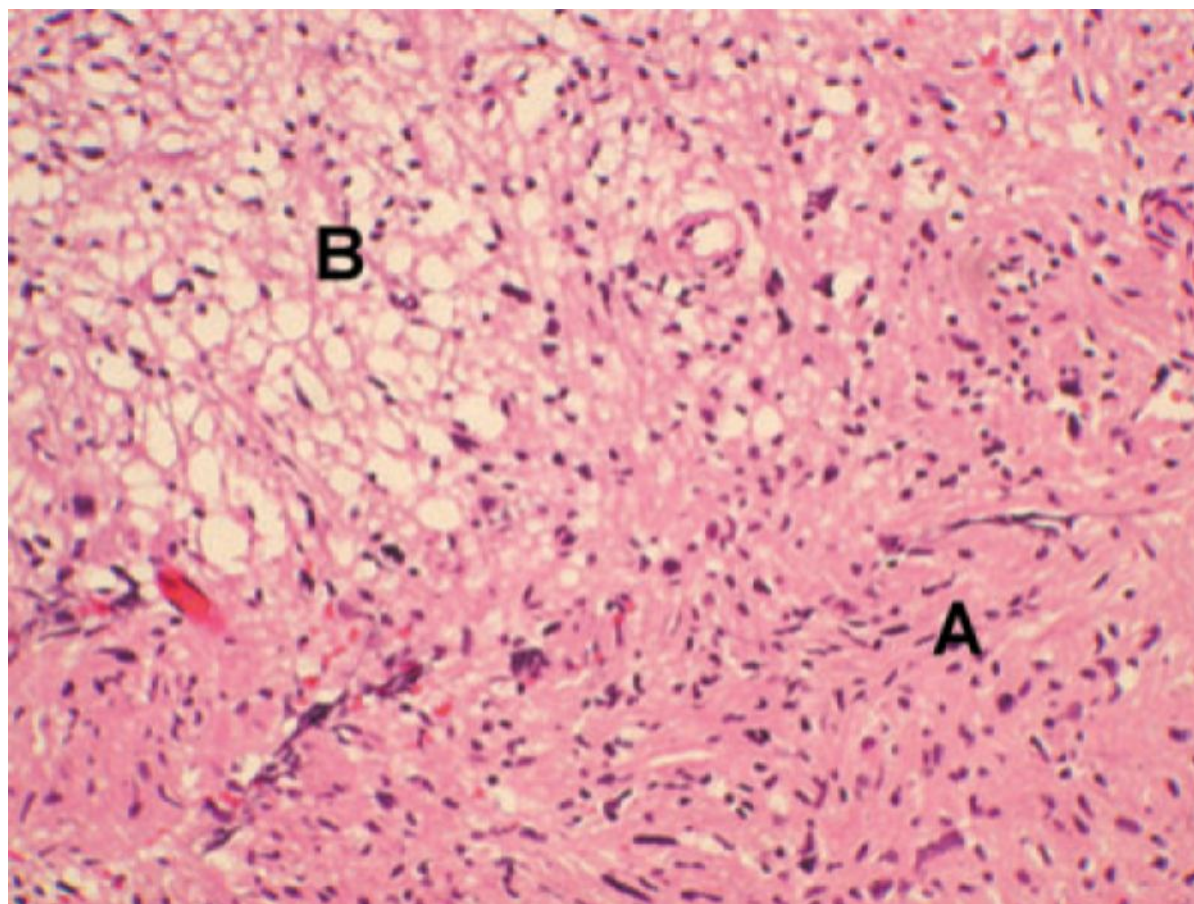


Figure 121-1 Histopathologic patterns of cellular distribution in facial neuroma. A, Antoni A (cellular, more organized); B, Antoni B (loose stroma, fewer cells, myxoid change).

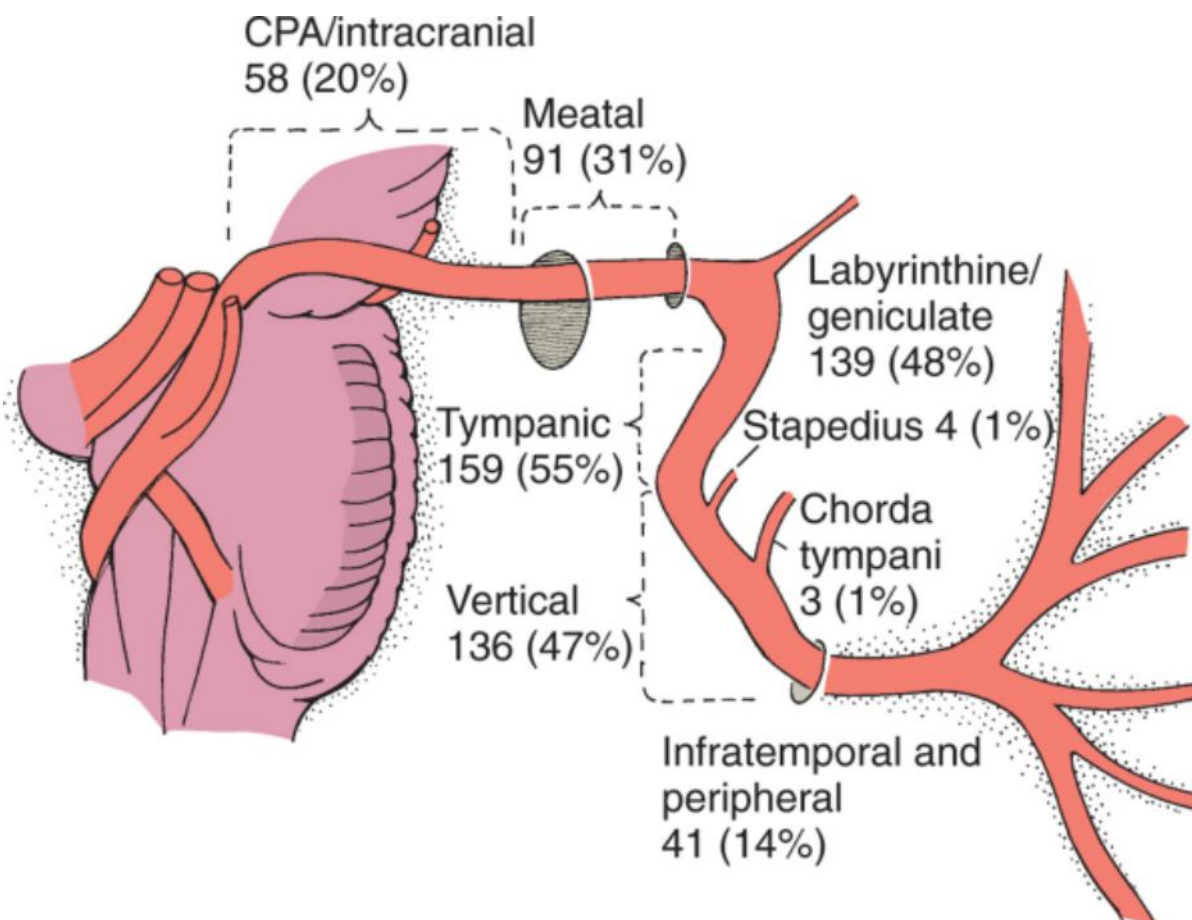


Figure 121-2 Segmental distribution and frequency of facial neuromas in 287 cases. These tumors characteristically involve multiple nerve segments. CPA, cerebellopontine angle.

PATIENT SELECTION

Complete examination of the head and neck is necessary. Examination of the face must include all branches of the facial nerve, with attention directed to gross and fine motor function. Evidence of synkinesis, mass motion, and fasciculations is sought. These signs may be further elicited by asking the patient to squeeze the eyelids closed and perform a sustained exaggerated smile or grimace. These maneuvers evoke latent fasciculations or demonstrate subsequent weakness. Based on the degree of paresis and synkinesis, the level of facial function is defined by the House-Brackmann grading system (Table 121-1).^[13] This system was devised to describe recovery of facial function after surgery, inflammation, or trauma. It has been adapted to describe abnormalities ranging from mild weakness to complete paralysis at initial examination.

Table 121-1 -- HOUSE-BRACKMANN FACIAL NERVE GRADING SYSTEM

| Grade | Description | Characteristics |
|-------|----------------------|--|
| I | Normal | Normal facial function in all areas |
| II | Mild dysfunction | <i>Gross:</i> Slight weakness noticeable on close inspection; may have very slight synkinesis <i>At rest:</i> Normal symmetry and tone <i>Motion:</i> Forehead—moderate to good function; eye—complete closure with minimal effort; mouth—slight asymmetry |
| III | Moderate dysfunction | <i>Gross:</i> Obvious but not disfiguring difference between the two sides; noticeable but not severe synkinesis, contracture, or hemifacial spasm <i>At rest:</i> Normal symmetry and tone |

| Grade | Description | Characteristics |
|-------|-------------------------------|---|
| | | <i>Motion:</i> Forehead—slight to moderate movement; eye—complete closure with effort; mouth—slightly weak with maximal effort |
| IV | Moderately severe dysfunction | <i>Gross:</i> Obvious weakness and/or disfiguring asymmetry <i>At rest:</i> Normal symmetry and tone <i>Motion:</i> Forehead—none; eye—incomplete closure; mouth—asymmetrical with maximal effort |
| V | Severe dysfunction | <i>Gross:</i> Only barely perceptible motion <i>At rest:</i> Asymmetry <i>Motion:</i> Forehead—none; eye—incomplete closure; mouth—slight movement |
| VI | Total paralysis | No movement |

The parotid gland and neck are palpated for evidence of tumor or adenopathy, respectively. The fifth cranial nerve is tested for evidence of motor or sensory abnormalities. Findings on otoscopy are usually normal, although a mass in the middle ear aural polyp may be evident. The results of tuning fork testing would be reversed (bone greater than air or a negative Rinne test) when ossicular chain movement is impeded by the tumor. Significant asymmetrical sensorineural hearing loss may also be identified with tuning fork testing.

Radiologic imaging is critical in establishing a presumptive diagnosis. Both high-resolution CT and MRI can demonstrate a primary tumor of the facial nerve. Thorough and precise radiologic imaging is mandatory for tracing the entire course of the facial nerve from the brain stem to the parotid gland. CT is better able to show bone expansion or changes such as those seen with ossifying hemangioma. This honeycomb appearance with evidence of bone remodeling and ossification is highly suggestive of hemangioma (Fig. 121-3). In addition, the structures of the otic capsule and middle ear are more readily identified on CT, and their degree of involvement can be defined (Fig. 121-4). Radiologic diagnosis of facial nerve neuromas can be based on the location of nerve enlargement, surrounding bone changes, or evidence of enhancement. MRI is more sensitive for demonstrating such enhancement. Tumor expansion of the geniculate ganglion area will be evident on both MRI and CT, but diffuse facial nerve involvement is readily apparent on gadolinium-enhanced MRI (Fig. 121-5).

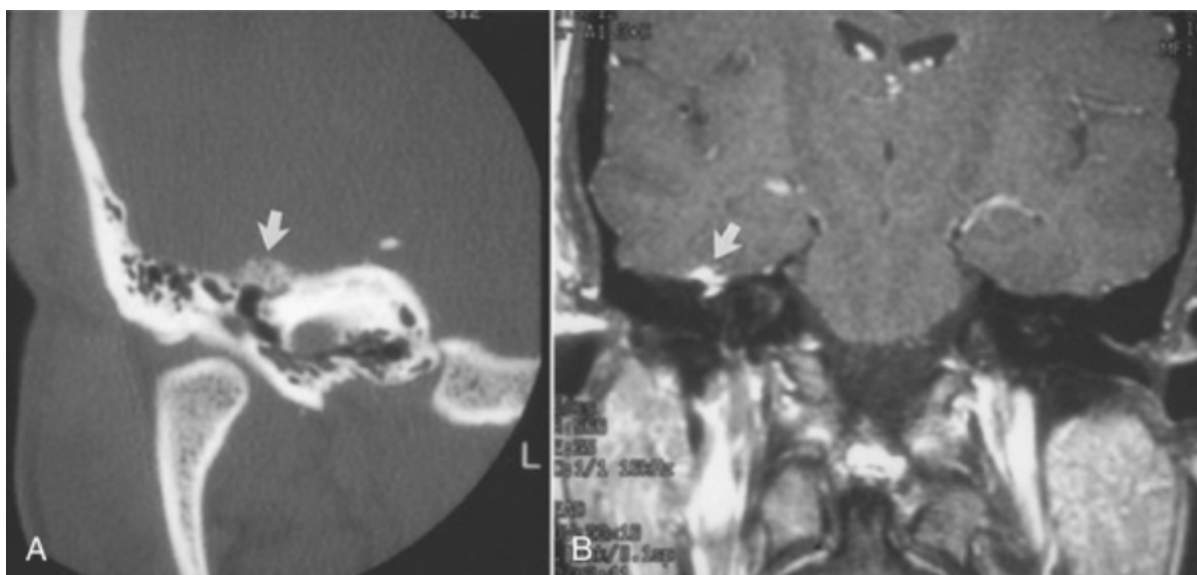


Figure 121-3 The patient is a 32-year-old woman with facial weakness and synkinesis. **A**, Bone-windowed computed tomography scan demonstrating a tumor of the geniculate ganglion with honeycomb trabeculation (*arrow*). **B**, T1-weighted contrast-enhanced magnetic resonance image showing enhancement of the geniculate ganglion region (*arrow*).

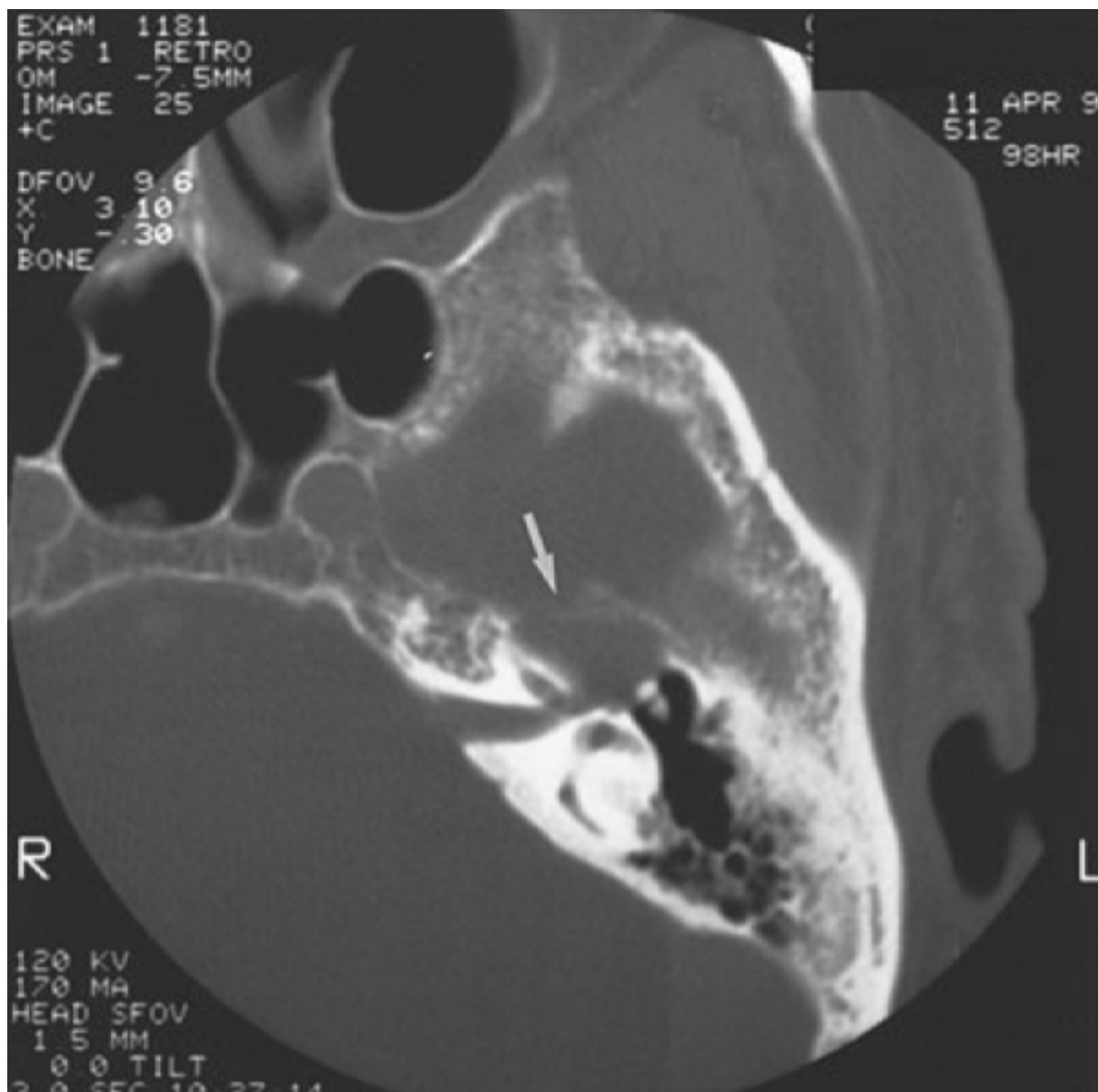


Figure 121-4 Bone-windowed computed tomography scan showing a facial nerve neuroma eroding the cochlea and impinging on the head of the malleus (*arrow*).

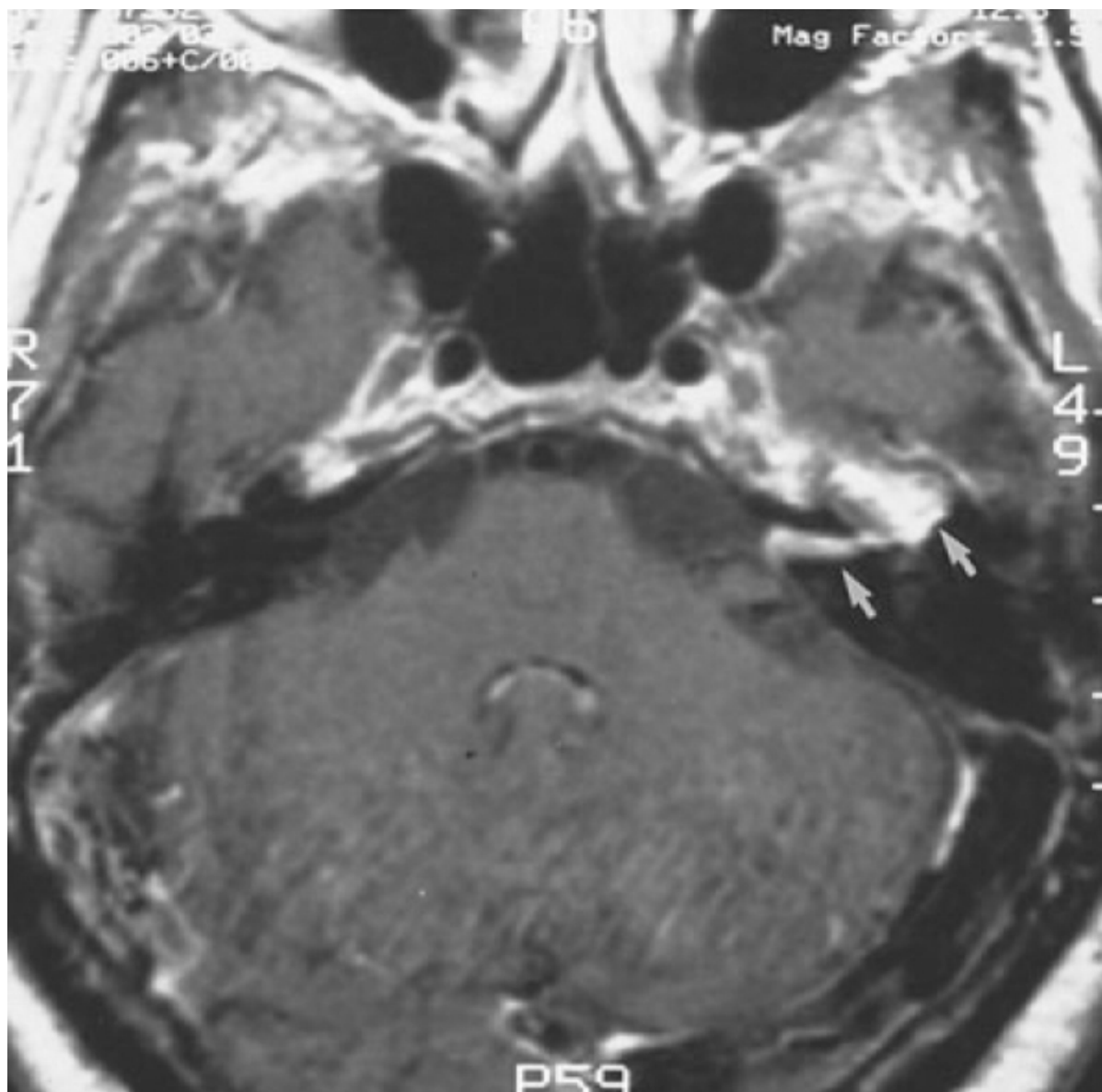


Figure 121-5 T1-weighted enhanced magnetic resonance image (same patient as in Fig. 121-4) demonstrating facial nerve neuroma of the geniculate ganglion and diffuse involvement of the facial nerve from the porus to the geniculate ganglion (arrows).

Intracranial or intrameatal (IAC) neuromas of the facial nerve are more frequently diagnosed as acoustic neuroma. However, lateral extension into the labyrinthine segment and geniculate ganglion areas should alert the radiologist and otolaryngologist to the likelihood of a primary tumor of the facial nerve. Certainly, enlargement of the tympanic (horizontal) and mastoid (vertical) segments of the facial nerve is suggestive of neuroma of the facial nerve. Secondary involvement from a primary tumor of the parotid gland or metastatic cancer must be ruled out. Primary neuroma of the facial nerve in the extratemporal (parotid) portion of the facial nerve has been reported.^[14]

Options for management include surgical resection, decompression, stereotactic radiosurgery, or observation. Removal of the bony compartment encasing the facial nerve (fallopian canal) permits tumor expansion with less entrapment, thereby allowing facial weakness to progress more slowly over time. The detriment in waiting for complete paralysis to develop is that ongoing, irreversible degenerative changes are occurring in both the proximal and distal portions of the nerve along with atrophy of the denervated facial muscles. Optimal results with facial reanimation procedures occur when paresis/paralysis is of shorter duration. The longer the duration of paralysis, the worse the outcome. To formulate a treatment plan, one attempts to predict the natural history and progression of an untreated tumor whose presumptive histopathologic diagnosis is based on the interpretation of radiologic imaging.

The status of the patient's hearing must also be considered. It must be anticipated that a patient will probably lose residual hearing if the tumor erodes into the otic capsule or is causing retrocochlear hearing loss. In patients with poor contralateral hearing, removal of an ipsilateral tumor of the facial nerve should not be recommended if injury to the cochlear nerve or otic capsule is likely. In this situation, surgical decompression, stereotactic radiosurgery

(SRS), or watchful waiting is suggested unless the tumor has imminent potential to create other significant neurologic sequelae. Similarly, if tumor growth results in severe ipsilateral sensorineural hearing loss, resection is undertaken and amplification or cochlear implantation is performed on the contralateral side.

The physician may be faced with various options that require careful deliberation regarding the optimal management of facial nerve tumors. The most straightforward situation occurs in a patient with symptoms of slowly progressive facial paralysis and evidence of synkinesis, hemifacial spasm, or a tic developing over a period of a few weeks to months. Similarly, a patient with complete facial paralysis in whom Bell's palsy is diagnosed and who fails to demonstrate any return of motor function within 6 months should be presumed to have a tumor.

Recurrent episodes of Bell's palsy should also arouse suspicion of a tumor of the facial nerve, especially when complete return of function does not occur and each episode results in subsequent worsening or permanent dysfunction. Patients often experience twitching, tics, or fasciculations with concomitant synkinesis and mass movement and may complain of eye symptoms because of an inability to blink or close the eye. The patient's facial nerve function grade is recorded.

The ideal surgical candidate is a patient who has slowly progressive facial paresis with House–Brackmann grade III or worse and normal hearing on the contralateral side. Assuming that the patient has no medical conditions posing unacceptable surgical risks or contraindications to a potentially lengthy general anesthetic, the patient is a candidate for surgical removal of the tumor. The radiographic size, location, and involvement of nearby structures will dictate the most advantageous surgical approach.

One of the more difficult decisions to be made is recommending treatment of a presumed tumor of the facial nerve when a patient has normal facial function or is presenting with subtle facial paresis, synkinesis, or fasciculations. This situation arises when a patient is scanned for other symptoms, such as sensorineural hearing loss, unrelenting pain, pulsatile tinnitus, or atypical vestibular complaints. If a tumor of the facial nerve is presumptively diagnosed because of its location and enhancement characteristics, a comprehensive discussion of treatment options, risks, and benefits should take place with the patient and family. When the tumor is small, there is a greater likelihood of complete removal with anatomic preservation of the facial nerve. This affords the patient the greatest chance of normal or nearly normal facial function. A small hemangioma located in the area of the labyrinthine segment, geniculate ganglion, or horizontal segment can frequently be removed with preservation of the underlying facial nerve. However, both the patient and the surgeon must be prepared for the possibility that the tumor has infiltrated the facial nerve and requires resection and nerve grafting. This would obviously result in complete paralysis, which is a marked change requiring psychological adjustment in view of the fact that minimal dysfunction was present preoperatively. Larger hemangiomas filling the IAC are more likely to require facial nerve sacrifice and repair.

Thus, patients with normal facial function and a presumed tumor of the facial nerve must decide whether they wish to proceed with surgical removal or wait until facial paresis/paralysis develops. It must be emphasized to such patients that the longer facial paralysis has been present, the poorer the outcome after primary nerve repair. The timing for intervention is influenced by the surgeon's technical skills and experience in managing tumors in this site. Special mention should be made of the rare cystic facial neuroma. Rapid growth of the cyst can make facial weakness worse or accentuate other symptoms. A recent publication advocates drainage and marsupialization of the cyst through a retrosigmoid approach to manage this subset of patients.^[15]

The surgeon may incidentally discover a primary tumor of the facial nerve when surgery is being carried out for other indications. Otologic procedures during which the horizontal or vertical segments of the facial nerve may be visualized include exploratory tympanotomy, tympanomastoidectomy, and cochlear implantation. In this situation, the planned procedure should be completed if the anatomy permits. Rather than removing the tumor in a patient with normal preoperative facial function, the findings and subsequent evaluation should be discussed once the patient is fully awake. Although there are rare exceptions to this principle, the patient and family should understand the nature of the pathology and participate in the subsequent course of management given the potential for facial nerve paralysis.

Medium to large primary tumors of the facial nerve are frequently accompanied by facial weakness. Occasionally, in a patient with normal facial function preoperatively, a tumor of the facial nerve is identified intraoperatively during surgery for a posterior fossa tumor. In particular, during surgery for an enhancing IAC and CPA tumor presumed to be an acoustic neuroma, it may be discovered that the tumor is indeed a neuroma or, less likely, a hemangioma of the facial nerve. It is usually well into the tumor removal stage that the origin of the tissue is clearly identified. Decompression may buy additional time for paralysis to occur, but once the tumor is partially resected, facial paresis will probably result. Because facial paralysis is a known risk and complication of acoustic neuroma surgery, the surgeon should proceed with tumor removal. In this situation, the patient is informed after tumor resection and reconstruction have been completed. However, if it can be demonstrated early in the procedure that a small tumor originates from the facial nerve, decompression of the surrounding bone provides additional time for less restricted tumor growth. Retrospective review of the radiologic images may demonstrate findings that in the future would raise suspicion of a primary facial nerve tumor.

Dissatisfaction with the possibility of worsening facial weakness after microsurgical resection has led to a search for treatment alternatives for facial nerve neuroma. Recent reports in the literature have discussed the use of SRS for nonacoustic schwannomas, including facial nerve neuroma. SRS has been well established as a treatment modality for acoustic neuroma that affords excellent tumor control with a low rate of new cranial nerve deficits. Several publications have discussed the use of both gamma knife- and linear accelerator-based radiosurgery to treat nonacoustic neuromas. Pollock and colleagues reported on the treatment of 11 patients with either trigeminal or jugular foramen schwannomas.^[16] Tumor growth was controlled in 100% of patients with trigeminal schwannomas and 75% of patients with jugular foramen schwannomas at mean follow-ups of 21 and 10 months, respectively. None of the patients experienced worsening of cranial nerve deficits after treatment. Mabanta and associates published a series of 18 patients with nonacoustic schwannomas who were treated with linear accelerator SRS.^[17] Two of the 18 patients had a facial neuroma; the remaining patients had a trigeminal or jugular foramen tumor. The authors reported a 100% control rate. Three patients suffered complications, including progression of preexisting facial weakness, new hearing loss, and ataxia. However, five patients experienced improvement in neurologic symptoms. A French series of nine patients with facial neuroma treated with gamma knife radiosurgery was published in 2004.^[18] Follow-up ranged from 2 to 7 years, during which time no patient experienced worsening of facial function. Additionally, two case reports from the Japanese literature discuss the use of SRS for facial neuroma.^[19,20]

PREOPERATIVE PLANNING

Complete examination of the head and neck with attention directed to facial nerve function is performed. The House–Brackmann grading system is used to record the degree of facial nerve weakness or paralysis. The patient is instructed to attempt complete and forceful eye closure. If the patient demonstrates a poor Bell phenomenon and is unable to adequately protect the cornea, the benefit of insertion of a gold weight should be addressed. Various gold weights are temporarily taped to the upper eyelid to determine whether improved eye closure and comfort are achieved. If ectropion is present, a lower lid-tightening procedure should be planned. The ear is inspected with an operating microscope. The status of the external auditory canal, tympanic membrane, and middle ear is noted. Audiometric analysis will determine the hearing status of both ears. Identification of ipsilateral conductive hearing loss suggests that ossicular chain involvement may require disarticulation and reconstruction and that the middle ear will require exploration. If poor hearing is identified in the contralateral ear and it is anticipated that hearing will be compromised on the side with the tumor, careful deliberation must be given to the options of tumor management. In this situation, observation, radiation therapy, or surgical decompression to provide more room for tumor growth are all reasonable management alternatives.

Stapes reflex testing may be helpful in determining the site of the lesion. An intact stapes reflex in the presence of facial paralysis would indicate facial nerve involvement in the distal vertical segment or its extratemporal course. Vestibular testing does not provide enough information to alter the treatment strategy.

Preoperative electrophysiologic facial nerve tests are occasionally helpful. When facial paresis is present, electroneuronography (ENoG) demonstrates decreased responses. The ENoG data obtained from patients with normal facial function or minimal paresis may occasionally be of benefit. Elicitation of 50% or greater reduction in the ENoG amplitude provides additional support demonstrating compromised neural integrity. Patients with normal facial function and surgeons may be more willing to entertain surgical excision when objective verification of facial nerve involvement is established. However, ENoG is rarely useful when complete facial paralysis has been present for more than a few weeks. In the setting of facial paresis or paralysis, facial electromyography of the peripheral facial muscles may provide evidence of fibrillation (denervation) and polyphasic voluntary motor unit (reinnervation) potentials. This provides confirmatory evidence that an ongoing destructive and reparative process is occurring.

Preoperative diagnosis of a presumed tumor of the facial nerve depends on radiologic imaging. The surgeon must carefully review the scans to ensure that complete data have been compiled. Both contrast-enhanced CT and MRI are often needed to determine the extent and size of the lesion, whether the otic capsule is eroded, and which segments of the facial nerve are affected. Facial nerve neuromas are prone to multiple areas of involvement. Imaging will identify whether the labyrinthine segment of the nerve enhances or is widened, whether the greater superficial petrosal nerve is involved, and whether middle ear growth has affected the ossicular chain. The MRI or CT scanning protocol should extend beneath the skull base to trace the course of the facial nerve through the parotid. This information is needed to determine which operative approach is most appropriate, who should be part of the surgical team, what surgical and ancillary equipment is necessary (such as facial nerve monitoring), and whether other autologous tissue is needed for nerve repair and wound closure. These latter areas include the neck for a greater auricular nerve graft, the lateral lower part of the leg for a sural nerve graft, or the abdomen for a free fat graft.

The appropriate surgical approach is dictated by the size and location of the tumor and the type and degree of hearing loss. Although unusual and difficult to diagnose, presumed tumors of the facial nerve isolated to the CPA are removed through a retrosigmoid craniotomy. The more common location for these tumors is between the lateral IAC and the vertical segment of the facial nerve. A middle fossa approach is necessary for tumors of the

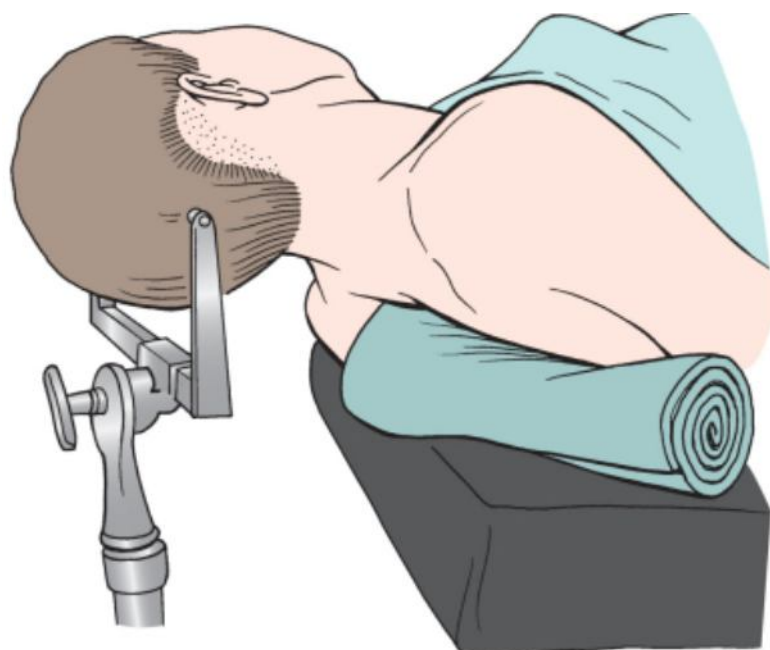
facial nerve in the IAC, labyrinthine segment, and geniculate ganglion areas if hearing is to be preserved. Tumors confined to the horizontal or vertical fallopian canal are managed by a transmastoid approach. A combined middle fossa–transmastoid approach is necessary for larger tumors centered around the geniculate ganglion because access to the proximal and distal nerve stumps for repair will be needed. When hearing in the ipsilateral ear is compromised (speech reception threshold >50 dB, discrimination <50%), a translabyrinthine or transcochlear approach permits direct access to the tumor and facilitates primary repair of the facial nerve.

Hemangiomas of the facial nerve are, by definition, vascular tumors. However, they rarely achieve sufficient size to warrant arteriography and embolization. Nevertheless, blood typing and screening are performed when an intracranial procedure is planned.

A comprehensive discussion of the natural course of the tumor and the risks and benefits of surgical resection must take place with the patient and significant family members. The patient should understand that temporary or permanent facial paralysis will probably occur and that subsequent care will be dictated by the intraoperative management of the tumor. Unilateral complete hearing loss may result despite undertaking a hearing preservation approach.

SURGICAL TECHNIQUES

All procedures are performed with the patient under general endotracheal anesthesia with inhalational and intravenous narcotic agents. In patients with preoperative facial function, muscle relaxants are avoided to permit facial nerve monitoring. The patient is placed in the supine position with the head turned toward the contralateral ear when undergoing transmastoid, translabyrinthine, middle fossa, or a combined procedure. The more medial access needed for the retrosigmoid approach often requires placement of support rolls beneath the patient's shoulder and hip to rotate the patient into a semidecubitus position. A Mayfield head holder and pins are secured to the patient and the table when a retrosigmoid approach is used (Fig. 121-6A and B). The upper part of the neck is included in the operative field to provide access to the greater auricular nerve. The ipsilateral leg is also prepared and draped if the tumor is known to be extensive and could require a longer nerve graft. The left lower quadrant of the abdomen is prepared for a fat graft when a translabyrinthine or middle fossa approach is planned. Perioperative antibiotics are given when opening the dura is anticipated.



A





B

Figure 121-6 **A**, Positioning of a patient for a retrosigmoid approach to the posterior fossa. **B**, Photograph of a patient in a three-pin Mayfield head holder.

Transmastoid Approach

A curvilinear incision is made 1 cm posterior to the postauricular sulcus through the skin, subcutaneous tissue, and fibroperiosteal layer over the mastoid cortex. The periosteum is elevated posteriorly and anteriorly until the level of the bony external auditory canal is encountered. Self-retaining retractors are placed, and a transcortical mastoidectomy is performed. The antrum and horizontal semicircular canal serve as important initial surgical landmarks for this approach. The sinodural angle is delineated. Bone over the sigmoid sinus and posterior bony canal wall is thinned to provide the necessary exposure to the vertical portion of the facial nerve.

The short process of the incus is identified. It serves as a helpful landmark for the facial recess, which must be surgically defined. Regardless of whether the tumor is located in the horizontal or vertical portion of the facial nerve, the middle ear and mastoid portions are exposed to obtain access to the margins. The tumor should be evident at this time. Using continuous suction irrigation and diamond burrs, the bone overlying the facial nerve and tumor is thinned. Access to the horizontal (tympanic) segment of the facial nerve often requires removal of the incus (Fig. 121-7), which is accomplished through the facial recess unless the tumor is so large that it obliterates this space. In this situation, the ear is returned to its anatomic position and a tympanomeatal flap is elevated through the ear canal. Bone from the posterosuperior canal wall is removed by curette to provide exposure of the

incudostapedial joint. The joint is separated and the incus is removed (see Chapter 117)  (see [Video 121-1](#) ).

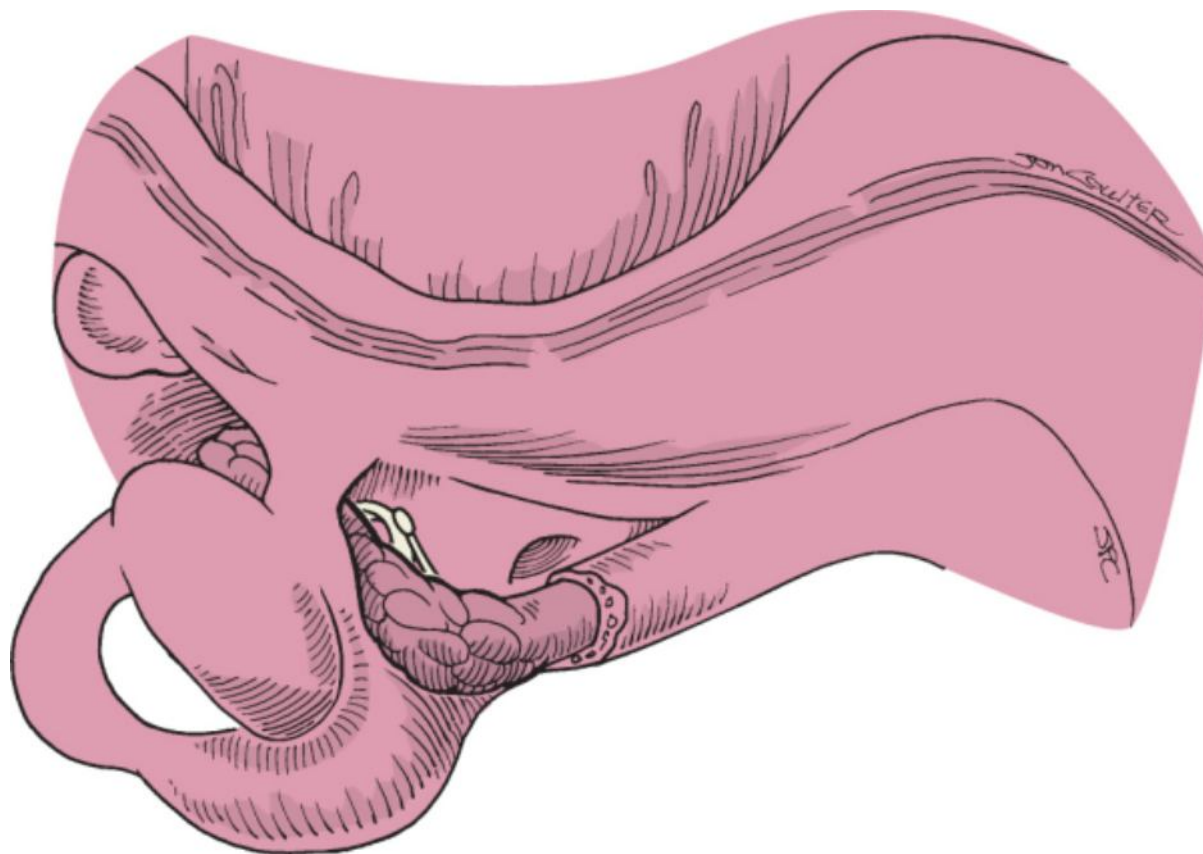




Figure 121-7 A complete mastoidectomy via a facial recess approach provides exposure of the epitympanum and the vertical segment of the facial nerve. The incus has been removed to reveal the tumor. The head of the malleus is intact.

This approach provides access to the facial nerve from the geniculate ganglion to the stylomastoid foramen. If additional exposure is necessary at the proximal horizontal segment of the facial nerve, the head of the malleus is removed. The eggshell-thinned bone of the fallopian canal is removed. If the tumor extends toward the distal aspect of the vertical segment of the facial nerve, the chorda tympani nerve is sacrificed and dissection of the facial recess is extended. The tumor is removed with a 59S Beaver blade by sharply dividing the tumor from the nerve. Fine spring scissors facilitate this dissection. If the tumor cannot be separated from the nerve, sharp transection is performed to remove the nerve segment and tumor together (see Videos 121-2  and 121-3 .

Frozen section examination is used to examine the margins of resection and histologically confirm complete removal of the tumor. The nerve is repaired with an interposition graft from the greater auricular nerve (see the next section, "Greater Auricular Nerve Graft"). The graft is carefully positioned in the fallopian canal without tension, between the remaining segments of the facial nerve. It is difficult and unnecessary to suture the ends together. A small piece of fascia or fibrin glue may be placed over each anastomosis to minimize mobility. If the head of the malleus has not been removed, the incus can be returned to its anatomic position and supported by Gelfoam. Absence of the head of the malleus requires ossicular chain reconstruction by means of an incus interposition. This procedure is detailed in Chapter 114. If the ear canal was entered for exposure or ossicular chain reconstruction, the tympanic membrane is returned to its anatomic position and supported with a silk sleeve or Gelfoam. The postauricular incision is closed in the usual fashion, and a sterile dressing is applied to the mastoid and neck.

Greater Auricular Nerve Graft

Various characteristics of the greater auricular nerve make it an ideal donor for facial nerve repair. The nerve is in close proximity to the surgical field. Although it supplies sensation to the pinna and preauricular area, sacrifice of this nerve leaves only a small area of anesthesia. The anatomy of the nerve is relatively constant, thus providing consistent localization.

Two methods are used for localizing the greater auricular nerve (Fig. 121-8). Dividing the sternocleidomastoid (SCM) muscle into thirds, the nerve is identified at the posterior border of the SCM, between the upper and the middle thirds. The other method locates the nerve midway along a line connecting the mastoid tip and the angle of the mandible. An incision in a skin crease over this area is taken through subcutaneous tissue. This may be incorporated with the mastoid incision or, preferably, through a separate incision. The greater auricular nerve is found lying lateral to the SCM, exiting from its posterior surface. Retrograde dissection along the nerve by

retracting the SCM anteriorly permits more proximal harvesting of the nerve from its origin in the cervical plexus.

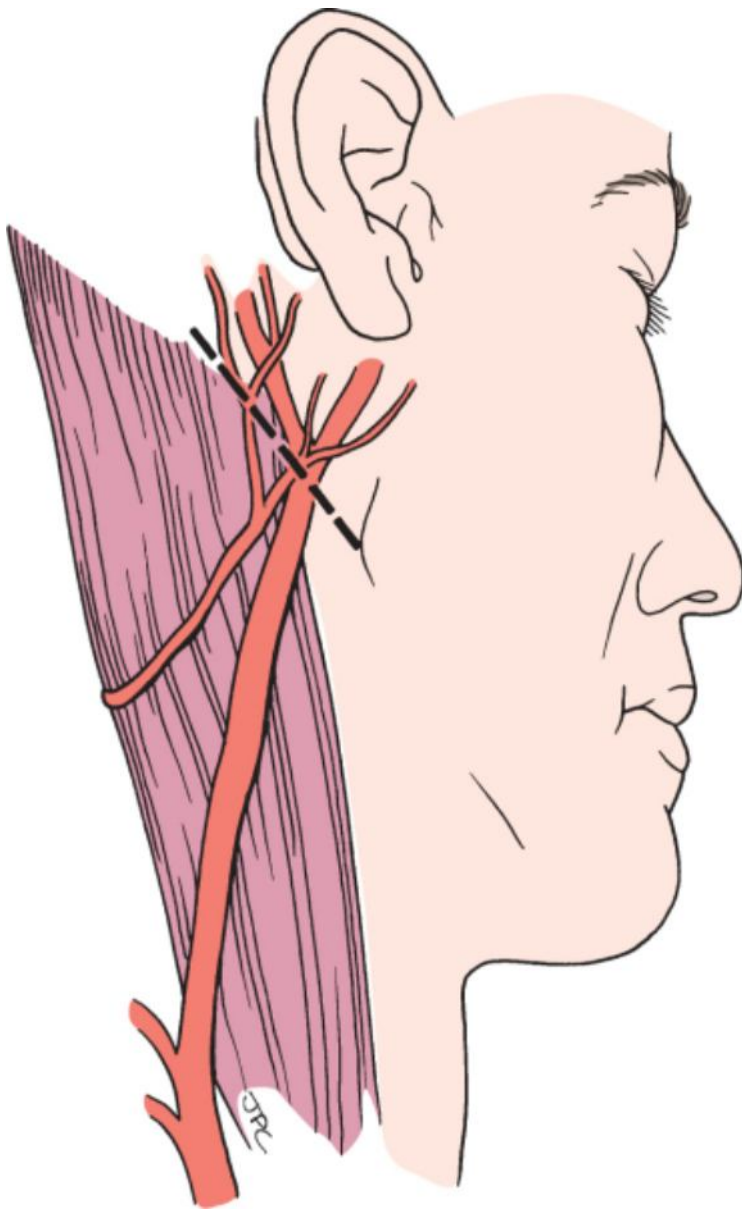


Figure 121-8 Methods for identifying the greater auricular nerve with pertinent anatomic landmarks. The greater auricular nerve exits posterior to the sternocleidomastoid muscle at the junction of its upper and middle thirds. The greater auricular nerve can also be found midway between the angle of the mandible and the mastoid tip.

Translabyrinthine Approach

A postauricular curvilinear incision is positioned approximately 3 to 4 cm posterior to the postauricular crease. A transcortical mastoidectomy is performed as described previously. Similar to the approach for an acoustic neuroma, bone posterior to the sigmoid sinus is removed and the sigmoid sinus is skeletonized. This facilitates retraction of the posterior fossa dura and sigmoid sinus. A labyrinthectomy is performed to expose the IAC. The bone surrounding the IAC and porus is removed. Tumor extending distal to the labyrinthine segment is exposed (Fig. 121-9). Involvement of the horizontal segment may require removal of the head of the malleus and the incus to get to the distal tumor margin. The facial nerve is decompressed beyond the area of enlargement. All drilling should be completed before opening the dura of the IAC and posterior fossa.

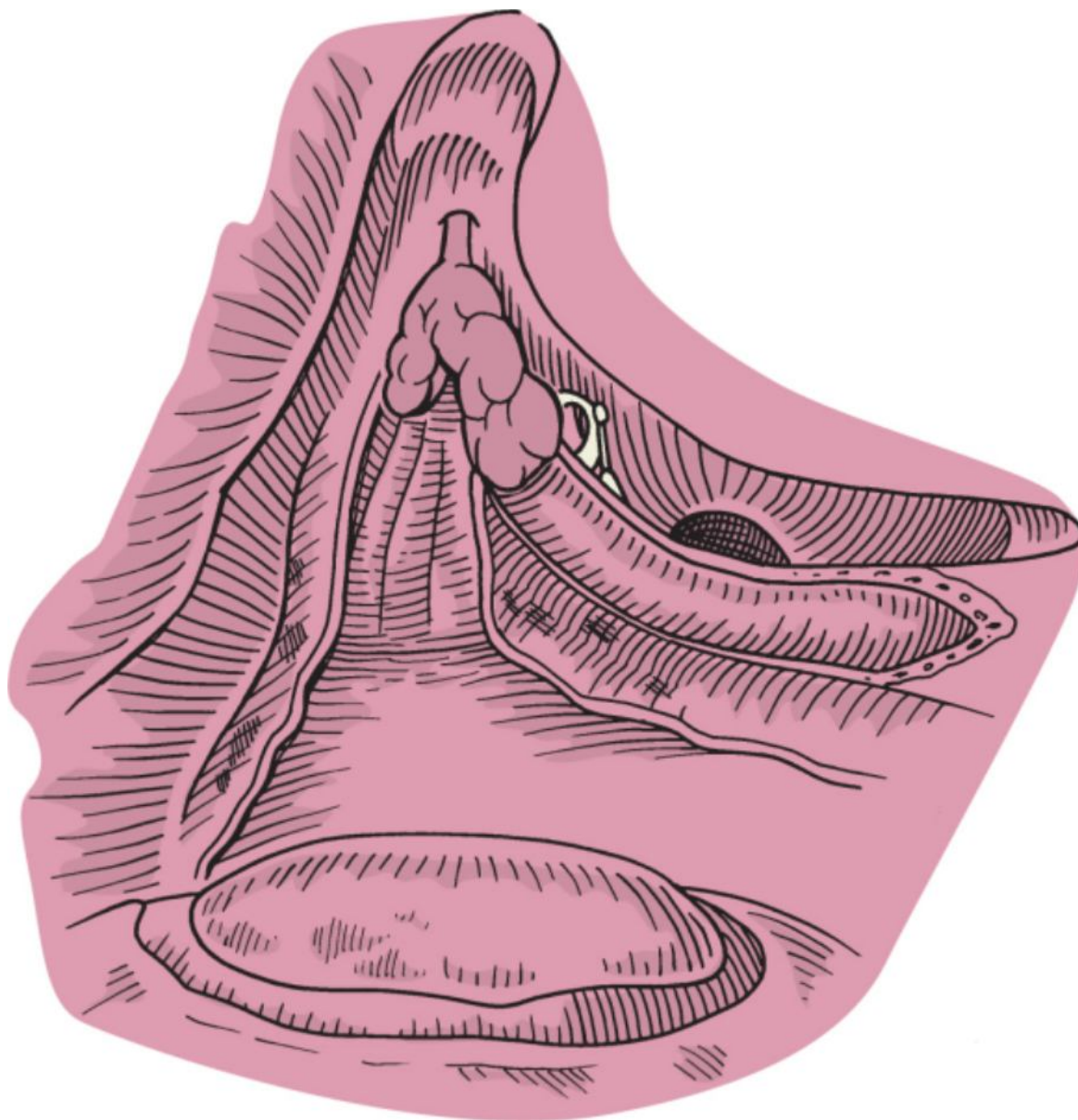


Figure 121-9 Translabrynthine approach to a tumor of the facial nerve proximal to the geniculate ganglion. An island of bone remains over the sigmoid sinus. The dura of the internal auditory canal is intact. The tumor extends from the fundus to the second genu.

The posterior fossa and IAC dura are opened. The labyrinthine segment of the facial nerve is a critical anatomic landmark in translabrynthine surgery. However, it may not be evident when the nerve is encased by tumor. Its location in the anterosuperior quadrant of the IAC and medial to the eighth nerve complex in the CPA is traced proximally and confirmed with a nerve stimulator if facial function was present preoperatively. The vestibular nerves are sharply divided and sacrificed. Tumor that is extrinsic to the nerve is sharply dissected from the underlying facial nerve. Hemangioma of the facial nerve is vascular with a gritty texture because of the bony trabeculations that are often present in the labyrinthine segment and geniculate ganglion areas. Sacrifice of the facial nerve is necessary if the tumor cannot be sharply dissected from the nerve. The tumor is removed and the margins checked by frozen section examination.

The method of nerve repair depends on the length of facial nerve graft resected. If a small section of nerve has been sacrificed, primary repair is accomplished by mobilizing the horizontal and vertical portions of the facial nerve from the fallopian canal. This posterior transposition provides 1 to 2 cm of additional length to perform the anastomosis (Fig. 121-10). The proximal nerve stump is supported with Gelfoam. Placement of 9-0 or 10-0 nylon suture may be difficult near the porus or CPA. Satisfactory reapproximation with one suture is sufficient. Because of the lack of epineurium covering the proximal facial nerve, a single suture passed through the diameter of the nerve stabilizes the anastomosis.^[21] Resection of a larger segment of the facial nerve may require a cable graft. The greater auricular nerve usually provides both adequate length and similar diameter to that of the facial nerve. If there is a noticeable difference in diameter between the two ends of the nerve graft, with the proximal end being larger, the distal end of the nerve graft is sutured to the proximal end of the facial nerve to ensure that all graft fascicles at this anastomosis are present in the distal nerve. The distal anastomosis is secured with 8-0 nylon

suture and has the additional support of the fallopian canal (Fig. 121-11).

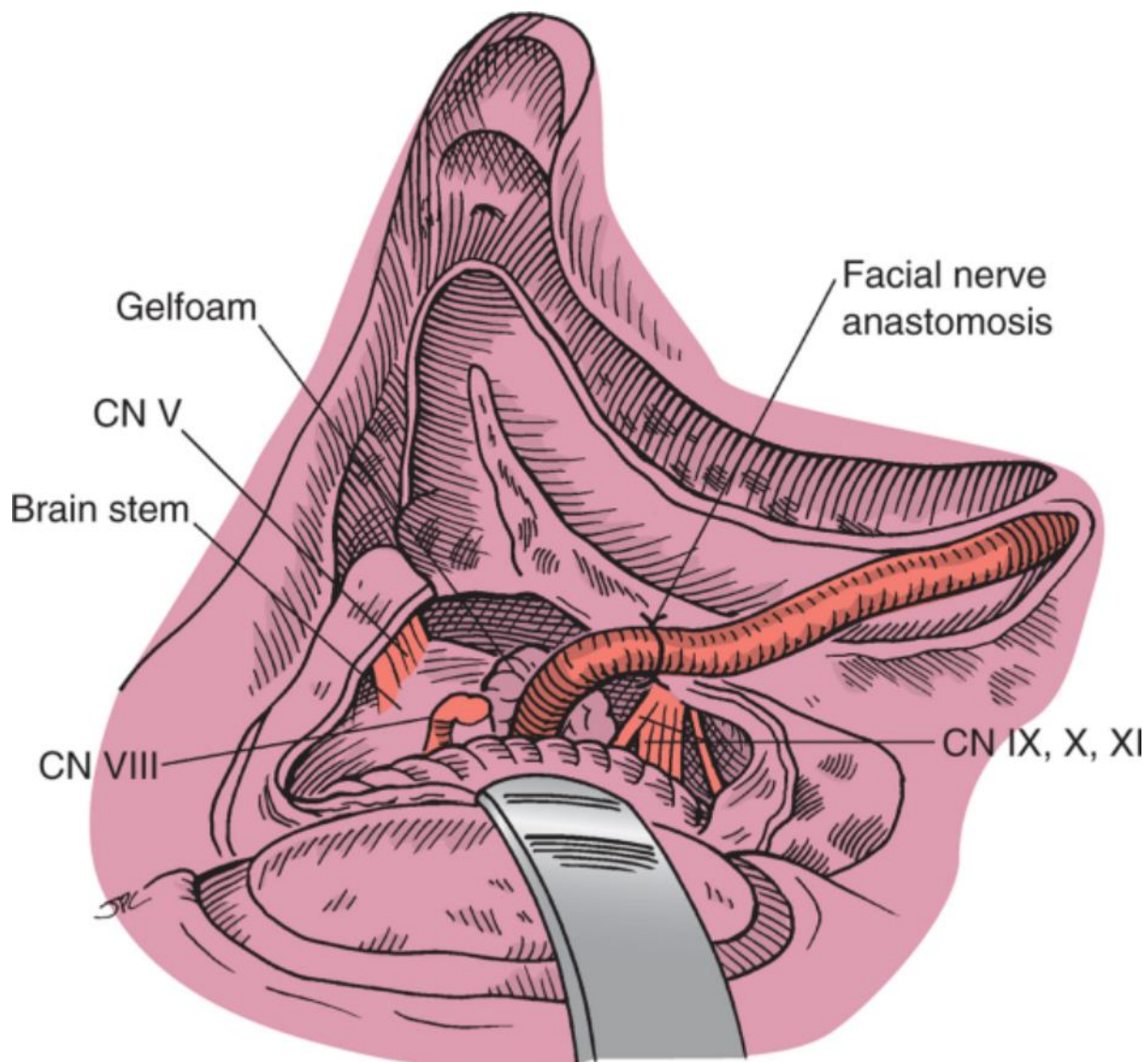


Figure 121-10 Horizontal and vertical segments of the facial nerve mobilized to permit primary repair of the facial nerve (cranial nerve [CN] VII). The brain stem, trigeminal nerve (CN V), cochleovestibular nerves (CN VIII), and lower cranial nerves (CN IX, X, XI) are demonstrated. Gelfoam supports the proximal nerve for suturing.

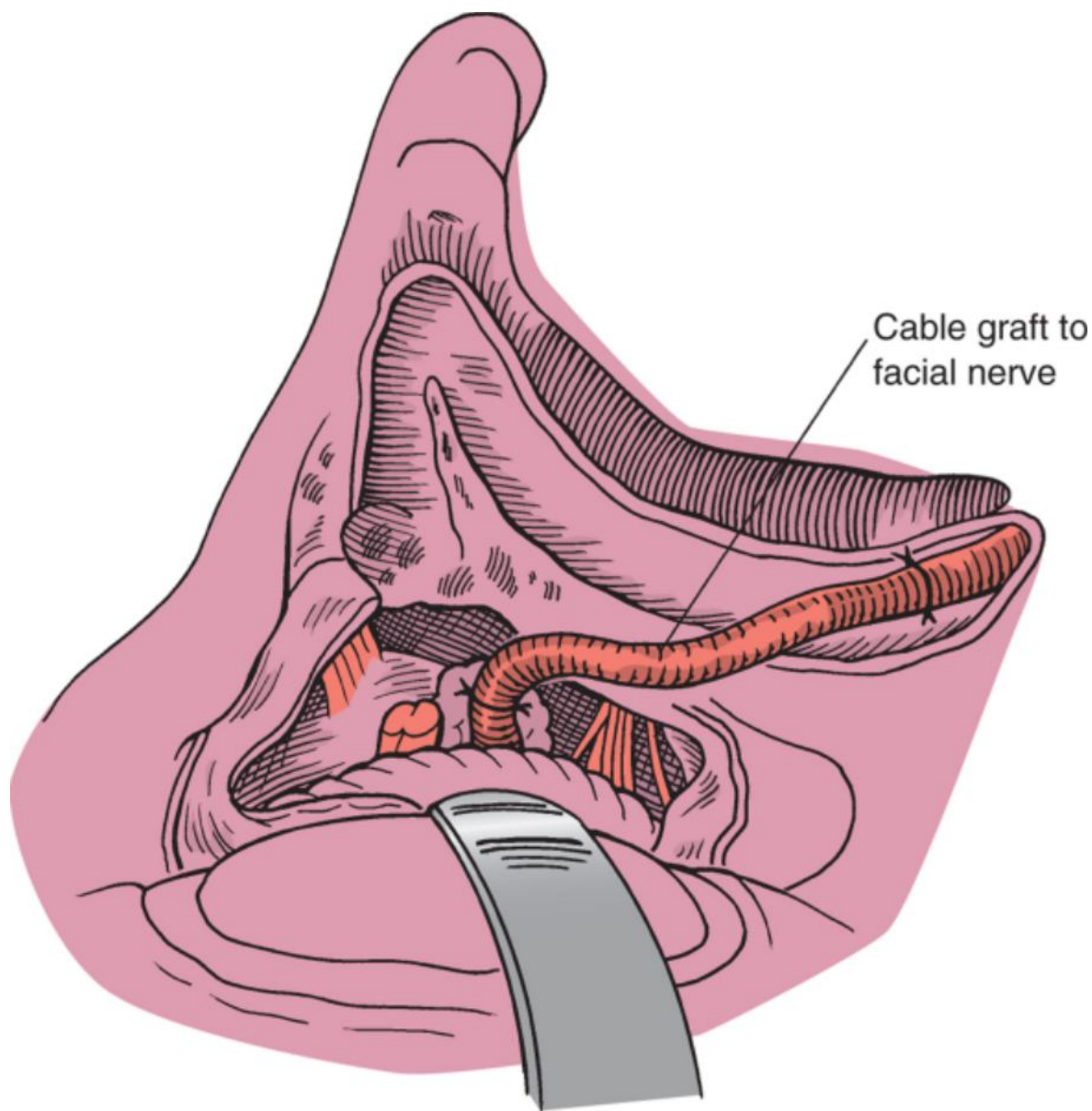


Figure 121-11 Greater auricular nerve interposition cable graft to the facial nerve from the porus to the distal vertical segment.

Fat is harvested from the left lower quadrant of the abdomen. The abdominal wound should be closed over a Penrose drain. The abdominal fat is soaked in bacitracin solution, and the wound is copiously irrigated with bacitracin solution. Strips of abdominal fat are placed around the nerve to support the anastomosis. The remainder of the cavity is filled with abdominal fat. The wound is closed in three-layer fashion, and a sterile mastoid pressure dressing is applied.

Middle Fossa Approach

Detailed surgical techniques involving the middle fossa approach are comprehensively described in Chapter 124. The patient is placed in a supine position on the operating table with the head fully turned toward the contralateral ear. A preauricular incision begins in a skin crease anterior and superior to the tragus. The incision is gently curved to the superior temporal line to expose the temporal fascia. The skin flaps are undermined posteriorly and anteriorly, and a self-retaining retractor is placed. A temporalis muscle flap pedicled on its inferior origin is elevated to the level of the zygomatic root. A middle fossa temporal craniotomy measuring 4 × 5 cm is created, and the bone flap is removed. The middle fossa dura is elevated from the temporal bone. It is typically necessary to remove additional bone inferiorly to a level flush with the floor of the middle fossa. When the floor of the middle fossa is reached, it is safer to elevate the dura in a posterior-to-anterior direction to avoid injury to the greater superficial petrosal nerve. Hemangiomas of the facial nerve involving the geniculate ganglion should be evident at this time. Another important landmark is the arcuate eminence. Unfortunately, this structure is inconsistently present and may be confused with other ridges on the floor of the middle fossa. Drilling medially over the meatal plane toward the superior petrosal sinus is safe and avoids injury to the otic capsule. Drilling with suction irrigation

is directed inferiorly until the roof of the IAC is encountered. The underlying periosteum and dura are left intact until all bone work is completed. The IAC is demarcated laterally to expose its roof until the labyrinthine segment of the facial nerve is identified. Tumor extending distal to the geniculate ganglion requires further bony dissection. The tegmen tympani is removed with caution because of the underlying ossicular chain. If additional access to the epitympanum and horizontal facial nerve is needed, a transmastoid approach should be performed as well. Preoperative imaging should have prepared the surgeon to anticipate the need for a combined approach.

Once adequate bone exposure is achieved, the IAC dura is opened to expose the tumor and proximal facial nerve (Fig. 121-12). The tumor is sharply dissected from the underlying facial nerve, if possible. Often during removal of a hemangioma, a plane can be developed between the facial nerve and the tumor. Sometimes a neuroma of the facial nerve, if located eccentrically, may be excised while continuity of the underlying nerve is maintained. If the nerve must be sacrificed because of tumor involvement, reconstruction will require a graft. For this short distance, the greater auricular nerve is readily accessible and of appropriate length and diameter.

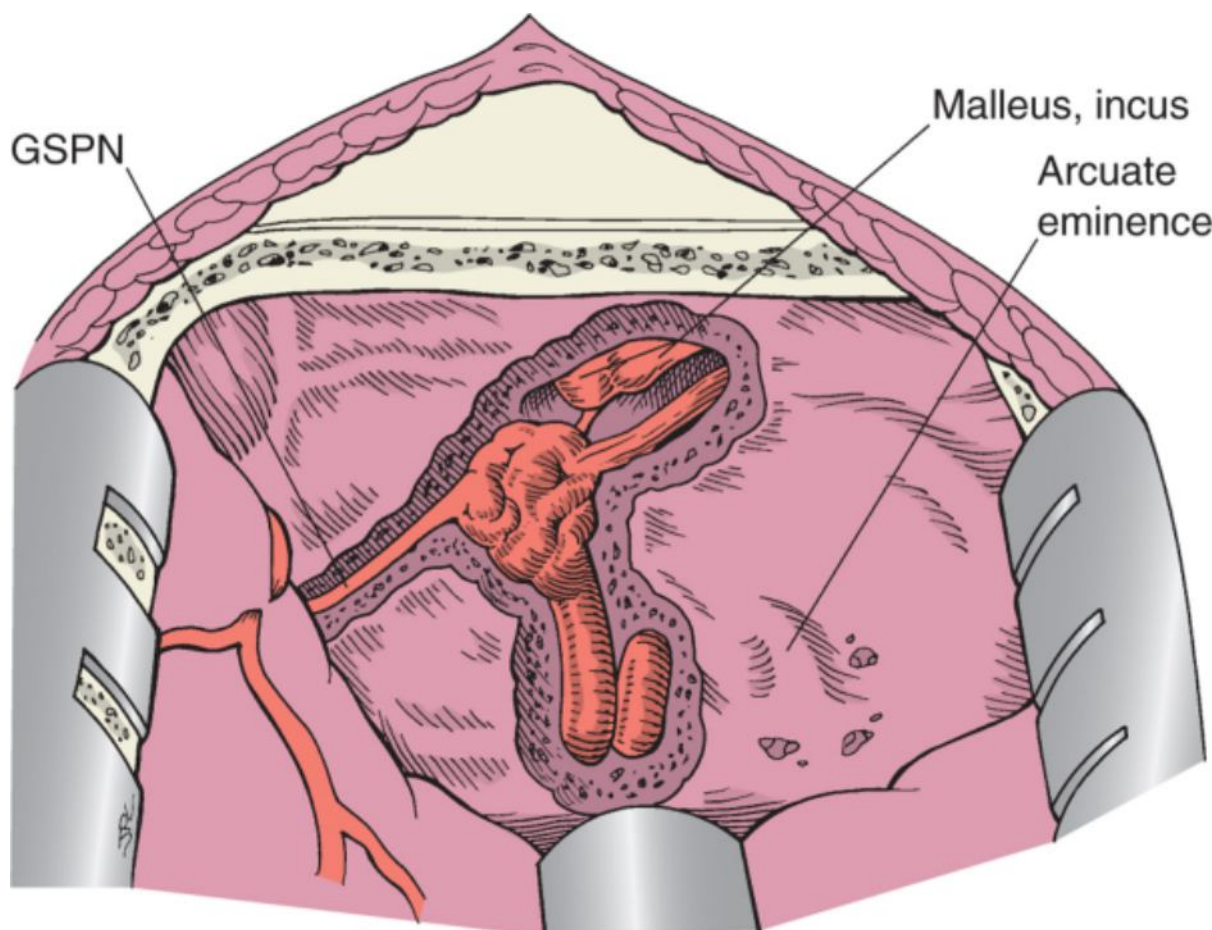


Figure 121-12 Middle fossa approach to a tumor of the facial nerve located at the geniculate ganglion. The labyrinthine and proximal horizontal segments are exposed. GSPN, greater superficial petrosal nerve.

If there is a minimal weakness of facial nerve function, decompression of the involved segment of the nerve is a treatment strategy. The overlying bone of the IAC, labyrinthine segment, and geniculate ganglion is drilled to eggshell thickness and carefully removed. Despite bleeding that may occur from a hemangioma, the use of bipolar cautery should be avoided around the facial nerve.

Proximal anastomosis of the facial nerve is technically challenging through a middle fossa approach. Size 9-0 or 10-0 nylon suture is secured to the proximal end of the nerve graft. A single suture is then passed into the cut end of the proximal facial nerve. The distal end of the nerve graft is positioned without tension next to the distal facial nerve. If the tumor is located laterally in the fundus and geniculate ganglion areas, the greater auricular nerve graft is placed in a bone trough connecting the labyrinthine and proximal horizontal segments of the fallopian canal. Suturing in this area is usually unnecessary (Fig. 121-13). Either a free temporalis muscle graft or a small piece of abdominal fat is used to plug the IAC to prevent a cerebrospinal fluid (CSF) leak.

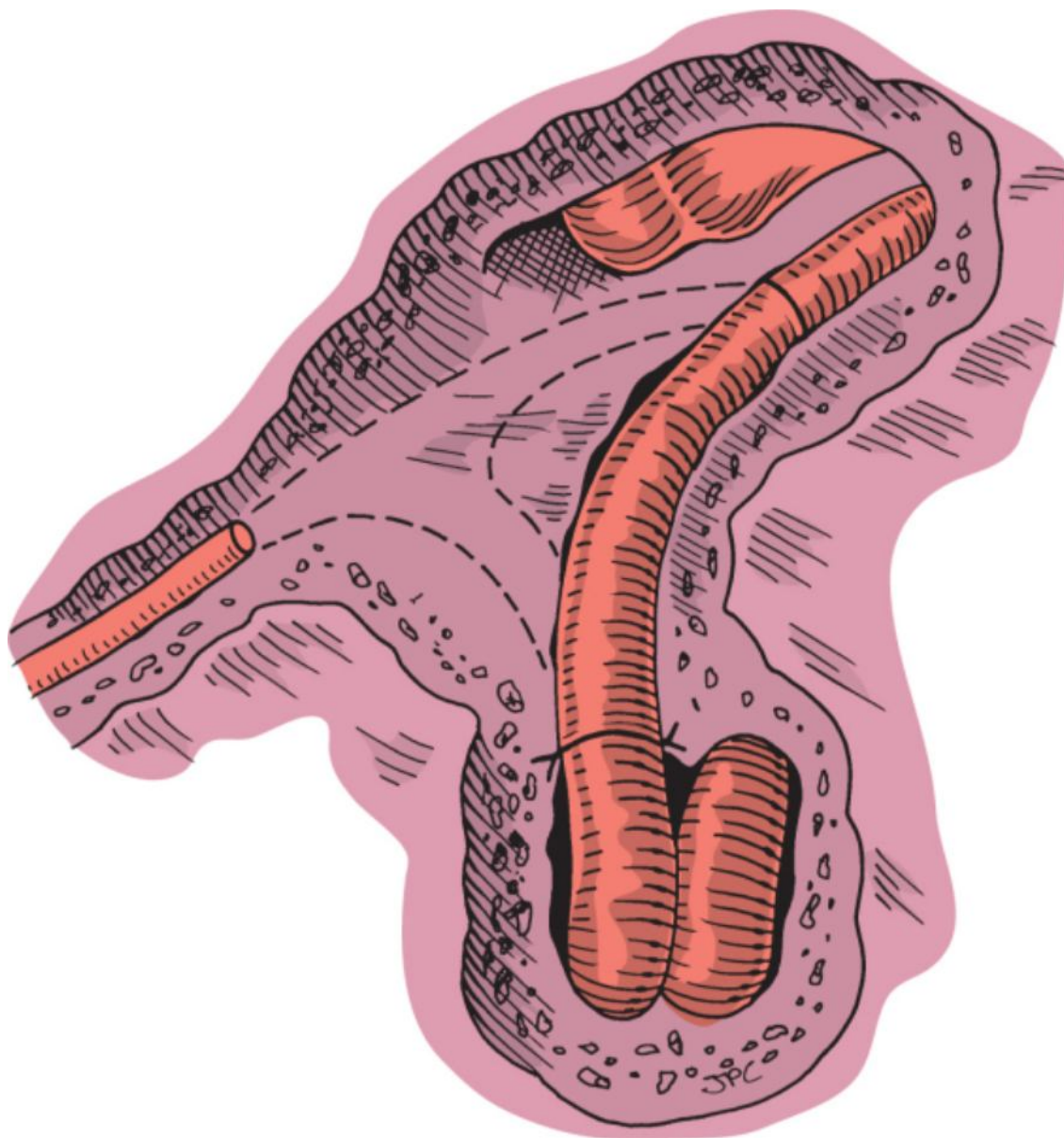


Figure 121-13 Interposition cable graft repair from the facial nerve in the distal internal auditory canal to the horizontal segment.

During closure, attention must be focused on the tegmen tympani. If it is absent, support is provided by sagittally splitting the craniotomy bone plate and positioning a bone segment over the epitympanum. The dura is tacked with 4-0 Nurodon to the edges of the craniotomy defect. The craniotomy bone plate is replaced and secured with 2-0 absorbable suture or miniplates. The temporalis muscle is returned to its anatomic position and secured with 2-0 and 3-0 absorbable suture. The skin is closed in two layers, and a compression dressing is applied.

Combined Middle Fossa–Transmastoid Approach

This approach is used when a tumor of the facial nerve is proximal to the geniculate ganglion and distal to the head of the malleus in the horizontal segment of the nerve. This approach is also undertaken if hearing is to be preserved. Otherwise, a translabyrinthine approach is more direct and efficient. The incision for the combined approach is the standard postauricular one. The superior limb extends beyond the route of the helix to the preauricular area. The incision continues anteriorly and superiorly, parallel to the hairline toward the temporal area. The incision courses superiorly and curves posteriorly, parallel to the superior temporal line. It forms the shape of a question mark (Fig. 121-14). The posteriorly based temporal skin flap is retracted with either suture or large self-retaining retractors. This combined approach provides additional access to the horizontal and vertical portions of the facial nerve, as described in the section “Transmastoid Approach,” earlier in this chapter.

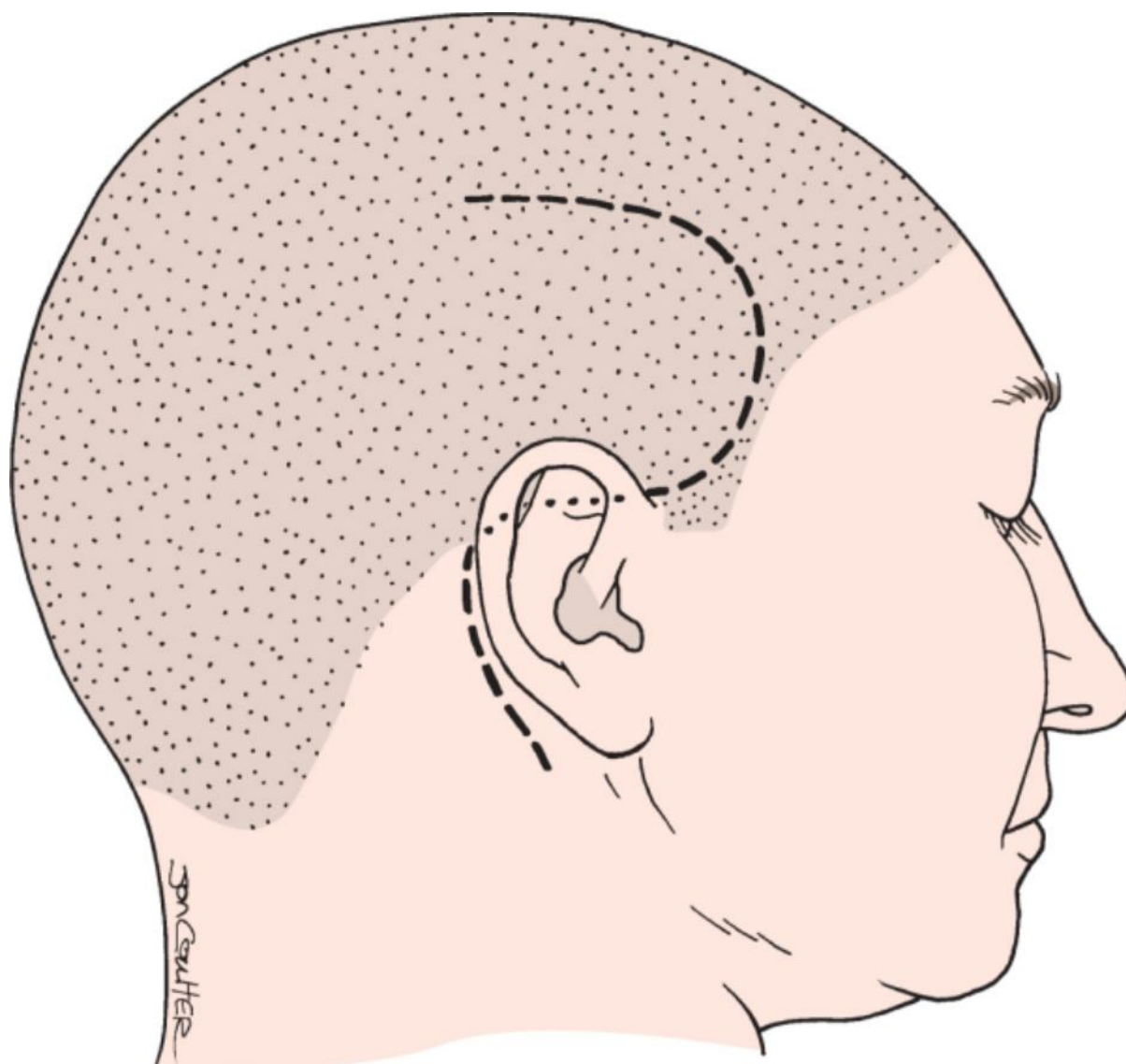


Figure 121-14 Incision for a combined middle fossa–transmastoid approach.

The choice of donor nerve is dictated by the length of the resected facial nerve. Usually, the greater auricular nerve provides adequate length for repair. However, should a longer segment be needed, a sural nerve is harvested (see the next section, “Sural Nerve Graft”). The surrounding areolar and adventitial layers should be débrided from the

end of the nerve graft and the thin epineurium left for suturing



(see [Video 121-4](#)



).

In this combined approach the tegmen tympani is removed to expose the tumor and the course of the facial nerve. As mentioned in the previous section, this area is repaired with bone taken from the middle fossa craniotomy plate. Care must be taken to not compress the facial nerve graft or to impede ossicular chain reconstruction. Again, the IAC must be diligently packed with fat or muscle to prevent leakage of CSF from the IAC into the middle ear space. Wound closure follows as described in the previous section.

Sural Nerve Graft

The sural nerve is a cutaneous sensory nerve that innervates the posterolateral aspect of the foot and back of the leg (Fig. 121-15). It is identified 2 cm posterior to the lateral malleolar process and deep to the lesser saphenous vein. The sural nerve is of greater diameter than the auricular nerve and has a higher neural population. There are two methods for harvesting the nerve. The first is by multiple stepped transverse incisions over the posterolateral aspect of the leg and blind dissection of the nerve with a vein stripper (Fig. 121-16A and B). The other technique dissects the nerve with a continuous vertical incision. This method creates more postoperative morbidity but provides better control of bleeding and minimizes trauma to the nerve.

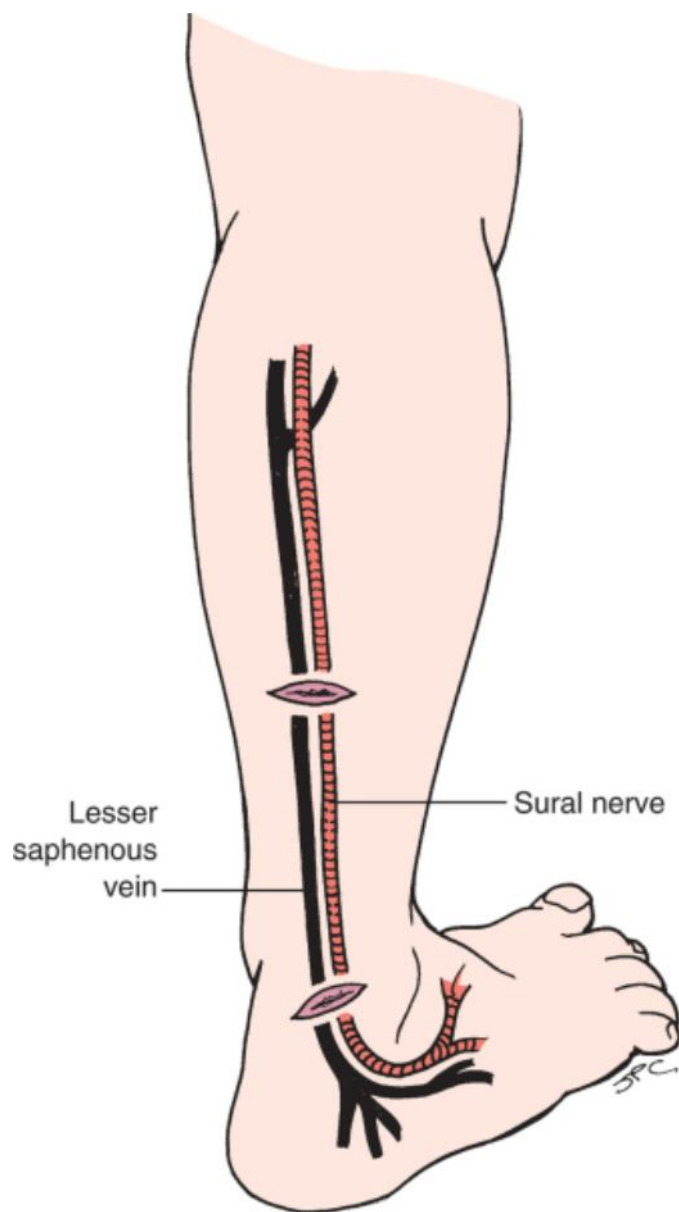


Figure 121-15 Landmarks and anatomy of the sural nerve. Note the relationship to the lesser saphenous vein and lateral malleolus.

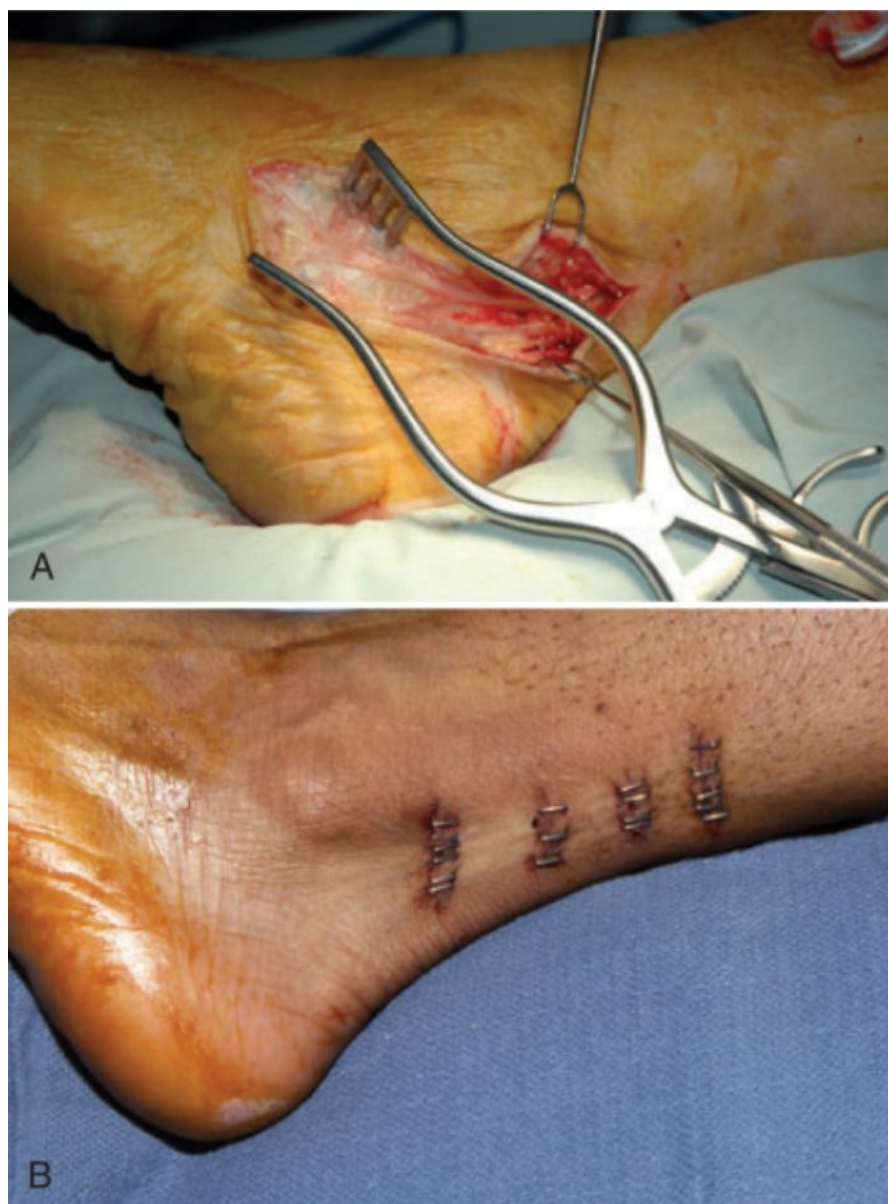


Figure 121-16 A, Surgical exposure for harvesting a sural nerve graft. B, Closure of incisions with a stair-step approach.

Retrosigmoid Approach

It is unusual to preoperatively diagnose a neuroma of the facial nerve that is isolated to the posterior fossa and IAC. An enhancing tumor in this area would be most consistent with an acoustic neuroma. The operative approach and techniques are detailed in Chapter 124. Primary anastomosis is not feasible when surgical exposure is limited to the posterior fossa. A translabyrinthine approach would be required to mobilize the facial nerve in the temporal bone. Cable nerve grafting in this area is also technically difficult, especially when tumor extends out to the fundus, and may necessitate a combined transmastoid and retrosigmoid approach.

Stereotactic Radiosurgery

Fractionated external beam radiation therapy has been the conventional method for radiation treatment. Newer techniques using a frame-based linear accelerator, gamma radiation from cobalt delivered through a gamma knife, or electron beam from a mobile linear accelerator such as the CyberKnife are available for single or multiple focused treatments (fractions) and provide millimeter accuracy in delivery of treatment. We prefer the CyberKnife system for various reasons. It is a mobile linear accelerator that delivers photon beams measured in megavolts (Fig. 121-17). It is a frameless system that avoids the minor morbidity of placing a head frame on the patient, thus making it ideal for fractionated treatment. More important, the system allows treatment of tumors that extend below the skull base. The gamma knife system has its field of delivery limited to the head and skull base.



Figure 121-17 CyberKnife Robotic Radiosurgery system manufactured by Accuray, Sunnyvale, CA.

Both CT and MRI are performed, fused, and displayed by overlapping the images. The tumor map is contoured so that the field of interest is outlined by using information from both the CT and MRI scans, although treatment is based on CT confirmation. The soft tissue signal from MRI allows the tumor to be outlined. The bony anatomy of the temporal bone is also used to verify the location of the tumor and identify other important structures to be protected from the full dose of delivered radiation. These are termed critical structures. At our institution, this process of contouring the tumor is done jointly by both the surgeon and the radiation oncologist. The planned treatment is then reviewed with the radiation physicist. The tumor is irradiated with 18 to 21 Gy to the 80% isodose line in three fractions. For 18 Gy, each treatment fraction would be 6 Gy given during three sessions delivered every other day. The dose at the margin of the tumor is usually close to the prescription dose of 18 to 21 Gy, unless there is a critical structure immediately adjacent to the tumor. Minimizing treatment to a critical structure would lower the dose at the margin of the tumor. The maximum tumor dose is 22.5 to 26.25 Gy at the 100% dose. A mask made of Aquaplast is fit tightly to the patient's head, face, and neck to provide immobilization. Each treatment is given for approximately 30 minutes delivered every other day. No sedation, intravenous fluids, or hospitalization is necessary.

Conventional external beam radiation therapy may incur long-term complications such as osteonecrosis, chronic otitis externa, breakdown of skin, eustachian tube dysfunction, and the potential development of a malignant tumor. Acute cranial nerve dysfunction (hearing loss, imbalance or vertigo, facial paresis, vocal fold weakness, dysphagia) can occur and may be transient. The use of focused stereotactic radiation therapy should minimize these risks.

POSTOPERATIVE MANAGEMENT

Unless medical conditions indicate otherwise, patients undergoing transmastoid or extratemporal surgery do not require intensive care management in the postoperative period. In contrast, patients undergoing a translabyrinthine, suboccipital, or middle fossa approach should be observed in an intensive care setting. Neurologic symptoms and potential complications are monitored for 24 hours. The compression dressing is maintained for 3 days. If an abdominal drain was placed at the site of a fat graft, it is removed when drainage is minimal.

Patients are relatively immobilized when a sural nerve graft is taken. An elastic wrap is left in place for approximately 1 week. Active ambulation in the immediate postoperative period is uncomfortable and discouraged. Physical therapy consultation and the use of a walker are most helpful for early rehabilitation. Sutures or staples

are removed 7 to 10 days after surgery.

Paresis or paralysis of the face is often present postoperatively. Critical attention is directed toward protection of the cornea during the recovery period. Lubrication with artificial tears during the day and ointment at night is prescribed. A moisture chamber is also beneficial to maintain humidity around the eye. If a nerve-grafting procedure was undertaken, facial recovery is not anticipated for at least 6 months, depending on the site of nerve sacrifice and repair. The patient is asked to attempt eye closure while noting the degree of scleral and corneal show (Fig. 121-18). If eye symptoms are bothersome and corneal protection is compromised, a gold weight is temporarily affixed to the upper lid to determine whether lid closure is facilitated and irritation is alleviated. A variety of gold weights (0.6 to 1.6 g) are sampled to determine the optimal size (Fig. 121-19). Insertion of the gold weight is subsequently performed during the hospital stay. Topical tetracaine hydrochloride (Pontocaine) ophthalmic drops are placed in the eye. A scleral shield is placed over the cornea for protection. Intravenous sedation is given, and a local anesthetic is injected into the upper eyelid.



Figure 121-18 Left facial paralysis with incomplete eye closure. A good Bell phenomenon is present, with the lower cornea just visible.



Figure 121-19 A gold weight (1.2 g) is taped to the upper eyelid to provide complete eye closure.

The incision in a supratarsal crease is taken through the skin and orbicularis oculi muscle. Westcott scissors are used to dissect a plane deep to the orbicularis oculi muscle and superficial to the tarsal plate. A pocket is created to insert the appropriate size of gold weight, as determined preoperatively (Fig. 121-20). A 1.2-g gold weight is well tolerated by most patients. The gold weight is sutured to the tarsal plate with 8-0 nylon. The orbicularis oculi muscle is reapproximated with 6-0 Dexon, and the skin is closed with running fast-absorbing catgut. A 1/8-inch Steri-Strip is applied over the incision.

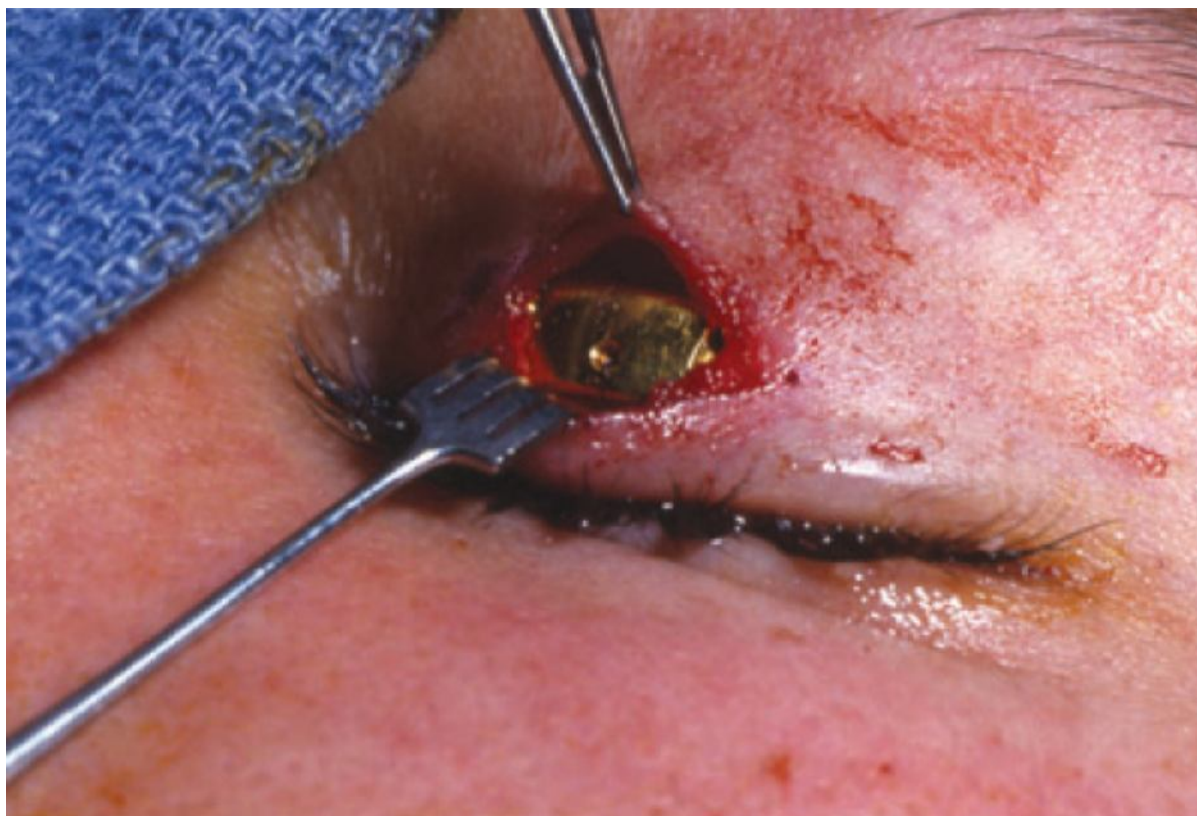


Figure 121-20 A pocket is created deep to the orbicularis oculi muscle and superficial to the tarsal plate for insertion of a gold weight.

Patients not complaining of or demonstrating preoperative vestibular symptoms may experience acute nausea and become vertiginous if the vestibular nerve has been sacrificed or the otic capsule is entered during the procedure. Supportive care is provided over the first few days, pending central compensation for the unilateral vestibular injury.

Patients who are stable and able to ambulate are discharged within 2 to 6 days postoperatively, depending on whether an intracranial approach was undertaken. Hearing is preliminarily assessed by gross measures such as whispering or using a telephone. Tuning forks are also useful. If the middle ear was not involved but surgery was in close proximity to the cochlear nerve or artery or the otic capsule, an audiogram is obtained while the patient is in the hospital. If ossicular chain reconstruction was performed, a formal audiogram is obtained 4 to 6 weeks after surgery. Sutures are removed from the operative site 7 to 10 days after surgery.

There are several choices regarding the optimal radiologic method for monitoring the tumor postoperatively. If details regarding the bony anatomy need to be critically assessed, CT scanning is appropriate. Otherwise, MRI provides important information on soft tissue densities. Fat suppression techniques will reduce the signal of adipose tissue and more readily differentiate tumor from a fat graft. A baseline scan is obtained 3 months postoperatively and repeated 1 year later. Any postoperative changes should be less intense and contracted than on the baseline scan. The schedule for future scans is based on whether complete tumor removal was achieved and the findings on the 15-month postoperative scan.

Hearing status is assessed during the postoperative period. An audiogram is performed to determine whether a conductive, sensorineural, or mixed hearing loss remains. Significant conductive loss can be repaired electively if the middle ear space was not obliterated during wound closure. Hearing aid amplification may also be offered for conductive or sensorineural loss if word recognition is good.

Return of facial function is monitored over time. Evidence of reinnervation is initially manifested by an increase in facial tone and redefinition of the nasolabial fold. Recovery may occur within 6 weeks if tumor removal permitted preservation of the facial nerve. Facial recovery can take 12 to 15 months if nerve or cable grafting was performed in the IAC/CPA region. When return of function is delayed close to one year, needle electromyographic recordings may demonstrate polyphasic potentials indicating that reinnervation is taking place. This reassures both the surgeon and the patient that more time is needed to observe for ongoing recovery.

COMPLICATIONS

Vascular Injury

Intraoperative arterial or venous bleeding should be readily recognized and managed. The surgeon should be aware of the anatomic variability of the sigmoid sinus and jugular bulb. The sigmoid sinus may be located extremely laterally or anteriorly within the mastoid as the facial nerve is approached. This applies to any transmastoid procedure. Furthermore, approaches that require dissection medial to the plane of the vertical segment of the facial nerve may encounter the jugular bulb. Constant awareness of this structure will minimize inadvertent damage. Injury to the sigmoid sinus usually responds to local compression with Surgicel and neuroplasty. The carotid artery should not be subject to injury with any of the approaches described unless the cochlea has been extensively eroded by tumor. This would be evident on preoperative images, and appropriate meticulous surgical technique should be used. During a translabyrinthine or retrosigmoid approach, the anterior inferior cerebellar artery should be identified and protected. Injury to this vessel results in anacusis, vertigo, facial paralysis, hemiparesis, and possibly death.

Cerebrospinal Fluid Leak

The potential morbidity of CSF otorrhea, rhinorrhea, or a cutaneous fistula is the development of meningitis. Meningitis is a serious complication that if not recognized and treated early, may prove fatal. High clinical suspicion for a leak or meningitis is maintained throughout the postoperative period. The patient is instructed to avoid straining or sneezing with a closed mouth and to report any suspicious watery drainage. A leak through the wound is repaired by placing interrupted sutures across the dehiscence site. Topical collodion liquid is another adjunctive method for sealing the incision. A compression dressing is reapplied. If the wound is not ballotable and rhinorrhea is present, a subarachnoid lumbar drain is placed for 3 to 5 days. Drainage is maintained at 6 to 10 mL/hr (~150 to 240 mL/day). Almost all CSF leaks respond to this regimen. Failure to stop the leak will require re-exploration and possible obliteration of the eustachian tube.

Hearing Loss and Vertigo

Depending on the location of the tumor and the operative approach used, hearing loss may occur. Complete sensorineural loss (anacusis) is a known risk rather than a complication when a tumor is located in the IAC and CPA. Similarly, if the CT scan demonstrates otic capsule erosion, there is a greater likelihood of hearing compromise. Persistent conductive hearing loss would occur from unrecognized ossicular chain involvement, unsuccessful ossicular chain reconstruction, or persistent fluid in the middle ear space.

Unless there has been ongoing vestibular loss, sudden sacrifice of the vestibular nerves or injury to the labyrinth results in acute vestibular vertigo. Supportive measures are provided, and assuming that complete denervation has occurred, central compensation should provide return of balance function over the next 6 weeks. Patients experiencing difficulty in recovery of balance are referred to a physical therapist trained in vestibular rehabilitation.

Facial Nerve Repair

Certain principles must be adhered to when facial nerve repair is undertaken. The ends of the nerve or cable graft should be sharply freshened before anastomosis. The proximal end of the facial nerve in the CPA, bathed in CSF, is not likely to become desiccated. Immediately after tumor removal, if the facial nerve is resected, isolating plus wrapping the proximal stump in moistened Gelfoam protects the nerve and eliminates confusion regarding its true identity when the anastomosis is performed. There should be no tension on the anastomosis. If during a translabyrinthine approach a transposed nerve is reapproximated, it should be further supported with the fat used for obliteration and packing. Monofilament nylon suture (9-0 or 10-0) is used for the proximal neurorrhaphy when the dural extension or epineurium is absent. An epineurium-to-epineurium repair is performed more peripherally with 8-0 suture.

PEARLS

- Up to one half of all patients with primary tumors of the facial nerve will have normal facial nerve function.
- Both CT and MRI with gadolinium enhancement are essential in establishing the diagnosis of primary tumors of the facial nerve.
- Resection of the nerve and cable grafting are often required to achieve complete tumor resection, and the surgeon should always be prepared for this possibility.
- MRI with fat suppression should be used postoperatively to evaluate for tumor recurrence.
- Stereotactic radiosurgery appears to be an effective means of tumor control for facial nerve neuromas and should be considered in elderly patients, patients who are higher surgical risks, patients with good facial nerve function, and those with only one hearing ear.

PITFALLS

- Patients with a primary tumor of the facial nerve and good preoperative facial function present a difficult management problem, and consideration should be given to surgical decompression, stereotactic radiosurgery, or observation.
- Attempts to resect a tumor of the facial nerve in an only-hearing ear should be avoided if the tumor involves the IAC or cochlea.
- Resection of a hemangioma of the facial nerve may involve significant bleeding, but use of bipolar cautery should be avoided near the facial nerve.
- A primary tumor of the facial nerve may be identified intraoperatively after having been diagnosed preoperatively as an acoustic neuroma; in this case, the surgeon should notify the patient's family and proceed with tumor resection.
- Failure to plug the IAC with fat or muscle during a middle fossa approach may lead to CSF rhinorrhea or otorrhea.
- Tumor may involve a long length of the nerve not detected by MRI.

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