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# Section 15 – TEMPORAL BONE

# Chapter 123 – Carcinoma of the Temporal Bone

#### Barry E. Hirsch, C.Y. Joseph Chang, Stephanie Moody Antonio

Primary malignant tumors arising in the temporal bone are rarely seen in the typical community practice of otology. The external auditory canal (EAC) is the most common site of origin. Squamous cell carcinoma of the EAC has a reported incidence of about 1 per one million per year and is the major focus of this chapter. Primary malignancies of the temporal bone may also arise in the middle ear and mastoid and rarely in the jugular foramen and petrous apex. Secondary tumors of the temporal bone commonly develop as a result of spread from adjacent structures such as the concha, pinna, and parotid gland. Metastasis to the temporal bone from distant sites can also occur, often at the petrous apex. Each region of the temporal bone is affected by unique tumor types, clinical manifestations, and examination findings. Because it is common for a tumor to involve overlapping anatomic regions, no area can be considered in isolation, and broad understanding of the temporal bone and its surrounding structures is vital.

Basal cell carcinoma is the most common tumor of the pinna and concha and occurs four times as frequently as squamous cell carcinoma. Melanoma and Merkel cell cancer are less common. Melanoma of the pinna and EAC account for about 10% of head and neck melanomas.<sup>[1]</sup> Malignancies arising from the medial EAC and middle ear include squamous cell carcinoma, adenocarcinoma, adenoid cystic carcinoma, and rarely, Merkel cell carcinoma, lymphoma, and malignant melanoma. Rhabdomyosarcoma originates in the middle ear and mastoid and is the most common malignancy of the temporal bone in children. Other tumor types occurring in the middle ear and mastoid include melanoma, squamous cell carcinoma, endolymphatic sac tumor (papillary adenocarcinoma), adenocarcinoma, and jugular foramen. Unique tumor types in the petrous apex include chondrosarcoma, neuroendocrine carcinoma, meningioma, and hemangiopericytoma. Adenoid cystic carcinoma, mucoepidermoid carcinoma, sarcoma, plasmacytoma, fibrosarcoma, multiple myeloma, and osteosarcoma have also been reported in the temporal bone. Metastasis to the temporal bone from breast, colon, and renal cell carcinomas have all been reported.

A patient with long-standing otorrhea, otalgia, or a mass in the EAC should be presumed to have carcinoma until proved otherwise. Bleeding, hearing loss, facial paralysis, a periauricular swelling or mass, lymphadenopathy of the parotid or upper part of the neck, and lower cranial nerve palsies may indicate a cancer lesion in the temporal bone. It is important to be mindful of the possibility of malignant otitis externa because its clinical picture is very similar to cancer in this site.

Sun exposure contributes to the development of basal cell and squamous cell carcinomas of the pinna and lateral EAC. A genetic predisposition to skin cancer may also exist and is manifested by the development of skin cancer in sites not exposed to sunlight, as well as in sun–exposed areas. Chronic otitis media and cholesteatoma are common in patients with cancer of the temporal bone and have been implicated as etiologic factors. Squamous metaplasia resulting from chronic inflammation could result in dysplastic changes and lead to cancer.<sup>[2–4]</sup> Human papillomavirus has been implicated in squamous cell carcinoma of the middle ear.<sup>[5–7]</sup> Included in these series are cases of temporal bone cancer induced by radiation exposure. Patients with these cancers had a particularly poor outcome.

# PERTINENT TEMPORAL BONE ANATOMY

The pinna and temporal bone have traditionally been divided into anatomic subunits that correspond to the depth of resection of the various procedures used for surgical correction. The EAC is divided into cartilaginous (lateral) and bony (medial) portions. The cartilaginous portion is a poor barrier against the spread of neoplasm. Tumors in this area can erode through skin, cartilage, and soft tissue to invade the parotid gland anteriorly or the concha and postauricular sulcus posteriorly (Fig. 123-1A and B [1 and 2]). The fissures of Santorini are vertical fissures in the cartilaginous EAC that allow direct access of cancer from the skin of the EAC to periparotid tissues (Fig. 123-2). In contrast, the bony canal provides a more resistant barrier to direct invasion. Cancer in this area tends to track medially through the tympanic membrane into the middle ear and mastoid spaces (see Fig. 123-1A and B [3]). The foramen of Huschke provides a potential pathway between the bony EAC and the soft tissues of the parotid area

and temporomandibular joint (TMJ). This structure is a defect in the anterior inferior tympanic ring that usually closes by 5 years of age but persists in 7% of adults (Fig. 123-3).[8]





Figure 123-1 Axial (A) and coronal (B) temporal bone anatomy identifying pathways of the spread of cancer. Cancer can spread (1) anteriorly through the cartilaginous ear canal into the parotid gland; (2) through the concha into the postauricular sulcus; (3) through the tympanic membrane into the middle ear; (4) posteriorly into the mastoid; (5) into the anterior mesotympanum to the carotid artery and eustachian tube; (6) into the inner ear through the round window or otic capsule; (7) along the facial nerve into the infratemporal fossa; (8) through the mastoid, posterior fossa dura, and sigmoid sinus; and (9) beneath the skull base to the jugular fossa, carotid artery, and lower cranial nerves.



Figure 123-2 The fissures of Santorini are vertical fissures in the cartilaginous external auditory canal. Tumor spreads easily through these pathways into the periparotid soft tissues.



Foramen of Huschke

**Figure 123-3** Foramen of Huschke. During development, two prominences (greater and lesser tympanic spines) extend from the tympanic ring and separate the space into the external auditory canal and the foramen of Huschke. These processes approach each other and fuse during the first year of life. With continued growth of bone from the inferior part of the tympanic bone, the foramen eventually fills with bone. Occasionally, the foramen remains patent.

(Adapted from Anson BJ, Donaldson JA: Surgical Anatomy of the Temporal Bone. The Middle Ear. Philadelphia, WB Saunders, 1967, p 122.)

The middle ear and mastoid cavities provide minimal resistance to the spread of cancer. Anteriorly, cancer may invade through tissue surrounding the eustachian tube and involve the internal carotid artery (ICA), Meckel's cave, and the cavernous sinus (see Fig. 123-1A and B [5]). Superiorly, cancer can penetrate the tegmen tympani and tegmen mastoideum to involve the middle fossa dura and temporal lobe (see Fig. 123-1A and B [4]). Medially, the otic capsule provides a good barrier to the spread of cancer, but several areas are vulnerable. The round and oval windows can be penetrated and result in invasion through the vestibule into the internal auditory canal (IAC) and

posterior fossa (see Fig. 123-1A and B [6]). In addition, the facial nerve can provide a pathway for the spread of cancer intracranially through the petrous apex, as well as into the infratemporal fossa (ITF) through the stylomastoid foramen (see Fig. 123-1A and B [7]). Posteriorly, cancer can invade the posterior fossa plate, dura, and sigmoid sinus (see Fig. 123-1A [8]). Finally, the air cell system of the temporal bone provides a multidirectional highway for the spread of cancer. Cancer within the middle ear and mastoid follows air cells as a pathway into the petrous apex and IAC and inferiorly into the jugular bulb and can penetrate the ITF and posterior fossa (see Fig. 123-1A and B [5, 6, and 9]).

Lymphatic drainage of the EAC extends anteriorly to the parotid and periparotid lymph nodes, inferiorly to the internal jugular lymph nodes, and posteriorly to the mastoid lymph nodes. The lymphatic drainage pathways from the middle ear and mastoid have not been well elucidated, but they are believed to be of only minor importance, because nodal metastasis from a malignancy limited to the middle ear and mastoid is rare.

# STAGING OF TEMPORAL BONE MALIGNANCIES

A systematic and useful staging system for cancer of the temporal bone has not been universally accepted. A major obstacle to the development of a useful preoperative staging system in the past was the difficulty of cancer mapping because the medial portion of the cancer could not be visualized and spread to the skull base and intracranial cavity was difficult to evaluate. The development of multiaxial imaging modalities such as computed tomography (CT) and magnetic resonance imaging (MRI) has helped overcome this problem. A useful system proposed by the University of Pittsburgh uses imaging to determine the extent of tumor involvement and has become widely accepted for squamous cell carcinoma.<sup>[9–14]</sup>

The staging system for cancer of the temporal bone involves the same tumor-node-metastasis (TNM) model used for staging cancers in other anatomic sites. Tumor extent is defined as T1 through T4, depending on the degree of bone, cartilage, and soft tissue involvement of the ear canal and tympanic, mastoid, and petrous bones (Table 123-1). The N and M portions of the classification and the staging system follow the same format as for other sites according to the American Joint Committee on Cancer.[15] In our recent series of patients with squamous cell carcinoma of the temporal bone staged in this manner, the 2-year disease-free survival rate was 100% for T1, 100% for T2, 56% for T3, and 17% for T4, thus demonstrating that this staging system can be predictive of outcome.<sup>[9]</sup> We modified the Pittsburgh staging system after further review of patients from an extended series.<sup>[16]</sup> In the modified staging system, facial nerve weakness is considered an indicator of T4 cancer and has a worse prognosis than T3 cancer. We observed that facial nerve paresis did not occur in cancers classified as limited T1, T2, or T3 lesions. Involvement of the facial nerve would otherwise be classified as T4 based on the anatomic area of involvement, including the medial wall of the middle ear (horizontal segment), extensive bony erosion in the mastoid (vertical segment), or involvement of the stylomastoid foramen. In the T4 group, survival was similar in patients with and without facial paralysis. A few reports have used the modified staging system, [16,17] but further study on the importance of facial paralysis in cancer of the temporal bone is needed to support standardization of this staging system.

#### Table 123-1 -- CARCINOMA OF THE TEMPORAL BONE: TUMOR STAGING SYSTEM

T1 Tumor limited to the EAC; no bone erosion or soft tissue extension

T2Tumor with limited bone erosion to the EAC or <0.5 cm of soft tissue involvement

T3Tumor with full-thickness EAC bone erosion, <0.5-cm soft tissue involvement, or tumor in the middle ear or mastoid

T4 Tumor eroding the cochlea, petrous apex, medial wall of the middle ear, carotid canal, jugular foramen, or dura; or >0.5-cm soft tissue involvement; or facial nerve paresis

EAC, external auditory canal.

# TREATMENT

The first systematic discussion of the management of cancer of the temporal bone was presented by Politzer in 1883.<sup>[18]</sup> Refinements in this approach have evolved over the past century. Radical mastoidectomy with radiation therapy was previously the recommended treatment, but cure rates were quite low. In 1954, Parsons and Lewis described the technique of en bloc subtotal temporal bone resection.<sup>[19]</sup> Lederman presented the first large series of patients treated by radiation therapy in 1965 and espoused the concept of com-bined therapy at a time when the role of radiation in treating cancer of the temporal bone was not yet well accepted.<sup>[20]</sup> In 1984, Graham and colleagues reported total en bloc resection of the temporal bone, including the ICA.<sup>[21]</sup>

Historically, overall 5-year survival with temporal bone cancer was low. In Conley and Novack's series of patients

undergoing surgical resection, the cure rate was 18%, but the operative mortality rate was 27%.[22] Lederman reported a cure rate of 33% at 5 years for combined surgical and radiation treatment versus 11% for radiation alone.<sup>[20]</sup> Despite advocating en bloc subtotal temporal bone resection and occasional adjuvant radiation therapy, Lewis and Page achieved a 28% cure rate.<sup>[23]</sup> The cure rate remains low despite the innovative work of surgeons and radiation oncologists and the use of aggressive therapy. Patients with cancer limited to the EAC have been reported to have an 85% to 100% disease-free survival rate with surgery with or without radiation therapy. <sup>[10,16,24,25]</sup> Cancer extending to the middle ear, mastoid, and soft tissues is associated with a poor outcome, and even with extensive surgery and radiation therapy, the survival rate is little more than 50%.

It has not been clearly established what treatment modalities provide the best outcome given the extent of disease. The relatively low incidence of cancer of the temporal bone makes randomized clinical trials impossible. As a consequence, every published study is essentially an analysis of a case series, with inconsistent treatment protocols often being used. This, along with the lack of a universal staging system and variability in surgical nomenclature, limit making use of published data in a meaningful analysis.

#### Surgical Therapy

Primary surgical therapy should be performed to remove the cancer in its entirety. The issue of whether piecemeal resection as opposed to en bloc resection compromises survival has not been resolved. It is clear that piecemeal resection of the temporal bone medial to the EAC is a more controlled operation than the en bloc technique is. Once the cancer has spread to the medial wall of the middle ear and invades the mastoid air cells, the prognosis becomes quite poor unless clear margins are achieved with radical therapy. A positive margin is associated with a very poor chance of cure.

The treatment program depends primarily on the extent of the cancer. The margins of resection for lateral, subtotal, and total temporal bone resection are illustrated in Figure 123-4. Cancer limited to the EAC (T1, T2) should be treated by lateral temporal bone resection with superficial parotidectomy and preservation of the facial nerve. Rarely, the cancer may be small, isolated in the lateral cartilaginous aspect of the EAC, and not eroding bone, thereby permitting sleeve resection of the external canal skin, lateral to the tympanic membrane. In practice, sleeve resection is probably not oncologically adequate. Cancer that has invaded the middle ear and mastoid cavities (T3) should be addressed by subtotal temporal bone resection. Total temporal bone resection (sometimes called radical temporal bone resection) is performed when cancer extends to the petrous apex. Cancer extending to the TMJ, neck, dura, or ITF (T4) will require resection of these structures. Although involvement of dura and brain is considered by some to indicate unresectable disease, Moffat and coauthors reported on two patients with squamous cell cancer involving the brain who survived after resection and radiation therapy.<sup>[13]</sup>



**Figure 123-4** Axial (A) and coronal (B) illustrations demonstrating (1) lateral resection of the temporal bone, (2) subtotal resection of the temporal bone, and (3) total resection of the temporal bone. The carotid artery is preserved in this illustration.

# Adjuvant Therapy

Radiotherapy as a single modality has not been very successful in curing any stage of carcinoma of the temporal bone. Although the additional benefit of postoperative radiation therapy has been questioned for limited tumors excised with adequate margins and for larger tumors with positive margins,<sup>[26]</sup> it is generally believed that combined therapy consisting of primary surgical resection followed by radiation therapy provides the best chance to control the cancer and improve survival.<sup>[10,13,16]</sup> We routinely recommend postoperative radiation therapy unless the cancer is well isolated in the EAC with very little bone erosion. Fifty to 60 Gy are delivered to the primary site, parotid region, ITF, and ipsilateral neck.

The role of chemotherapy is evolving. Traditionally, it has not been effective for primary treatment. A group of patients treated by preoperative chemoradiation therapy and temporal bone resection had better survival than did patients treated by surgery or radiation therapy alone.<sup>[27]</sup> In cases in which medical conditions preclude a safe procedure or the extent of disease prohibits a reasonable chance of adequate resection, palliative radiation therapy and chemotherapy may be offered.

#### Management of the Parotid Lymph Nodes

Parotidectomy is routinely performed in conjunction with temporal bone resection. In this way, the first echelon of lymphatic drainage for the anterior EAC may be reviewed. Ideally, the parotid is resected en bloc with the temporal bone. However, for T1 and T2 cancers, superficial parotidectomy is sufficient. For T3 and T4 cancers

and if the facial nerve is to be resected, total parotidectomy is performed.

#### **Neck Dissection**

Supraomohyoid neck dissection is performed in most cases. The lymphatic drainage of pinna and EAC lesions is not entirely predictable but will often extend to the upper jugular nodal chain. Also, exposure of cervical structures provides safe control of the great vessels, if needed. In a clinically N0 neck, neck dissection can be done as a staging procedure to assist in the decision as whether to proceed with postoperative radiation therapy. The survival benefit provided by elective neck dissection is unclear. Metastasis to the lymph nodes is a significant indicator of a poor prognosis. In Moffat and associates' report of 39 patients with squamous cell carcinoma of the temporal bone, 9 (23%) had positive nodes in the neck. All these patients succumbed to the disease within 27 months.<sup>[13]</sup> In our experience, neck dissection and parotidectomy are commonly carried out at the time of temporal bone resection, which provides good local/regional control but does not seem to affect survival.<sup>[16]</sup>

# **PATIENT SELECTION**

In general, less extensive cancers are more readily cured than advanced cancers that have invaded the middle ear and mastoid. A major difficulty in the management of cancer of the temporal bone has been the small percentage of patients in whom the diagnosis is made early in the course of their disease. Because chronic otitis media or otitis externa with purulent otorrhea is very common, otorrhea caused by cancer is often assumed to be secondary to inflammatory disease. Other symptoms of hearing loss and tinnitus are not specific to cancer of the temporal bone. All too frequently, biopsy is delayed until the disease is no longer limited to the EAC (Fig. 123-5).



Figure 123-5 This patient with pathology of the right external auditory meatus failed to respond to medical therapy. Early biopsy in this case is warranted.

The signs and symptoms of cancer arising in the temporal bone, especially early in its course, may be difficult to distinguish from those of chronic otitis externa and otitis media. However, the presence of persistent, deep-seated, unrelenting pain may be a differentiating symptom because appropriate treatment of routine chronic ear infections usually provides relatively rapid relief of discomfort. Chronic petrositis (Gradenigo's syndrome) is associated with deep chronic pain and otorrhea, although a mass lesion is absent. Spontaneous bleeding is also an unusual feature in purely infectious ear disease unless canal cholesteatoma has caused bone erosion with neovascularization. Malignant external otitis, which can closely mimic the signs and symptoms of malignancy, can be difficult to distinguish from cancer. Cases of malignant external otitis that were eventually diagnosed as cancer have been reported, thus underscoring the importance of early biopsy in suspicious cases.<sup>[28]</sup> In summary, any condition of the EAC that does not respond to topical and systemic antibiotic treatment should be suspected of harboring a malignancy, and biopsy should be performed early. Biopsy is performed by direct incision; a staging mastoidectomy is not appropriate because it reduces the reliability of staging radiography and may compromise the surgical margins of resection.

Depending on the stage of disease, surgical treatment of cancer arising in the temporal bone is potentially a major undertaking. The risk of morbidity and mortality increases with factors such as advanced age and medical conditions such as coronary artery disease, pulmonary disease, diabetes mellitus, and peripheral vascular disease. However, because combined-modality therapy that includes major surgery appears to be the only treatment that offers any reasonable chance of control of cancer, there is no absolute medical contraindication to surgical treatment. Aggressive preoperative, perioperative, and postoperative care must be given to minimize the chance of a major medical complication. Each patient and physician will need to decide whether the proposed treatment and its potential benefit are worth the risk. For example, a patient with cancer of the temporal bone who has a life expectancy of less than one year for other medical reasons will probably derive minimal benefit from aggressive treatment.

The second issue regarding the decision to proceed with treatment is the extent of the cancer. Extension into certain areas such as the cavernous sinus and brain parenchyma makes total tumor extirpation nearly impossible,<sup>[29]</sup> and the possibility of cure becomes unlikely, even with the addition of aggressive adjuvant radiation therapy and chemotherapy. In these cases there is little potential for improving the patient's prognosis. Involvement of the internal carotid canal is also problematic but it is not an absolute contraindication to total resection of cancer. Some patients will be able to tolerate resection of the ICA but will require preoperative studies to determine eligibility, as described in a later section.

# PREOPERATIVE PLANNING

History, physical examination, and radiologic imaging remain the most important methods of determining the extent of the cancer. Evidence of the spread of cancer beyond the anatomic limits of the EAC is evaluated. The cancer and associated inflammation often obliterate the EAC, thereby restricting examination of the tympanic membrane and middle ear. However, the presence of severe conductive hearing loss, unilateral sensorineural hearing loss, otitis media, vertigo, taste dysfunction, or facial nerve paralysis may indicate invasion into the middle ear, eustachian tube, and otic capsule. The presence of Horner's syndrome, transient ischemic attacks, or syncope may indicate involvement of the ICA. Trismus, TMJ dysfunction, and lower cranial nerve palsies suggest the spread of cancer into the jugular foramen ITF. The presence of seizures, dysphasia, and other cognitive deficits warrant investigation for intracranial spread of tumor. The patient should be examined carefully for physical evidence of a mass in the neck, parotid gland, soft palate, and postauricular region. An audiogram is obtained to establish the type and degree of hearing loss. The status of the hearing is important because preservation of hearing is possible only if lateral resection of the temporal bone is performed. However, the amount of resection is determined by the extent of the cancer and not by the degree of residual hearing function.

The full extent of the cancer should be mapped in accordance with physical findings and multiaxial imaging consisting of CT for delineation of the bony anatomy of the temporal bone and MRI for visualization of soft tissues and intracranial structures. The initial radiologic study for definition of the tumor is high–resolution CT with and without contrast enhancement. It can accurately map tumors in the temporal bone and predict invasion of cancer outside the EAC. CT is especially well suited for detecting bony abnormalities. Extension of tumor into the bone of the EAC, middle ear, mastoid, labyrinth, eustachian tube, carotid canal, sigmoid sinus, ITF, and intracranial cavity can be seen (Fig. 123-6). MRI with gadolinium enhancement is superior to CT in certain respects, especially in delineating soft tissue detail. MRI is more accurate in differentiating effusion in the middle ear and mastoid from cancer, although such differentiation may be difficult with either modality. MRI is significantly better than CT in determining the interface between cancer and normal tissue. Therefore, MRI is quite sensitive in detecting invasion of cancer into the dura and soft tissues of the ITF (Fig. 123-7) and temporomandibular region (Fig. 123-8). Usually, both MRI and CT will be required to adequately stage the disease process.



**Figure 123-6** Computed tomography scan of extensive squamous cell carcinoma of the temporal bone with erosion through the bone of the right external auditory canal into the anterior soft tissue (arrow), middle ear, and mastoid (arrowheads). (Courtesy of M. Arriaga, M.D.)



**Figure 123-7** Magnetic resonance image of the same patient as in Figure 123-2 with carcinoma of the temporal bone. A T1-weighted contrast-enhanced image demonstrates enhancement of the middle ear and infratemporal fossa (*arrows*). (*Courtesy of M. Arriaga, M.D.*)





Once a tumor map has been created, the need for further studies will become apparent. Encroachment of cancer in the region of the ICA should be investigated with angiography to help determine whether invasion into the vessel has occurred. Because there is a significant risk of injury to the carotid artery if tumor resection is performed near this vessel, the patient's tolerance of permanent occlusion of the ICA should be determined preoperatively by balloon test occlusion with xenon/CT.<sup>[30]</sup> The venous phase of the angiogram demonstrates the venous anatomy of the brain. Specifically, the location of the vein of Labbé and the dominance of the ipsilateral sigmoid and jugular drainage are noted. In a few patients, the ipsilateral venous drainage is so dominant that sacrifice of the jugular vein or sigmoid sinus on this side can lead to venous cerebral infarction. Thus, careful consideration of the patency of the contralateral side must be undertaken if it is anticipated that the sigmoid sinus or jugular vein may be sacrificed.

The presence of metastatic cancer, especially in the neck, should be detected before therapy. Metastasis to the neck is unusual in cancer of the temporal bone, but a thorough physical examination and CT or MRI with contrast enhancement should detect lymph node involvement. Spread of cancer to the lymph nodes in the superficial lobe of the parotid gland is more common, so the superficial lobe of the parotid gland is usually removed during surgery with preservation of the facial nerve. If the pathology is squamous cell carcinoma, a metastatic evaluation consisting of chest radiography and liver function testing is adequate. In malignant tumors with a high predilection for systemic spread such as melanoma, CT scanning of the chest, abdomen, and pelvis, as well as whole-body bone scanning, should be performed. The recent introduction of positron emission tomography (PET)/CT scanning has made identification of metastasis more accurate.

Preoperative staging determines the scope of the planned procedure and the necessity for a team approach. Neurosurgical participation is appropriate when surgery requires intracranial resection. Similarly, wound closure after tumor removal may necessitate transfer of regional or distant tissue, thus warranting consultation with a reconstructive surgeon.

Surgical treatment of cancer of the temporal bone usually requires a long duration of anesthesia and can entail major blood loss and fluid shifts, intracranial manipulation, and postoperative stress, including fluid and electrolyte abnormalities, pulmonary and cardiac stress, coagulation abnormalities, and the possibility of aspiration. The patient's medical status should be optimized before surgery. Significant conditions such as coronary artery disease, chronic obstructive pulmonary disease, diabetes mellitus, bleeding disorders, and malnutrition should be identified and treated preoperatively to minimize potential major medical complications.

# SURGICAL TECHNIQUES

The surgical approach is dictated by the location and extent of cancer relative to the temporal bone. Lateral resection of the temporal bone is performed for cancer limited to the EAC (T1, T2). Subtotal resection of the temporal bone is performed for cancer that has penetrated into the middle ear and mastoid cavities (T3). The medial extent of resection incorporates the otic capsule. Extratemporal dissection is dictated by the areas of cancer infiltration and is specifically focused toward the ITF, jugular bulb, or dura, as indicated. Invasion into the petrous apex by cancer is managed by total resection of the temporal bone.

General oral endotracheal anesthesia is achieved with inhalational agents, intravenous narcotics, and hypnotics. Neuromuscular blocking agents are avoided during the procedure to permit monitoring of motor nerve activity. A short–acting agent, such as succinylcholine, is frequently used at the start of the procedure for safe induction and intubation. The patient is placed supine with the head turned toward the contralateral ear. Sequential compression stockings are worn to prevent deep venous thrombosis. Cranial nerves VII, IX, X, and XI are monitored if they need to be identified and preserved. Monitoring for cerebral and brain stem traction or injury during subtotal or total temporal bone resection is achieved with somatosensory evoked potentials and contralateral auditory brain stem response recordings, respectively. Regions to be prepared include not only the primary surgical site but also any areas needed to provide graft material, such as the abdomen for fat; neck or leg for nerve grafts; and chest, abdomen, or back for pedicled or free flaps. A third–generation cephalosporin with cerebrospinal fluid (CSF) penetration, such as ceftriaxone, is administered perioperatively. Furosemide (Lasix), 10 mg, and mannitol, 0.25 to 0.5 g/kg, are given intravenously 30 minutes before the intracranial portion of the procedure to decompress the subarachnoid space if retraction of the temporal lobe or cerebellum is anticipated.

#### Lateral Temporal Bone Resection

The anatomic unit removed with lateral resection of the temporal bone is the EAC. Boundaries of resection include the middle ear cavity medially, the TMJ capsule anteriorly, the zygomatic root superiorly, the mastoid cavity posteriorly, and the ITF inferiorly. Laterally, the resection can include the concha, as well as portions of the pinna, depending on the extent of the cancer. Occasionally, the entire pinna must be removed. Lateral resection of the temporal bone is appropriate for cancer limited to the EAC without penetration medially through the tympanic membrane. Spread of cancer anteriorly or inferiorly into the TMJ or ITF can be treated by extension of the surgical resection into these areas. Significant spread of cancer into the middle ear or mastoid cavity requires more extensive resection of the temporal bone.

The lateral margins of resection depend on the location of the cancer. In general, if the cancer is situated within the EAC, only the conchal bowl is included. The proposed incision sites are injected with 2% lidocaine with 1:100,000 epinephrine for hemostasis. A circumferential incision is made around the concha for cancer involving only the EAC, and the tragus is left with the skin flap if the cancer does not involve the lateral and anterior external auditory meatus. Otherwise, the tragus is included with the lateral circumferential skin margin (Fig. 123-9). If the subcutaneous layer of the concha, tragus, and antihelix is involved by cancer, more of the central portion of the pinna is resected (Fig. 123-10). If a substantial portion of the pinna is involved, the entire pinna is resected.



Figure 123-9 Postauricular and meatal incisions for resection of the temporal bone. This illustration demonstrates inclusion of the tragus with the specimen.



Figure 123-10 Skin incision when tumor infiltrates the tragus and conchal bowl.

A postauricular incision is made 3 cm behind the postauricular sulcus and extended superiorly toward the temporal area and inferiorly over the mastoid tip into a high cervical skin crease. The neck incision is brought more inferiorly if neck dissection is anticipated. The postauricular skin incision is raised a short distance anteriorly. The dissection is then directed medially through the fibroperiosteal layer over the mastoid cortex. This facilitates obtaining a tight three–layer closure if CSF is encountered. The soft tissue flap is dissected in a subperiosteal plane until the posterior part of the conchal incision is reached. To avoid inadvertent entry into the EAC, a hemostat is placed through the posterior aspect of the conchal incision into the postauricular wound. Once this plane of dissection has been established, the conchal incision is connected circumferentially with the postauricular wound, thus isolating the conchal bowl, EAC, and tumor from the skin flap containing the remainder of the pinna (Fig. 123-11). If there is any doubt about the adequacy of the lateral skin margin, a crescent–shaped segment of skin from the conchal bowl of the pinna is sent for frozen section analysis. The lateral part of the EAC is sutured closed to prevent tumor spillage. The skin flap is dissected anteriorly while staying superficial to the deep temporalis fascia superiorly, the sternocleidomastoid fascia inferiorly, and the parotid fascia anteriorly (Fig. 123-12) until the masseter muscle is identified. This develops an anterior-based skin flap that is retracted forward to expose the entire parotid gland (Fig. 123-13). The skin flap is secured with sutures and rubber bands.



Figure 123-11 The anteriorly based skin flap containing the pinna is separated from the core of the external auditory meatus. The meatus has been oversewn to prevent tumor spillage.



Figure 123-12 The anteriorly based skin flap containing the pinna (*black arrow*) is reflected anteriorly, with the external auditory canal (*white arrow*) and surrounding soft tissue left behind.



**Figure 123-13** Most of the pinna remains with the specimen (see Fig. 123-10). The skin flap is secured anteriorly to expose the parotid gland *(arrow)*. The superior limb of the incision is extended toward the forehead in preparation for an infratemporal fossa approach.

Using a large cutting burr, a complete mastoidectomy is performed to expose the sinodural angle posteriorly, the

tegmen mastoideum superiorly, the bony ear canal anteriorly, and the digastric ridge inferiorly. Care is taken to not thin the bony ear canal to the point of exposing the cancer. The horizontal semicircular canal is identified for orientation, and the bone of the zygomatic root is drilled to expose the epitympanum and bony plate of the tegmen tympani. Drilling in this area is directed anteroinferiorly toward the posterior aspect of the glenoid fossa. Dissection directed anteriorly poses the risk of penetrating the dura of the middle fossa. The anterior limit of this dissection is the capsule of the TMJ.

A large diamond burr is used to identify the vertical portion of the facial nerve from the second genu to the stylomastoid foramen while maintaining the horizontal semicircular canal and digastric ridge as landmarks. Once the facial nerve has been identified, the facial recess is opened with a small cutting burr, followed by a diamond burr (Fig. 123-14). The middle ear is inspected through the facial recess to verify the absence of penetration of the cancer through the tympanic membrane. The incudostapedial joint is disarticulated and the incus is removed. The tensor tympani tendon is transected. The facial recess is then extended inferiorly by sacrificing the chorda tympani nerve to expose the posterior portion of the hypotympanum. Using a cutting burr, the inferior portion of the tympanic ring is drilled toward the anterior wall of the EAC (Fig. 123-15). Care is taken to not thin the bony EAC extensively to avoid exposure of the cancer. The more medial orientation of the anterior tympanic membrane requires infratympanic drilling directed anteromedially to avoid inadvertent entry into the anterior sulcus of the EAC. This is accomplished by continuing the dissection of the facial recess anteromedially while always keeping the hypotympanum in view. The anterior limit of this dissection is the periosteum between the temporal bone, the ITF, and the capsule of the TMJ. In this area, the surgeon must remain oriented to the location of the eustachian tube orifice to avoid injury to the carotid artery.



Figure 123-14 The facial recess is dissected to expose the chorda facial angle and the incudostapedial joint.



**Figure 123-15** The operative field is demonstrated. The facial recess is extended inferiorly and then anteriorly by dissecting medial to the tympanic annulus. The stylomastoid foramen is exposed, but the nerve is not dissected free from its surrounding fascia and fibrous tissue.

Once this bony dissection has been completed, the EAC is attached anteriorly by anteromedial section of bone just lateral to the eustachian tube. A superficial parotidectomy is performed by identifying the facial nerve distal to its exit from the stylomastoid foramen. After retracting the tail and body of the parotid gland, the main trunk of the facial nerve is traced peripherally to isolate the lateral or superficial portion of the gland. Details of this technique can be found in Chapter 62. Dissection of the facial nerve in the stylomastoid foramen can be difficult because the nerve is surrounded by a thick cuff of fibrous tissue. Meticulous dissection in this area and isolation of the nerve often result in facial paresis or paralysis, which is usually temporary. It is preferable to maintain this cuff of tissue lateral to the facial nerve because less aggressive dissection can allow preservation of facial function. Once the superficial lobe of the parotid gland has been dissected, the EAC and parotid specimens are removed in continuity with the extratemporal portion of the facial nerve under direct vision.

An osteotome is inserted through the extended facial recess to the bone just lateral to the eustachian tube orifice. A few gentle taps on the osteotome will complete the osteotomy, and the EAC can be dissected from the TMJ capsule (Fig. 123-16). During this maneuver care must be taken to avoid injury to the ICA by the osteotome, which lies just medial to the eustachian tube. At this point the specimen is attached only to the parotid gland, the superficial lobe of which can be removed in continuity with the specimen and the facial nerve preserved.



Figure 123-16 The remaining anterior bony attachment is freed with a small osteotome positioned medial to the anterior tympanic annulus.

If the operative resection creates a large and deep cavity, the posterior one half of the temporalis muscle is rotated into the surgical defect and secured with 2-0 absorbable suture (Fig. 123-17). The skin flap is returned to its anatomic position. Hemovac drains are placed both superiorly and inferiorly while avoiding suction trauma to the facial nerve (Fig. 123-18). Small to medium defects are closed with a split-thickness skin graft that is secured with bolster packing consisting of silk ligatures tied over a Xeroform bolster. This technique is described under "Reconstruction" later in this chapter. Larger skin and cavity defects are closed with free or pedicled myocutaneous flaps. A compression dressing is applied unless microvascular anastomosis has been performed.



Figure 123-17 A superficial parotidectomy is performed to remove the parotid with the lateral temporal bone specimen. The posterior one half of the temporalis muscle is rotated into the mastoid cavity.



Figure 123-18 Skin closure with Hemovac drains in place. The central cavity defect will be closed with a skin graft.

#### Subtotal Temporal Bone Resection

Cancer extending medial to the tympanic membrane and invading the middle ear, hypotympanum, otic capsule, facial nerve, or mastoid is managed by subtotal resection of the temporal bone. In the past, an en bloc technique of resection was advocated to encompass the tumor. It has been noted that en bloc resection of the EAC with surrounding soft tissue structures followed by piecemeal resection of tumor and structures medial to the middle ear provides better operative control and probably does not negatively influence the cure rate.<sup>[24]</sup> The strategy is to perform a largely extradural and subperiosteal resection of the temporal bone with margins at the middle fossa dura superiorly, the posterior fossa dura and sigmoid sinus posteriorly, the ICA anteriorly, the base of the skull and jugular bulb inferiorly, and the petrous apex medially. Extension of cancer beyond these boundaries signifies a grave prognosis, but the resection can be extended in certain cases, as discussed later.

The approach for lateral resection of the temporal bone is performed as described previously, with examination of the middle ear through the facial recess and mastoid cavity for cancer. If cancer is present, the more encompassing resection is warranted. If facial nerve function is normal preoperatively and it is visibly clear of tumor as noted during the procedure, a decision is made, based on the extent of cancer, whether the nerve should be sacrificed and included with the specimen. If the nerve is to be preserved, piecemeal resection of the cancer is planned. If the facial nerve is uninvolved, it will be mobilized and preserved as the dissection proceeds. Bone is removed over the sigmoid sinus and posterior fossa dura. The tegmen and posterior fossa plates of bone are first thinned with a cutting burr and then resected piecemeal after dural elevation. These segments of bone are sent for permanent section. Care is taken to not injure the superior petrosal sinus at this stage. Translabyrinthine dissection exposes the IAC and jugular bulb. If cancer is not present at the jugular bulb, no further resection is performed in this area.

If the facial nerve is to be preserved, it is skeletonized along the labyrinthine, geniculate, horizontal, and vertical segments down to the stylomastoid foramen. The eggshell-thin pieces of bone on the anterior, lateral, and posterior aspects of the nerve are removed with a dissector. The dura of the IAC is opened to allow mobilization of the facial nerve at the porus acusticus. The greater superficial petrosal nerve is transected from the geniculate ganglion, and the facial nerve is gently dissected out of its bony canal and mobilized posteriorly (Fig. 123-19). If the facial nerve is to be sacrificed, the dissection and mobilization are omitted, but the IAC is still opened. Both the facial and the vestibulocochlear nerves are transected, and the proximal ends of these nerves are examined by frozen section analysis to ensure a tumor–free medial margin of resection. The remaining tympanic ring inferiorly is

then drilled to the periosteum of the skull base. This periosteum protects vascular and neural structures in the ITF and is not resected unless cancer is identified in this area. Bone resection continues along the periosteum anterosuperiorly until the capsule of the TMJ is encountered. Medially, bone dissection is continued until the vertical portion of the ICA is encountered. Care is taken to keep the periosteal covering over this artery intact while drilling with a diamond burr.





If cancer has not infiltrated into the protympanum, the anterior limit of resection can be performed without resection of the mandibular condyle. Absence of cancer in this area can be confirmed intraoperatively by obtaining a frozen section of the mucosal edge near the eustachian tube orifice. The bone lateral and posterior to the vertical portion of the ICA above the jugular bulb is drilled with a diamond burr to isolate the artery from the otic capsule. Once this has been accomplished, the remainder of the otic capsule containing the cochlea is drilled (Figs. 123-20 and 123-21). The vaginal process between the jugular bulb and the ICA is also drilled out, with only the petrous apex remaining.



**Figure 123-20** Subtotal resection of the temporal bone with the medial margin through the internal auditory canal and cochlea. The vertical portion and genu of the carotid artery are dissected. The vaginal process between the jugular bulb and the carotid artery has been drilled. The extratemporal facial nerve is intact.



Figure 123-21 Intraoperative dissection through the internal auditory canal and cochlea modiolus for subtotal resection of the temporal bone.

#### Dissection of the Jugular Foramen

If cancer has infiltrated into the jugular bulb, total removal of the cancer requires resection of the sigmoid sinus. If only the lateral aspect of the sigmoid sinus or jugular bulb is involved, resection can be achieved without much added morbidity. Resection of cancer involving the medial wall or pars nervosa of the jugular foramen results in injury to the lower cranial nerves and possibly a larger dural defect.

For cancer that involves only the lateral wall of the sigmoid sinus and jugular bulb, total resection of tumor can be achieved with an ITF approach similar to the Fisch type A. The subtotal temporal bone resection is completed after dissection and isolation of the internal jugular vein (IJV) and cranial nerves IX, X, XI, and XII in the neck. The facial nerve has been either transected or mobilized out of the way. The soft tissues between the posterior belly of the digastric muscle and the IJV are dissected in layers to expose the vein from the skull base to the jugular bulb. The IJV is doubly ligated in the neck with 2-0 silk and divided. The sigmoid sinus inferior to the takeoff of the superior petrosal sinus is tied with 2-0 silk by making stab incisions in the dura on either side of the sigmoid sinus, passing a curved aneurysm needle into and out of the dural opening, and passing the tie around the sigmoid sinus (see Chapter 125, Figs. 125-10 and 125-11). The dural incisions are repaired primarily or with plugs of muscle secured with 4-0 Nurolon. The sigmoid sinus can also be controlled proximally by extraluminal packing with oxidized cellulose.

The retrosigmoid dura is decompressed to the jugular foramen with a diamond drill. The vaginal process has already been resected during the subtotal temporal bone resection. The cancer in the region of the jugular bulb is debulked without entering the lumen. To resect the lateral wall of the sigmoid sinus and jugular bulb, the inferior petrosal sinus feeding into the lumen must be controlled. A large incision is made on the lateral aspect of the jugular bulb, and the medial wall is quickly packed with oxidized cellulose to control bleeding. Once adequate hemostasis has been achieved, the lateral wall of the sigmoid sinus from the superior ligation to the jugular foramen is removed along with the IJV. The medial wall of the lumen is inspected for residual cancer, and the packing in the jugular bulb is reinforced as needed (Fig. 123-22).





If cancer is present on the medial wall of the jugular bulb, the resection must be extended into the pars nervosa. If a decision is made to do this, the medial wall of the jugular bulb is removed, and the contents of the par nervosa are resected, including cranial nerves IX, X, and XI. The adequacy of margins of resection of these nerves proximally and distally is verified by frozen sections, and further resection is completed as necessary. A positive margin proximally will require nerve resection in the posterior fossa, which can be performed through a retrosigmoid approach or through the exposure created by resection of the jugular bulb. The residual bone anteromedial to the jugular foramen can be drilled to the ICA anteriorly and the hypoglossal canal anteromedially. The inferior petrosal sinus may need to be packed again during this maneuver. Any dural defects should be repaired with fascia or pericranium.

If the lower cranial nerves were functional preoperatively and required transection intraoperatively, a tracheotomy is performed for pulmonary toilet and care of aspiration, and a nasogastric feeding tube is placed. Medialization of the vocal cord via a type I thyroplasty or arytenoid adduction can also be performed intraoperatively (see Chapter 41).

# **Dural Resection**

If inspection of the dura reveals residual cancer after subtotal resection of the temporal bone, resection of the dura is performed in collaboration with a neurosurgeon. For cancer in the subtemporal dura, an incision is made in the dura laterally while being careful to not injure the vein of Labbé. The temporal lobe is retracted superiorly to expose the involved dura inferiorly, which is excised with adequate margins. The defect is closed with a fascial or pericranial graft. The presence of invasion into brain parenchyma should have been detected preoperatively. Resection of involved brain tissue may be technically possible, but the probability of cure is exceedingly low.

Involvement of the posterior fossa dura by cancer is treated similarly. Adequate exposure can frequently be obtained through a presigmoid dural incision. The cerebellum is retracted, and the involved dura is excised with

appropriate margins. If the medial wall of the sigmoid sinus is involved, the craniectomy is extended posteriorly with a drill to make a retrosigmoid opening in the dura. The lateral wall of the sigmoid has already been resected, and hemostasis at the inferior petrosal sinus has been achieved. The cerebellum is retracted, the involved dura is resected with margins, and the defect is repaired with a fascial or pericranial patch.

### Infratemporal Fossa Resection

When cancer infiltrates into the protympanum, additional anterior exposure is needed to transpose the carotid artery and excise soft tissue around the eustachian tube. The superior portion of the incision is extended anterosuperiorly toward the frontotemporal hairline to expose the temporalis muscle. Elevation of the skin flap is performed superficial to the deep temporal fascia until the region of the frontal branch of the facial nerve is reached, at which point the dissection is carried deep to the fascia in the superficial temporal fat pad while avoiding injury to the frontal branch of the facial nerve. This dissection is carried to the lateral rim of the orbit anteriorly and the zygomatic arch inferiorly. Using electrocautery, the periosteum is incised along the lateral edge of the orbital rim and the superior edge of the zygomatic arch. The soft tissue lateral, medial, and inferior to the zygomatic arch is dissected in a subperiosteal plane to release it from the masseter muscle. A V–shaped osteotomy is made in the malar eminence, which consists of the anterior zygoma and lateral maxilla. The zygomatic arch is removed and saved after the osteotomy is completed in the posterior zygomatic root. The temporalis muscle is then dissected off the underlying bone and remains pedicled on the coronoid process of the mandible (Fig. 123-23).





Total parotidectomy is performed by dissecting out the branches of the facial nerve. If the nerve is to be sacrificed and included with the temporal bone dissection, the peripheral branches are identified, tagged, and transected. The superficial temporal artery is divided and the soft tissues overlying the condylar portion of the mandible are resected. Using an oscillating saw, the mandibular condyle is transected at the level of the coronoid notch. The frontal branch of the facial nerve may require transection to provide access to the mandible.

With the temporalis muscle reflected inferiorly, the contents of the ITF are separated from the skull base by dissection in a subperiosteal plane starting anteriorly. The lateral pterygoid plate is encountered first. The foramen ovale is located approximately 1 cm posterior and just medial to this structure, and the foramen spinosum is situated about 0.5 cm posterior to the foramen ovale (Fig. 123-24). The middle meningeal artery is coagulated with bipolar cautery and transected. Now, with the vertical portion of the ICA in view at the skull base, the periosteum of the skull base is elevated in a posterior–to-anterior direction to connect to the dissection at the foramen ovale.



Figure 123-24 Exposure of the petrous apex and infratemporal fossa. A total parotidectomy has been completed. The middle meningeal artery is divided, and the trigeminal nerve (V3) is exposed.

If any tumor is seen within the ITF, it may be resected at this time because the important structures in the region have been identified. The lateral pterygoid muscle is transected in the region of the mandibular osteotomy. The dissection is performed in layers while taking care to not lacerate the internal maxillary artery inadvertently. Any vascular structures that are in the way can be divided and controlled with ties or clips. The venous plexus in the pterygoid region can be the source of significant bleeding, which must be controlled by bipolar cautery and packing with oxidized cellulose.

A temporal craniotomy is performed to provide access to the floor of the middle fossa. The middle fossa dura is

elevated to expose the IAC posteriorly, the superior petrosal sinus medially, and the foramen ovale and V3 anteriorly. The floor of the middle fossa is then resected posterior to V3 and medial to the eustachian tube with a cutting burr or rongeurs. If tumor invades the eustachian tube with extension into the petrous apex, total resection of the temporal bone is performed.

### **Total Temporal Bone Resection**

Total en bloc resection of the temporal bone with clear margins is conceptually the procedure needed for oncologic control. The vital structures that surround the temporal bone demand that a meticulously controlled approach be performed to avoid catastrophic vascular and neurologic consequences. Even if the carotid artery is to be sacrificed with the specimen, proximal and distal control must be achieved. Similarly, the brain, dura, and cranial nerves must be dissected and isolated. Although a "no-touch" tumor approach remains the technique to which we aspire, it is often not accomplished to safely obtain access to the petrous portion of the temporal bone.

After completion of subtotal temporal bone dissection, proximal control of the ICA is obtained either at the skull base or in the neck. Distal control is achieved in the floor of the middle cranial fossa via middle fossa craniotomy. The carotid artery posterior to V3 is exposed so that a clip can be placed on it for distal control if necessary. The bone lateral to the ICA at the genu is removed, and the ICA is dissected free from its canal from the skull base to the foramen ovale. A thick, fibrous ring of tissue surrounds the ICA at the carotid foramen. When mobilizing this portion of the ICA, it is safer to first dissect circumferentially around the fibrous tissue rather than along the carotid itself because the risk of carotid laceration is high. The fibrous ring can be left intact while the ICA is mobilized. Once adequate exposure has been achieved, the remainder of the otic capsule containing the cochlea is drilled. Anteriorly, the petrous apex is drilled to complete total resection of the temporal bone (Fig. 123-25). The eustachian tube lies inferolateral to the level of the foramen ovale. If cancer is found anterior to this area, nasopharyngeal resection and possibly cavernous sinus dissection will be necessary to achieve total resection, which is beyond the scope of this chapter. The transected end of the eustachian tube is sutured closed to prevent CSF rhinorrhea.



**Figure 123-25** Total temporal bone resection. The mandibular condyle is resected. The carotid artery has been spared and is retracted to provide safe access to the petrous apex. Removal of the otic capsule has left a large dural defect. V3 has been divided and the eustachian tube has been suture-ligated. The infratemporal fossa is dissected down to the pharyngobasilar fascia.

#### Reconstruction

The goals of soft tissue reconstruction after temporal bone resection include alleviation of cosmetic deformity, prevention of CSF leak, and promotion of adequate healing. In most cases, these objectives are best accomplished with a vascularized flap. To prevent communication between the nasopharynx and the middle ear space after lateral temporal bone resection, the eustachian tube is obliterated by scarifying the mucosa and filling the lumen with ossicles, bone dust, Surgicel, or a plug of muscle.

The resultant defect after lateral resection of the temporal bone is similar to that after radical mastoidectomy, and there is usually little risk of CSF leak, so adequate tissue coverage can be accomplished with a split-thickness skin graft to create a mastoid cavity. However, because most patients require radiation therapy after surgery or have previously undergone radiation therapy, we believe that a vascularized flap to promote adequate healing provides the optimal result. The posterior one half of the temporalis muscle is separated from the anterior portion, and while pedicled inferiorly, the posterior portion of the muscle is rotated into the mastoid cavity.

The surgical defect after subtotal temporal bone resection extends medially through the otic capsule and poses a greater likelihood of a CSF leak. If ITF dissection has not been performed, the resultant defect is not significantly larger than one resulting from lateral temporal bone resection. Usually, a pedicled posterior temporalis flap provides adequate coverage. Other options include a lower island trapezius or pectoralis major myocutaneous flap. If these flaps cannot be mobilized superiorly to cover the temporal bone defect adequately, a free flap with microvascular anastomosis to the superficial temporal or facial artery system can be used. The most commonly used free flap is the rectus abdominis, although a scapular or latissimus dorsi free flap can also be used. The TMJ is not reconstructed routinely.

If the facial nerve has been resected and there is an adequate proximal stump, it is reconstituted with a cable nerve graft. Most frequently, we use the sural nerve (see Chapter 121). The greater auricular nerve can be used if the length required is less than 6 cm. Otherwise, a sural nerve graft is necessary to obtain a longer donor graft for anastomosis between the proximal and distal ends of the facial nerve (Fig. 123-26). If facial nerve resection has been performed distal to the pes anserinus, both the upper and lower branches are reconstructed. If the proximal stump of the facial nerve has been resected at the brain stem with an inadequate length left for anastomosis, a hypoglossal-to-facial (XII to VII) nerve anastomosis can be performed.



**Figure 123-26** Right total temporal bone resection, including the condyle of the mandible. The facial nerve is repaired with a long sural nerve graft (*arrows* at the upper and lower divisions of the remaining nerve).

If a CSF leak is a potential problem, the wound should be closed in at least three layers with two layers of 2-0 Vicryl or Dexon for the deep tissues followed by a running and locking horizontal mattress suture consisting of 4-0 Surgilon for the skin. If a conchal defect is closed with a skin graft, this area must be isolated from the remainder of the wound before skin grafting by completely obliterating the space between the pinna flap and the underlying muscle flap with 2-0 and 3-0 absorbable suture.

A split-thickness skin graft (0.015 inch) is secured to 180 degrees of the pinna defect. Sutures of 3-0 silk are kept long to serve as bolster ties. A piecrust technique is used, similar to that for grafting oral cavity lesions. The central aspect of the skin graft is contoured within the cavity defect, and the remaining skin is trimmed to the appropriate size and further secured to the wound margins (Fig. 123-27). The graft is usually packed with Xeroform gauze and secured with a tie-over bolster dressing (Fig. 123-28).



Figure 123-27 A split-thickness skin graft is sutured to the posterior skin margins with 3-0 silk.



Figure 123-28 The skin graft is contoured to the cavity, immobilized with Xeroform gauze, and secured with 3-0 silk sutures tied over the packing.

After wound closure, a mastoid dressing is placed and the patient is extubated before transport to the recovery area. Unless a watertight CSF closure has been achieved, drains for the wound are placed to gravity drainage. Continuous suction is applied when the likelihood of CSF permeating the wound is negligible.

# POSTOPERATIVE MANAGEMENT

Most patients who undergo lateral resection of the temporal bone require postoperative care comparable with that after routine mastoidectomy, except that the flap requires monitoring. The head should be elevated 30 degrees, and diet and ambulation are advanced as tolerated. The mastoid dressing is removed on the first postoperative day, and any skin graft bolsters are removed on the fifth day. The remaining pinna may have a tenuous blood supply, especially if a large, anteriorly based skin flap was raised. This is usually manifested by increased venous congestion, which can compromise the auricle and the remainder of the wound. If the pinna appears to be in jeopardy, we have used medical-grade leeches to assist venous decompression and mobilization of fluid (Fig. 123-29).





If a significant dural defect was created or a considerable amount of brain retraction was necessary during surgery, the patient is managed in a monitored care situation for the first 24 hours for frequent neurologic evaluation and monitoring of vital signs. The level of care needed, which is similar to that after translabyrinthine acoustic tumor surgery, can be given in an intensive care unit or a step-down unit. The patient's intake and output, as well as blood count and electrolytes, are monitored. The mastoid dressing is kept in place for 3 to 5 days, although on the second postoperative day the dressing is removed to check the wound and then replaced. The patient is monitored for CSF rhinorrhea or CSF leak through the wound, and any suspicious fluid is sent for β2-transferrin testing. The patient is instructed to not strain or perform a Valsalva maneuver for 3 weeks. We do not routinely place a lumbar drain intraoperatively, although it has been done on occasion if a large dural defect is expected or if neck dissection has been performed and there is possible communication between the neck wound and the subarachnoid space. The patient can be transferred to surgical floor care if no significant intracranial complications have occurred.

If ITF dissection with resection of the mandibular condyle has been performed, the patient is encouraged to mobilize the mandible as soon as possible to promote maintenance of a proper occlusal relationship and to decrease the risk of fibrous ankylosis. Our colleagues in oral surgery are consulted to oversee this aspect of the patient's recovery.

If a free flap has been performed, the patient and wound are closely monitored by clinical findings and Doppler ultrasound examination of the vascular anastomosis. Care is taken to not place any ties around the patient's neck that could potentially constrict the vascular pedicle to the flap. Any evidence of significant flap ischemia should be detected early so that intervention to salvage the flap, which may include revising the anastomoses, can be performed immediately.

Patients who have facial nerve palsy or paralysis postoperatively are treated with eye drops during the day and ointment at night to avoid exposure keratitis and corneal ulceration. A plastic eye shield (moisture chamber) is useful in protecting the eye. If facial nerve function is expected to return within 4 to 6 weeks, as occurs after facial nerve transposition, the patient usually requires no further treatment. If recovery from facial nerve dysfunction is expected to take longer or if the dysfunction may be permanent, a gold weight is placed in the upper eyelid and a lateral tarsal strip is performed if ectropion becomes a problem. These procedures are performed in the early postoperative period (see Chapter 121). If facial nerve function fails to recover, a hypoglossal-to-facial (XII to VII) nerve anastomosis can subsequently be performed.

A patient with new iatrogenic lower cranial nerve palsy or paralysis is at significant risk for aspiration and resultant pulmonary infection. The patient should be placed in a step-down unit for frequent suctioning through the tracheostomy. Nutrition is provided through a feeding tube while the patient undergoes swallowing rehabilitation coordinated by a speech pathologist. This rehabilitation includes clinical assessment as well as evaluation by modified barium swallow and performance of various swallowing exercises customized for each patient. Once the patient is handling secretions adequately with competent glottic function, the tracheotomy tube is removed to enhance recovery of deglutition. The feeding tube is removed when the patient is able to ingest adequate nutrition by mouth. If adequate swallowing function cannot be attained, a gastrostomy tube is placed.

Immediate postoperative scans are not performed unless the patient is showing evidence of an intracranial complication such as hemorrhage, abscess, hydrocephalus, or pneumocephalus. Baseline postoperative scans consisting of MRI or CT, or both, are obtained within 3 months after surgery to help monitor for recurrent disease. These scans are repeated every 6 months for the next 2 years and yearly thereafter.

Reconstruction with a split—thickness skin graft over a vascularized muscle flap creates minimal cosmetic deformity (Fig. 123-30). If the entire pinna has been resected, reconstruction options are a prosthetic ear or multistaged procedures in a surgical bed that is compromised. Given the problems inherent in reconstruction of the pinna, our preference is for a prosthesis. Using either the preoperative ipsilateral ear or a reverse moulage of the contralateral ear, a synthetic prosthesis can be created that is both functionally and cosmetically acceptable (Fig. 123-31).



**Figure 123-30** A well-healed split–thickness skin graft at the meatal opening. The size of the defect does contract to provide an acceptable cosmetic appearance.



Figure 123-31 A, Total auricular resection with only the tragus remaining. B, A prosthetic pinna provides excellent cosmetic appearance and function.

Radiation treatment is begun approximately 6 weeks after surgery, when adequate wound healing has occurred. The most common area of recurrence of cancer is locally, in the skull base, dura, or ITF, so these areas are treated specifically. The neck is also treated, especially zones II and III, although the risk of failure here is much

less than the risk of failure locally.

# **COMPLICATIONS**

The goal of surgical treatment of cancer of the temporal bone is to achieve total removal of the cancer with minimal morbidity. Avoidance of complications is critical to successful rapid rehabilitation of the patient postoperatively. Aggressive temporal bone resection may be associated with major intracranial complications. Extensive brain retraction can result in lower cranial nerve palsy with aspiration, cerebellar or temporal lobe edema and dysfunction, and cerebral infarction. The vein of Labbé is particularly vulnerable, and compression of this vein can lead to temporal lobe venous infarction. Dissection of the ICA can result in embolic stroke, injury with bleeding, and stroke. The dura must be carefully reconstructed because CSF leaks in this setting can be difficult to control and are a major risk for meningitis.

#### Vascular

Major arterial injuries can cause disastrous consequences, and all precautions should be taken to prevent these injuries. Most moderate-sized vessels such as branches of the internal maxillary artery can be controlled adequately, even in the event of inadvertent laceration. However, the ICA requires more complex management. The need for possible surgical dissection near the ICA should be ascertained preoperatively by CT and MRI and by angiography if necessary. If it is determined that carotid mobilization or dissection may be required, preoperative balloon test occlusion with xenon/CT is performed. The data obtained provide an estimate of ipsilateral regional cerebral blood flow while the carotid is temporarily occluded for 15 minutes. These results place patients in one of three risk groups for the development of major cerebral ischemia in the event of abrupt carotid interruption: high, intermediate, and low. Approximately 80% of patients will be in the low–risk group. These patients will not tolerate any duration of carotid occlusion, so operative therapy involving carotid manipulation should be avoided unless bypass to the middle cerebral artery is performed initially. The remaining 10% of patients will be in the intermediate–risk group. These patients require carotid revascularization if the ICA is sacrificed and the use of adjuvant methods to minimize cerebral ischemia, such as induced hypertension, barbiturate coma, and hypothermia.<sup>[30]</sup>

Intraoperatively, before manipulation of the ICA, proximal control of the vessel in the neck or at the skull base and distal control in the horizontal portion of the intrapetrous carotid artery should be obtained. In this way, should an inadvertent injury to the ICA occur, rapid control of the situation can be achieved so that the injury can be managed in an expeditious and controlled fashion. If a laceration of the ICA occurs, it is managed by either primary repair, repair with a patch, bypass grafting using the saphenous vein, or sacrifice of the ICA without reconstruction.

Venous complications are also possible after temporal bone resection, especially if manipulation or resection of the sigmoid sinus is performed. The vein of Labbé is an important route of venous outflow from the temporal lobe, and damage to this vein can lead to significant temporal lobe infarction with aphasia if on the dominant side and seizures. The best strategy for managing this complication is prevention. When packing or ligating the sigmoid or transverse sinus it is important to stay far anterior to the insertion of the vein of Labbé into the transverse sinus. Once an infarct occurs, therapy consists of speech and language rehabilitation, neurologic support, and administration of antiseizure medications such as phenytoin (Dilantin) and phenobarbital.

#### **Cerebrospinal Fluid Leak**

The main morbidity of a CSF leak is increased risk for the development of meningitis. Because meningitis can be a life-threatening condition, prevention plus control of CSF leaks is very important. CSF leaks occur when there is inadequate closure of the dura in conjunction with a path for egress of CSF through the soft tissues to the external environment. The usual sites of leakage are the eustachian tube, incision site, and remnant of the EAC.

Prevention is the most efficacious method of management. All dural closures should be made as watertight as possible. Any potential dead spaces should be obliterated by placement of muscular flaps or autologous fat grafts. The final barriers to CSF flow, skin closure and the eustachian tube, should be closed or obliterated in watertight fashion. A drain is not placed in the wound. Postoperatively, the patient's head is kept elevated and the patient is instructed to avoid straining or performing a Valsalva maneuver. A pressure dressing is maintained for 5 days.

Despite preventive treatment, CSF leaks can occur, and when they do, prompt management can prevent serious consequences. The critical factor is early diagnosis. The patient should be instructed to report any egress of clear fluid from the nose or any drip down the nasopharynx. Oftentimes, the diagnosis is obvious and no further testing is required.  $\beta_2$ -Transferrin testing of fluid will identify CSF.[31] However, if the physician is clinically suspicious that a CSF leak has occurred, action must be taken despite test results to the contrary.

If the leakage occurs through an incision, the area of dehiscence is reinforced with horizontal mattress sutures.

Topical collodion liquid is also useful. Most CSF leaks that persist for more than 24 hours despite maintenance of a pressure dressing and head elevation can be controlled with the placement of a lumbar drain. The lumbar drain is allowed to drain approximately 150 mL/day, or 50 mL per 8-hour shift. Care must be taken to not overdrain CSF, because severe headaches may develop and there is the potential that the negative pressure may permit air and other nonsterile fluids to enter the subarachnoid space from the wound. The use of antibiotics prophylactically in the setting of a CSF leak is controversial. In the event of a CSF leak into a clean surgical wound, the authors think that antibiotic prophylaxis does not reduce the incidence of meningitis and may in fact increase the risk of having a resistant organism in patients in whom bacterial meningitis does develop.

If the CSF leak persists after 3 to 5 days of lumbar drainage, a surgical procedure to stop the leak is indicated. Usually, the uncontrolled CSF leak is through the eustachian tube. A transmastoid procedure through the previous incision is performed to access the eustachian tube and obliterate it in a more complete fashion. This can be accomplished by further scarification of the mucosa followed by insertion of bone dust, Surgicel, and muscle plugs. Occasionally, the source of a CSF leak in the dural repair will be visible. In such cases, the dural closure can be reinforced with patches of fascia or muscle sewn over the dural defect. The wound is closed in multiple layers, as described previously, in watertight fashion, and a mastoid dressing is placed for 5 days. Lumbar drainage is continued for the next 3 days and the drain removed if there is no further CSF leak.

A very difficult problem occurs if an extensive neck procedure is performed at the time of temporal bone resection with dural resection. Because the neck requires drainage temporarily after surgery, a CSF leak into the neck can occur quite readily. This becomes a potentially serious problem when a tracheostomy is in place. Great effort is necessary to ensure that the tracheostomy wound does not communicate with the neck dissection. Otherwise, CSF will readily pass subcutaneously through the neck into the tracheo and exit out the tracheotomy wound. Similarly, tracheal secretions colonized with bacteria can infect the neck and CSF space by retrograde contamination and create a very serious infection.

#### Infection

Wound infections occur infrequently after resection of the temporal bone. Appropriate treatment is no different from treatment elsewhere in the head and neck. A more feared complication is meningitis or other intracranial infection such as a brain abscess. The risk of meningitis developing after resection of the temporal bone and requiring intracranial surgery is low in the absence of a CSF leak, so prompt and effective treatment of such leaks is critical. Symptoms of meningitis include fever, severe headache, backache, and altered sensorium. There may or may not be focal neurologic deficits. The diagnosis is made by lumbar puncture. A third–generation cephalosporin is administered intravenously after samples are obtained for culture. A CT scan with contrast enhancement is obtained to rule out an intracranial abscess. It is difficult to differentiate aseptic meningitis from bacterial meningitis based on clinical findings and laboratory values, so antibiotics are continued until culture results are available in 2 days. If the cultures are negative and clinical suspicion of bacterial meningitis is not high, the antibiotics are stopped and steroids (dexamethasone [Decadron], 4 mg intravenously every 6 hours) are given and tapered over a period of several weeks. If the cultures are positive or the patient's clinical status is suggestive of bacterial meningitis, the antibiotics are tailored to the culture results and continued for 10 to 14 days.

Aspiration pneumonia is a possible complication after temporal bone resection, especially if previously functional lower cranial nerves are sacrificed or injured during surgery. In these cases, a type I thyroplasty with or without vocal cord medialization and a tracheotomy are usually performed at the time of surgery. The patient is fed through a feeding tube until aspiration stops and the patient is able to swallow. Aggressive pulmonary toilet is provided while the patient undergoes rehabilitation for deglutition and airway protection. Aspiration pneumonia is treated with culture–specific antibiotic therapy and further aggressive pulmonary toilet.

#### Intracranial Hemorrhage and Hypertension

Although neurosurgical procedures have become relatively safe, there is still a risk of acute intracranial complications after temporal bone resection involving intradural manipulation. One of the most rapidly progressive and potentially fatal complications is acute intracranial hemorrhage, which if not treated promptly can lead to irreversible cerebral injury with uncontrolled cerebral edema or brain stem herniation and even death. Preventing this complication obviates the urgent management necessary for this event. All patients should be screened by history and physical examination for bleeding disorders. If detected, the disorder should be treated adequately before surgery. Intraoperatively, all bleeding sites must be well controlled before closure of the wound. Postoperatively, the patient should remain normotensive and refrain from vigorous activities.

The signs of acute intracranial hemorrhage are due to increased intracranial pressure, as manifested by altered sensorium or loss of consciousness. Focal neurologic deficits, including fixed and dilated pupils and systemic signs such as bradycardia and hypertension, can also occur, but lack of these changes does not rule out an intracranial hemorrhage. The location of hemorrhage can be extradural, subdural, or intraparenchymal. Most extradural hematomas are due to bleeding from dural vessels or dural sinuses. Subdural hemorrhage occurs as a result of

dural bleeding, as well as uncontrolled bleeding from intracranial vessels. Intraparenchymal hemorrhage is rare after the resection of extra-axial lesions.

Once an intracranial hemorrhage is suspected, a CT scan without contrast enhancement is performed to confirm the diagnosis. The patient's condition is temporized by the administration of mannitol, 0.25 to 0.5 g/kg intravenously, and hyperventilation after intubation. If the patient's vital signs are unstable or there is evidence of brain stem herniation, the patient is brought directly to the operating room without scanning. A lumbar puncture should never be performed in this setting for fear of inducing brain stem herniation through the foramen magnum. A ventriculostomy can be performed to rapidly decompress the increased intracranial pressure. The craniectomy or craniotomy is reopened, and the blood clot is evacuated. The source of bleeding is sought and controlled.

Postoperatively, the patient is monitored in an intensive care unit setting with maintenance of ventriculostomy pressure under 15 mm Hg. A CT scan is obtained to rule out further hematoma, and the patient is allowed to recover.

#### Wound

Management of wound complications after temporal bone resection is similar to that in other areas of the head and neck, except that CSF leak is a potential problem, as discussed previously. The integrity of the flap used to obliterate the operative defect is vital to keep the subarachnoid space separated from the outside environment. Complications that can compromise this function are ischemia and separation of the flap from the wound.

It is unusual for ischemia to develop in pedicled flaps, but it occurs occasionally if the flap is stretched too far or if strangulation of the vascular pedicle occurs as a result of significant constriction placed inadvertently around the neck. Injury to the vascular pedicle intraoperatively can also occur but is rare. If the pedicled flap becomes nonviable, it should be replaced immediately with an alternative flap, whether it is another pedicled flap or a free flap. Free flaps are more susceptible to ischemic complications than pedicled flaps are, and careful monitoring in the postoperative period by clinical evaluation and Doppler ultrasound is performed to detect any impending vascular pedicle compromise. If vascular compromise does occur, the patient is taken back to the operating room to revise the vascular anastomosis.

Flap separation leading to wound dehiscence occurs most frequently at the distal edge of a pedicled flap. The cause is usually placement of the flap under too much tension or local necrosis of the distal portion of the flap. If an intact bed of granulation exists within the wound and there is no evidence of a CSF leak, conservative wound management with dressing changes to allow healing by secondary intention is adequate. In the presence of a CSF leak, the wound should be revised immediately. If the tip of the flap is necrotic but an adequate extra length of flap is available, the flap revision is performed by advancing the new distal end to cover the entire wound. If there is not enough length, a second pedicled or free flap should be used to either close the residual wound or replace the old flap entirely.

# SUMMARY

Cancer of the temporal bone is a challenging disease to treat effectively. Modern management of this disease consists of combined–modality therapy, including surgery to remove all evident disease and radiation therapy to treat the margins of resection. The surgical procedure of choice depends on the extent of disease and consists of lateral resection of the temporal bone; subtotal resection of the temporal bone; subtotal resection of the temporal bone, which also includes removal of the petrous apex. In the event of facial nerve sacrifice, primary reconstruction with a cable graft is performed. Postoperative radiation therapy should encompass the resection margins, residual parotid gland, ITF, and ipsilateral neck.

#### PEARLS

- Always consider the possibility of cancer in a patient with chronic otorrhea, especially if the patient has severe pain, a mass or ulcer, or facial paralysis or if the patient does not respond appropriately to medical management.
- Perform both CT and MRI to assess the bone and soft tissue anatomy and the extent of disease.
- Management of temporal bone cancer includes adequate resection, usually with lateral or subtotal temporal bone resection, and in most cases, postoperative radiation therapy.
- The cure rate is 80% to 100% for T1 and T2 cancers but remains limited for late-stage disease, with a mortality rate of 50% or higher.
- Compromise of facial nerve function requires diligent care of the eye.

## PITFALLS

- Performance of a mastoidectomy for staging or biopsy is never appropriate.
- Failure to achieve adequate margins will probably ultimately result in treatment failure.
- Large defects should not be closed by local tissue advancement; free flaps are more appropriate in selected patients.
- Failure to obliterate the eustachian tube may result in nasopharyngeal reflux and infection or CSF rhinorrhea when the dura is incompletely closed.
- Insufficient venous drainage may compromise the vascular integrity of the remaining pinna.

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