

## **Chapter 154: Infections of the External Ear**

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### **Applied Anatomy and Physiology of the External Ear**

The external ear consists of the auricle, external ear canal, and the lateral (skin-covered) surface of the tympanic membrane. The auricle or pinna is a flexible appendage of skin and subcutaneous tissue with a cartilaginous skeleton. The cartilage is elastic and forms the flared lateral extremity of an incomplete funnel that is set into the side of the head. Its medial portion is more attenuated and continues as the cartilaginous ear canal. Although cartilage forms the framework of the auricle, it is absent in the lobule, which consists of skin and fat. The cartilage is covered by perichondrium, which nourishes and regenerates it. Whereas the cartilage has no intrinsic blood supply or innervation, the perichondrium is both richly vascularized and innervated.

The auricle is covered by skin; subcutaneous tissue of varying thickness intervenes between the deep dermis and the perichondrium. Rudimentary muscle slips may be found on the posterior surface of the auricle and adjacent mastoid process.

In many lower mammals, the auricle is a motile acoustic sensor that amplifies sound and interpretes its direction. By comparison, the human ear is atavistic in structure and function.

The external ear canal is a passage that connects the auricle to the tympanic membrane. It originates in the embryo as an invagination from the surface of the head. Once it is fully developed, it becomes a deep, skin-lined sinus. Topologically, the external ear canal and the lateral surface of the tympanic membrane should be considered as part of the outer body surface.

The external ear canal gains its shape and structural support from its cartilaginous and bony walls. The lateral one third of the ear canal is formed by a funnel of cartilage. The cartilage is an extension of the auricular cartilage, which forms the skeleton of the pinna. The cartilage is incomplete in its circumference, with an anterosuperior dehiscence. This hiatus, which begins between the crus of the helix and the tragus, continues medially into the external canal. This deficiency of cartilage renders the auricle more flexible because a circumferentially fused funnel of cartilage would be stiff and prone to fracture. The flexibility of the outer ear is further increased by several transverse slits in the cartilaginous floor of the external canal. These fissures of Santorini transmit an inconsistent number of blood and lymphatic vessels.

The bony walls of the deep external canal are formed by the tympanic ring and a small portion of the squamous temporal bone. The tympanic ring is in reality U-shaped and forms the massive floor of the bony canal and its lateral walls. The superior deficiency between the arms of the U is bridged by the scutum, a plate of bone that separates the deep canal from the attic.

The length, caliber, orientation, and cross-sectional dimension of the external ear canal changes with growth. In the infant, the canal is shorter and straighter, with the cartilaginous portion dominating. During postnatal development the canal deepens and grows relatively more tortuous. This is due primarily to the increasing mass of the tympanic bone, the downward and anterior overgrowth of the mastoid process, and later the elongation of the craniofacial skeleton. In the adult, the canal assumes a lazy "S" configuration. It curves in two directions and ends by obliquely abutting the tympanic membrane (Plate 18, A).

The bony and cartilaginous walls of the ear canal are lined by skin and subcutaneous tissue. Although the skin is contiguous with the rest of the body surface, it demonstrates certain unique localized adaptations. The skin of the cartilaginous canal is thick and replete with exocrine glands and hair follicles. These glands secrete sweat, sebum, and cerumen and function to lubricate the hair follicles and canal skin as well as to aid in the removal of debris. The cartilaginous canal has a relatively thick dermis and subdermal connective tissue layer. The bony canal by contrast is lined by a thin layer of skin that lacks epidermal appendages. Its dermal and deeper soft tissue layers are sparse, and lie directly on the periosteum of the bony canal wall (Plate 18, B). The skin of the ear canal is amply vascularized. Its lymphatics drain to nodal substations that lie circumferentially around the external ear.

Aging brings about several important changes in the structures of the ear canal. There is atrophy of the subcutaneous tissues and thinning of the overlying skin. Decreased secretion by the glands of the ear canal results in skin that is dry, attenuated, and prone to trauma or breakdown. Cerumen becomes more concentrated and often hard and impacted.

The primary function of the ear canal is to carry ambient sound waves to the tympanic membrane. In order to perform this role, the canal's lumen must be kept patent and maintain a lining that is intact and healthy. The epithelial lining of the ear canal continually sheds kerating debris which, if left to accumulate, can obstruct the lumen of the canal and harbor potential pathogens. To prevent the accumulation of desquamated debris, the tympanic membrane and ear canal have developed a unique self-cleansing mechanism. There is a constant centrifugal migration of kerating flakes from the center of the tympanic membrane. The migration continues peripherally and then laterally along the bony canal wall. At the junction of bony and cartilaginous canals, the kerating layer encounters the hairs of the lateral canal. These hair shafts point laterally. The rows of hairs act as ramps to separate the kerating squames from the underlying skin. This sloughing is aided by the ample lubrication of the hair shafts and, possibly, by as yet unidentified enzymes that break the attachment between the kerating squames. The narrowing of the canal at the isthmus throws the separating sheet of keratin up into wrinkles and ridges that further aid in shearing the retained layer away from the underlying living epidermis (Johnson and Hawke, 1988). The keratin debris is shed into the lumen of the canal, where it is trapped by cerumen and eventually expelled from the ear.

The self-debriding mechanism of the ear canal is unique, but only one of several ways by which the ear canal keeps itself free of infection. Other important protective features include the acidic pH of the ear canal (6.5-6.8), which is below the optimal pH for most pathogenic bacteria; the water-resistant and tightly bound skin surface lining the ear canal; the ample blood supply and lymphatic supply to the skin and subcutaneous layer of the canal; and the antibacterial properties of cerumen, which like other exocrine secretions, contains

lysozyme. Thus in considering the various infections that may develop in the external ear canal, it is important to keep in mind that the ear canal is generally well protected against most pathogens. The onset of an infection therefore often coincides with the breakdown of the ear canal's protective mechanisms.

## **Bacterial Infections**

### **Acute diffuse bacterial external otitis (swimmer's ear)**

#### ***Pathogenesis***

Despite the numerous protective mechanisms listed above, the external ear canal is more prone to bacterial infection than skin surfaces elsewhere. The resident microbial flora, primarily *Staphylococcus epidermidis*, thrives on the skin surface and in the shed keratin. Other microorganisms, including occasional fungal spores, may be found in the canal lining or the cerumen, as benign colonizers or commensal flora. The ear canal may retain moisture and a humid microenvironment may persist, which can encourage microbial growth. Persistent moisture leaches out the acid ions of the skin, raising the surface pH to a neutral or alkaline level. This higher pH is more conducive to the growth of pathogenic bacteria. Keratin imbibes moisture, and the wet keratin layer creates a nourishing bacterial culture medium. With prolonged contact between the wet keratin and the underlying living skin, maceration with breakdown of local defenses may occur, resulting in a frank infection of the canal's lining.

Patients who are prone to external otitis are swimmers, patients with a tendency to retain water in the canal (due to stenosis, exostoses, or impacted debris), patients who live in humid climates, and patients with trauma (often self-inflicted) to the skin of the ear canal. Patients who wear tightly fitted hearing aid molds create their own tropical microenvironment. Submersion of the head, particularly in contaminated or infected water, is such a common cause of bacterial external otitis, that the condition is commonly called *swimmer's ear*. Swimmer's ear, however, may occur as the result of a self-inflicted scratch or abrasion of the canal, particularly in humid climates. During World War II this condition was so common among soldiers in the Pacific that it carried the moniker *Singapore ear*.

The predominant pathogen in swimmer's ear is *Pseudomonas aeruginosa*. This is an ubiquitous gram-negative rod that commonly thrives on moist surfaces. The bacteria can be air-borne. In the ear canal, it may occasionally be present as a normally benign local commensal. More commonly, it enters by introduction of contaminated material such as water. Acute bacterial otitis externa may also arise if pathogens infect the canal skin through an abrasion or laceration.

#### ***History and physical findings***

The clinical history is one of initial discomfort or itching of the ear canal, which rapidly progresses to tenderness and pain. In severe cases, pain may be constant and exacerbated by even trivial manipulation of the auricle. There may be a sensation of pressure and fullness and hearing loss may occur. This is due to edema of the canal with occlusion of the lumen. There is typically no otorrhea although some moisture may be present because of weeping from the skin surface. The infection is localized and systemic manifestations such

as fever or chills are absent.

Physical findings depend on the degree of infection. Discomfort can be elicited by palpation of the ear canal in most cases. This is best done by pushing up on the concha underneath the lobule. Eliciting tenderness by pulling back on the helix is often suggested, but this is a less sensitive test.

The auricle is usually unaffected by this condition, although the cavum conchae may show a slight "peau d'orange" appearance. This lymphatic stasis is due to inflammation of the canal. If the outer part of the ear is visibly involved, the examiner should suspect either a deeper infection or an allergic reaction to ear drops that the patient may have used. Extending deeper infection (such as erysipelas, discussed later) has no preferred direction, whereas ear-drop allergy typically affects the more dependent portions of the auricle.

Otoscopic findings in the canal itself vary. In early cases the canal is minimally red and dull. Absence of cerumen is a classic finding. As the infection progresses, the canal becomes narrowed due to edema of its lining (Plate 18, C). In severe cases, the canal may become pinpoint in caliber. Scant sticky secretion, which may be clear or turbid, may occlude the remaining lumen. The surface is extremely tender, making examination difficult. Although the infection is localized to the ear canal, regional lymphadenopathy is variably present.

### *Treatment*

Treatment of pseudomonal otitis depends on the severity of the disease, and may include topical therapy, systemic antibiotics, and adjunctive measures.

Although basic principles dictate that therapy should be based on culture, treatment is usually begun immediately based on the presumption that *Pseudomonas aeruginosa* is the chief pathogen. If a culture is not taken at the initial visit, it may be obtained subsequently if the ear is not responding to appropriate antipseudomonal medications. In all cases, the patient is cautioned to avoid wayer in the ear and to abstain from cleaning or manipulating the ear canal. Early cases usually respond to topical drops alone. A variety of otic drops are available, and they are usually combinations of antibiotics such as polymyxin B and neomycin, steroids, and a wetting agent such as propylene glycol, buffered to an acid pH.

Although most drops are effective, the otologist should understand the components of this therapy. The acidic pH of ear drops is an important part of treatment. It was shown years ago (Jones, 1965) that dilute acetic acid kills *Pseudomonas aeruginosa*. In folk medicine, vinegar has been used to good effect. Acetic acid drops, with or without steroids, such as VoSol and VoSol-HC, are available and may be prescribed when topical antibiotics must be avoided (eg, in a patient with topical neomycin sensitivity). Burow's solution (3% aluminum acetate) is another alternative; this is a soothing solution that decreases canal edema, restores physiologic pH and kills *Pseudomonas* (Dibb, 1985). Most ear drops contain a combination of antibiotics, and topical application allows the use of antibiotics that would be too toxic to be used systemically. The disadvantage of antibiotic drops is twofold: patients may develop cutaneous sensitivity, particularly to neomycin, and the prolonged use of broad-spectrum topical antibiotics may predispose to the growth of resistant strains or fungi. Similarly, steroids, which are added for their antiinflammatory effect, may have an adverse effect in

prolonging the condition and allowing for the overgrowth of other organisms. Finally, propylene glycol (used as a vehicle in most compounded drops) increases the adherence of the medication to the infected skin. It also raises the viscosity of the solution, which may be a problem in a canal with a pinpoint lumen.

Ophthalmic drops may be useful in certain situations. Gentamicin is currently available only in the ophthalmic formulation and is not formulated for use in the ear. The rationale for this, which is that gentamicin is ototoxic, is questionable, particularly because the much more toxic neomycin is commonly used, even in the presence of perforated tympanic membranes. Ophthalmic drops are also buffered to a higher pH, which may be a consideration in patients who find otic drops irritating. The lower viscosity of aqueous ophthalmic drops makes them useful when the lumen is narrowed by edema, or where a wick is used to carry the medication to the deep canal.

Whereas in milder cases the drops may be placed directly into the canal, on more severe cases a wick may be useful. The wick draws the drops down into the deep canal by capillary action. A wick is made by teasing out a few fibers from an absorbent cotton ball and saturating it in medicated solution. One end of the wick is inserted into the ear canal. Even a narrowed lumen usually accommodates the smallest pediatric ear speculum. When the canal is swollen shut, it can be partially pried open with a pediatric nasal speculum. The wick is grasped with an alligator forceps and gently insinuated as deeply as possible. It should be kept in the ear for 3 to 5 days and saturated with drops several times a day. If prefabricated sponge wicks are used, they may have to be trimmed; the forceful insertion of a rather large and dry sponge is painful in the presence of severe swelling. If the swelling prevents insertion of even the finest wick, the patient is instructed to use Burow's solution soaks on the auricle several times a day. Once the swelling has decreased, a wick can be inserted.

The role of systemic antibiotics is controversial. Most cases of pseudomonal external otitis respond to topical treatment, and until recently there has been no reliable oral anti-pseudomonal drug available. The recent introduction of ciprofloxacin gives the otologist the option to treat this condition both topically and systemically. The beginning emergence of resistant pseudomonal strains dictates that this drug should be used appropriately. At this time we suggest that ciprofloxacin be reserved for severe case only, and given at full dose (500 mg bid) for 10 days. Ciprofloxacin should not be used in patients under 17 years of age because of possible damage to developing cartilage.

Analgesics should be given for pain, which may be considerable. Codeine-containing medications are useful. Once the infection begins to subside, the wick is removed and drops are continued. As the canal opens, it can be cleaned using a microscope or otoscope. Where the infection had been severe, a considerable amount of macerated keratin and sloughed debris can be removed. Again the patient is cautioned to keep water out of the ears for at least 6 weeks. Patients prone to recurrent external otitis should consider using ear plugs when bathing or swimming. Over-the-counter drops of acetic or boric acid and alcohol may also be used routinely after swimming. Hearing-aid users who are prone to otitis externa should consider having the mold vented to improve aeration of the external ear canal.

### **Acute localized external otitis (furunculosis)**

A furuncle of the ear canal may present as acute otitis externa. Furuncles are small abscesses that form in the hair follicles. In the ear canal, they typically occur in the meatus superiorly, just inside the canal (Plate 18, D). The patient complains of tenderness on touching the area above the tragus. There is no hearing loss or aural discharge. The infection is localized, and distant signs or symptoms are unusual. Unlike in patients with swimmer's ear, palpation under the lobule is not painful. On insertion of the otoscope, however, there is exquisite tenderness. As the lateral canal comes into view, a red raised area becomes apparent, usually at the junction of the tragus and the anterior crus of the helix. Depending on the time of evaluation, the furuncle may be deep and diffuse or superficial and pointing. The medial canal is normal in appearance.

Furunculosis of the external canal is caused by *Staphylococcus aureus*. Treatment involves local and systemic measures. If the abscess is pointing, it may be nicked open with the beveled tip of a 14-gauge needle. Once the abscess is opened and pus expressed, antibiotic ointment can be applied. If the lesion is not pointing, application of topical heat may be useful, either with a heating pad or warmed oil-based ear drops. Systemic antibiotics should be given in the earlier stages but are not necessary if the abscess has been opened and evacuated. The patient is cautioned to avoid touching or cleaning the area. Attempts to squeeze an immature furuncle may lead to spread of the infection to deeper tissues.

### **Impetigo of the external ear**

Impetigo is a highly contagious infection that involves the superficial layers of the epidermis. The infective organism is *Staphylococcus aureus*, or less commonly, *Streptococcus pyogenes*. Impetigo of the ear canal is commonly seen in children, and there is often impetigo elsewhere, such as at the corner of the mouth. Although this infection may be associated with neglect (such as institutionalized children), it can occur in anyone.

Impetigo is usually carried to the ear by the dirty finger. For this reason, the typical early lesions are found at the entrance to the external canal. Unlike furunculosis, impetigo is a superficial spreading infection that may extend to the concha and even the auricle. The initial lesion forms a small blister that ruptures to exude straw-colored infected fluid. The exudate dries to a golden crust. As the infection spreads, confluent areas of weeping and crusting are seen (Plate 8, E). Impetigo of the ear should be treated by debridement of the infected area. This can be done with cotton-tipped swabs moistened with antiseptic solution or hydrogen peroxide. The infected areas are then covered with antibiotic ointment. Neomycin-containing ointments are useful, as is mucopirin (Bactroban), a single-agent ointment with anti-staphylococcal activity. The patient should be cautioned against touching the ear. The nails should be cut short and the hands cleaned with antibacterial soap. Systemic antibiotics are usually not necessary, unless the area of infection spreads. If the impetigo fails to resolve with strict local measures, dermatologic consultation may be necessary.

## **Erysipelas**

Erysipelas is a streptococcal cellulitis that may occasionally involve the ear. The cause is unknown, but it may result from a scratch or self-inoculation as the patient tries to clean his or her ear. Unlike swimmer's ear and impetigo, which are epidermal infections, erysipelas involves the dermis and at times the deeper tissues.

The clinical features of erysipelas are pain and swelling. The lesion is a spreading red area of cellulitis with an irregular perimeter that is raised and clearly demarcated from adjacent normal skin (Plate 18, F). If erysipelas begins in the ear canal or on the auricle, it typically spreads anteriorly over the face with no respect for anatomic boundaries. Erysipelas is further distinguished by its associated systemic features. The patient feels ill, with chills, fever, and malaise. Systemic involvement is not seen in the more superficial infections. The treatment of erysipelas includes topical soaks and systemic antibiotics. High doses of oral anti-streptococcal medications may be tried, but if the patient fails to show significant response within 48 hours, intravenous antibiotics effective against beta-hemolytic streptococcus should be considered.

### **Perichondritis and chondritis of the auricle**

Perichondritis and chondritis of the auricle are rare infections that involve the perichondrium and cartilage of the auricle respectively. Although there are noninfectious causes for inflammation of these tissues (such as relapsing polychondritis), on rare occasions a bacterial infection may be found. These infections are almost invariably the result of a laceration of the auricle, either traumatic or surgical. If the perichondrium is separated from the cartilage or if the cartilage is exposed, local devitalization in a dirty wound may result in perichondritis or chondritis.

The clinical findings include a history of recent trauma or surgery. In the absence of an infected open wound, there may be diffuse swelling of auricle. Exquisite tenderness on deflecting the cartilage is due to inflammation of the perichondrium and distinguishes these deeper infections from those that involve the skin only. The patient may have fever and chills.

Treatment of perichondritis and chondritis requires surgery and intravenous antibiotics. If an abscess is present, the area must be drained and left open to heal by secondary intent. If the wound is open, it should be reexplored and debrided. Devitalized cartilage must be cut back to living tissue. Packing with iodine gauze or topical soaks may be useful. Indwelling irrigating catheters have been suggested for local perfusion of antibiotics. Systemic antibiotics should be administered and should include anaerobic as well as aerobic coverage. The antibiotics should be adjusted based on culture as soon as feasible. If the infection is the result of an accident or a bite, appropriate coverage for tetanus or rabies should be considered.

## Chronic external otitis

Chronic external otitis is a diffuse low-grade infection of mixed etiology. The patient typically complains of itching and irritation in the canal rather than of pain. The appearance of the canal is usually dry and somewhat atrophic. Asteatosis (lack of cerumen) is common, and may be a cause rather than a result of this condition. Longstanding chronic external otitis may cause the canal skin to thicken, with resultant narrowing of the lumen (Plate 19, A).

Although there is often an infective element present in chronic external otitis, other factors predispose. These include asteatosis (inherent or caused by frequent cleaning), senile atrophy of the epidermis, and chronic irritation caused by manipulation or allergy. Because of the dryness of the skin, the ear canal may be difficult to culture in chronic external otitis. A cotton swab may need to be moistened with sterile saline before a culture is taken. The culture results are often nonspecific or are reported as containing "normal flora". Even commensals, such as *Staphylococcus epidermidis*, can exert a pathogenic effect in chronic otitis externa, and a pure culture of such flora should not be ignored. Treatment of chronic otitis externa is difficult because the underlying factors of dryness, atrophy, and chronic irritation need to be addressed along with the infection. Steroid- and antibiotic-containing cream may be used but should be discontinued if acute infection, heralded by pain, develops.

## Otomycosis

Fungal infections of the external ear canal are becoming more common. Otomycosis is usually a chronic superficial infection that affects the deeper ear canal and the tympanic membrane. The fungi involved are most commonly *Aspergillus* and *Candida* species, although other fungi may also be found. *Aspergillus* is a sporulating mold that forms hyphae. *A. flavus*, *A. niger*, and *A. fumigatus* are the three species most commonly found in the ear. They form spores that are yellow, black/brown, and gray, respectively. *Candida* is a dimorphic fungus. It can exist as a pseudohyphenate form and as a budding yeast. *C. albicans* and *C. parapsilosis* are the usual species in candidal otomycosis.

The environmental prevalence of fungi means that susceptible ear canals may become inoculated by air-borne spores. Three kinds of otomycosis should be considered: nonpathogenic colonization, superficial infection, and deep (invasive) mycosis.

Nonpathogenic colonization by *Aspergillus* species is commonly found in neglected mastoid cavities. Tufts of fungal hyphae may be seen growing on cerumen or skin debris. The self-cleansing mechanism of the ear has been surgically altered in these cavities, and the stagnant lining is vulnerable to fungal colonization.

The treatment for these cases is simple debridement. Medication is usually not necessary, but all the fungal material must be removed by using the microscope.

Superficial mycosis is the most common form of fungal infection in the ear. Several etiologic factors may predispose to mycotic infections of the ear canal. These include: (1) chronic ear infection with prolonged use of antibiotic-steroid ear drops, (2) evidence of yeast or fungal infection elsewhere in the body, (3) diabetes or (4) other diseases that may alter the



body's immune response.

Patients with chronic otitis externa or otitis media may have used topical drops to excess. The bacterial flora of the ear canal is lost and the skin pH is lowered. An acidic milieu is harmful to most bacteria, but allows an overgrowth of fungi. If a patient with chronic infection no longer responds to ear drops or has a recurrence of symptoms, otomycosis should be suspected.

It is not uncommon for patients with *Candida* otomycosis to have other forms of mucocutaneous candidiasis. We have seen patients with *Candida* otomycosis and vaginal moniliasis who experience flare-ups of both infections during their menstrual periods. Hormonal and pH changes may be responsible. Patients with candidiasis of the ear canal may also have a chronic infection of their nails, which may reinoculate the ear canal, and should be inspected for thickening and onycholysis.

Patients with hyperglycemia are more prone to fungal infections. In addition to otomycosis, they may also have intertriginous infections. The clinical course of the mycosis sometimes fluctuates with the level of sugar in the blood.

Physical examination in these cases reveals that the infection is confined to the ear canal only. With *Aspergillus*, the canal may be filled with macerated infected keratin that looks like wet tissue paper. The debris is speckled with black or yellow dots (Plate 19, B). Microscopic examination may disclose discrete clumps of hyphae with conidiophores. *Candida* infection has a more variable appearance. The deep canal may be filled with a whitish cheesy material. At times there is no exudate, but the skin of the deep canal wall is edematous, dull, and red. Discrete organisms are not usually seen.

Whereas the above descriptions are suggestive of otomycosis and call for antifungal treatment, the appearance of the ear canal may at times be quite nondescript. Fungal infection should therefore be suspected in all cases of chronic external otitis that have not responded to conventional topical therapy. Even allergic or other noninfectious inflammations usually show a good response to topical steroid-containing medications. If the response is short and is followed by a more flagrant relapse, otomycosis should be considered regardless of appearance. In the absence of hyphae or other diagnostic features, a KOH preparation of a superficial scraping from the canal wall may yield the diagnosis. Cultures may at times show a mixed infection of bacteria and yeasts, and complete management requires the treatment of both infective agents.

The treatment of superficial otomycosis begins with eliminating the predisposing factors. Conventional ear drops are discontinued. If there is mycosis elsewhere in the body, appropriate measures are taken. The patient is asked to cut his or her nails short. Female patients may need referral to a gynecologist for treatment of vaginal moniliasis. In patients with recurrent or persistent otomycosis, the clinician should consider a fasting blood glucose or glucose tolerance curve. Topical treatment begins as the ear canal is thoroughly debrided of all fungal elements. Antifungal drops or cream may be applied. Mycostatin or ketoconazole cream can be layered into the deep canal using a 5-cc syringe and a large-bore needle. Topical Cresylate (M-cresyl acetate) or Vioform drops have been found useful. We have also had success using tolnaftate (Tinactin) (Liston and Siegel, 1986). This preparation is sold over

the counter for the treatment of athlete's foot and is available as a cream, solution, or powder. If the patient is a hearing-aid user, the mold should be kept out of the ear canal as much as possible, cleaned prior to each reinsertion, and vented if feasible.

Topical treatment can be supplemented with systemic medication at the clinician's discretion (Zelen, 1985). If ketoconazole or mycostatin tablets are given, they should be continued for at least 2 weeks. Invasive otomycosis is rarely found in the ear canal (Phillips et al, 1990). We have recently seen a few patients with AIDS and fungal pneumonia who also developed invasive aspergillus otomycosis. The deep ear canal in these patients was filled with granulation tissue that was adherent, painful, and friable. Treatment of such invasive otomycosis requires intravenous amphotericin B (Bickley et al, 19880).

### **Malignant otitis externa**

The term *malignant otitis externa (MEO)* refers to a progressive and necrotizing *Pseudomonas* infection of the ear. This condition, originally described in the temporal bone by Melzer and Kelemen, was brought to the general attention of otolaryngologists by Chandler (1989). Malignant otitis externa occurs in immunocompromised patients, primarily those with diabetes (Britigan and Blythe, 1987). MOE begins as a diffuse *Pseudomonas* external otitis. Because of defective immune defenses (and perhaps microvessel pathology), the infection does not remain localized to the skin of the ear canal. It extends to the deeper tissues and invades medially along the floor of the ear canal. Extension medially and posteriorly leads to invasion of the mastoid, the facial nerve, and the base of the skull. If control cannot be achieved, malignant otitis externa may be fatal.

This condition should be suspected in any person with diabetes who also has a persistent external otitis. Poor control of diabetes is the most significant predisposing factor. Once the infection begins, control of hyperglycemia may be worsened as a vicious circle develops. The condition is seen less often in other immune-deficient patients. We have not yet seen a significant incidence of malignant otitis externa with AIDS. This would suggest that microvascular obliteration or other disorders seen with diabetes may play a more important role than the immune defect alone.

The usual patient with malignant otitis externa is elderly and has diabetes and otalgia. The pain in this condition is generally worse than in cases of uncomplicated swimmer's ear and is typically most severe at night. Examination of the ear canal may reveal the usual features of skin inflammation, with redness, swelling, and tenderness. A classic though variable feature of malignant external otitis is a nubbin of granulation tissue found on the floor of the canal (Plate 19, C). This tissue arises at the junction of the bony and cartilaginous canal and is the result of osteitis of the lateral lip of the tympanic bone. Probing in this area will often reveal a small pocket of necrotic soft tissue surrounding the osteitic bone. It is in this area that the infection invades into the deeper tissues, although some believe that the initial invasion gains access via the fissures of Santorini.

At a later stage, malignant external otitis may present with facial palsy. This is the result of the infection extending posteriorly to the stylomastoid foramen. If unchecked, the infection spreads up along the facial canal into the mastoid. If the infection is untreated, osteitis of the skull base develops with multiple cranial nerve palsies. Diagnosis of this

condition is clinical: the clinical impression may be confirmed by radionuclide scans of bone and soft tissue (Behjati et al, 1987; Garty et al, 1985) or a CT scan (Mendelson et al, 1983). Magnetic resonance (MRI) studies have also been used (Gherini et al, 1986). Bone scans may show increased uptake over the tympanic bone and skull base because of periosteitis and osteitis. CT scans may show clouding of the mastoid, or, at a later stage, rarefaction of the skull base. It must be stressed, however, that a negative bone scan does not rule out early MEO.

There are three aspects to the treatment of MEO. The most important part of therapy is gaining control of the patient's diabetes. The internist therefore must be involved early and on an ongoing basis. Patients normally on oral hypoglycemics may need to be hospitalized and placed on insulin.

Local debridement of the infection is also important and must be gauged accordingly to the extent of the infection. In early MEO curettage of the osteitic lateral lip of the tympanic bone may be adequate. Mastoidectomy or partial resection of the skull base may be needed if the facial nerve or the lower cranial nerves are involved. This is not an abscess-forming infection and the surgeon will find granulation tissue and phlegmon, rather than coalescent suppuration. Adequate surgical debridement both removes infection and improves the control of the diabetes. Hyperbaric oxygen is a useful adjunct in reducing the anaerobic milieu in the ear canal. It has been used successfully even in patients whose allergies have limited the use of antibiotics (Pilgramm et al, 1986; Shupak et al, 1989).

Antibiotics should be given early, in adequate amounts, and for a long enough time. Although several recent studies have shown that patients in the early stages of MEO respond well to oral ciprofloxacin (Lang et al, 1990), the mainstay of therapy remains intravenous antibiotics. A combination of drugs, usually an aminoglycoside and a semisynthetic penicillin are given. Peak and trough levels are monitored, along with renal and auditory function. Topical gentamicin or acetic-acid preparations can also be used by means of drops or an indwelling wick.

The earliest sign of response to therapy is a decrease in pain. Even before there is a visible change in the ear, the nurses may report that the patient demands analgesics less often. Better diabetic control is another early sign of recovery. Treatment for MEO should be continued until the infection has completely cleared (Uri et al, 1984). This may require prolonged hospitalization and up to 6 weeks of intravenous antibiotic therapy. Some clinicians will discharge patients earlier on topical drops and ciprofloxacin, if the organism is sensitive to that drug.

### **Herpes Infections**

Although primary herpes simplex may involve the auricle, it is more common for herpes zoster to develop in this area. Herpes zoster (shingles) is a reactivation of a varicella infection that has lain dormant in the nerve roots. The cause for this reactivation is not always known. It may accompany a loss of immune function, as with leukemia. The coincidence of stress and shingles is anecdotally well known and may be another manifestation of a transiently decreased immune response. Herpes zoster is found in some cases to herald an occult malignancy.

Herpes zoster appears as clumps of blisters. The blisters are confined to specific dermatomes, reflecting the segmental innervation of the body. In the ear, they are seen most often in the concha or superficial ear canal (Plate 19, D). At times they involve the lower part of the auricle and lobule and the adjacent upper neck. The blisters begin as raised reddish papules that vesiculate and then crust. Immunosuppressed patients may have a more generalized distribution.

Patients with herpes zoster of the ear may also have hearing loss, vertigo, or facial paralysis. Ramsay Hunt syndrome (herpes zoster oticus) presents with hearing loss and facial paralysis caused by herpes virus. The blisters in the ear are an early and transient finding that may be missed by both patient and clinician.

The treatment for herpes infection of the external ear consists of topical debridement; antiviral agents are usually reserved for more extensive infections. A neglected herpes zoster infection of the ear canal may become crusted and secondarily infected by bacteria. We have seen this condition misdiagnosed as *malignant otitis externa*. Careful cleaning revealed the crusting blisters and the correct diagnosis was made. The use of oral prednisone is recommended by some clinicians, primarily to reduce the incidence of postherpetic neuralgia.

### **Bullous Myringitis**

Bullous myringitis (myringitis bullosa hemorrhagica) is a viral infection that involves the tympanic membrane and adjacent deep canal. The condition is associated with a viral upper respiratory infection and is more common in the winter. The patient complains of severe ear pain and, at times, some decrease in hearing ability. Examination reveals reddish vesicles on the surface of the tympanic membrane that enlarge to form bullae (Plate 19, E). The bullae are filled with a straw-colored fluid that may be tinged with blood, and they may become confluent. Myringitis bullosa often affects both ears in succession. In some cases a sympathetic middle ear effusion develops and occasionally a reversible sensorineural hearing loss occurs.

The causative organism responsible for bullous myringitis is not known. A viral etiology is most likely although in some instances *Mycoplasma pneumoniae* has been cultured.

The management of bullous myringitis involves topical measures, systemic antibiotics, and analgesics. Opening the blisters with a beveled needle or myringotomy knife ("blebotomy") may relieve the pain in some cases, although most of the pain probably occurs during the early formation of the bullae. Topical analgesic drops containing benzocaine and lidocaine have been used. If mycoplasmal infection is of concern, oral erythromycin may be given. Pain relief often requires oral narcotics in moderately high doses.

### **Granular Myringitis**

Granular myringitis represents a spectrum of conditions. The findings range from a nubbin of granulation tissue on the surface of the tympanic membrane to a carpet of granulation tissue covering the entire membrane and deep canal (Plate 19, F). Discomfort and a mild hearing loss may be accompanied by some scant weeping of the raw surfaces.

A variety of infective agents have been cultured from the granulating surface, including *Staphylococcus*, *Pseudomonas*, and *Candida*. Despite the bacteriologic findings, the etiology of granular myringitis is not clear. The condition may be the result of trauma or it may be an idiopathic inflammatory disorder with a secondary infection. Granular myringitis is treated by physical measures and antibiotic drops. Limited areas of granulation may be removed by curettage, or cauterized with silver nitrate. Topical application of other denaturing agents, including formaldehyde, have also been recommended. The management of more extensive granular myringitis is less satisfactory. Despite aggressive management with topical antibiotics, physical measures, and oral steroids, some cases persist unchanged. Others progress to an obliterative inflammation of the deep ear canal with subsequent epithelialization and the formation of a "false fundus" (Hawke and Jahn, 1987).

### Fig. 154a

Plate 18. A, Latex case of adult external ear canal. Note the complex curvature of the canal and the oblique relationship of the canal to the tympanic membrane. B, Axial section of deep external ear canal. The skin and sparse connective tissue lie directly on the bony walls of the deep canal. C, Acute external otitis (swimmer's ear). The inflamed canal has narrowed to a slit because of edema. Note the absence of cerumen and the *peau d'orange* appearance of the canal. D, Furuncle of the ear canal. The small abscess forms a localized swelling at the entrance of the ear canal. The furuncle is pointing and has begun to rupture. E, Impetigo of the external ear canal. This superficial staphylococcal infection has caused confluent areas of golden-brown crust in the dependent part of the concha and lobule. F, Erysipelas. Note the diffuse erythema and swelling of the auricle and the advancing margin of cellulitis involving the face. This line of demarcation between involved and uninvolved areas is clearly defined.

### Fig. 154b

Plate 19. A, Chronic external otitis. The hallmarks are atrophy of the skin of the ear canal and stenosis due to chronic irritation. This patient was a Q-tip abuser and debris from the canal with cotton fibers has been pushed medially against the tympanic membrane. B, Otomycosis. This florid infection with *Aspergillus flavus* has infected the deep canal with tufts of fungus crowned by yellow conidiophores. C, Malignant otitis externa. A nubbin of granulation tissue sits on the floor of the ear canal at the junction of the bony and cartilaginous portions. D, Herpes zoster. Note the infectious vesicles with erythematous margins involving the concha, as well as a crusted older lesion over the mastoid. The patient also had facial palsy. E, Bullous myringitis. The deep ear canal and tympanic membrane are acutely inflamed. A large bulla is seen over the posterior tympanic membrane. F, Granular myringitis. A tongue of granulation tissue is seen over the umbo and extending to the posterior margin of the tympanic membrane.