Paparella: Volume II: Otology and Neuro-Otology

Section 3: Diseases of the Ear

Part 2: External Ear

Chapter 21: Trauma and Infections of the External Ear

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The External Ear Canal

Otitis Externa

Otitis externa is one of the most common diseases encountered by the otolaryngologist. The treatment of otitis externa may be simple and easily accomplished in one office visit, or it may be protracted and frustrating with a fatal outcome. Successful treatment depends on a thorough understanding of the pathophysiology of each patient, a proper diagnosis, and treatment tailored to the individual case.

Pathophysiology

The external auditory canal is anatomically designed so that it is protected against the entrance of foreign bodies and infections. The tragus helps to shield the orifice of the canal from foreign bodies. The anterior direction of the canal from lateral to medial, narrow isthmus, hairs of the outer third of the canal, and sebaceous and apocrine glands that empty into the hair follicles and produce cerumen also protect the external auditory canal. The cerumen covering the squamous epithelial lining has an acid reaction. It collects in the outer third of the ear canal, providing a chemical barrier to infection and an additional obstruction to the entrance of foreign bodies.

Many local factors may interfere with the normal defenses against infection. Fastidious individuals, who feel that the presence of cerumen in the ear canal is a sign of uncleanliness, carefully clean cerumen from their own and their children's external auditory canals. Removing the cerumen eliminates an important barrier to infection. The lipid content of cerumen tends to prevent moisture within the external canal from entering the pilosebaceous units, thereby preventing maceration of the squamous epithelium of the external auditory canal. Its acid pH inhibits the growth of bacteria and fungi. Recurrent douching of the external auditory canal with water, which occurs when swimming or scuba diving, tends to remove the cerumen from the ear canal and dissolve some of its more water-soluble elements. This increases the possibility of penetration by bacteria (Senturia, 1957). Washing the ear canal with soapy water leaves a film of alkali along the canal wall. This also predisposes the patients to otitis externa. A narrow canal or excessive cerumen may permit the accumulation of water within the external canal during swimming and lead to recurrent otitis externa.

In addition to removing cerumen, cleaning the external auditory canal, whether performed with cotton-tipped swabs, bobby pins, fingernails, or the tip of a pen or pencil, may cause abrasion of the epithelial barrier and permit the entrance of organisms into the

tissues. Many patients, in times of emotional stress, have a habit of scratching their ears with fingernails or other objects. The feeling of fullness caused by serous otitis media may cause a patient to scratch and dig at the ears and lead to otitis externa (Morrison and MacKay, 1976). The mild inflammation caused by scratching produces itching that leads to further scratching, with this cycle continuing until the skin becomes infected. Epidermolysis bullosa may affect the external auditory canal when the skin is traumatized (Thawley et al, 1977).

Many systemic conditions, such as anemia, vitamin deficiency, endocrine disorders, and various forms of dermatitis, lower host resistance to infection. Seborrheic dermatitis is a systemic disease that may predispose a patient to otitis externa. It usually presents with a history of infantile eczema, severe dandruff, and seborrhea corporis. Characteristic findings in patients with seborrheic dermatitis involving the external auditory canal include retroauricular eczema and scaling within the outer third of the canal, which may extend into the cavum conchae. Psoriasis may also occur in the external auditory canal and must be considered predisposing cause of otitis externa.

Circumscribed neurodermatitis, which is occasionally present in the external auditory canal, produces spasmodic itching associated with anxiety. Contact dermatitis involving the auricle and the external auditory canal is very common. It may be secondary to the use of hair sprays or of chemical leached from the earpieces of eyeglasses or from the earmolds of hearing aids. Contact dermatitis commonly occurs secondary to drug instillation into the external canal. Neomycin is probably the most common sensitizing drug used in the ears. Allergies to bacteria may also play a role in external otitis when, as in the case of chronic otitis media, the external auditory canal is subjected to constant purulent drainage. In these cases the organisms and their metabolites elicit an allergic response in the skin of the external canal. Last, the heat and humidity and increased swimming in warmer climates tends to increase the moisture within the external auditory canal, leading to the growth of bacteria and fungi.

The normal ear canal is sterile in about 30 per cent of individuals. In 50 per cent of normal individuals, however, *Staphylococcus albus* is cultured along or in combination with other microorganisms, usually diphtheroids. *S. aureus* and *Pseudomonas aeruginosa* are isolated uncommonly in cultures from healthy ears (Stewart, 1951). In cases of otitis externa, bacteriologic cultures more commonly show a mixed flora, with *P. aeruginosa* predominating (Feinmesser, 1982). This microorganism is especially common in infections in tropical and subtropical regions (Singer et al, 1952). Mycologic studies suggest that fungi have limited etiologic significance in otitis externa (Jones, 1971; Singer et al, 1952). Singer found diverse mycologic flora in both normal and infected ears. In his study, 31 per cent of normal ears and 38 per cent of infected ears yielded fungal isolations. The most common fungi isolated in both normal and infected ears was *Aspergillus* (SInger et al, 1952). Fungi appear to be more common in ears treated previously with topical antibiotics. Water quality, as measured by bacterial counts, is not related to the incidence of otitis externa in swimmers (Calderon, 1982).

Diagnosis

In general, the diagnosis of otitis externa presents no problem to the experienced otolaryngologist. The patient usually presents with a characteristic picture suggesting infection, which includes pain, discharge from the external auditory canal, tenderness upon

movement of the tragus and auricle, and occasionally fever and cellulitis. However, the predominant symptoms may be itching, hearing loss, aural fullness, or discharge. These symptoms, as well as the physical findings, are helpful in determining the specific factors that play an etiologic role in each case of otitis externa. Dry, scaly skin associated with an itchy external canal suggests the presence of seborrheic dermatitis, especially in the presence of severe dandruff or other signs of seborrhea. Itching may also be characteristic of superficial fungal infection or a contact dermatitis or eczema. The weeping, crusting, and itching associated with eczematous otitis externa is diagnosed by examining the ear and by noting the related dermatoses elsewhere on the skin. Neurodermatitis, which is presumably caused by chronic rubbing or scratching of the skin, is usually diagnosed by the anxiety that the patient exhibits, by the thickened skin of the external auditory canal, and by other patches of neurodermatitis on the body. A feeling of fullness with itching in the ear may represent otomycosis in the external auditory canal. In the usual case of *Aspergillus niger* infection, examination shows the characteristic grayish membranes.

Treatment

There is no single therapeutic approach that will cover all cases of otitis externa. The principles of therapy for otitis externa are as follows:

1. Relief of pain or discomfort.

2. Elimination or control of predisposing causes.

3. Thorough cleansing of the aural canal.

4. Acidification of the external auditory canal.

5. The judicious, limited use of specific medication.

6. Termination of therapy, particularly specific therapy, as promptly as possible.

The need for the relief of pain seems obvious but is often overlooked in the rush to apply medications to the external auditory canal. Otitis externa can be a severely painful and debilitating condition, requiring the use of codeine or meperidine hydrochloride for a short period of time while the disease is being controlled.

Successful treatment of otitis externa must begin with an understanding of the causes of the particular case at hand. Systemic conditions, including anemia, hypovitaminosis, endocrine disturbances, and dermatologic conditions should be addressed. It is often necessary to control severe dandruff of the scalp before a refractory external otitis will respond to local treatment. Persistent cleaning or digging at the ears by the patient must be controlled before other therapy will be effective.

The most important factor in the treatment of most cases of otitis externa is the thorough and repeated debridement of the external auditory canal by the physician. Medications placed in the canal in the form of drops are unlikely to reach the surface epithelium unless the canal is thoroughly cleansed of desquamated epithelium, pus, and

cerumen. Thorough cleansing of the ear canal is usually necessary at least weekly until the infection is controlled. Because the canal may be extremely painful and tender, it is preferable to flush the canal with Burow's solution. In particularly refractory cases, the patient is given a 60-mL syringe and a prescription for Burow's powders or tablets. The patient is instructed to dissolve two tables in a pint of water at home and irrigate the ear three or four times daily. The solution must be warmed to body temperature before irrigating the ear to avoid a caloric effect; this may be accomplished by diluting the previously described solution in half, using very warm tap water, prior to each instillation. Usually, the powders are first dissolved in boiling water. The patient can tell when the ear is being irrigated adequately, as it will feel similar to the irrigation performed in the office by the physician. A member of the family can usually perform this maneuver for the patient over the sink. Although this procedure is messy and inconvenient, it has a high patient acceptance if the physician explains the necessity for its use.

When the ear responds to this therapy, usually after 1 or 2 weeks, the patient may be given any one of a number of proprietary preparations to place in the ear once or twice daily. These preparations should have an acid pH and be in a nonsensitizing base such as propylene glycol. Burow's drops with acetic acid and Vo Sol otic solution are two such combinations of medications. Whichever agent is selected it must be applied in sufficient quantity to cover the whole surface of the external auditory canal. Therefore, the patient should be instructed to place a whole dropperful in the ear canal, keeping the head to the side to allow the drops to remain in the ear canal and coat the surface before straightening the head and allowing the excess medication to run out.

Culturing of the ear canal is usually performed on the second or third visit if the otitis externa appears to be an infectious process and is not responding to therapy. If the external auditory canal is infected and there is no accumulation of pus or debris in the canal, one of the many proprietary ear drops containing a combination of antibiotics effective against staphylococcus and gram-negative organisms, a topical steroid, and an acid pH may be prescribed. This therapy is easier to implement than irrigation. However, the physician must be constantly alert to the development of hypersensitivity to one of the antibiotics, particularly neomycin. This reaction may be partially masked by the steroid that is usually included in such preparations. Occasionally, in severe infections, the skin and subcutaneous tissue of the external auditory canal are swollen to such a degree that medication cannot be instilled into the ear canal. Debris has collected and is trapped within the canal. In these cases, a cotton wick must be gently inserted into the canal and medication placed on the wick to be carried medially by capillary attraction. After 1 or 2 days, the canal usually opens, permitting cleaning and instillation of the medication.

Fungal infection of the external auditory canal is usually caused by *Aspergillus niger* infection. This infection may be recognized by the velvety-grayish membranes that line the medial two-thirds of the external auditory canal and frequently fill the canal. Suction removal of the membranes reveals an inflamed epithelial lining of the canal. The canal must be thoroughly cleansed by suction and irrigation and then treated with a fungicidal drug. Gentian violet solution is usually effective topically but has the disadvantage of staining the tissues of the cavum conchae and sometimes the patient's clothes. It may be applied in the office after thorough cleansing of the ear canal. The patient may then be treated at home with an acidic alcohol solution (8 drops vinegar qs to 10 mL with 10 per cent isopropyl alcohol), or

Burow's drops. In particularly refractory cases, Lotrimin drops instilled three times daily with weekly or biweekly cleaning of the ear canal in the office has been successful. If this approach does not work, the external auditory canal may be filled at 2-week intervals with an ointment containing antibacterial and fungicidal preparations. This approach frequently succeeds because it provides constant contact of the medicine and prevents water and contaminated particles from entering the external auditory canal. When the ear canal is healed and the infection cleared, therapy should be terminated. If the symptoms recur the patient should return to the physician rather than begin treating himself with a prescription from a previous infection.

Systemic antibiotics with a broad spectrum of bactericidal effect are helpful when there is a surrounding cellulitis, cervical lymphadenopathy, or signs of systemic infection. Although ampicillin is the medication of choice in most instances, oral tetracycline or cephalosporin may also be effective. Because of the nature of the mixed flora cultured in cases of otitis externa, the systemic antibiotic is selected empirically and not on the basis of the culture.

Furuncles may form in the outer third of the external auditory canal. These are staphylococcal infections of the pilosebaceous units that present with the symptoms previously described for otitis externa. Although they are usually treated with analgesics, systemic antibiotics, local heat, and antibiotic solutions, incision and drainage may be necessary.

In rare instances, chronic, irreversible changes occur on the skin and within the subcutaneous tissue of the external auditory canal. These changes may render the canal susceptible to persistent, chronic otitis externa. In these cases the skin is markedly thickened and often polypoid. The pilosebaceous units are usually nonfunctioning, and the cerumen and acid mantle are not present to aid in the prevention of infection. In these instances of chronic, intractable otitis externa, excision of all infected skin with a meatoplasty, with or without skin grafting, has been successful (Paparella, 1981; Proud, 1966).

Prevention

Prevention of recurrent otitis externa may be accomplished only by strict attention to the predisposing causes. Seborrheic dermatitis, psoriasis, eczema, and contact dermatitis must all be treated adequately. Trauma to the external canal must be avoided. Patients with a history of recurrent attacks of external otitis after swimming are given directions to use acidic alcohol drops in their ears prophylactically after exposure to water. A 10-mL bottle is filled with rubbing alcohol and 8 drops of vinegar. A dropperful of this solution is placed in each ear after the day's swimming. Divers exposed to water regularly and for long periods of time find this particularly helpful in preventing otitis externa.

Malignant Otitis Externa

Malignant otitis externa is a progressive, debilitating, and occasionally fatal infection of the external auditory canal, surrounding tissue, and base of the skull. Although uncommon, it is seen perhaps once or twice yearly in a busy otolaryngology practice. First described by Meltzer and Kelemen (1959) and later elaborated upon by Chandler (1968), it is an uncommon infection caused by *P. aeruginosa* in patients with low resistance to infection.

Pathophysiology

Typically the disease occurs in the elderly diabetic. The vasculitis of diabetes enhances the vasculitis produced by the *Pseudomonas* organism (Chandler, 1977). Other predisposing conditions include arteriosclerosis, immunosuppression by chemotherapy, steroid administration, hypogammaglobulinemia (Meyerhoff et al, 1977), neutropenia with decreased neutrophil chemotactic activity (Ichimura et al, 1983), and other disorders of cellular immunity (Yust et al, 1980). The *Pseudomonas* organism is cultured from the external auditory canal in pure or mixed culture. This organism produces exotoxins, including a neurotoxin. It contains enzymes, including lecithinase, hemolysin, lipase, esterase, and a variety of proteases (Lowbury, 1975). These enzymes cause a necrotizing vasculitis that aids the organism by destroying local tissues and resisting phagocytosis. Leukocidin and hemolytic toxin also produced by *Pseudomonas* organisms destroy white blood cells and inhibit their phagocytic chemotactic activity (Ichimure et al, 1983; Corberand et al, 1982). The infection begins in the external auditory canal, probably as a mixed infection, and later becomes a pure *Pseudomonas* infection. The disease may spread from the external auditory canal through Santorini's fissures in the conchal cartilage to invade the periauricular tissue, including the parotid gland, the temporomandibular joint, and the soft tissue at the base of the skull. From there, the infection normally progresses along the base of the skull. This infection causes paralysis of the seventh cranial nerve at the stylomastoid foramen; the ninth, tenth, and eleventh cranial nerves at the jugular foramen; and the hypoglossal nerve at the hypoglossal canal. The jugular vein may become thrombosed, progressing to a lateral sinus thrombosis. Alternatively, the infection may progress from the external auditory canal through the tympanic membrane into the middle ear and throughout the mastoid air cell system. From here it has access to the petrous apex and the intracranial structures and brain stem. The infection may progress into the mastoid air cell system by producing osteitis in the bony external auditory canal wall. The progressive cranial polyneuropathy is indicative of a very poor prognosis, with impending meningitis and lateral sinus and cavernous sinus thrombosis. Recent literature reported a 67 per cent mortality in patients with malignant external otitis and facial nerve paralysis and an 80 per cent mortality in patients with other cranial nerve involvement (Meyerhoff et al, 1977).

Diagnosis

The diagnosis is based upon a history of refractory, progressive otitis externa in an elderly diabetic or immunosuppressed patient. Invariably, there is granulation tissue at the junction of the cartilaginous and bony external auditory canal, with bare or necrotic cartilage occasionally seen at this site. The tympanic membrane is usually intact. Culture reveals *P. aeruginosa.* The severe pain produced by this infection is monitored in following the progress of treatment. Recurrent pain after apparent control is an ominous sign and warrants aggressive management. The disease may require several attempts at control (Caruso et al, 1977). Although the diagnosis of malignant external otitis is made using clinical criteria, the extent of the disease is difficult to determine. Additional information is needed regarding the exact extent of infection in the soft tissue and bone at the base of the skull.

Conventional x-rays and computed tomography are insensitive in determining the presence or extent of cellulitis or early osteomyelitis. In contrast, both technetium and gallium scans are very sensitive (100 per cent positive), and accurate in tracing the extent of disease

in patients with early malignant otitis externa (Strashum et al, 1984). They are normal in patients with benign otitis externa. Although technetium scans remain positive long after the osteomyelitis has cleared, gallium scans become negative as the infection clears. Thus, gallium scans offer a means to follow the progression of disease or recovery (Noyek et al, 1984). Because uncertainty about the extent of the disease results in undertreatment, with increased morbidity and mortality (Lucente et al, 1983), these scans provide an important contribution to successful treatment.

Treatment

The principles of successful treatment include control of the diabetes, administration of antibiotics, and aggressive local wound care, usually with surgical debridement. Currently the drugs of choice are tobramycin plus ticarcillin. These two drugs are used in combination because of the propensity of the *Pseudomonas* organism to develop resistance to either drug when given alone. In patients allergic to penicillin, desensitization may be carried out in order to use ticarcillin. Since tobramycin is both nephrotoxic and ototoxic, these parameters of function must be monitored before and during therapy. Aminoglycoside blood levels should be monitored. Alternative drugs are amikacin, gentamicin, and colistimethate. Moxalactam is active against *P. aeruginosa* and has been used successfully in malignant otitis externa (Haverkos et al, 1982).

Local wound care usually demands aggressive debridement of the infected tissue, sometimes including bone, cartilage, and the parotid gland. Each case should be treated individually, and a mastoidectomy should be avoided if the mastoid air cell system is not involved. Following debridement, topical wound care may be accomplished using antibiotics effective against *Pseudomonas* (eg, colistin, polymyxin B, and neomycin); 4 per cent acetic acid soaks are also effective. Patients who have had this disease must be followed closely, and close cooperation is necessary between the infectious disease experts and the otolaryngologist.

Trauma

Abrasions and Lacerations

Abrasions and lacerations of the external auditory canal are common and may be caused by the patient or by the physician. These injuries heal spontaneously unless grossly contaminated. The status of the tympanic membrane and facial nerve should be recorded and the patient followed closely for signs of infection. Proprietary ear drops containing antibiotics are usually effective in treating infected superficial lacerations and abrasions of the external auditory canal. Deeper lacerations or avulsion of skin may require stenting the external auditory canal with petrolatum gauze covered with neomycin-containing ointment. This allows lacerations to heal without infection or stenosis. Partial avulsion of the skin usually does not require grafting because the remaining skin of the external auditory canal regenerates quickly if there is no infection.

Temporal Bone Fractures

Temporal bone fractures may follow head trauma and may be of the transverse, longitudinal, or mixed variety. The *transverse* fracture is usually produced by a blow to the occiput. It runs translabyrinthine, traversing the internal auditory canal and cochlea, and ending in the foramen lacerum or spinosum. It may also run posterolaterally across the entire labyrinth and fallopian canal, ending in the middle ear. In transverse fractures, the external auditory canal is usually spared; sensorineural hearing loss, facial nerve paralysis, nystagmus, and hemotympanum are the usual findings. In the *longitudinal* variety, the fracture runs through the bone surrounding the carotid artery, passes through the middle ear, causing a rent in the tympanic membrane, and terminates as a fracture in the roof of the external auditory canal. Patients with a longitudinal fracture usually manifest conductive hearing loss due to ossicular and tympanic membrane injury and bloody (occasionally mixed with cerebrospinal fluid) otorrhea. Facial nerve paralysis will be present in approximately 25 per cent of cases. The *mixed type* of temporal bone fracture may include any combination of the above findings.

Treatment of temporal bone fractures is usually nonsurgical, with systemic antibiotics and careful cleansing of the external auditory canal when otorrhea is present. Facial nerve paralysis of immediate onset may dictate exploration (Gros, 1967).

Temporal bone fractures may result in aural meningoceles or in meningoencephaloceles. Surgical correction is performed through a mastoidectomy approach if the patient has not had previous mastoid surgery (Scott and Merrell, 1974). If a radical mastoidectomy has been performed previously, repair is done through a middle cranial fossa approach.

Foreign Bodies

Foreign bodies in the external auditory canal may be a challenge for the otolaryngologist, although he usually has an operating microscope and several microsurgical instruments, including ring curets, wire loops, and hooks of various sizes. Inanimate objects that are lateral to the isthmus of the canal are usually removed easily. Objects medial to the isthmus of the canal are more difficult to remove because they must be brought through the isthmus, where the canal wall skin is thin and quite sensitive. Often, hematomas and maceration are produced in the skin of the inner two thirds of the external auditory canal by minimal trauma. Care must be taken to avoid injuries to the tympanic membrane and the ossicles when removing larger objects medial to the isthmus. Several drops of oil are useful to kill insects prior to their removal. Compressed air may be used to flush the ear canal with Burow's solution or hydrogen peroxide when removing smaller objects from the ear canal. This procedure causes little discomfort to the patient. Irrigation should not be used to flush out vegetable seeds and beans, as these may expand in a position medial to the isthmus, making their extraction more difficult.

Older children and adults may be anesthetized locally prior to a difficult extraction. In young children or in cases of difficult extractions in adults, it is best to begin the attempted extraction under general anesthesia in the operating room. One or two drops of blood, some maceration of the canal wall skin, and some pain may make the procedure very difficult and uncomfortable for the patient.

Osteoma

Osteomas of the external auditory canal most frequently are associated with repeated cooling of the bone of the external auditory canal. This occurs from swimming repeatedly in cold water (DiBartolomeo, 1979). It is uncommon in warm climates unless the patient has lived previously in northern climates. If large enough, osteomas may prevent water from exiting from the sulcus lateral to the tympanic membrane after swimming or may prevent the egress of desquamated epithelium. Patients with this condition may have recurrent external otitis and a collection of epithelial debris medial to the osteoma. In these cases, the osteoma should be removed surgically.

The Auricle

Trauma

Sharp Trauma

A detailed review of the reconstructive techniques employed for repairing the injured auricle is beyond the scope of this chapter; however, several excellent reviews on this subject are available (Brent, 1977; Lacher and Blitzer, 1982; Larsen and Pless, 1976; McNichol, 1950; Potsic and Naunton, 1974; Spira and Hardy, 1963). The principles of managing the acutely injured auricle are reviewed briefly here.

As in other soft tissue trauma, injuries to the auricle must be carefully treated in the emergency room to obtain the best possible long-term cosmetic result. The blood supply in this area is plentiful, and debridement should be judicious and minimal. Every effort is made to preserve perichondrium, since cartilage devoid of perichondrium is subject to necrosis. Local flaps and skin grafts have been used to cover exposed cartilage and perichondrium. Incomplete avulsion injuries to the ear are treated conservatively to preserve tissue. Free composite grafts of auricular tissue will probably survive repair when not too heavily contaminated. Large pieces of auricular cartilage that are devoid of perichondrium may be buried under postauricular skin for later use in a reconstruction, using a pedicled flap. Complete avulsion of the auricle has been treated successfully by immediate reanastomosis. Iced saline has been used to decrease the metabolic needs of the composite graft, and dextran, heparin, and antibiotics have been used to prevent intravascular coagulation and infection in the flap (Potsic and Naunton, 1974).

Although keloids usually occur in blacks, they also may occur in whites. Commonly found on the lobule of the ear, they may be a result of ear piercing. Keloids usually respond to intralesional injection of triamcinolone at weekly intervals. To prevent recurrence, large keloids may require excision followed by triamcinolone injection and low-dose irradiation. Earrings worn on pierced ears may be pulled through the earlobe. This often occurs when women are holding children who pull on large, attractive earrings, and in patients who wear unusually heavy earrings. A variety of methods exist for repair of a defect resulting from earlobe keloid removal or a cleft earlobe due to pierced earrings (Hamilton and LaRossa, 1975).

Blunt Trauma

The most common complication of blunt trauma to the ear is the formation of hematomas. When these form below the perichondrium they must be evacuated to prevent infection, or "cauliflower ear" deformity (Ohlsen et al, 1975; Pandya, 1973). Aspiration and application of pressure may be successful but often have to be repeated. If a hematoma occurs more than once, incision and drainage is performed, followed by application of a pressure dressing. The "cauliflower ear" deformity is a thickening of cartilage in the location of an earlier hematoma. It is repaired by thinning the cartilage under a skin-perichondrial flap (Pandya, 1973).

Burns

Burns of the auricle are common and occur in about 90 per cent of patients presenting with facial burns at a large burn hospital. Of these patients, about 25 per cent develop suppurative chondritis; however, this occurrence is not necessarily related to the severity of the burned auricle (Dowling et al, 1968). Burns may be caused by fire, hot liquids, sun exposure, or electrical current. First-degree burns are treated conservatively with pain medications. Second- and third-degree burns may be treated with silver sulfadiazine or silver nitrate and Surgifix mesh dressing (Lawrence et al, 1972). Full-thickness auricular burns may lead to demarcation and autoamputation of the affected portion of the auricle. Third-degree burns may be extensively debrided and grafted or closed (Grant et al, 1969). However, more conservative management usually leads to less tissue loss and may carry no higher incidence of suppurative chondritis. In conservative management, granulation tissue is allowed to form over the full-thickness burned area and split-thickness grafts are used for cover (Dowling et al, 1968). Chemical burns are treated by flushing the surface with saline, half-strength vinegar (2 per cent acetic acid), or a mild alkali (1 teaspoonful of baking soda in a 12-oz glass of water), depending on whether the burn is alkaline or acidic.

Frostbite

Frostbite is a condition in which tissue is frozen. Temperatures below 10°C block sensory nerve input, depriving the patient of advanced warning of impending danger. Once the temperature falls below freezing, the frostbite injury begins. Initially, vasoconstriction produces a pallor. Ice formation occurs in extracellular fluid, resulting in a hypertonic state in the remaining fluid and intracellular dehydration. As the affected area thaws, subcutaneous edema due to extravasated fluid causes bullae to form. Later, erythema occurs around the demarcating tissue, which is distinguishable over a period of weeks or months (Holms and Vanggard, 1974). Treatment consists of rapidly rewarming the frostbitten area with moist cotton pledgets at a temperature of 38 to 42 degrees centigrade. Silver nitrate (0.5 per cent) soaks are applied to superficial infections only. Ordinarily, the auricle is kept sterile, but no dressings are used (Sessions et al, 1971). The use of sympathectomy, hyperbaric oxygen, and low molecular weight dextran remains controversial. Rubbing with snow or exposure to radiant heat is contraindicated.

Inflammation

Cellulitis and Perichondritis

Cellulitis of the auricle, which is usually secondary to otitis externa, is managed with mild, local heat and systemic antibiotics. It may also occur as the result of contact dermatitis. Perichondritis is an inflammation of perichondrium that complicates burns or trauma to the auricle. It is treated vigorously with surgical debridement and drainage to prevent a mutilating loss of cartilage structure.

Perichondritis usually occurs from 3 to 5 weeks following a burn and presents initially with severe pain. This is followed within several hours by redness, swelling, tenderness, and elevated temperature in the affected area. Mild cases may be treated by aggressive debridement, and severe cases by the bivalve technique. Diabetes may be a predisposing factor in patients with perichondritis of the auricle, especially when there is no history of burns.

Relapsing Polychondritis

Relapsing polychondritis is an episodic, inflammatory disease of the connective tissue and various cartilages in the head and neck and upper respiratory tract. The etiology is probably that of an autoimmune reaction. It presents most commonly as an acute inflammation of the cartilage of the ears, larynx, and nasal septum, but it may affect the trachea, causing respiratory obstruction. "Saddle nose" and "cauliflower ear" deformities are late signs commonly seen in the disease. Fever is the most common clinical symptom but is nonspecific. Auricular manifestations occur in about half of the cases. Steroids are used to control the acute attacks and suppress recurrence (Rabuzzi, 1970). A high index of suspicion is necessary for early diagnosis of this disease.

Varicella-Herpes Zoster

Varicella-herpes zoster virus has two primary clinical forms. The varicella form has an incubation period of 14 to 17 days followed by prodromal malaise and fever of 1 to 2 days in duration and subsequent maculopapular cutaneous eruption. Pneumonia, encephalitis, neuritis, and myelitis occur rarely. In the zoster form of the disease the incubation period is unknown. The cutaneous eruption is usually unilateral and confined to one or more dermatomes corresponding to the distribution of the extramedullary cranial nerve ganglia (cranial nerve VIII in Ramsay Hunt syndrome). The cutaneous lesion is painful but self-limiting, and local treatment is usually unnecessary. Facial nerve paralysis may occur.