Introduction

Swimming and other aqua activities are important human activities. Millions of people swim, dive, scuba dive, surf or boat, either professionally or recreationally, in different depths of water in swimming pools, ponds, rivers, lakes and oceans. When a person’s ears are exposed directly to water without protection, the external ear canal and tympanic membrane can be contaminated and pressure can easily be transmitted into the middle and inner ear and cause several problems, especially infections and trauma. The external, middle, and inner ear are all susceptible to these problems. This article discusses ear problems that are frequently encountered at otolaryngology clinics, including external, middle, and inner ear problems related to swimming and other aqua activities.

External Ear

**Acute diffuse otitis externa**

Otitis externa can be inflammatory, eczematoid, or seborrheic in nature. Inflammatory otitis externa can be subclassified into the following types: acute localized, acute diffuse, and chronic diffuse. Acute diffuse otitis externa (swimmer’s ear) and otomycosis are the most common problems in swimmers.

The external ear comprises the auricle and external ear canal. The average length of the adult external auditory canal is 2.5 cm. Because of the oblique

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position of the tympanic membrane, the posteroinferior part of the canal is about 6 mm shorter than the anteroinferior portion. The lateral 40% of the external auditory canal is cartilaginous and contains a thin layer of subcutaneous tissue between the skin and cartilage. The medial 60% is osseous, formed primarily by the tympanic ring, and contains very scant soft tissue among the skin, periosteum and bone. The junction of the cartilaginous and bony portions of the canal is a narrowed section termed the isthmus. The external ear canal is lined with keratinized, stratified squamous epithelium. There are hair follicles and sebaceous and apocrine glands that produce cerumen, which has an acid reaction and maintains the normal canal pH of 5. This acid environment inhibits bacterial and fungal growth. The lipid content of cerumen protects the surface of the squamous epithelium and pilosebaceous units and prevents maceration and breakdown of the epithelium. It thus provides both a chemical and mechanical protective barrier to infection. The normal external auditory canal has a flora that principally includes Staphylococcus albus, S. epidermidis, Corynebacterium spp. and small quantities of S. aureus and Streptococcus viridans.\(^1\,^2\)

Heat and humidity cause swelling of the stratum corneum in the skin. Introduction of extraneous moisture from swimming or bathing increases maceration of the canal skin, encourages destruction of the protective barrier, and creates a condition favorable to bacterial growth. These changes may also cause itching in the external auditory canal, thus adding the possibilities of scratching and subsequent infection.

It is well known that the predominant pathogen in swimmer’s ear is Pseudomonas aeruginosa. Other organisms involved are Proteus vulgaris, Escherichia coli, S. aureus, S. epidermidis, streptococci, diphtheroids, Enterobacter aerogenes, Klebsiella pneumoniae and Citrobacter spp.\(^3\)

The risk of otitis externa is reported to be approximately 5 times greater in swimmers than nonswimmers, and that of otalgia is reported to be 2.4 times greater in swimmers than nonswimmers.\(^3\)

Besides ear problems, swimmers and people who enjoy aquatic activities may develop gastrointestinal, respiratory, dermatologic, and ear, nose and throat infections.\(^4\,^6\) The duration and type of exposure, concentration of pathogens, and host immunity determine the risk of infection. Pathogens undetectable by conventional methods may remain viable in marine waters, and both plankton and marine sediments may serve as reservoirs for pathogenic organisms, which can emerge to become infective when conditions are favorable. Water quality is related to otitis externa. The more polluted the water, the easier otitis externa may occur.\(^5\,^7\,^9\) Ear problems can be transmitted via recreational contact with marine waters contaminated with sewage. Fecal coliform exposure may be predictive of ear ailments: an estimated threshold for infection was a bather exposure level of > 100 fecal coliforms per 100 mL of water.\(^10\)

The very early symptom of otitis externa is itching; aural discharge, otalgia and tenderness then develop. Accumulation of discharge and debris in the external ear canal may lead to aural fullness and conductive hearing loss.

Simple manipulation of the pinna or pushing the tragus may elicit severe pain in acute diffuse otitis externa. The external ear canal and tympanic membrane should be examined carefully with an otoscope or microscope under good illumination. The examination reveals a swollen and edematous canal. Initially, the canal is often filled with copious serous discharge, which then turns into a foul-smelling purulent discharge with debris causing occlusion. If any discharge is noted on examination, car swabs should be obtained for bacterial culture and sensitivity tests to guide antibiotic use. Lymphadenopathy can occur in postauricular, subauricular and, occasionally, periauricular parotid areas. Sometimes, inflammation may spread to the auricle and involve the entire auricle.

Acute diffuse otitis externa should be differentiated from other forms of otitis externa, such as herpes zoster oticus, or eczematoid, seborrheic or malignant forms. Patients with swimmer’s ear have a history of water exposure and sometimes may also have a history of trauma to the ear canal. Herpes zoster is herpes zoster infection with ear-canal and auricular involvement. Vesicles and serous discharge are noted in the ear canal and auricle. Eczematoid otitis externa includes various hypersensitivity reactions of the canal skin due to contact dermatitis or neurodermatitis. Seborrheic otitis externa is associated with seborrheic dermatitis of other regions, particularly the scalp. The lesions comprise yellowish, greasy scaling of the ear canal. Malignant (necrotizing) otitis externa, which is characterized by severe otalgia, is a type of temporal bone osteomyelitis. The most common pathogen is P. aeruginosa, and generally, only immunocompromised hosts, such as patients with diabetes mellitus, are affected. The treatment is aggressive parenteral antibiotics against P. aeruginosa and control of the underlying disease. Acute diffuse otitis externa must also be differentiated from carcinoma involving the external ear canal. It is often mistaken at the earliest stage for infection and treated inappropriately.
Ear problems in swimmers

The management of acute diffuse otitis externa includes frequent inspection and cleansing of the ear canal, control of pain, use of appropriate medications, either oral or topical, acidification of the ear canal, and control of predisposing factors. Cleansing the ear canal is perhaps the single most important aspect of treatment. Frequent inspection and drying of the canal are also important. Cleansing can be done by irrigation, gentle suction, and gentle application of cotton swabs, under direct visualization. Removal of discharge and debris can facilitate the application of topical medications. Drying of the ear canal can be done with 70% alcohol. Acute diffuse otitis externa caused by edema and inflammation can be very painful, and can be controlled by nonsteroidal anti-inflammatory drugs, or narcotics such as codeine or hydrocodone. The short-term use of corticosteroids can be useful for pain control if not contraindicated.

Together with cleansing, topical medication is usually effective as an initial treatment, and can be administered directly or by a wick. Acidifying agents can acidify the canal environment to inhibit bacterial and fungal growth. Topical corticosteroids can reduce inflammation, edema and itching, and antibiotic ointment can attack specific organisms. Neomycin is effective against Proteus and Staphylococcus spp., and polymyxin is effective against Pseudomonas spp. Chloramphenicol is effective against Bacteroides fragilis, an anerobe that is less common. Chemical agents such as aqueous gentian violet 2% and silver nitrate 10% are bactericidal and may be applied directly to canal skin. Systemic antibiotics should be used in patients with lymphadenopathy, in patients taking systemic corticosteroids, and in immunocompromised patients, such as those with diabetes. The initial, oral antibiotic therapy may be an empiric selection against common offending pathogens; subsequently, a change to appropriate therapy is required based on culture and sensitivity results. Patients in whom the condition is severe may require parenteral antibiotic therapy against P. aeruginosa.

For the prevention of acute diffuse otitis externa, swimming avoidance is effective, but is impractical for swimmers or participants in aqua activities. Various protective devices, including commercial rubber or silicon ear plugs, cotton wool coated with paraffin jelly or Vaseline, and swimming caps, are advised and may be useful. Swimming in clean water, such as in a chlorinated swimming pool, or at non-polluted beaches, rivers or lakes, will decrease the risk of infection. Keeping the ear canal dry may also be helpful, since the incidence of otitis externa can be minimized by eliminating moisture in the canal. The ear canal can be dried using a hair dryer after each period of swimming. Ear-canal cleaning with cotton-tip applicators should be avoided as it will traumatize the canal skin and compromise the mechanical barrier of the canal, thus increasing the possibility of otitis externa.

**Otomyosis**

Fungal infection is also a common external ear problem in swimmers that can be facilitated by heat and moisture. Physical examination may show swelling and hyperemia of the canal skin, and fungal hyphae or characteristic cheese-like grayish debris in the canal. The treatment is similar to that for acute diffuse otitis externa, including frequent cleansing, preventing moisture, and drying the canal. Topical application of antifungal drugs is also helpful, but recurrence is common.

**Exostoses**

The most common benign bony tumors of the external ear canal are exostoses and osteomas. Prolonged exposure to cold water in activities such as swimming, surfing or diving not only increases the risk of developing exostoses but also increases the severity of the condition. The symptoms of exostoses include debris accumulation in the ear canal, otorrhea secondary to otitis externa, and conductive hearing loss. The bony tumor may block the ear canal and obstruct cleansing of the ear canal medial to the tumor. The accumulation of debris medial to the tumor increases the risk of infection leading to ototrahea. The tumor itself and the accumulated debris also cause conductive hearing loss. Exostoses should be differentiated from osteoma. Exostoses are usually bilateral, broad-based lesions that arise from the medial aspect of the bony ear canal near the tympanic annulus, along the tympanomastoid and tympanosquamous suture lines. In contrast, osteomas are typically solitary and unilateral. These pedunculated bony tumors are less common than exostoses and are found in the outer half of the ear canal. The treatment for exostoses is transmeatal surgical removal.

**Middle Ear**

**Traumatic eardrum perforation**

Swimming and water sports, especially water skiing and scuba diving, may lead to traumatic eardrum perforation, which is one of the principal types of non-explosive blast injury to the ear. Individuals with previous recurrent otitis media, atrophic scarring of the tympanic membrane, and poor Eustachian tube
function, are predisposed to traumatic eardrum perforation. In scuba diving, tympanic membrane rupture may occur from 4–7 feet if there is no equalization of pressure via the Eustachian tube. The common symptoms are hearing loss, otalgia, otorrhea, tinnitus and vertigo. The hearing loss is usually conductive, but sometimes sensorineural. The treatments include topical or oral antibiotics and analgescics. The perforations are, in most cases, small and will heal spontaneously in 1 month. If not, they can be repaired by tympanoplasty.

**Swimming, chronic otitis media and mastoidectomy**

Swimming is not advised for patients with chronic otitis media with active drainage, but is allowed for patients with eardrum perforation without discharge if ear protection (e.g. earplugs) is used. For patients who have undergone mastoidectomy or tympanoplasty with mastoidectomy, swimming is allowed if there is no discharge or cavity problem. The cavity should be lined with healthy epithelium, and ear protection is needed when swimming. If the patient has a discharging cavity or granulation tissue in the cavity, swimming should be restricted. For patients with a large open cavity, swimming may cause vertigo because of caloric effects. However, patients who have undergone surgical obliteration of the tympanomastoid compartment and external auditory canal may participate in swimming, diving, and all other aquatic sports.

**Children with ventilation tubes**

Can children with ventilation tubes be allowed to swim without protection? This common question asked by parents in ear, nose and throat clinics is controversial and has been debated for decades. Some clinicians consider it safe for such children to swim without protection, whereas others consider it necessary for the children to swim with earplugs, and others stipulate that swimming should be strictly forbidden. Other contentious issues include the use of antibiotic ear drops after swimming, the feasibility of diving, and the possibility of differences between locations, such as chlorinated swimming pools, rivers, lakes, and beaches. Derkay et al conducted a questionnaire survey of 1,266 otolaryngologists in the southern and eastern USA. Of all respondents, 14.1% prohibited swimming, 3.1% had no water precautions, and 68% limited their patients’ swimming. The most frequently recommended protection was earplugs. Interestingly, 94% of respondents said they would be willing to alter their current practice based on new information generated from a clinical trial; this suggests that most otolaryngologists were not confident with their current practice.

Pashley and Scholl showed, *in vitro*, that it took 11.45–22.57 cmH₂O to force water through a tympanostomy tube. While length and position of the tube did not have any effect, soapy water or liquids with decreased surface tension entered the tube more readily. The investigators described 3 necessary parameters for water to enter the middle ear: Eustachian tube opening, fixation of the tympanic membrane, and increased external canal pressure. Hebert et al designed a model of the human ear with pinna, external ear canal, tympanic membrane with ventilation tube, middle ear cleft, and mastoid cavity, and subjected the model to various conditions. Showering, hair rinsing and head submersion in clean tap water did not promote water entry into the middle ear. However, head submersion in soapy water and swimming deeper than 60 cm produced a significant number of positive test results (water entry into the middle ear). Morgan used fluorescent powder to show the extent of water penetration into the external ear canal and tympanic membrane. He showed that water on the tympanic membrane was found in 13.5% of cases of hair washing, and in 52% cases of head submersion in water for 4 minutes.

Several prospective studies and meta-analyses showed no difference in the incidence of otorrhea among swimmers and nonswimmers in a group of children with ventilation tubes. All of these studies and analyses concluded that swimming should not be prohibited in children with ventilation tubes.

Generally, diving is not advised for children with ventilation tubes. Lounsbury studied the effects of unprotected swimming in patients with ventilation tubes. Divers had a significantly increased rate of infection versus nondivers (1 infection per 100 days of swimming vs 1 infection per 600 days of swimming).

Whether or not earplugs are used for swimming seems to make no difference to the incidence of ear infection in children with ventilation tubes. Becker et al reported an infection rate of 16% in individuals swimming without earplugs, compared with 30% in individuals swimming with earplugs. Salata and Derkay also showed no difference in the rate of otorrhea in children with tympanostomy tubes swimming with or without earplugs.

Smelt and Monkhouse irrigated the middle ears of guinea pigs with normal saline, bath water, sea water, and swimming pool water. They then sacrificed the animals and assessed histologic changes in the middle ear mucosae. Reactive changes with swimming pool water and seawater were no greater than with
normal saline; however, a greater degree of inflammation was noted in ears irrigated with bath water, possibly because of easier contamination with enteral bacteria. 39

In our recent, unpublished, study, we included 9 children with ventilation tubes in a total of 15 ears. After surface swimming for 1 hour without any ear protection in a clean chlorinated swimming pool, the children had their ears checked by videotelescope at the poolside. Eight ears were dry, 4 had water in the outer third of the external ear canal, and 3 had water on the tube or tympanic membrane, but none had water penetration into the middle ear. No otorrhea was found, even after 2 weeks. Thus, water penetration through ventilation tubes into the middle ear is unlikely to occur with surface swimming, so children with ventilation tubes can enjoy swimming without protection in clean chlorinated swimming pools.

Inner Ear

Inner ear injury on diving
Sudden sensorineural hearing loss and some degree of vertigo may occur after diving because of rupture of the round or oval window. 40 Rupture of the round window may occur after diving, even if the dive is performed from a low height and no contact is made with the bottom of the pool. Besides direct contusion to the external ear and barotrauma, other causes such as whiplash have to be considered. The treatments are diagnostic tympanotomy with sealing of the round and oval window membranes, and vasoactive rheologic therapy combined with corticosteroid treatment.

Conclusion
The most common ear problem related to aqua activities is acute diffuse otitis externa (swimmer’s ear), in which the most common pathogen is P. aeruginosa. Acute diffuse otitis externa is more frequent after swimming in polluted water and when the chemical and mechanical protective barrier in the ear canal is breached. The symptoms are itching, otalgia, otorrhea and conductive hearing loss. The treatment includes frequent inspection and cleansing of the ear canal, pain control, use of appropriate medications, either oral or topical, acidification of the ear canal, and control of predisposing factors. Ear protection with earplugs may be helpful. Swimming in polluted water and ear-canal cleaning with cotton-tip applicators should be avoided. Exostoses and osteomas are most commonly seen in benign tumors of the ear canal, and are usually noted in people swimming in cold water. The symptoms are accumulated debris, otorrhea, and conductive hearing loss. The treatment for exostoses is transmeatal surgical removal. Traumatic eardrum perforations may occur during water skiing or scuba diving. Previous recurrent otitis media, atrophic scarring of the tympanic membrane, and poor Eustachian tube function may be predisposing factors. The common symptoms are hearing loss, otalgia, otorrhea, tinnitus and vertigo. Tympanoplasty may be needed if the perforations do not heal spontaneously. Swimming is not advised for patients with chronic otitis media with active drainage, but is allowed for patients without discharge or cavity problems after mastoidectomy with or without tympanoplasty.

In children with ventilation tubes, it is not easy for water to penetrate into the middle ear during surface swimming, so earplugs are not required; diving is not recommended. If middle ear infection or otorrhea occurs, it is not difficult to manage. It is safe for children with ventilation tubes to enjoy surface swimming in clean chlorinated swimming pools. Sudden sensorineural hearing loss and some degree of vertigo may occur after diving because of labyrinthine fistula due to rupture of the round or oval window membrane.

References


