Clinical practice guideline: Acute otitis externa

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OBJECTIVE: This guideline provides evidence-based recommendations to manage diffuse acute otitis externa (AOE), defined as generalized inflammation of the external ear canal, which may also involve the pinna or tympanic membrane. The primary purpose is to promote appropriate use of oral and topical antimicrobials and to highlight the need for adequate pain relief.

STUDY DESIGN: In creating this guideline, the American Academy of Otolaryngology–Head and Neck Surgery Foundation (AAO-HNSF) selected a development group representing the fields of otolaryngology–head and neck surgery, pediatrics, family medicine, infectious disease, internal medicine, emergency medicine, and medical informatics. The guideline was created with the use of an explicit, a priori, evidence-based protocol.

RESULTS: The group made a strong recommendation that management of AOE should include an assessment of pain, and the clinician should recommend analgesic treatment based on the severity of pain. The group made recommendations that clinicians should: 1) distinguish diffuse AOE from other causes of otalgia, otorrhea, and inflammation of the ear canal; 2) assess the patient with diffuse AOE for factors that modify management (nonintact tympanic membrane, tympanostomy tube, diabetes, immunocompromised state, prior radiotherapy); and 3) use topical preparations for initial therapy of diffuse, uncomplicated AOE; systemic antimicrobial therapy should not be used unless there is extension outside of the ear canal or the presence of specific host factors that would indicate a need for systemic therapy.

The group made additional recommendations that: 4) the choice of topical antimicrobial therapy of diffuse AOE should be based on efficacy, low incidence of adverse events, likelihood of adherence to therapy, and cost; 5) clinicians should inform patients how to administer topical drops, and when the ear canal is obstructed,

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delivery of topical preparations should be enhanced by aural toilet, placing a wick, or both; 6) when the patient has a tympanostomy tube or known perforation of the tympanic membrane, the clinician should prescribe a nonototoxic topical preparation; and 7) if the patient fails to respond to the initial therapeutic option within 48 to 72 hours, the clinician should reassess the patient to confirm the diagnosis of diffuse AOE and to exclude other causes of illness. And finally, the panel compiled a list of research needs based on limitations of the evidence reviewed.

CONCLUSION: This clinical practice guideline is not intended as a sole source of guidance in evaluating patients with AOE. Rather, it is designed to assist clinicians by providing an evidence-based framework for decision-making strategies. It is not intended to replace clinical judgment or establish a protocol for all individuals with this condition and may not provide the only appropriate approach to the diagnosis and management of this problem.

SIGNIFICANCE: This is the first, explicit, evidence-based clinical practice guideline on acute otitis externa, and the first clinical practice guideline produced independently by the AAO–HNSF. © 2006 American Academy of Otolaryngology–Head and Neck Surgery Foundation, Inc. All rights reserved.

The primary purpose of this guideline is to promote appropriate use of oral and topical antimicrobials for diffuse acute otitis externa (AOE) and to highlight the need for adequate pain relief. The target patient is aged 2 years or older with diffuse AOE, defined as generalized inflammation of the external ear canal, with or without involvement of the pinna or tympanic membrane. As the first clinical practice guideline developed independently by the AAO-HNSF, a secondary purpose was to refine methods for future efforts. Additional goals were to make possible an AOE performance measure and to make clinicians aware of modifying factors that can or may alter management (eg, diabetes, immunocompromised state, prior radiotherapy, tympanostomy tube, nonintact tympanic membrane).

This guideline does not apply to children under age 2 years or to patients of any age with chronic or malignant (progressive necrotizing) otitis externa. AOE is uncommon before age 2 years, and very limited evidence exists with respect to treatment or outcomes in this age group. Although the differential diagnosis of the “draining ear” will be discussed, recommendations for management will be limited to diffuse AOE, which is almost exclusively a bacterial infection. The following conditions will be briefly discussed but not considered in detail: furunculosis (localized AOE), otomycosis, herpes zoster oticus (Ramsay Hunt syndrome), and contact dermatitis.

The guideline is intended for primary care and specialist clinicians, including otolaryngologists–head and neck surgeons, pediatricians, family physicians, emergency physicians, internists, nurse-practitioners, and physician assistants. The guideline is applicable to any setting in which children, adolescents, or adults with diffuse AOE would be identified, monitored, or managed.

INTRODUCTION

Acute otitis externa (AOE) as discussed in this guideline is defined as diffuse inflammation of the external ear canal, which may also involve the pinna or tympanic membrane. A diagnosis of diffuse AOE requires rapid onset (generally within 48 hours) in the past 3 weeks of symptoms and signs of ear canal inflammation as detailed in Table 1. A hallmark sign of diffuse AOE is tenderness of the tragus, pinna, or both, that is often intense and disproportionate to what might be expected based on visual inspection.

Also known as “swimmer’s ear” or “tropical ear,” AOE is one of the most common infections encountered by clinicians. The annual incidence of AOE is between 1:100 and 1:250 of the general population, with regional variations based on age and geography; lifetime incidence is up to 10%. The direct cost of AOE is unknown, but the ototopical market in the United States is approximately 7.5 million annual prescriptions with total sales of $310 million (IMS/Verispan 2004, personal communication). Additional medical costs include physician visits and prescriptions for analgesics and systemic medications, such as antibiotics, steroids, or both. The indirect costs of AOE have not been calculated but are likely to be substantial because of severe and persistent otalgia that limits activities.

AOE is a cellulitis of the ear canal skin and subdermis, with acute inflammation and variable edema. Nearly all (98%) AOE in North America is bacterial. The most common pathogens are Pseudomonas aeruginosa (20% to 60% prevalence) and Staphylococcus aureus (10% to 70% prevalence), often occurring as a polymicrobial infection. Other pathogens are principally gram negative organisms (other than P aeruginosa), which cause no more than 2% to 3% of cases in large clinical series. Fungal involvement is distinctly uncommon in primary AOE but may be more

Table 1

<table>
<thead>
<tr>
<th>Elements of the diagnosis of diffuse acute otitis externa</th>
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<td>1. Rapid onset (generally within 48 hours) in the past 3 weeks, AND</td>
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<td>2. Symptoms of ear canal inflammation that include:</td>
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<td>● otalgia (often severe), itching, or fullness,</td>
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<tr>
<td>● WITH OR WITHOUT hearing loss or jaw pain,* AND</td>
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<tr>
<td>3. Signs of ear canal inflammation that include:</td>
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<td>● tenderness of the tragus, pinna, or both</td>
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<td>● OR diffuse ear canal edema, erythema, or both</td>
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<tr>
<td>● WITH OR WITHOUT otorrhea, regional lymphadenitis, tympanic membrane erythema, or cellulitis of the pinna and adjacent skin</td>
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*Pain in the ear canal and temporomandibular joint region intensified by jaw motion.
common in chronic otitis externa or after treatment of AOE with topical, or less often systemic, antibiotics.13

Topical antimicrobials are beneficial for AOE, but oral antibiotics have limited utility.14 Nonetheless, about 20% to 40% of patients with AOE receive oral antibiotics, often in addition to topical therapy.2,15,16 The oral antibiotics selected are usually inactive against P aeruginosa and S aureus, may have undesirable side effects, and, because they are widely distributed, serve to select out resistant organisms throughout the body.17,18 Bacterial resistance is of far less concern with topical antimicrobials, because the high local concentration of drug in the ear canal will generally eradicate all susceptible organisms plus those with marginal resistance.4

The cause of AOE is multifactorial. Regular cleaning of the ear canal removes cerumen, which is an important barrier to moisture and infection.19 Cerumen creates a slightly acidic pH that inhibits infection (especially by P aeruginosa) but can be altered by water exposure, aggressive cleaning, soapy deposits, or alkaline eardrops.20,21 Debris from dermatologic conditions may also encourage infections,6,22 as can local trauma from attempts at self-cleaning, irrigation,23 and wearing hearing aids.24,25 Other factors such as sweating, allergy, and stress have also been implicated in the pathogenesis of AOE.26

AOE is more common in regions with warmer climates, increased humidity, or increased water exposure from swimming.27,28 Most, but not all, studies have found an association with water quality (in terms of bacterial load) and the risk of AOE. The causative organisms are present in most swimming pools and hot tubs; however, even those that comply with water quality standards may still contain AOE pathogens.29-32 Some individuals appear more susceptible to AOE on a genetic basis (those with type A blood group) and the subspecies of Pseudomonas causing AOE may be different from those causing other Pseudomonas infections.33,34

Strategies to prevent AOE are aimed at limiting water accumulation and moisture retention in the external auditory canal, and maintaining a healthy skin barrier. No randomized trials have compared the efficacy of different strategies to prevent AOE. Available reports include case series and expert opinion that emphasize the prevention of moisture and water retention in the external auditory canal. Recommendations to prevent AOE include removing obstructing cerumen; the use of acidifying ear drops shortly before swimming, after swimming, at bedtime, or all 3 times; the use of a hair dryer to dry the ear canal; the use of ear plugs while swimming; and the avoidance of trauma to the external auditory canal.35-38

Variations in managing AOE and the importance of accurate diagnosis suggest a need for an evidence-based practice guideline. Failure to distinguish AOE from other causes of “the draining ear” (eg, chronic external otitis, malignant otitis externa, middle ear disease) may prolong morbidity or cause serious complications. Because topical therapy is efficacious, systemic antibiotics are often prescribed inappropriately.14,39 When topical therapy is prescribed confusion exists about whether to use an antiseptic, antibiotic, corticosteroid, or a combination product. Selecting an antibiotic creates additional controversy, particularly with respect to the role of newer quinolone drops. And finally, the optimal methods for cleaning the ear canal (aural toilet) and drug delivery are undefined.

The primary outcome considered in this guideline is clinical resolution of AOE. Additional outcomes considered include minimizing the use of ineffective treatments; eradicating pathogens; minimizing recurrence, cost, complications, and adverse events; maximizing the health-related quality of life of individuals afflicted with AOE; increasing patient satisfaction40; and permitting the continued use of necessary hearing aids. The relatively high incidence of AOE and the diversity of interventions in practice (Table 2) make AOE an important condition for the use of an up-to-date, evidence-based practice guideline.

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**Table 2**

<table>
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<th>Interventions considered in AOE guideline development</th>
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<tr>
<td><strong>Diagnosis</strong></td>
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<tr>
<td>History and physical examination</td>
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<tr>
<td>Otoscopy</td>
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<tr>
<td>Pneumatic otoscopy</td>
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<td>Otomicroscopy</td>
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<tr>
<td>Tympanometry</td>
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<td>Acoustic reflectometry</td>
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<tr>
<td>Culture</td>
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<tr>
<td>Imaging studies</td>
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<tr>
<td>Audiometry (excluded from guideline)</td>
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<tr>
<td><strong>Treatment</strong></td>
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<tr>
<td>Aural toilet (suction, dry mopping, irrigation, removal of obstructing cerumen or foreign object)</td>
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<tr>
<td>Nonantibiotic (antiseptic or acidifying) drops</td>
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<tr>
<td>Antibiotic drops</td>
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<tr>
<td>Steroid drops</td>
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<td>Oral antibiotics</td>
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<td>Analgesics</td>
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<tr>
<td>Complementary and alternative medicine</td>
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<tr>
<td>Ear canal wick</td>
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<td>Biopsy (excluded from guideline)</td>
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<td>Surgery (excluded from guideline)</td>
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<td>Water precautions</td>
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<td>Prophylactic drops</td>
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<tr>
<td>Environmental control (eg, hot tubs)</td>
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<tr>
<td>Avoiding neomycin drops (if allergic)</td>
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<tr>
<td>Addressing allergy to ear molds or water protector</td>
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<tr>
<td>Addressing underlying dermatitis</td>
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<tr>
<td>Specific preventive measures for diabetics or immunocompromised state</td>
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METHODS

General Methods and Literature Search
To develop an evidence-based clinical practice guideline on managing AOE, the American Academy of Otolaryngology–Head and Neck Surgery Foundation (AAO-HNSF) assembled a working group who represented the disciplines of otolaryngology–head and neck surgery, pediatrics, infectious disease, internal medicine, family medicine, emergency medicine, biostatistics, and medical informatics. Several group members had significant prior experience in the development of clinical practice guidelines.

A MEDLINE search from 1966 through July 2005 was performed on PubMed with the terms “otitis externa” (MeSH term) and “swimmer’s ear.” Titles and abstracts unrelated to AOE were excluded, leaving 240 articles that were collated under these headings: risk factors (n = 30), microbiology (n = 24), pharmacologic intervention (n = 118), other interventions (n = 17), epidemiology and practice patterns (n = 14), potential harms (n = 30), and otomycosis (n = 9). Citations and abstracts were distributed to all group members to assist in formulating and prioritizing evidence-based statements. Members performed additional targeted MEDLINE searches through September 2005 to supplement the initial broad search.

A meta-analysis was performed with an a priori protocol to compare the following topical treatments: antimicrobial vs placebo, antiseptic vs antimicrobial, quinolone antibiotic vs nonquinolone, steroid-antimicrobial vs antimicrobial, or antimicrobial-steroid vs steroid. Search of MEDLINE from 1966 through July 2005, without language restrictions, identified 2860 articles, of which 509 were potential randomized trials. Review of these studies, plus 7 others found in the Cochrane Database, yielded 43 articles that were assessed by 2 reviewers independently for relevance, study quality, and data extraction. The final data set included 20 articles that had random allocation, were limited to diffuse AOE (or had subgroup data), gave results by subjects (or, if results by ears, 90% or higher must be unilateral disease), and had 2 or more parallel groups related to the above comparisons.

In a series of conference calls, the working group defined the scope and objectives of the proposed guideline. During the 7 months devoted to guideline development ending in November 2005, the group met twice with interval electronic review and feedback on each guideline draft to ensure accuracy of content and consistency with standardized criteria for reporting clinical practice guidelines. At the first meeting, all members disclosed relationships with pharmaceutical manufacturers and discussed what impact, if any, potential conflicts of interest might have on guideline validity. The group concluded that none of the relationships precluded involvement.

In September and November 2005, the Guidelines Review Group of the Yale Center for Medical Informatics used GEM-COGS, the Guideline Implementability Appraisal (GLIA) and Extractor software, to appraise adherence of the draft guideline to methodologic standards, to improve clarity of recommendations, and to predict potential obstacles to implementation. AOE guideline development group members received summary appraisals before their second meeting and modified an advanced draft of the guideline.

The final draft practice guideline underwent extensive external peer review. Comments were compiled and reviewed by the group chairperson. The recommendations contained in the practice guideline are based on the best available published data through September 2005. Where data are lacking, a combination of clinical experience and expert consensus was used. A scheduled review process will occur at 5 years from publication or sooner if new compelling evidence warrants earlier consideration.

Classification of Evidence-Based Statements
Guidelines are intended to reduce inappropriate variations in clinical care, to produce optimal health outcomes for patients, and to minimize harm. The evidence-based approach to guideline development requires that the evidence that supports a policy be identified, appraised, and summarized and that an explicit link between evidence and statements be defined. Evidence-based statements reflect both the quality of evidence and the balance of benefit and harm that is anticipated when the statement is followed. The definitions for evidence-based statements are listed in Tables 3 and 4.

Guidelines are never intended to supersede professional judgment; rather, they may be viewed as a relative constraint on individual clinician discretion in a particular clinical circumstance. Less frequent variation in practice is expected for a strong recommendation than might be expected with a recommendation. Options offer the most opportunity for practice variability. Clinicians should always act and decide in a way that they believe will best serve their patients’ interests and needs, regardless of guideline recommendations. Guidelines represent the best judgment of a team of experienced clinicians and methodologists addressing the scientific evidence for a particular topic.

Making recommendations about health practices involves value judgments on the desirability of various outcomes associated with management options. Values applied by the AOE guideline development group sought to minimize harm, diminish unnecessary and inappropriate therapy, and reduce the unnecessary use of systemic antibiotics. A major goal of the working group was to be transparent and explicit about how values were applied and to document the process.

AOE GUIDELINE EVIDENCE-BASED STATEMENTS

Each evidence-based statement is organized in a similar fashion: evidence-based statement in bold face type, fol-
1a. DIFFERENTIAL DIAGNOSIS: Clinicians should distinguish diffuse AOE from other causes of otalgia, otorrhea, and inflammation of the external ear canal.

Recommendation based on observational studies with a preponderance of benefit over risk.

1b. MODIFYING FACTORS: Clinicians should assess the patient with diffuse AOE for factors that modify management (nonintact tympanic membrane, tympanostomy tube, diabetes, immunocompromised state, prior radiotherapy). Recommendation based on observational studies with a preponderance of benefit over risk.

Differential Diagnosis. A diagnosis of diffuse AOE requires rapid onset with signs and symptoms of ear canal inflammation (Table 1). Symptoms of AOE include otalgia (70%), itching (60%), or fullness (22%), with or without hearing loss (32%) or ear canal pain on chewing. A hallmark sign of diffuse AOE is tenderness of the tragus (when pushed), pinna (when pulled up and back), or both. The tenderness is
often intense and disproportionate to what might be expected based on visual inspection. Otoscopy will reveal diffuse ear canal edema, erythema, or both, either with or without otorrhea or material in the ear canal. Regional lymphadenitis or cellulitis of the pinna and adjacent skin may be present in some patients.6,51

AOE can mimic the appearance of acute otitis media (AOM) because of erythema that involve the tympanic membrane. Distinguishing AOE from AOM is important because the latter may require systemic antimicrobials.52 Pneumatic otoscopy will demonstrate good tympanic membrane mobility with AOE but will show absent or limited mobility with AOM and associated middle-ear effusion. Similarly, tympanometry will show a normal peaked curve (type A) with AOE, but a flat tracing (type B) with AOM. The validity of acoustic reflectometry with AOE is unknown.

Anything that disrupts the epithelium of the ear canal can permit invasion by bacteria that cause diffuse AOE. Common predisposing factors for AOE26 are humidity or prolonged exposure to water, dermatologic conditions (eczema, seborrhea, psoriasis), anatomic abnormalities (narrow canal, exostoses), trauma or external devices (wax removal, insertion of earplugs, use of hearing aids), and otorrhea caused by middle-ear disease. AOE may also occur as a result of ear canal obstruction by impacted cerumen, a foreign object, or a dermoid or sebaceous cyst. Clinical history should identify predisposing factors and assess swimming behavior. Other causes of otalgia, otorrhea, and inflammation should be distinguished from diffuse AOE because management will differ.

Furunculosis is the presence of an infected hair follicle on the outer third of the ear canal, sometimes referred to as localized otitis externa. Clinical findings include otalgia, otorrhea, and localized tenderness. Treatment may include local heat, incision and drainage, or systemic antibiotics that cover \( S\) aureus, the most common causative agent.

Eczema, seborrhea, and other inflammatory dermatoses that involve the ear canal and surrounding tissues are relatively common and may predispose to acute infection. In contrast, contact dermatitis of the ear canal is an allergic reaction to antigens such as metals (nickel, silver), chemicals (cosmetics, soaps, detergents, shampoo, hairspray), plastics, rubber, leather, or drugs. Nickel is the most common contact allergen, affecting around 10% of women with pierced ears.53-55 Contact allergy also occurs in some patients who wear hearing aids as a reaction to the plastics and other chemicals used in hearing aid molds.56,57

Sensitization to topical treatment (secondary contact otitis) can result from prolonged or repeated use of topical antimicrobials. Many otic preparations (antibiotics and vehicle substances) have been reported to cause sensitization. Neomycin is the most common substance and causes reactions in about 5% to 15% of patients with chronic external otitis.58 Patch testing has demonstrated that 13% of normal volunteers are hypersensitive to neomycin.59 A maculopapular eruption on the conchal bowl and in the ear canal is consistent with an allergic reaction to a topical agent; an erythematous streak may extend down the pinna where drops contact the auricular skin. Management involves removal of the sensitizing agent and application of a topical steroid.

Viral infections of the external ear, caused by varicella, measles, or herpesvirus, are rare. Herpes zoster oticus (Ramsay Hunt syndrome) causes vesicles on the external ear canal and posterior surface of the auricle, severe otalgia, facial paralysis or paresis, loss of taste on the anterior two-thirds of the tongue, and decreased lacrimation on the involved side.60 Management involves antiviral therapy, with or without systemic steroid.

Modifying Factors. Key components of the clinical history that can modify management of diffuse AOE include 1) diabetes, HIV infection, or other immunocompromised states, 2) a history of radiotherapy, and 3) the presence of tympanostomy tubes or nonintact tympanic membrane.

Malignant (progressive necrotizing) otitis externa is an aggressive infection that predominantly affects elderly, diabetic, or immunocompromised patients.61 \( P\) aeruginosa is isolated from exudate in the ear canal in more than 90% of cases. Initial signs and symptoms are those of the initiating AOE, but untreated disease develops into a skull base osteomyelitis that can invade soft tissue, the middle ear, inner ear, or brain. Facial nerve paralysis may be an early sign, with the glossopharyngeal and spinal accessory nerves less frequently involved. Granulation tissue is classically seen on the floor of the canal and at the bony-cartilaginous junction. Clinical diagnosis can be confirmed with a raised erythrocyte sedimentation rate plus an abnormal computed
tomography or magnetic resonance imaging scan\(^{61,62}\); other imaging modalities include gallium scan, indium-labeled leukocyte scan, technetium bone scan, and single-photon emission tomographs (SPECT). Treatment includes systemic antibiotics adequate to cover pseudomonal and staphylococcal infection, including methicillin-resistant \(S\) \(aureus\). Biopsy may be necessary to detect neoplasia if the diagnosis of malignant otitis externa is uncertain or response to therapy is incomplete.

Otomycosis, or fungal infection of the external ear canal, is common in tropical countries, humid locations, after long-term topical antibiotic therapy, and in those with diabetes, HIV infection, or an immunocompromised state. \(Aspergillus\) species (60% to 90%) and \(Candida\) species (10% to 40%) are often cultured.\(^{63}\) Symptoms include pruritus and thickened otorrhea, which may be black, gray, bluish green, yellow, or white. Candidal otitis externa results in white debris sprouting hyphae, best seen with an otologic microscope. \(Aspergillus\) niger appears as a moist white plug dotted with black debris (“wet newspaper”).\(^{64}\) Management may include debridement plus topical antifungal therapy, systemic antifungal therapy, or both.

Radiotherapy can damage the external ear by causing acute and late skin reactions that involve the pinna, external canal, and periauricular region.\(^{65}\) Acute events include erythema, desquamation, or ulceration of the auricle and ear canal, thus leading to pain and otorrhea. Late skin changes include atrophy, necrosis or ulceration, external otitis, and external canal stenosis. Damage to the epithelium of sebaceous and apocrine glands can diminish cerumen secretion. Management of AOE in patients after radiotherapy may require systemic antimicrobials.

Middle-ear disease can modify treatment of AOE. Patients with a tympanostomy tube or tympanic membrane perforation may develop diffuse AOE because of purulent middle-ear secretions that enter the ear canal. This condition has been called infectious eczematoid dermatitis because the skin changes resemble eczema as well as infection.\(^{51}\) As discussed later in the guideline, clinicians should prescribe a nonototoxic topical preparation when the tympanic membrane is not intact. Management of the underlying middle-ear disease may also require systemic antimicrobials, imaging studies, or surgery.

Evidence Profile for 1a: Differential Diagnosis.

- Aggregate evidence quality: C, observational studies and D, expert opinion
- Benefit: improved diagnostic accuracy
- Harm: none
- Cost: none
- Benefits-harm assessment: preponderance of benefits over harm
- Value judgments: importance of accurate diagnosis

\(Evidence\) Profile for 1b: Modifying Factors.

- Aggregate evidence quality: C, observational studies
- Benefit: optimizing treatment of AOE through appropriate diagnosis and recognition of modifying factors
- Harm: none
- Cost: additional expense of diagnostic tests or imaging studies to identify modifying factors
- Benefits-harm assessment: preponderance of benefits over harm
- Value judgments: avoiding complications in at-risk patients
- Role of patient preferences: none
- Policy level: recommendation

2. PAIN MANAGEMENT: The management of diffuse AOE should include an assessment of pain. The clinician should recommend analgesic treatment based on the severity of pain. Strong recommendation based on well-designed randomized trials with a preponderance of benefit over harm.

Pain relief is a major goal in the management of AOE. Frequent use of analgesics is often necessary to permit patients to achieve comfort, rest, and to resume normal activities.\(^{66-68}\) Ongoing assessment of the severity of discomfort is essential for proper management. Use of a faces,\(^{69}\) Oucher,\(^{70}\) or visual analog\(^{71}\) scale may help determine the level of pain, particularly for children and non-English speaking patients.

Adequate pain control requires knowing the dose, timing, routes of delivery, and possible adverse effects of an analgesic.\(^{66-68,72}\) Mild to moderate pain usually responds to acetaminophen or nonsteroidal anti-inflammatory drugs given alone or in fixed combination with an opioid (eg, acetaminophen with codeine, oxycodone, or hydrocodone; ibuprofen with oxycodone). Administering a nonsteroidal anti-inflammatory drug during the acute phase of diffuse AOE significantly reduces pain compared with placebo.\(^{73}\)

Convenience, ease of use, and cost make orally administered analgesics the preferred route of administration whenever possible. Rarely, parenteral analgesia may be necessary to achieve adequate pain relief in a timely fashion. In all cases, analgesic therapy should be guided by the recognition that pain is easier to prevent than treat. Thus, early treatment at an appropriate starting dose is always indicated. When frequent doses are required to maintain adequate pain relief, the administration of analgesics at fixed intervals rather than on a pro re nata (prn) basis may be more effective. Nonpharmacologic therapies such as heat or cold, relaxation, and distraction are of unproven value but have stood the test of time.
Acute analgesia and occasionally procedure-related sedation may be required to accomplish adequate aural toilet in patients with severe inflammation and tenderness of the canal. In 1 study, analgesic cream has been applied to the ear canal in adults and cooperative children to relieve pain and anesthetize the external auditory meatus if the tympanic membrane is intact. Opioids such as fentanyl citrate, morphine sulfate, and hydromorphone hydrochloride are indicated for procedure-related pain and moderate to severe around-the-clock pain.

Benzocaine otic solution, with or without antipyrine, is available for topical anesthesia of the ear canal. There are no clinical trials that show efficacy of these medications in AOE, and the use of these drops may mask progression of underlying disease while pain is being suppressed. Topical benzocaine may cause contact dermatitis that can worsen or prolong AOE. If a topical anesthetic drop is prescribed for temporary pain relief, the patient should be reexamined within 48 hours to ensure that AOE has responded appropriately to primary therapy.

The addition of a topical steroid to topical antimicrobial drops has been shown to hasten pain relief in some randomized trials, but others have shown no significant benefits. Benzocaine otic solution, with or without antipyrine, is available for topical anesthesia of the ear canal. There are no clinical trials that show efficacy of these medications in AOE, and the use of these drops may mask progression of underlying disease while pain is being suppressed. Topical benzocaine may cause contact dermatitis that can worsen or prolong AOE.

Evidence Profile for 2: Pain Management.

- Aggregate evidence quality: B, 1 randomized controlled trial limited to AOE; consistent, well-designed randomized trials of analgesics for pain relief in general
- Benefit: increase patient satisfaction, allows faster return to normal activities
- Harm: adverse effects of analgesics
- Cost: direct cost of medication
- Benefits-harms assessment: preponderance of benefit over harm
- Value judgments: preeminent role of pain relief as an outcome when managing AOE
- Role of patient preferences: choice of analgesic, degree of pain tolerance
- Policy level: strong recommendation

3. INITIAL THERAPY: Clinicians should use topical preparations for initial therapy of diffuse, uncomplicated AOE. Systemic antimicrobial therapy should not be used unless there is extension outside the ear canal or the presence of specific host factors that would indicate a need for systemic therapy. Recommendation based on randomized controlled trials with minor limitations and a preponderance of benefit over harm.

The recommendation for initial topical therapy applies to the otherwise healthy patient with diffuse AOE that is not complicated by osteitis, abscess formation, middle ear disease, or recurrent episodes of infection. Topical therapy should be supplemented by systemic antibiotics if the affected individual has a condition, especially diabetes that is associated with markedly increased morbidity, or HIV infection/AIDS with immune deficiency that could impair host defenses; if the infection has spread beyond the confines of the ear canal into the pinna, skin of the neck or face, or into deeper tissues such as occurs with malignant external otitis; or if there is good reason to believe that topical therapy cannot be delivered effectively (see below Drug Delivery). Topical preparations are recommended as initial therapy for diffuse, uncomplicated AOE because of safety, efficacy over placebo in randomized trials, and excellent clinical and bacteriologic outcomes in comparative studies. There are no data on the efficacy of systemic therapy with the use of appropriate antibiotics and stratified by severity of the infection. Moreover, orally administered antibiotics have significant adverse effects that include rashes, vomiting, diarrhea, allergic reactions, altered nasopharyngeal flora, and development of bacterial resistance. Societal consequences include direct transmission of resistant bacterial pathogens in homes and child care centers.

Three randomized trials have compared topical antimicrobial versus placebo for treating diffuse AOE. Meta-analysis of the 2 trials with similar methods yields a combined absolute rate difference (RD) of 0.46 based on 89 patients (95% CI, 0.28 to 0.63), which suggests that only 2 patients needed to be treated (NNT) with topical antimicrobial to achieve 1 additional cure. Bacteriologic efficacy (RD, 0.61) was higher than clinical efficacy. Another trial reported significantly less edema and itching 3 days after therapy was initiated, and less edema, itching, redness, scaling, and weeping 7 days after therapy was initiated. Conversely, another study showed no benefit for an antimicrobial-steroid drop vs placebo, but patients with chronic otitis externa, otomycosis, and furunculosis were also included.

No randomized, controlled trials have directly compared oral antibiotic therapy with topical therapy. Reviews of survey data, however, show that about 20% to 40% of subjects with AOE receive oral antibiotics, often in addition to topical antimicrobials. Many of the oral antibiotics selected are inactive against and S. aureus, the most common pathogens identified in cases of AOE. Further, treatment with penicillins, macrolides, or cephalosporins increases disease persistence (rate ratios, 1.56 to 1.91), and treatment with cephalosporins also increases recurrence (rate ratio, 1.28; 95% CI, 1.03 to 1.58).

One additional study directly addresses the use of oral antibiotics in treating diffuse AOE. When patients were randomized to topical ointment plus oral antibiotic (trimethoprim-sulfamethoxazole) vs topical ointment plus placebo, there was no significant difference in cure rates at 2 to 4 days (RD, −0.01; 95% CI, −0.21 to 0.18) or at

Evidence Profile for 2: Pain Management.

- Aggregate evidence quality: B, 1 randomized controlled trial limited to AOE; consistent, well-designed randomized trials of analgesics for pain relief in general
- Benefit: increase patient satisfaction, allows faster return to normal activities
- Harm: adverse effects of analgesics
- Cost: direct cost of medication
- Benefits-harms assessment: preponderance of benefit over harm
- Value judgments: preeminent role of pain relief as an outcome when managing AOE
- Role of patient preferences: choice of analgesic, degree of pain tolerance
- Policy level: strong recommendation

3. INITIAL THERAPY: Clinicians should use topical preparations for initial therapy of diffuse, uncomplicated AOE. Systemic antimicrobial therapy should not be used unless there is extension outside the ear canal or the presence of specific host factors that would indicate a need for systemic therapy. Recommendation based on randomized controlled trials with minor limitations and a preponderance of benefit over harm.

The recommendation for initial topical therapy applies to the otherwise healthy patient with diffuse AOE that is not complicated by osteitis, abscess formation, middle ear disease, or recurrent episodes of infection. Topical therapy should be supplemented by systemic antibiotics if the affected individual has a condition, especially diabetes that is associated with markedly increased morbidity, or HIV infection/AIDS with immune deficiency that could impair host defenses; if the infection has spread beyond the confines of the ear canal into the pinna, skin of the neck or face, or into deeper tissues such as occurs with malignant external otitis; or if there is good reason to believe that topical therapy cannot be delivered effectively (see below Drug Delivery). Topical preparations are recommended as initial therapy for diffuse, uncomplicated AOE because of safety, efficacy over placebo in randomized trials, and excellent clinical and bacteriologic outcomes in comparative studies. There are no data on the efficacy of systemic therapy with the use of appropriate antibiotics and stratified by severity of the infection. Moreover, orally administered antibiotics have significant adverse effects that include rashes, vomiting, diarrhea, allergic reactions, altered nasopharyngeal flora, and development of bacterial resistance. Societal consequences include direct transmission of resistant bacterial pathogens in homes and child care centers.

Three randomized trials have compared topical antimicrobial versus placebo for treating diffuse AOE. Meta-analysis of the 2 trials with similar methods yields a combined absolute rate difference (RD) of 0.46 based on 89 patients (95% CI, 0.28 to 0.63), which suggests that only 2 patients needed to be treated (NNT) with topical antimicrobial to achieve 1 additional cure. Bacteriologic efficacy (RD, 0.61) was higher than clinical efficacy. Another trial reported significantly less edema and itching 3 days after therapy was initiated, and less edema, itching, redness, scaling, and weeping 7 days after therapy was initiated. Conversely, another study showed no benefit for an antimicrobial-steroid drop vs placebo, but patients with chronic otitis externa, otomycosis, and furunculosis were also included.

No randomized, controlled trials have directly compared oral antibiotic therapy with topical therapy. Reviews of survey data, however, show that about 20% to 40% of subjects with AOE receive oral antibiotics, often in addition to topical antimicrobials. Many of the oral antibiotics selected are inactive against and S. aureus, the most common pathogens identified in cases of AOE. Further, treatment with penicillins, macrolides, or cephalosporins increases disease persistence (rate ratios, 1.56 to 1.91), and treatment with cephalosporins also increases recurrence (rate ratio, 1.28; 95% CI, 1.03 to 1.58).

One additional study directly addresses the use of oral antibiotics in treating diffuse AOE. When patients were randomized to topical ointment plus oral antibiotic (trimethoprim-sulfamethoxazole) vs topical ointment plus placebo, there was no significant difference in cure rates at 2 to 4 days (RD, −0.01; 95% CI, −0.21 to 0.18) or at
5 to 6 days (RD 0.08; 95% CI, –0.15 to 0.30). The ointment (Kenacomb) contained an antifungal, an antibacterial active against gram-negative organisms, an antibiotic active against gram-positive organisms, and a steroid.

The most compelling argument against the use of oral antibiotics for diffuse AOE limited to the ear canal is the efficacy of topical treatments that do not include antibiotics. Effective topical treatments include acetic acid, boric acid, aluminum acetate, silver nitrate, and an endogenous antiseptic N-chlorotaurine. Topical steroids are also effective, as a single agent, or in combination with acetic acid, or an antifungal preparation. When the success of these nonantibiotic therapies is considered, it is likely that for cases of uncomplicated AOE, oral antibiotics, particularly those with no activity against \( P. \) aeruginosa or \( S. \) aureus, are unnecessary.

An advantage of topical therapy is the very high concentration of antimicrobial that can be delivered to infected tissue, often 100 to 1000 times higher than can be achieved with systemic therapy. For example a 0.3% solution of antibiotic (a typical concentration in commercial otic drops) has a concentration of 3000 mcg/mL. Any organisms known to cause AOE, even those considered “resistant,” will be unlikely to survive contact with this antibiotic concentration. Because there are between 10 to 20 drops/mL, depending on the nature of the liquid (solution vs suspension, viscosity, etc), each dose of 3 to 5 drops contains about 0.5 to 1.5 mg of antibiotic.

Topical therapy avoids prolonged exposure of bacteria to subtherapeutic concentrations of antibiotic, and may therefore be less likely than systemic therapy to result in selective pressure for resistant organisms. The avoidance of antibiotic exposure of host bacteria resident outside the ear canal, as occurs with systemic therapy, provides a further advantage to the reduction of the selection of resistant microorganisms. Restrictive use of oral antibiotics for AOE is important because of the increased resistance among common AOE pathogens, especially \( S. \) aureus and \( P. \) aeruginosa.

Along with prescribing topical antimicrobials, clinicians should advise patients to resist manipulation to minimize ear trauma and should discuss issues that pertain to water restrictions during treatment. The insertion of earplugs or cotton (with petroleum jelly) before showering or swimming can reduce the introduction of moisture into the ear. The external auditory canal can be dried after swimming or bathing with a hair dryer on the lowest setting.

Patients with AOE should preferably abstain from water sports for 7 to 10 days during treatment. A swimming pool, as long as prolonged submersion is avoided, can be allowed in mild cases. Competitive swimmers sometimes return to competition 2 to 3 days after treatment, or if they use well-fitting ear plugs, after the pain has resolved. Patients with hearing aids or ear phones should limit insertion until pain and discharge (if present) have subsided.

Evidence Profile for 3: Initial Therapy.

- Aggregate evidence quality: B, randomized controlled trials with minor limitations; no direct comparisons of topical vs systemic therapy
- Benefit: avoid side effects by not using unnecessary systemic medications, avoid increased disease persistence rates and disease recurrence rates seen when inappropriate systemic antibiotics are used, reduce antibiotic resistance by avoiding systemic antibiotics, and potential for increased patient adherence to therapy
- Harm: adverse effects of topical antimicrobials
- Cost: oral or topical antimicrobials range in cost from a few dollars to more than $100
- Benefits-harms assessment: preponderance of benefit over harm
- Value judgments: desire to decrease the use of ineffective treatments, societal benefit from avoiding the development of antibiotic resistance
- Role of patient preferences: the selection of the specific therapy may involve patient preferences in this decision process based on cost, adherence to therapy, and other factors
- Policy level: recommendation

4. TOPICAL THERAPY: The choice of topical antimicrobial for initial therapy of diffuse AOE should be based upon efficacy, low incidence of adverse events, likelihood of adherence to therapy, and cost.

Recommendation based on randomized trials with some heterogeneity and a preponderance of benefit over harm.

A variety of topical preparations are approved by the US Food and Drug Administration (FDA) for treating AOE (Table 5). Most of those currently available in the United States provide antimicrobial activity through: 1) an antibiotic, which may be an aminoglycoside, polymyxin B, a quinolone, or a combination of these agents; 2) a steroid, such as hydrocortisone or dexamethasone; or 3) a low pH antiseptic, such as aluminum acetate solution or acetic acid.

Efficacy of Topical Therapy. Clinicians should use a topical drug that is efficacious for diffuse AOE. Efficacy is best summarized with meta-analysis of randomized controlled trials, but at the time of this writing (November 2005) there were no published meta-analyses that concern diffuse AOE. Therefore, we conducted a systematic review that yielded 20 randomized trials (Table 6) meeting inclusion criteria (see “Methods” section). Two trials did not report data suitable for statistical pooling. Full details of the meta-analysis are reported separately in this supplement, but relevant summary data are reported herein.

The randomized trials in Table 6 have varying methods and quality. Sample size ranges from 28 to 842 patients;
50% of trials included both children and adults. Two studies reported outcomes by ears not patients, but were included in the meta-analysis because 90% of patients had unilateral disease. Study quality varied, with 50% having a Jadad quality score less than 3; only 2 achieved a maximum quality score of 5. Most (56%) studies were not double-blind, because of obvious differences in drug appearance or dosing schedule. The quality of 1 study could not be fully assessed because it was an abstract.

### Table 5
Common topical otic preparations approved by the FDA for treating diffuse AOE

<table>
<thead>
<tr>
<th>Active drug(s)</th>
<th>Trade name</th>
<th>Bottle size, mL</th>
<th>Cost, US$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetic acid, aluminum acetate</td>
<td>Otic Domeboro</td>
<td>60.0</td>
<td>31</td>
</tr>
<tr>
<td>Acetic acid, hydrocortisone</td>
<td>VoSol HC</td>
<td>10.0</td>
<td>110</td>
</tr>
<tr>
<td>Ciprofloxacin, hydrocortisone</td>
<td>Cipro HC</td>
<td>10.0</td>
<td>125</td>
</tr>
<tr>
<td>Ciprofloxacin, dexamethasone</td>
<td>Ciprodex</td>
<td>7.5</td>
<td>125</td>
</tr>
<tr>
<td>Neomycin, polymyxin B, hydrocortisone</td>
<td>Cortisporin Otic</td>
<td>10.0</td>
<td>89</td>
</tr>
<tr>
<td>Ofloxacin</td>
<td>Floxin Otic</td>
<td>5.0</td>
<td>71</td>
</tr>
</tbody>
</table>

Adapted from Fairbanks.

### Table 6
Randomized controlled trials included in the meta-analysis of AOE treatment

<table>
<thead>
<tr>
<th>Author, year, country</th>
<th>N</th>
<th>Age, y</th>
<th>Topical treatment groups</th>
<th>Aural toilet</th>
<th>Follow-up, %</th>
<th>Jadad score†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arnes and Dibb, 1993, Norway</td>
<td>30</td>
<td>≥18</td>
<td>Cipro vs oxytetracycline/polymyx/Hydrocortisone</td>
<td>Wick</td>
<td>100</td>
<td>1</td>
</tr>
<tr>
<td>Cannon and Grunwaldt, 1967, USA</td>
<td>40</td>
<td>2-68</td>
<td>Neo/methylprednisolone vs placebo</td>
<td>Yes</td>
<td>73</td>
<td>4</td>
</tr>
<tr>
<td>Cannon, 1970, USA</td>
<td>43</td>
<td>≥4</td>
<td>Acetic/glyceryl vs placebo</td>
<td>Yes</td>
<td>100</td>
<td>4</td>
</tr>
<tr>
<td>Clayton et al, 1990, UK</td>
<td>66</td>
<td>ns</td>
<td>Alum-acetate vs gentamicin</td>
<td>No</td>
<td>73</td>
<td>4</td>
</tr>
<tr>
<td>Emgard and Helsmoort, 2005, Sweden</td>
<td>51</td>
<td>19-67</td>
<td>Betamethasone vs oxytetracycline/polymyx/Hydrocortisone</td>
<td>No</td>
<td>98</td>
<td>3</td>
</tr>
<tr>
<td>Freedman, 1978, USA</td>
<td>91</td>
<td>4-76</td>
<td>Neo/collistin/Hydrocortisone vs placebo</td>
<td>Wick</td>
<td>100</td>
<td>2</td>
</tr>
<tr>
<td>Goldenberg et al, 2002, Israel</td>
<td>120</td>
<td>18-52</td>
<td>Cipro vs tobramycin</td>
<td>No</td>
<td>NS</td>
<td>1</td>
</tr>
<tr>
<td>Jones et al, 1997, USA</td>
<td>601</td>
<td>≥1</td>
<td>Ofloxacin vs neo/polymyx/Hydrocortisone</td>
<td>No</td>
<td>79</td>
<td>3</td>
</tr>
<tr>
<td>Kime et al, 1978, USA</td>
<td>102</td>
<td>6-73</td>
<td>Acetic/Hydrocortisone vs neo/collistin/Hydrocortisone</td>
<td>Wick</td>
<td>81</td>
<td>3</td>
</tr>
<tr>
<td>Lambert, 1981, Cyprus</td>
<td>126</td>
<td>1-44</td>
<td>Alum-acetate vs neo/polymyx/Hydrocortisone</td>
<td>Yes</td>
<td>93</td>
<td>1</td>
</tr>
<tr>
<td>Neher et al, 2004, Austria</td>
<td>50</td>
<td>8-89</td>
<td>NCT vs neo/polymyx/Hydrocortisone</td>
<td>Yes</td>
<td>100</td>
<td>2</td>
</tr>
<tr>
<td>Ordonez et al, 1978, USA</td>
<td>181</td>
<td>10-82</td>
<td>Acetic/Hydrocortisone vs neo/polymyx/Hydrocortisone</td>
<td>Yes</td>
<td>61</td>
<td>4</td>
</tr>
<tr>
<td>Pistorius et al, 1999, USA</td>
<td>842</td>
<td>2-85</td>
<td>Cipro vs ciproflaxacin vs neo/polymyx/Hydrocortisone</td>
<td>Yes</td>
<td>83</td>
<td>1</td>
</tr>
<tr>
<td>Psifidis et al, 2005, Greece</td>
<td>91</td>
<td>22-61</td>
<td>Cipro vs ciproflaxacin vs neo/polymyx/Hydrocortisone</td>
<td>No</td>
<td>NS</td>
<td>1</td>
</tr>
<tr>
<td>Roland et al, 2004, USA</td>
<td>468</td>
<td>1-90</td>
<td>Ciproflaxacin vs neo/polymyx/Hydrocortisone</td>
<td>Yes</td>
<td>85</td>
<td>2</td>
</tr>
<tr>
<td>Ruth et al, 1990, Sweden</td>
<td>53</td>
<td>11-74</td>
<td>HC butyrate vs oxytetracycline/polymyx/Hydrocortisone</td>
<td>No</td>
<td>87</td>
<td>2</td>
</tr>
<tr>
<td>Sabater et al, 1996, Spain</td>
<td>54</td>
<td>≥18</td>
<td>Cipro vs gentamicin</td>
<td>No</td>
<td>NS</td>
<td>2</td>
</tr>
<tr>
<td>Slack, 1987, UK</td>
<td>28</td>
<td>ns</td>
<td>Boric/ethyl vs neo/polymyx/Hydrocortisone</td>
<td>Yes</td>
<td>86</td>
<td>3</td>
</tr>
<tr>
<td>Tsikoudas et al, 2002, UK</td>
<td>39</td>
<td>≥18</td>
<td>Betamethasone vs betamethasone/neomycin</td>
<td>Yes</td>
<td>67</td>
<td>5</td>
</tr>
<tr>
<td>van Balen et al, 2003, The Netherlands</td>
<td>213</td>
<td>≥18</td>
<td>Acetic vs acetaminophen vs neo/polymyx/Hydrocortisone</td>
<td>Yes</td>
<td>93</td>
<td>5</td>
</tr>
</tbody>
</table>

Acetic, acetic acid 2%; alum-acetate, aluminum acetate 8%; boric, boric acid 4%; ciprofloxacin; dex, dexamethasone; ethyl alcohol 25%; HC, hydrocortisone; glyceryl, glyceryl acetate 88%; methylprednisolone; NCT, N-chlorotaurine (antiseptic); neo, neomycin; NS, not stated; oflox, ofloxacin; oxytetracycline; polymyx, polymyxin B; triamcin, triamcinolone. †Indicates a double-blind comparison. ‡Quality score ranging from 1 (lowest) to 5 (highest).
The trials identified by systematic review permit the following statistical comparisons of topical therapy that are summarized in Table 7:

- antimicrobial (antibiotic or antiseptic) vs placebo, 2 trials (discussed in preceding section)
- antiseptic vs antimicrobial, 8 trials
- quinolone antibiotic vs nonquinolone antibiotic(s), 7 trials
- steroid-antimicrobial vs antimicrobial alone, 3 trials
- antimicrobial-steroid vs steroid alone, 2 trials

We found no significant differences in clinical outcomes of AOE (Table 7) for antiseptic vs antimicrobial, quinolone antibiotic vs nonquinolone antibiotic(s), or steroid-antimicrobial vs antimicrobial alone. Regardless of topical agent used, about 65% to 90% of patients had clinical resolution within 7 to 10 days. One potential explanation is that differences among agents may have been missed because of low statistical power; however, all analyses had a combined sample size above 300 patients, making low power unlikely. Further, the magnitude of differences observed between treatments is very modest, with a maximum rate difference of 0.11 (NNT of 9 patients). Therefore, even if additional studies were performed to increase power, it is likely that differences among treatments remain small or negligible.

The only clinical comparison that achieved statistical significance in Table 7 is for steroid/antibiotic vs steroid alone (comparison 13). The reason for inferior outcomes with steroid/antibiotic is unclear, although in 1 of the studies,97 cited the steroid used for single-agent therapy had high potency (betamethasone) and the comparator had low potency (hydrocortisone). Conversely, the second study95 used a low potency steroid (hydrocortisone) in both groups. The combined analysis with both studies, however, includes only 92 patients and the broad confidence limits cannot exclude a trivial effect. Additional studies are needed to confirm this finding and to increase precision.

Another significant comparison in Table 7 is bacteriologic cure after nonquinolone therapy, with an 8% absolute increase when a quinolone antibiotic is used. The clinical significance of this modest effect (NNT of 12 patients) is uncertain, yet the addition of a quinolone antibiotic may be of benefit particularly if topical antibiotic therapy is insufficient.

Table 7

<table>
<thead>
<tr>
<th>Treatment group vs control group</th>
<th>References combined</th>
<th>N</th>
<th>Control rate†</th>
<th>RD (95% CI)‡</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antimicrobial vs placebo</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Clinical cure at 3-10 days</td>
<td>83,84</td>
<td>89</td>
<td>0.15</td>
<td>0.46 (0.29, 0.63)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2. Bacteriologic cure</td>
<td>83,84</td>
<td>$112</td>
<td>0.20</td>
<td>0.61 (0.46, 0.76)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Antiseptic vs antibiotic</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Clinical cure at 7-10 days</td>
<td>76,77,89,94</td>
<td>318</td>
<td>0.66</td>
<td>0.05 (−0.03, 0.12)</td>
<td>0.217</td>
</tr>
<tr>
<td>4. Clinical cure at 11-14 days</td>
<td>76,77,88,91</td>
<td>368</td>
<td>0.80</td>
<td>0.04 (−0.06, 0.13)</td>
<td>0.468</td>
</tr>
<tr>
<td><strong>Quinolone vs nonquinolone antibiotic(s)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Clinical cure at 3-4 days</td>
<td>106,107</td>
<td>476</td>
<td>0.15</td>
<td>0.11 (−0.06, 0.28)</td>
<td>0.192</td>
</tr>
<tr>
<td>6. Clinical cure at 7-10 days</td>
<td>9,10,78,105,107,108</td>
<td>1475</td>
<td>0.77</td>
<td>0.07 (−0.02, 0.16)</td>
<td>0.110</td>
</tr>
<tr>
<td>7. Clinical cure at 14-28 days</td>
<td>10,106,107</td>
<td>936</td>
<td>0.83</td>
<td>0.04 (−0.01, 0.08)</td>
<td>0.145</td>
</tr>
<tr>
<td>8. Improved at 7-10 days</td>
<td>10,105,107</td>
<td>890</td>
<td>0.89</td>
<td>0.05 (−0.05, 0.14)</td>
<td>0.292</td>
</tr>
<tr>
<td>9. Bacteriologic cure</td>
<td>9,10,78,105,106,107</td>
<td>980</td>
<td>0.87</td>
<td>0.08 (0.006, 0.16)</td>
<td>0.035</td>
</tr>
<tr>
<td>10. Any adverse event</td>
<td>9,10,107</td>
<td>1330</td>
<td>0.15</td>
<td>0.002 (−0.07, 0.08)</td>
<td>0.963</td>
</tr>
<tr>
<td><strong>Antimicrobial/steroid vs antimicrobial alone</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Clinical cure at 7 days</td>
<td>10,76,78</td>
<td>660</td>
<td>0.68</td>
<td>0.04 (−0.08, 0.16)</td>
<td>0.546</td>
</tr>
<tr>
<td>12. Bacteriologic cure</td>
<td>10,78</td>
<td>342</td>
<td>0.93</td>
<td>−0.02 (−0.15, 0.11)</td>
<td>0.761</td>
</tr>
<tr>
<td><strong>Steroid/antibiotic vs steroid alone</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. Clinical cure at 7-11 days</td>
<td>95,97</td>
<td>92</td>
<td>0.72</td>
<td>−0.20 (−0.38−0.03)</td>
<td>0.021</td>
</tr>
</tbody>
</table>

CI, Confidence interval; RD, absolute rate difference.
†Control rate is calculated by simple division of total events by total patients to aid in interpreting the RD.
‡Absolute change in outcomes for treatment vs. control groups, beyond the control group rate, based on random-effects meta-analysis.
§Analysis by ears not patients.
days. \(^7\) Two other studies showed differences in inflammation scores \(^9\),\(^10\),\(^10\) and I showed significantly less itching with the steroid-containing drop. \(^9\)

**Adverse Events, Adherence To Therapy, and Cost.** The lack of differences in efficacy, on average, among most topical antimicrobial and steroid preparations (Table 7) suggests that patient preference and clinician experience are important aspects in selecting therapy. Cost, adherence to therapy, and adverse effects must also be considered.

Only a few studies \(^9\),\(^10\),\(^10\) report detailed information on adverse events and show an overall low incidence and comparable rates among treatment groups. The most common problems are pruritus (about 7%) and site reaction (5%); other events with an incidence less than 2% include rash, discomfort, otalgia, dizziness, vertigo, superinfection, and reduced hearing. \(^9\),\(^10\) None of the randomized trials reported otomycosis after topical antibiotics, although otomycosis has been described anecdotally after topical ofloxacin therapy for AOE. \(^11\)

One study \(^16\) that compared 2 quinolone preparations to a neomycin product found that only 1.1% of patients had to discontinue the drug because of infection, nausea, or vomiting. Unfortunately, studies of antiseptics have reported limited to no information with respect to adverse events.

Contact dermatitis is a potential sequela of topical antimicrobial or steroid therapy, but it is rare after a single course of therapy for diffuse AOE. Three studies \(^9\),\(^10\),\(^10\) have compared a quinolone drop vs neomycin-polymyxin B-hydrocortisone drop for diffuse AOE, with no significant difference in adverse events individually or when combined (Table 7, comparison 10). Conversely, about 30% to 60% of patients with chronic or eczematous external otitis develop a contact dermatitis, most often to aminoglycosides such as neomycin and framycetin.\(^5\)\(^)\(^8\)\(^)\(^11\)\(^)\(^15\) No studies are limited specifically to patients with recurrent AOE, chronic external otitis, or eczematous external otitis, but it would appear prudent to avoid the use of aminoglycoside drops in these populations.

Remaining factors to consider when prescribing topical therapy include adherence to therapy and cost. Adherence to therapy and patient satisfaction are highest when drops are easy to administer,\(^4\) which would entail a less frequent dosing schedule, shorter duration of therapy, or both. There are no comparative studies, but drops administered 4 times daily (eg, neomycin, polymyxin, hydrocortisone) may be less acceptable to some patients. Cost varies widely among available otic preparations (Table 5) and range from a few dollars for antiseptics or generic products (eg, neomycin, polymyxin B, hydrocortisone) to more than $100 for quinolones, with or without a steroid.

Dosing schedules for AOE have not been studied systematically, but available data suggest that, at least with quinolone drops (and perhaps also with the other concentration-dependent drugs like the aminoglycosides), a twice-daily (bid) dose regimen is adequate. One open label study \(^11\) showed good clinical outcomes when ofloxacin was given once daily. The optimal duration of therapy has not been determined and varies from a few days up to several weeks in published trials. Recent studies suggest that 7 days of therapy are adequate, at least for quinolone antibiotics.\(^9\),\(^10\),\(^10\),\(^11\)

**Complementary and Alternative Therapies.** There are no data with respect to the efficacy of complementary and alternative therapies for AOE. Isopropyl (“rubbing”) alcohol, and 5% acetic acid (white vinegar) plus equal parts of isopropyl alcohol or water, are time honored “home remedies,” but have never been formally evaluated in clinical trials. The similarity of these preparations to some antiseptic or acidifying agents that have been studied suggests they may be effective. For example, most acetic acid preparations have a concentration of 2.5%, which equals a 50% dilution of white vinegar. Although “tea tree oil” has been found to be effective in vitro against 71% of organisms cultured from 52 patients with AOE,\(^11\) Pseudomonas was resistant in 75% of cases, and no controlled efficacy trials that evaluate this form of therapy have been described.

Ear candles should not be used in treating AOE. Ear candles have never been shown to be efficacious for AOE but have been shown to produce harm.\(^11\) Obstruction of the ear canal with paraffin and associated hearing loss and perforation of the tympanic membrane have been reported.\(^11\)

**Evidence Profile for 4: Topical Therapy.**

- **Aggregate evidence quality:** B, randomized controlled trials with some heterogeneity
- **Benefit:** effective therapy, appropriate adherence to therapy, and acceptable cost
- **Harm:** low incidence of adverse events
- **Cost:** direct cost of topical antimicrobials or steroids
- **Benefits-harms assessment:** preponderance of benefit over harm
- **Value judgments:** meta-analysis with cure rates (clinical and bacteriologic) used as primary measure of efficacy; heterogeneity among trials considered acceptable for statistical pooling with a random effects model
- **Role of patient preferences:** substantial role for patient preference in choice of topical therapeutic agent
- **Policy level:** recommendation

5. **DRUG DELIVERY:** Clinicians should inform patients how to administer topical drops.

When the ear canal is obstructed, delivery of topical preparations should be enhanced by aural toilet, placement of a wick, or both.

Recommendation based on observational studies with a preponderance of benefit over harm.

For topical treatment to be effective, the drug must be delivered to infected tissues. Although the majority of patients with uncomplicated AOE will require only topical medication, for some patients, additional management is needed to ensure the appropriate drug delivery. Ensuring
adequate delivery of the topical medication may require removal of a foreign body, performance of aural toilet to remove obstructing debris, placement of a wick to permit drug delivery through the length of the ear canal, or all three.

Drug delivery may be impaired by poor adherence to therapy, poor application (ie, “missing” the ear canal), debris filling the canal, or edema closing the canal. Poor adherence to therapy and ineffective administration must be dealt with by providing clear instructions. Self-administration of eardrops is difficult because it must be done by feel. Only 40% of patients who self-medicate do so appropriately during the first 3 days, often tending to under-medicate. Adherence to therapy increases significantly when someone other than the patient applies the drops, which makes this the preferred method of administration when feasible.

Ototopical drops should be applied with the patient lying down and the affected ear upward. Drops should be run along the side of the canal until it is filled. The amount required will vary with the age and size of the patient. Gentle to-and-fro movement of the pinna is often necessary to eliminate trapped air and to assure filling, particularly when a viscous solution is used. The patient should remain in this position for about 3 to 5 minutes. Use of a timer to mark the minutes is often helpful to facilitate the cooperation of young children. After the placement of drops, the canal is best left open to dry to avoid trapped moisture and infected debris.

The ear canal should be cleared of inflammatory debris, obstructing cerumen, or any foreign object. There are no randomized studies of the use of aural toilet in AOE, but some investigators have proposed that aural toilet by itself (without antimicrobials) is therapeutic. Aural toilet may be done with a gentle lavage using body-temperature water, saline solution, or hydrogen peroxide. Alternative methods of aural toilet include physically removing the obstructing debris with suction or dry mop (blotting with cotton). Adequate visualization for suctioning may be facilitated by using an otoscope with an open head or a binocular otologic microscope.

There are no randomized trials that address the safety of aural lavage in diabetics or immunocompromised patients with AOE. Lavage of the ear canal for cerumen impaction in elderly or diabetic patients, however, has been implicated as a contributing factor in malignant otitis externa. The pathophysiology of malignant (necrotizing) otitis externa is poorly understood, but aural water exposure is a potential iatrogenic factor. Patients with risk factors such as diabetes or immunocompromised state, as well as those with established malignant otitis externa, may require atraumatic cleaning with aural suctioning under microscopic guidance.

Clinicians may place a wick in the ear canal if there is edema that prevents drop entry or if most of the tympanic membrane cannot be visualized. The wick should preferably be made of compressed cellulose because it expands when exposed to moisture, facilitates drug delivery, and reduces ear canal edema. Alternatively, ribbon gauze can be used. Once a dry wick is placed in the ear canal, some experts recommend moistening the wick with an aqueous solution (water, saline solution, aluminum acetate) before the first application of an otic suspension or a nonaqueous viscous medication. A wick should not be made of a simple cotton ball since the cotton can fall apart and become lodged in the ear canal.

Many treatment studies uniformly use a wick to improve drug delivery (Table 6), but there are no trials of wick efficacy. Consequently, the benefit of a wick is questioned by some clinicians, especially in managing uncomplicated AOE. However, following first principles, if the anatomy (narrow or edematous canal) makes delivery of the topical medicine problematic, the use of a wick seems prudent. A wick is unnecessary once the ear canal edema subsides, which may occur within 24 hours or a few days of topical therapy. The wick may fall out spontaneously, may be removed by the patient, or may be removed by a clinician at a scheduled follow-up visit.

Evidence Profile for 5: Drug Delivery.

- Aggregate evidence quality: C, observational studies and expert opinion
- Benefit: improved adherence to therapy and drug delivery
- Harm: pain and local trauma caused by inappropriate aural toilet or wick insertion
- Cost: wicks (inexpensive)
- Benefits-harms assessment: preponderance of benefit over harm
- Value judgments: none
- Role of patient preferences: choice of self-administering drops vs using assistant
- Policy level: recommendation

6. NONINTACT TYPANIC MEMBRANE. When the patient has a tympanostomy tube or known perforation of the tympanic membrane, the clinician should prescribe a nonototoxic topical preparation. Recommendation based on reasoning from first principles and on exceptional circumstances where validating studies cannot be performed and there is clear preponderance of benefit over harm.

Special consideration must be given to the individual with known or suspected perforation of the tympanic membrane. The external auditory canal, including the tympanic membrane, is lined with keratinizing squamous epithelium, but the middle ear is lined with mucosa. This mucosa forms the lateral portion of the round window membrane, which separates the middle-ear space from the fluids of the inner ear. Antibiotics placed into the middle ear can cross the round window membrane and reach the inner ear. Ototoxic anti-
biotics delivered into the middle ear space of experimental animals, including primates, consistently cause severe hearing loss and ototoxic injury to the organ of Corti.126-128

Clinical experience with topical ototoxic antibiotics in patients with tympanic membrane perforation suggests that hearing loss does not occur after a short course of therapy129,130; however, severe hearing loss has been observed after prolonged or excessive administration of topical drops.130,131 The validity of these and other clinical reports is limited by retrospective design, incomplete follow-up, and inconsistent audiologic testing. Given the ethical limitations of randomizing patients with a nonintact tympanic membrane to an ototoxic drop, it is unlikely that definitive evidence (validating studies) is forthcoming.

Careful examination of the tympanic membrane will reveal a perforation in some cases of AOE. The ear canal and auricle may be so tender or swollen, however, that the tympanic membrane cannot be visualized without undue pain or discomfort. If swelling or discomfort do not preclude its use, tympanometry can sometimes be helpful to establish the presence of an intact tympanic membrane. When tympanometry shows a normal type A tracing (peaked curve with normal pressure), the tympanic membrane is assumed to be intact unless there is a reason to believe it is not (eg, an indwelling tympanostomy tube).

A perforation may be suspected if the patient has a positive history, unless the most recent examination before the episode of AOE has verified that the perforation has closed. Children with tympanostomy tubes are a special instance within this category. Most tympanostomy tubes remain in the tympanic membrane for at least 6 to 12 months; therefore a patent tube should be assumed to be present within the tympanic membrane of any individual who had it placed less than a year earlier, unless tube extrusion and subsequent closure of the tympanic membrane have been documented. Individuals who taste substances, presumably medicinals, placed into their ear, or who can expel air out their ear canal by pinched nose blowing, can be assumed to have a perforation.

If the tympanic membrane is known or suspected to be nonintact, topical drops that contain alcohol, have a low pH (most acidifying/antiseptic agents), or both should be avoided because of pain and potential ototoxicity. Substances with ototoxic potential (eg, aminoglycosides, alcohol) should not be used when the tympanic membrane is perforated and the middle ear space is open, because the risk of ototoxic injury outweighs the benefits compared with nonototoxic antimicrobials with equal efficacy.132 The only topical antimicrobials approved by the FDA (December 2005) for middle ear use are ofloxacin and ciprofloxacin/dexamethasone. Moreover, there is an explicit warning by the manufacturer that neomycin/polymyxin B/hydrocortisone not be used with a nonintact tympanic membrane:

“WARNINGS. Neomycin can induce permanent sensorineural hearing loss due to cochlear damage, mainly destruction of hair cells in the organ of Corti. The risk is greater with prolonged use. Therapy should be limited to 10 consecutive days (see PRECAUTIONS-General). Patients being treated with eardrops containing neomycin should be under close clinical observation. CORTISPORIN Otic Suspension should not be used in any patient with a perforated tympanic membrane (emphasis added).”

AOE can be secondary to acute otitis media. For example, mucopurulent exudate that flows through an acute tympanic membrane perforation from the middle ear can infect the tissues of the ear canal and create a secondary otitis externa. Less commonly, AOE will develop independently in an ear with acute otitis media (AOM). When AOM exists together with AOE, the AOM should be treated as an independent disease process according the current guidelines.52

Evidence Profile for 6: Nonintact Tympanic Membrane.

- Aggregate evidence quality: D, reasoning from first principles, and X, exceptional situations where validating studies cannot be performed
- Benefit: avoid pain and hearing loss
- Harm: none
- Cost: eardrops without ototoxicity are more costly
- Benefits-harms assessment: preponderance of benefit over harm
- Value judgments: importance of avoiding iatrogenic hearing loss from a potentially ototoxic topical preparation when nonototoxic alternatives are available; placing safety above economic cost
- Role of patient preferences: increased cost of nonototoxic preparation for patients who pay for their own medication vs risk of ototoxicity in less expensive alternatives
- Policy level: recommendation

7. OUTCOME ASSESSMENT: If the patient fails to respond to the initial therapeutic option within 48 to 72 hours, the clinician should reassess the patient to confirm the diagnosis of diffuse AOE and to exclude other causes of illness. Recommendation based on observational studies and a preponderance of benefit over harm.

Appropriate treatment of uncomplicated AOE should be followed by symptom improvement (otalgia, itching, fullness) within 48 to 72 hours (Fig 1). In clinical trials that evaluate patient outcomes of topical treatment with symptom diaries, significant decreases in patient-reported ear pain are generally seen after 1 day of treatment and most pain resolves within 4 to 7 days.76,97,116 One prospective cohort study40 that explored the relationship of patient-reported satisfaction with clinical outcomes showed that symptom relief was the factor most highly associated with patient satisfaction.
Figure 1  Flowchart for managing acute otitis externa.

- **Patient age 2y or older with diffuse AOE**
  - **Prescribe analgesics based on pain severity**

- **Extension outside ear canal or host factors requiring systemic therapy?**
  - **Yes**
    - **Prescribe systemic antimicrobial active against Pseudomonas aeruginosa and Staphylococcus aureus, with or without topical therapy, plus other management based on underlying condition**
  - **No**

- **Perforated tympanic membrane (known or suspected) or tympanostomy tube?**
  - **Yes**
    - **Prescribe topical therapy with a non-ototoxic preparation**
  - **No**
    - **Prescribe topical therapy based on benefits, cost, compliance, preference**

- **Obstructed ear canal?**
  - **Yes**
    - **Perform aural toilet to remove obstructing debris; place wick if edema prevents drug delivery**
  - **No**
    - **Educate patient or caregiver on how to administer topical drops**

- **Clinically improved in 48-72 hours?**
  - **Yes**
    - **Complete course of therapy**
  - **No**
    - **Re-assess patient**

- **Illness other than AOE?**
  - **Yes**
    - **Assess drug delivery, adherence to therapy, need to change therapy**
  - **No**
    - **Treat other illness**
Initial treatment failure of diffuse AOE may be caused by an obstructed ear canal, poor adherence to therapy, misdiagnosis, microbiologic factors, host factors, or contact sensitivity to eardrops. If topical antimicrobial therapy was prescribed, the clinician should reassess the patency of the ear canal to ensure that edema or debris are not impeding drug delivery. Any obstruction should be addressed with aural toilet, wick placement, or both (see preceding section, Drug Delivery), or, if the obstruction cannot be relieved, systemic therapy is begun with an oral antibiotic that covers *P. aeruginosa* and *S. aureus*.

The clinician should also assess adherence with therapy. Patients tend to over-administer ear drops when pain is greatest and to under-administer as symptoms resolve.40,120

Alternative causes of ear pain and associated otorrhea should be considered if the patient fails to respond to treatment, though the need for specialist referral is uncommon (3%) when AOE is treated appropriately.2 Fungi may be present as a copathogen in some patients with AOE, and cause persistent infection from overgrowth in the ear canal if the flora is altered after topical antibacterial therapy.5 A culture of the ear canal can identify fungi, resistant bacteria, or unusual causes of infection that require targeted topical or systemic therapy.

Initial treatment failures that are not related to drug delivery or microbiologic factors may reflect comorbidity or misdiagnosis.38,134 Persistent symptoms can be caused by dermatologic disorders that include dermatitis (atopic, seborrheic, or contact), psoriasis, dermatomycosis, or acne that involves the external auditory canal. The ear canal and tympanic membrane should be reexamined to detect an unrecognized foreign body, perforated tympanic membrane, or middle ear disease. Patients with severe refractory symptoms should be reassessed for malignant otitis externa or carcinoma of the external auditory canal, especially if granulation tissue is present.51,135

Contact sensitivity of the external auditory canal can result in refractory AOE in some patients. Delayed-type hypersensitivity reactions to topical antiseptic otic preparations are characterized by severe pruritus, skin inflammation, edema of the external auditory canal, and persistent otorrhea; blisters and vesicles may be present. The allergic reaction can extend beyond the ear canal to involve the skin around the ear and the neck. Neomycin-containing eardrops are most commonly noted to cause contact sensitivity, which has a 13% to 30% prevalence on patch testing of patients with chronic otitis externa.134,136,137

Contact sensitivity of the ear canal may also result from other topical antimicrobials (bacitracin, quinolones, gentian violet, polymyxin B sulfate), topical steroid preparations (hydrocortisone, triamcinolone), or topical anesthetics (benzocaine alone, or combined with dibucaine and tetracaine [caine mix]). Preservatives in topical otic preparations associated with at least a 1% incidence of contact sensitivity include propylene glycol, thimerosal, benzalkonium chloride, benzethonium chloride, and methyl-p-oxybenzoate. Fragrance additives may also cause similar reactions. Finally, contact sensitivity may be caused by silicone ear plugs or by hearing-aid molds that contain silicone or methyl-methacrylate.134,136,137

**Evidence Profile for 7: Outcome Assessment.**

- Aggregate evidence quality: C, observational studies
- Benefit: identify misdiagnosis and potential complications from delayed management; reduce pain
- Harm: none
- Cost: need for reevaluation by clinician
- Benefits-harms assessment: preponderance of benefit over harm
- Value judgments: none
- Role of patient preferences: limited
- Policy level: recommendation

**IMPLEMENTATION CONSIDERATIONS**

The complete guideline is published as a supplement to Otolaryngology–Head and Neck Surgery to facilitate reference and distribution. A full-text version of the guideline will also be accessible free of charge at the www.ent.net.org, the AAO–HNSF website. The AAO–HNSF has also given permission for members of the working group to have their professional medical societies publish all or part of the guideline in their journals or in electronic form. The guideline will be presented to AAO–HNSF members as a miniseminar at the annual meeting after publication. Existing brochures and publications by the AAO–HNSF will be updated to reflect the guideline recommendations.

Anticipated barriers to application of the recommendations in the guideline include: 1) difficulty of changing ingrained clinician habits toward prescribing ineffective systemic therapy for AOE, 2) inability or unwillingness of some clinicians to perform aural toilet or insert a wick into the ear canal, and 3) cost of some topical medications, especially the quinolone products recommended for use with a nonintact tympanic membrane. The first 2 can be addressed with educational events and workshops at continuing medical education events. The issue of cost should become less problematic in the next few years as generic versions of the quinolone otic drops become available.

The impact of the guideline on clinical practice will be assessed for otolaryngologists when a performance measure is developed. As noted above, one purpose of developing the guideline was to facilitate creation of a performance measure for maintenance of certification in otolaryngology–head and neck surgery. The guideline working group did not specifically discuss measuring impact on clinicians other than otolaryngologists.
RESEARCH NEEDS

1. Clinical trials to determine the efficacy of EMLA cream (lidocaine 2.5% and prilocaine 2.5%) and other topical anesthetic solutions for relief of pain caused by AOE
2. Clinical trials to determine the efficacy of topical steroids for relief of pain caused by AOE
3. Observational studies or clinical trials to determine optimal time to discontinue water precautions for AOE
4. Studies to assess the utility of drying the ear canal with a hair dryer or similar device after water exposure to prevent AOE
5. Increased ability to distinguish treatment failure from topical sensitivity when a patient with AOE fails to respond to topical therapy
6. High-quality randomized trials of comparative clinical efficacy for AOE that use an appropriate randomization scheme, explicit double-blind protocol, and fully describe dropouts and withdrawals
7. High-quality randomized trials that assess the benefit of systemic antimicrobial therapy vs topical therapy in patients stratified by severity of signs and symptoms
8. High-quality randomized trials of comparative clinical efficacy for AOE that provide clinical outcomes early in the course of therapy (eg, after 2 to 4 days of therapy) and compare time to symptom resolution in addition to categorical responses (eg, cure, improve, failure) for specific days
9. Comparative clinical trials of “home therapies” for (eg, vinegar, alcohol) vs antimicrobials for treatment of AOE
10. Studies to document the effect of pH on outcomes for topical therapies
11. Define the optimal duration of topical therapy for AOE and the role of patient preferences
12. Additional clinical trials to define the change in outcomes when a steroid is added to a topical antimicrobial
13. Define with greater precision the indications for aural toilet and wick placement
14. Determine the efficacy of aural toilet as an independent factor in treatment of AOE
15. Comparative clinical trials of wick vs no wick in administration of topical therapy
16. Determine the optimal composition and materials for a wick
17. Define the best methods of teaching clinicians, especially those in primary care settings, how to safely and effectively perform aural toilet and wick insertion
18. Determine the optimal method to assess tympanic membrane integrity in patients with AOE (eg, what is the utility of tympanometry?)
19. Assess the correlation between clinical cure and bacteriologic cure in clinical trials
20. Investigate the importance of bacteriologic cure and determine the natural history and clinical significance of bacteriologic failures
21. Assess the role of fungi to determine outcomes
22. Evaluate prevention strategies, including prophylactic use of vinegar with equal parts of isopropyl (rubbing) alcohol after swimming

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