THOMSON
AMERICAN HEALTH CONSULTANTS

Rapid Reference Card Enclosed

For the latest emergency medicine information, visit EMRonline.com.

The Practical Journal for Emergency Physicians

Emergency Medicine Reports®

Volume 25, Number 20 September 20, 2004

Disorders of the ear, nose, and throat are common complaints of patients presenting to the emergency department. Rarely are these problems life-threatening, but the potential for significant morbidity exists if certain illness are not diagnosed and treated in a timely manner. There is a trend toward no treatment for conditions such as otitis media in otherwise healthy older children. This article discusses basic problems of the ear, nose, and throat, and provides an update on the current trends in treatment of pervasive disorders such as otitis. Part I will cover ear and nose disorders, while part II will cover throat disorders.

—The Editor

Common Ear, Nose, and Throat Disorders Encountered in Emergency Practice: Expeditious Evaluation and Definitive Management

Part I: Otitis Media, Sinusitis, and Related Conditions

Authors: **Lisa Freeman, MD, FACEP**, Assistant Professor, Department of Emergency Medicine, University of Texas Medical School at Houston; **Katrin Takenaka, MD,** Assistant Professor, Department of Emergency Medicine, University of Texas Medical School at Houston; and **Brandt Delhamer, MD**, Resident, Department of Emergency Medicine, University of Texas Medical School at Houston.

Peer Reviewers: **Theodore C. Chan, MD**, Associate Professor of Clinical Medicine, Department of Emergency Medicine, University of California, San Diego; and **Ralph Riviello, MD**, Assistant Professor, Department of Emergency Medicine, Thomas Jefferson University, Philadelphia, PA.

Ear

Anatomy. The external ear consists of the pinna (auricle), external auditory canal (EAC), and the lateral tympanic membrane (TM). The middle ear consists of the remainder of the TM, the ossicles, eustachian tube orifice, air cells, facial nerve, and TM plexus. The inner ear is made of the membranous and bony labyrinth (including the cochlear and vestibular organs), and internal auditory canal and its contents.[1]

The sensory innervation of the external ear is complex and varies among individuals. There is significant overlap in this innervation that includes the mandibular branch of the trigeminal nerve; cervical nerve plexus (C2 and C3); auriculotemporal nerve; great auricular nerve; and the facial, glossopharyngeal, and vagus nerves. The middle ear is innervated by the glossopharyngeal nerve as part of the tympanic plexus. The inner ear is innervated by the vestibulocochlear nerve but it does not carry pain fibers, so significant inner ear pathology may develop without otalgia.[1]

Examination of the ear begins with visual inspection of the pinna for erythema, edema, ecchymoses, or warmth. The EAC then is examined by retracting the pinna upward and backward to

straighten the canal and introducing the otoscope with the largest speculum that the EAC will accommodate. (In small children, the pinna should be pulled gently downward.) Cerumen may be removed with a curette or via irrigation. Inspect the TM for color, opacity, and presence of effusion. Test the mobility of the TM with a pneumatic otoscope. Inspect and palpate the mastoid for erythema, edema, and tenderness.

For patients who complain of hearing problems, each ear is tested separately while covering the opposite ear. Ask the patient to repeat phonetically balanced words such as "send," "thick," and "daybreak." The patient should not miss more than one word in 10 with normal hearing. The Rinne and Weber tests also are helpful and are described in the section on hearing loss.

Common ear-related complaints can be divided into three categories: ear pain (otalgia), vertigo, and hearing loss.

Otalgia—Ear Pain. Middle ear infection is the most common cause of primary otalgia.[2] Other common causes are listed in Table 1. Referred pain also is a common cause of ear complaints. These will be discussed in a subsequent section.

Infections of the Ear

Otitis Externa. *Pathophysiology.* Otitis externa (OE) is an inflammation of the EAC. It most often is caused by a bacterial infection, but may have a fungal or noninfectious etiology. The EAC is an excellent medium for bacterial and fungal growth as it is warm, dark, and prone to becoming moist.[3] Moisture and debris easily can become trapped in the canal due to the curve of the EAC, by the presence of variably thick hair, or by excessively thick or viscous cerumen. Normally, cerumen acts as a defense in that it is acidic and hydrophobic, thus helping to protect the EAC from infectious growth and from water retention. Otitis externa develops when the EAC is damaged or the defenses break down. The most common precipitant of infection is excessive moisture that raises the pH and removes cerumen.[3] (*See Table 2.*) Once the cerumen is removed, the keratin debris absorbs water and creates a growth medium for bacteria and fungi. The most commonly implicated bacterial pathogens in otitis externa are *Pseudomonas aeruginosa* and *Staphylococcus aureus*.[4]

Clinical Presentation. The most common presentation of otitis externa is ear pain (otalgia) and drainage from the EAC (otorrhea). The pain can range from minimal discomfort or pruritis to severe pain. The pain is exacerbated by any movement of the ear or direct pressure. The otorrhea from acute bacterial OE typically is white and is variable in amount and consistency. It may be blood-tinged if the infection is chronic. If the OE has a fungal component, it may be black, gray, yellow, or green. Ten percent of cases of OE have fungal overgrowth.[5]

On examination, pain is elicited by pressure or movement of the tragus or of the entire pinna. The EAC is erythematous and edematous. The discharge may be excessive so as to preclude visualization of the tympanic membrane (TM). Avoid using a cerumen curette to remove the debris as the canal is more vulnerable to trauma when inflamed. Cleaning is best accomplished via suction or absorbtion with a cotton-tipped applicator. If the secretions are thick or adherent, it may be softened with hydrogen peroxide. Do not irrigate the EAC unless the tympanic membrane is visualized and is completely intact. Flushing the canal when the TM is disrupted can cause significant cochlear and vestibular damage and disrupt the ossicles. Tinnitus, hearing loss, and vertigo can result.[3] The remainder of the head and neck should be examined for alternate diagnoses and complications of otitis externa.

Treatment. Most cases of OE can be treated successfully with topical agents. *(See Table 3.)* Various antimicrobials, either alone or in combination with other agents, are available to treat OE. The first-line treatment for OE is a topical preparation such as 2% acetic acid or a topical antibiotic containing an aminoglyco-

Emergency Medicine Reports™ (ISSN 0746-2506) is published biweekly by Thomson American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Vice President/Group Publisher: Brenda Mooney
Editorial Group Head: Valerie Loner
Specialty Editor: Shelly Morrow Mark
Marketing Manager: Schandale Kornegay
GST Registration No.: R128870672

Periodicals postage paid at Atlanta, GA. **POSTMASTER:** Send address changes to ***Emergency Medicine Reports***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $31. Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

Multiple copy prices: One to nine additional copies, $359 each; 10 to 20 additional copies, $319 each.

Accreditation

Emergency Medicine Reports'™ continuing education materials are sponsored and supervised by Thomson American Health Consultants. Thomson American Health Consultants designates this continuing education activity for up to 60 hours in Category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

This CME activity was planned and produced in accordance with the ACCME Essentials.

Emergency Medicine Reports™ also is approved by the American College of Emergency Physicians for 60 hours of ACEP Category 1 credit and has been approved for 52 Category 2B credit hours by the American Osteopathic Association. Emergency Medicine Reports has been reviewed by the American Academy of Family Physicians as having educational content acceptable for Prescribed credit hours. This volume has been approved for up to 60 Prescribed credit hours. Term of approval covers issues published within one year from the beginning distribution date of 1/04. Credit may be claimed for one year from the date of this issue.

Thomson American Health Consultants (AHC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

This is an educational publication designed to present scientific information and opinion to health professionals, to stimulate thought, and further investigation. It does not provide advice regarding medical diagnosis or treatment for any individual case. It is not intended for use by the layman. Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. Clinical, legal, tax, and other comments are offered for general guidance only; professional counsel should be sought for specific situations.

This CME activity is intended for emergency physicians. It is in effect for 36 months from the date of the publication.

THOMSON
AMERICAN HEALTH CONSULTANTS

Subscriber Information

Customer Service: 1-800-688-2421

Customer Service E-Mail: customerservice@ahcpub.com
Editorial E-Mail: shelly.mark@thomson.com

World Wide Web page: http://www.ahcpub.com

Subscription Prices

1 year *with* 60 ACEP/60 AMA/60 AAFP Category 1/Prescribed credits (52 AOA Category 2B credits): $544
1 year *without* credit: $399
Resident's rate $199

All prices U.S. only.
U.S. possessions and Canada, add $30 plus applicable GST. Other international orders, add $30.

For Customer Service and CME questions,

Please call our customer service department at **(800) 688-2421.** For editorial questions or comments, please contact **Shelly Morrow Mark,** Specialty Editor, at shelly.mark@thomson.com or (954) 566-9203.

Statement of Financial Disclosure

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Drs. Freeman, Takenaka, Delhamer (authors), Chan, and Riviello (peer reviewers) report no relationships with companies related to the field of study covered by this CME program. Dr. Bosker (editor) is on the speaker's bureau for Pfizer, Sanofi-Synthelabo, Bristol-Myers Squibb, Roche Pharmaceuticals, and Schering Plough Corp. Dr. Bosker also acknowledges that he receives royalties, commissions, and other compensation relating to the sale of textbooks, reprints of articles, and other written materials to the following pharmaceutical companies: Pfizer, Genentech, Aventis, Pharmacia, and Bayer.

This publication does not receive commercial support.

Table 1. Common Causes of Ear Pain[1]

- Neoplasm
- Infectious
  - Otitis media
  - Mastoiditis
  - Dental abscess
  - Epidural abscess
  - Malignant otitis externa
  - Herpes zoster oticus (Ramsay Hunt syndrome)
  - Bell's palsy
  - Cellulitis/perichondritis
  - Chronic myringitis
  - Peritonsilar abscess
  - Pharyngitis
- Inflammatory
  - Otitis externa
  - Bullous myrinigitis
  - Muscle spasm
- Temporomandibular joint syndrome
- Environmental
  - Trauma
  - Temperature extremes
- Tympanic membrane perforation

side, polymyxin, or fluoroquinolone.[4,6,7]

Oral antibiotics are not needed in the vast majority of patients with otitis externa. The overuse of oral antimicrobial therapy is more likely to contribute to antimicrobial resistance than is topical antimicrobial therapy. In a recent study of prescribing patterns, 39% visits for OE resulted in a prescription for topical antibiotics, while 25% visits resulted in a prescription for oral antibiotics.[4]

Oral antibiotics should be used only if there is concomitant otitis media or there is fever or other signs of an invasive infection. Higher risk groups such as children younger than 2 years, diabetics, patients on steroids or those with other forms of immune compromise, or patients with chronic dermatitis should receive oral antibiotics.[3] If the TM is perforated, oral antibiotics should be considered. Quinolones, cephalosporins, and penicillinase-resistant penicillins are reasonable choices. Ofloxacin otic solution (Floxin Otic) is the only topical agent approved by the FDA for use when the TM is perforated.[3]

Complications. Malignant otitis media is a potentially life-threatening extension of OE into the mastoid or skull base that produces osteomyelitis. The most common causative organism is *Pseudomonas aeruginosa* in more than 98% of cases.[5] The elderly, especially diabetics, and immunocompromised patients are at highest risk for developing malignant otitis externa. Malignant OE is being seen more frequently in patients with AIDS with *Aspergillus fumigatus* and Pseudomonas as the responsible organisms.[8,9] Malignant OE should be suspected in a patient with OE who is not improving despite proper topical therapy. Often there is otalgia, otorrhea, and headache out of proportion to clinical findings. On examination, in addition to the typical clinical findings of otitis externa, there is granulation tissue in the floor of the EAC at the bony-cartilaginous junction.[10] The tympanic membrane is almost always intact. Cranial nerve palsies may be present, and this represents advancing infection. Other suppurative complications are rare but may be fatal and include meningitis, brain abscess, and dural sinus thrombosis.[11] Diagnosis of malignant otitis externa is made by consideration of the clinical and computed tomography (CT) or magnetic resonance imaging (MRI) findings. The patients usually are afebrile with a normal white blood cell count, although the ESR usually is elevated.[12] Cultures of the otorrhea should be taken prior to treatment to evaluate for resistant organisms.

Treatment for malignant otitis externa is intravenous antipseudomonal antibiotics, although oral quinolones may be adequate in some patients, and possible surgical debridement. Since the introduction of ciprofloxacin in the late 1980s, many patients in the early stages of pseudomonas malignant OE could be treated as outpatients. However, in recent years, ciprofloxacin-resistant pseudomonas has been emerging.[13] Antibiotics must be continued for 6-8 weeks as indicated for osteomyelitis. Malignant OE with a fungal etiology must be treated with 12 weeks of amphotericin B. There is no role for topical antimicrobials in the treatment of malignant otitis externa.

Fungi may be a primary etiologic agent of OE or present as overgrowth with a concomitant bacterial infection. The most common symptoms are pruritis and ear fullness, although otalgia may be present as well. The otorrhea typically is gray, black, or green. The primary organism is Aspergillus, but Candida is seen as well. Cleansing of the EAC and acidification with acetic acid drops for 5-7 days will affect a cure in most cases. If the infection doesn't resolve, clotrimazole (Lotrimin) 1% solution should be used if the TM is intact or tolnaftate 1% solution (Tinactin) in the case of a perforated TM. Resistant Aspergillus species may require treatment with oral itraconazole (Sporanox).

Noninfectious Causes of Otitis Externa. Primary dermatologic disorders are frequent precipitants of OE. Control of the underlying disease as a whole will reduce manifestations in the canal and represents the cornerstone of treatment.

Prevention. Avoiding known precipitants is key to preventing otitis externa. After swimming, drying drops should be used. For patients with recurrent OE, ear plugs should be used during swimming as well. The EAC should be kept dry during treatment for OE.

Otitis Media. *Epidemiology.* Otitis media (OM) is one of the most commonly diagnosed and most expensive childhood illnesses.[14-17] With the exception of the common cold, OM is the most common disorder for which children and their families seek pediatric care.[18] OM is responsible for more than 30 million clinic visits per year in the United States, with a total cost of more than $5 billion.[15-17] OM appears to be increasing in incidence (or at least being diagnosed with an increasing frequency),[15-17] as there has been a 150% increase in office visits for OM since 1975.[19] OM also is the leading indication for outpatient antimicrobial use in the United States,[20] accounting for 30% of all pediatric outpatient antimicrobial prescriptions.[21] OM is primarily a disease of early childhood, with the peak age-specific attack rate

Table 2. Precipitants of Otitis Externa[76]

EXCESSIVE MOISTURE
- Swimming
- Sweat
- High humidity/high temperature

MECHANICAL CERUMEN REMOVAL/TRAUMA

FOREIGN OBJECT INSERTION
- Cotton swabs
- Fingers
- Ear plugs

CHRONIC DERMATOLOGIC DISEASE
- Eczema
- Psoriasis
- Seborrheic dermatitis
- Acne

Table 3. Topical Antimicrobial Preparations[75]

TOPICAL ANTIMICROBIAL PREPARATIONS

Cipro HC Otic
Ciprofloxacin + hydrocortisone (suspension)
Ciprodex
Ciprofloxacin + dexamethasone
Cortisporin Otic
Polymyxin + neomycin + hydrocortisone (suspension or solution)
Cortisporin TC Otic
Neomycin + thonzonium + colistin + hydrocortisone (suspension)
Pediotic
Polymyxin + neomycin + hydrocortisone
Floxin Otic
Ofloxacin (solution)
Zoto HC
Chloroxylenol + pramoxine + hydrocortisone
Cortane B
(chloroxylenol = antimicrobial pramoxine = topical anesthetic)

TOPICAL PREPARATIONS WITHOUT ANTIMICROBIALS

Domeboro Otic
Acetic acid + aluminum acetate (solution)
Swim Ear
Isopropyl alcohol + anhydrous glycerins (solution)

occurring between 6-18 months of age. Nineteen percent to 62% will have their first episode of OM by age 1 year, and 50% will have experienced three or more episodes by age 3 years.[22,23]

Pathophysiology. Many factors, including genetic, infections, immunologic, and environmental, contribute to a predisposition to ear infections.[14] (*See Table 4.*) Children are at higher risk due to their anatomy. The Eustachian tube is shorter and angled less steeply than in adults, so reflux of organisms from the nasopharynx into the middle ear occurs more readily.[16] When the tube becomes congested, such as with viral upper respiratory infections, the normal tubal function becomes impaired and negative pressure within the middle ear causes secretions to accumulate and leads to the proliferation of pathogenic organisms.[22,24]

The bacterial pathogens most commonly implicated in OM are *Streptococcus pneumoniae*, *Haemophilius influenzae,* and *Moraxella catarrhalis*. These three organisms account for more than 90% of bacterial cases of OM.[25,26] Co-infection with viruses occurs in 30-40% of cases, but fewer than 10% of cases of OM are a result of a primary viral infection.[26,27]

Diagnosis. AOM is defined as the presence of fluid in the middle ear in association with signs or symptoms of acute local or systemic illness. There is no agreement on how to establish the diagnosis of AOM. AOM is estimated to be diagnosed incorrectly in approximately 50% of infants and young children.[28] A recent meta-analysis reviewed available data from several well-designed studies. The authors concluded that the presence of ear pain appears to be the only symptom that may be useful in making the diagnosis of AOM.[24] Symptoms consistent with a viral respiratory tract infection, such as cough and rhinorrhea, may precede the development of OM. Pulling or rubbing the ear is not sensitive nor specific for OM.[29] The absence of fever seems to confer little change to the likelihood of OM.[24]

Physical Examination. The presence of a slightly red TM only is not sufficient to diagnose OM. The findings most suggestive of AOM include a TM that is cloudy, bulging, or immobile. A TM that is strongly or moderately red is suggestive of OM. Normal color or normal mobility makes the probability of AOM less than 5%.[24]

Otitis media with effusion (OME) refers to the presence of fluid in the middle ear in the absence of signs and symptoms of acute infection. The natural history of appropriately treated AOM is for the middle ear effusion to persist for weeks to months. Approximately 70% of children will have fluid in the middle ear at two weeks, 50% at one month, and 10% at three months despite appropriate antibiotic therapy.[30-33]

Treatment. Does OM always need antimicrobial therapy? The development of bacterial resistance, expense, and the potential for side effects have led clinicians to reconsider the need for medical intervention in most cases of OM.[22] A recently published meta-analysis on the natural history of untreated OM that included 63 articles found that AOM symptoms improved within 24 hours without antibiotics in 61% of children (95% CI, 50-72%), rising to 80% by 2-3 days (95% CI, 69-90%). Suppurative complications were comparable if antibiotics were withheld (0.12%) or provided (0.24%).[34] It is common practice in many countries to medicate patients with AOM for pain only, observing the patient closely and reserving the use of antibiotics for refractory cases.[22,35,36] Delayed antibiotic prescribing is being used with increasing frequency in other countries. British researchers divided children between the ages of 6 months and 10 years with otitis media into two groups. One group was given an antibiotic prescription at the time of diagnosis and the other group was given a prescription with instructions to fill it at the parents' discretion after 72 hours if the child had not improved. Immediate antibiotic prescription provided symptomatic benefit mainly after the first 24 hours when symptoms were already resolving. Fewer than one-third of the parents in the delayed antibiotic group used antibiotics, and overall 77%

Table 4. Most Significant Risk Factors for Recurrent OM[22]

- Group daycare (> 6 children)
- Sibling history of recurrent OM
- Early occurrence of initial OM
- Lack of breastfeeding

Table 5. Risk Factors for Pneumococcal Resistance[76,77]

- Low-dose and prolonged treatment with beta-lactam antibiotics
- Recent exposure to antibiotics
- Attendance in large day-care setting
- Young otitis-prone infants
- Winter season

percent of the parents in the delayed antibiotic group were very satisfied. The authors concluded that for children who are otherwise well, a wait-and-see approach seems feasible and acceptable to parents to reduce the use of antibiotics for OM.[35] This strategy should not be employed in children younger than 2 years until further studies are done to document the safety of this approach; however, preliminary European studies suggest this strategy may work for younger children (as young as 6 months of age) as well.[35] The American Academy of Pediatrics and the American Academy of Family Physicians recently released evidence-based practice guidelines on the diagnosis and management of uncomplicated acute otitis media in children between 2 months and 12 years of age, which included a wait-and-see approach. See Table 7 for a summary of these guidelines.

A similar strategy can be employed with upper respiratory tract infections as well. A systematic review of the literature on delayed antibiotic prescribing done in 2003 noted that there was a consistent reduction in antibiotic usage and suggests that delayed prescription is an effective means for reducing antibiotic usage for acute respiratory infections.[37]

There are many antibiotics that are approved to treat AOM.[25] The agents with the greatest activity against the majority of otitic pathogens include high-dose amoxicillin-clavulanate (Augmentin) (up to 90 mg/kg/day) and ceftriaxone (Rocephin).[38-40]

Surveillance studies reveal that the prevalence of penicillin resistance for *S. pneumoniae* is approximately 50%. Of greater concern is the proportion of penicillin-resistant strains that also are resistant to other classes of antibiotics. (*See Table 5.*) Currently, 14% of strains of *S. pneumoniae* also are resistant to three or more classes of antibiotics.[29,41] The agents commonly used for treatment of OM that are most active against penicillin-resistant pneumococcus include high-dose amoxil (80-90 mg/kg/day), cefpodoxime (Vantin), clindamycin (Cleocin), and ceftriaxone.[25]

In 1999, the report issued by the Drug-Resistant *Streptococcus pneumoniae* Therapeutic Work Group concluded that amoxicillin is the most appropriate first-line therapy.[42] Recommended doses were 45 mg/kg/day for children at average risk for bacterial resistance and 80-90 mg/kg/day for children at high risk for resistant pneumococcus.[22] Beta-lactamase producing agents such as *M. catarrhalis* and *H. influenzae* are less likely to be eradicated by penicillin, but infections by these organisms are less severe and tend to resolve uneventfully. Treatment with amoxicillin-clavulanate usually is successful.[22] Second-line agents for suspected infection by a resistant organism include amoxicillin/clavulanate, cefuroxime, and ceftriaxone.[22] (*See Table 6.*)

The optimal duration of therapy is not universally agreed upon but should reflect both the severity of illness and the antibiotic selected. Single-dose therapy is appropriate with ceftriaxone in certain circumstances. A five-day course is reasonable in children older than 2 years.[22]

Bullous Myringitis. Bullous myringitis is a bacterial middle ear infection where blebs or blisters are present on the TM. It occurs alone or in association with otitis externa or otitis media. Middle ear disease is associated with bullous myrinigitis in 97% of patients.[43] It is not a result of Mycoplasma infection as previously thought. The same pathogens that are responsible for OM cause bullous myringitis, and *Streptococcus pneumoniae* is the major pathogen.[43] Treatment is with antimicrobials and pain control.

Complications of Otitis Media. Mastoiditis. Most cases of otitis media have an associated medical mastoiditis, which is an infection of the soft tissue surrounding the air spaces in the mastoid bone. This occurs because the air cell system is connected to the middle ear space. There have been studies indicating an increased incidence of mastoiditis in countries with lower rates of antibiotic use for acute OM.[44] The usual antibiotic therapy for OM is sufficient. Surgical mastoiditis is an osteitis and periosteitis that complicates OM. Surgical mastoiditis presents as edema, erythema, and tenderness over the mastoid bone. The swelling causes the pinna to be displaced anteriorly and inferiorly. Fever and facial weakness or paralysis may be present. CT will confirm the diagnosis. Intravenous antibiotics should be started and urgent otolaryngology consultation obtained because surgical debridement often is needed.[45]

Chronic Suppurative Otitis Media. Chronic suppurative OM is associated with chronic purulent otorrhea with middle ear inflammation in conjunction with a TM perforation. The offending bacteria include mixed flora, including *E.coli*, *S. aureus*, Proteus sp., *Pseudomonas aeruginosa,* and Bacteroides.[45]

The history is notable for variable degrees of hearing loss and vertigo. On examination, there is mucopurulent otorrhea and a defect in the TM. A cholesteatoma may be present. A cholesteatoma refers to the presence of normal squamous epithelium in the middle ear. A sac forms and enlarges to enclose the trapped desquamated cells. This provides a good medium for bacterial growth and can lead to destruction of the bone and ossicles and erosion into the cranial cavity.[45]

Treatment includes removal of the debris from the EAC and instillation of topical antibiotics. The same medications that are used to treat OE are indicated. Otolaryngology referral is indicated.[45]

Ear Foreign Bodies

Living insects account for most of the foreign bodies found in the ears of adults, while children place beads, rocks, food, and

Table 6. Antibiotics Approved to Treat Acute Otitis Media[25]

GENERIC NAME	BRAND NAME	COST PER REGIMEN (20 KG CHILD) IN DOLLARS	
Penicillins		5 days	10 days
• Amoxicillin	Amoxil	5.30	9.90
• Amoxicillin-clavulanate	Augmentin	44.10	77.20
Cephalosporins			
First-generation			
• Cephalexin	Keflex	70.04	140.08
Second-generation			
• Cefaclor	Ceclor	56.38	85.41
• Cefprozil	Cefzil	43.27	86.56
• Cefuroxime	Ceftin	56.81	85.21
• Loracarbef	Lorabid	47.19	87.67
Third-generation			
• Cefdinir	Omicef	34.93	90.37
• Cefixime	Suprax	34.64	69.73
• Cefpodoxime	Vantin	36.18	68.82
• Ceftibuten	Cedax	84.74	110.14
• Ceftriaxone	Rocephin	44.66	133.98 (3 doses)
Macrolide			
• Azithromycin	Zithromax	28.60	N/A
• Clarithromycin	Biaxin	33.20	61.48
Sulfa			
• Erythromycin-Sulfisoxazole	Pediazole	26.38	44.13
• Trimethroprim-Sulfamethoxazole	Bactrim	12.63	25.27

Table 7. AAP and AAFP Otitis Media Recommendations[78]

1. To diagnose AOM, the clinician should confirm a history of acute onset, identify signs of middle-ear effusion, and evaluate for the presence of signs and symptoms of middle-ear inflammation.
2. The management of AOM should include an assessment of pain. If pain is present, the clinician should recommend treatment to reduce pain.
3. a) Observation without the use of antibacterial agents in a child with uncomplicated AOM is an option for selected children based in diagnostic certainty, age, illness severity and assurance of follow-up.

   b) If a decision is made to treat with an antimicrobial agent, the clinician should prescribe amoxicillin for most children. When amoxicillin is used, the dosage should be 80 to 90 mg per kg per day.
4. If the patient fails to respond to the initial management option within 48-72 hours, the clinician must reassess the patient to confirm AOM and exclude other causes of illness. If AOM is confirmed in the patient initially managed with observation, the clinician should begin antimicrobial therapy. If the patient was initially managed with antibacterial agent(s), the clinician should change antibacterial agent(s).
5. Clinicians should encourage the prevention of AOM through reduction of risk factors.
6. No recommendations for complementary and alternative medicine for treatment of AOM are made based on limited and controversial data.

just about any object that will fit into their ears. An adult will complain of ear pain, fullness, or impaired hearing, while a child may not present until otitis externa with a purulent discharge manifests.[46] Suspect tympanic membrane perforation if there is associated tinnitus, bleeding from behind the object, or significant hearing loss.[46] After determining that a foreign body is present in the EAC, a reasonable number of attempts can be made to remove it. Repeated attempts at removal of a difficult object will serve only to traumatize the patient and the EAC, thus making subsequent attempts more difficult due to bleeding, swelling, and an uncooperative patient. Even the most cooperative patient may become difficult after feeling pain during manipulation of the ear canal.[46] Anesthesia of the ear canal is difficult to achieve, and conscious sedation of a pediatric patient may be the better choice if initial attempts at removal are unsuccessful.

The physician should be familiar with several removal techniques and have the proper equipment assembled. Irrigation is the least invasive option and works well for small rocks, dirt, or debris that is lying close to the TM. Do not attempt to irrigate vegetable matter that is deeply imbedded, since this may lead to swelling of the foreign body and complicate its removal.[46]

A suction tip catheter is useful for round objects that are difficult to grasp such as beads. The patient should be warned about the noise the suction will make so as to prevent sudden movements of the head. The Hognose (IQDr, Inc.) is a commercially available device that is used in combination with an otoscope and suction setup. It has a self-molding tip that can attach to objects.[46]

A speculum, alligator forceps, or right angle hook may be used to retrieve an object with edges that can be grasped, but care must be taken not to traumatize the wall of the EAC or further imbed the foreign body. Magnifying loupes often are required for adequate visualization of the object, as it is difficult to manipulate instruments around the otoscope head while it is in the EAC.[46]

Cockroaches are the most commonly found live insect in the ear. The bug must be immobilized or killed prior to its removal. Lidocaine or mineral oil is the best choice to incapacitate the bug. The carcass then can be extracted via grasping, irrigation, or suction.

After any foreign body removal from the EAC, the tympanic membrane should be examined and hearing should be tested. Minor lacerations or excoriations of the canal should heal quickly as long as the EAC is kept clean and dry. Document pre-existing trauma to the EAC or TM. Otolaryngology follow-up is indicated for perforated TM, significant trauma to the EAC, or concomitant infection of the EAC or middle ear.[46] If a foreign object is difficult remove and if no TM perforation or concomitant infection is present, it is reasonable to arrange follow-up with the

Table 8. Usual Hearing Status in Syndromes Associated with Vertigo[45]

HEARING LOSS USUAL	HEARING LOSS UNUSUAL
• Meniere's disease	• Vestibular neuronitis
• Labyrinthitis	• Multiple sclerosis
• Cholesteatoma	• Vertebrobasilar ischemia
• Ototoxicity	• Benign positional vertigo
• Schwannoma	• Basilar migraine

otolaryngologist in his or her office the next day (except for button batteries, which must be removed urgently).

Inner Ear Disorders and Vertigo

An exhaustive discussion of vertigo is outside the scope of this article. The primary task of the emergency physician who examines a patient with vertigo is to determine whether the vertigo is of central or peripheral origin, since some causes of acute vertigo can be life-threatening, such as cerebellar hemorrhage or infarction.[47] Patients with an acute peripheral vestibular lesion usually can stand, although they will lean to the side of the lesion, while patients with vertigo of central origin often are unable to stand without support. Associated neurologic signs such as dysarthria, incoordination, numbness, or weakness suggest a central origin.

Acute Labyrinthitis. Labyrinthitis is an inflammation of the inner ear structures resulting from infection or irritation of the inner ear by passage of toxic products from middle ear infections.[45] The etiology of labyrinthitis may be viral, bacterial, or vascular. Many viruses can cause labyrinthitis but the most common culprit is the mumps virus. Vertigo is uncommon from mumps infection but it may cause acute sensorineural hearing loss. If the hearing loss is unilateral, no specific treatment is required. *(See Table 8.*) Bacterial labyrinthitis may present as a complication of OM. Symptoms include hearing loss, severe vertigo, and nausea/vomiting. Treatment includes intravenous antibiotics and urgent otolaryngology consultation for myringotomy.[45]

Meniere's Disease. Meniere's disease is characterized by vertigo, hearing loss, tinnitus and aural fullness. The symptoms are due to distention of the membranous labyrinth (hydrops). The vertigo may be severe and usually is associated with nausea and vomiting. Episodes of vertigo typically last anywhere from 30 minutes to 12 hours. Decreased hearing is typical and is fluctuating sensorineural in character. Spontaneous remission occurs in 50-60% of patients, so treatment success is difficult to judge. Vestibular suppressants such as meclizine (Antivert) are used to control vertigo. Diazepam (Valium) is useful as well to control anxiety.[45,48]

Vestibular Neuronitis. Vestibular neuronitis is a self-limited condition characterized by vertigo, nausea and vomiting, ataxia, and nystagmus. Hearing is not impaired, but when there are similar symptoms with abnormal hearing, the syndrome is called labyrinthitis. Symptoms last 2-3 days. The cause of vestibular neuronitis is unknown but is thought to be due to a viral infection of the vestibular ganglion.[49]

Table 9. Vestibular Suppressants[49]

DRUG	DOSE	ADVERSE REACTIONS	DRUG CLASS
Meclizine	12.5-50 mg q4-6h	Sedation	Antihistamine Anticholinergic
Lorazepam	0.5 mg bid	Sedation/ Dependency	Benzodiazepine
Clonazepam	0.5 mg bid	Sedation/ Dependency	Benzodiazepine
Scopolamine	1.5 mg patch q3d	Topical allergy	Anticholingeric
Diphenhydramine	50 mg q4-6h	Sedating	Antihistamine Anticholinergic
Diazepam	2-10 mg (one dose)	Sedating	Benzodiazepine

No laboratory testing or imaging is indicated in straightforward cases. Vascular disorders such as labyrinthine artery infarction or vertebrobasilar insufficiency are difficult to exclude without imaging, and their diagnosis is suggested by an identical symptom complex combined with vascular risk factors.[49] The disease is self-limiting, with the most intense symptoms lasting for 2-3 days, but less intense symptoms may persist for 1-2 weeks. Treatment is with vestibular suppressants and antiemetics.[45] Commonly used vestibular suppressants are noted in Table 9.

Referred Otalgia. Dental etiologies are some of the most common causes of referred otalgia, specifically the TMJ syndrome. TMJ syndrome presents as pain and difficulty with chewing or opening/closing of the mouth. It is caused by pathology in the TMJ or adjacent musculature.[1] Otalgia with movement of the mandible is the presenting complaint of up to one-half of patients with TMJ syndrome.[50]

Other causes of referred otalgia include pathology of the nasal cavity such sinusitis, neuralgia, a neoplastic process, or gastroesophageal reflux disease.[1]

Acute Hearing Loss

Hearing loss is a common problem. (*See Table 10.*) It most often presents with a gradual onset and is slowly progressive. Acute, rapidly progressive hearing loss is uncommon. Physical examination, including the use of the Rinne and Weber tests, can classify the problem as conductive or sensorineural in origin. Most cases of chronic hearing loss can be managed as an outpatient, while acute, rapidly progressive symptoms require urgent otolaryngologic consultation.[51]

The Weber test is performed by striking a 512 hz tuning fork and placing it in the midline of the scalp, nose, or teeth. If the hearing loss is conductive, the sound will be heard best in the affected

Table 10. Common Causes of Hearing Loss[51]

CONDUCTIVE	SENSORINEURAL
Complete EAC occlusion by cerumen or foreign body	Autoimmune
Exostoses or osteoma	Meniere's disease
Perforated TM	Trauma
Otitis media with effusion	Viral infection
Cholesteatoma	Vascular insult

ear. If the loss is sensorineural, the sound will be heard best in the normal ear. If the sound is heard in the midline, hearing is normal. The Rinne test is performed by striking a tuning fork and placing it on the mastoid bone. When the patient can no longer hear the sound, place the fork adjacent to the ear canal. If the hearing is normal or there is sensorineural hearing loss, air conduction will be better than bone conduction. If there is conductive hearing loss, the bone conduction will be greater than air conduction.[51] If a tuning fork is not available, a gross evaluation of hearing can be made by whispering or placing a ticking watch near the patient's ear.

Nose Disorders

Epistaxis. *Epidemiology.* Patients who develop epistaxis often do not seek medical attention, making an estimate of its incidence and prevalence difficult; however, it is estimated that epistaxis occurs in 1 in 7 Americans. Mortality from epistaxis is rare, but more often tends to occur in patients who are frail and elderly and often is due to complications from hypovolemia or underlying medical conditions.[52-54]

Pathophysiology and Etiology. The most common etiologies of epistaxis are trauma and rhinosinusitis, but tumors, coagulopathies, hypertension, and atherosclerosis also may be responsible.[53-56] Children are most likely to develop epistaxis from nose-picking, but adults are more likely to have traumatic causes such as blunt force to the face. Also, allergic rhinitis is more likely in the adult populations. Cocaine use, nasal spray abuse, dry ambient air, and exposure to chemical irritants in the workplace also are etiologic factors to be considered.[52,53,57-60] Iatrogenic causes sometimes are seen in the emergency department as complications from indwelling nasogastric tubes or as latent complications from recent nasogastric tubes or nasotracheal intubation.[53]

Epistaxis occurs in two anatomically separate areas of the nose. Anterior epistaxis is responsible for 90% of all nosebleeds, and bleeding originates from Kiesselbach plexus, a network of small vessels on the anterior surface of the nasal septum immediately superior to the nasal vestibule.[53,54] Posterior epistaxis is far less common, accounting for only 5-10% of all nosebleeds and often is associated with the elderly who have hypertension and atherosclerotic disease. These bleeds most often are caused by a tear or rent in a branch of the sphenopalatine artery in the lateral nasopharyngeal wall.[53,54] Anterior epistaxis usually resolves with minimally invasive treatment; posterior epistaxis may continue, leading to hospital admission and possible mortality.[53-56]

Diagnostic Evaluation. Epistaxis is a condition that readily is diagnosed and for which treatment can be initiated very soon after the patient initially is seen, thus blurring the temporal separation between patient evaluation and treatment. However, for the sake of simplicity, the evaluation and management of these patients are discussed sequentially. Patients with anterior epistaxis typically complain of a slow nosebleed that will not stop, prompting them to come to the emergency room. Parents often give a history of nose picking in the child with nosebleeds, but adults who pick their noses may be less forthcoming about this habit. Also, patients may complain of having very itchy nose due to seasonal allergies or have been diagnosed with allergic rhinitis.[52] Patients also may state that they recently were involved in a motor vehicle accident or a physical altercation. Patients with posterior epistaxis are more likely to relate a history of copious bleeding that is running down their throats. Nausea and vomiting from swallowed blood is not uncommon. The patient may relate a history of hypertension for which he or she may or may not be compliant with medications, or the patient may state that he or she has a history of coronary artery disease, prompting consideration of the presence of atherosclerotic changes in the blood vessels.[52] To determine the location and extent of bleeding, the emergency physician must start with gathering historical information about the present illness, including the prior questions and whether the patient has ever had a nosebleed before and what the cause was; whether he or she ever has been diagnosed with a head or neck tumor and whether the patient has received radiation therapy for it; is the patient currently taking anticoagulant medications, aspirin, or nonsteroidal anti-inflammatory drugs (NSAIDs); and does the patient or a family member have a bleeding disorder or coagulopathy.[53-56] Answers to these questions and a good physical exam should inform the emergency physician about the source of the bleeding. Typically, anterior epistaxis presents unilaterally, and the patient will deny nausea from swallowing of blood, while posterior epistaxis often presents bilaterally, and the patient also may exhibit hemoptysis or hematemesis.[52-54] Before examining the patient closely, it is important to avoid exposures by exercising universal precautions and donning appropriate eye protection. To best visualize the dark interior of the nose, the use of an ENT headlamp with focusable beam is recommended, although the gentle use of an otoscope with an ear speculum also has been described.[53-55] Begin the exam of the nose by having the patient gently blow his or her nose to remove local clot and thus clear a path for direct visualization of the bleeding source, if possible. Apply a small amount of vasoconstrictive agent with anesthetic (*see Table 11*) to the end of a cotton swab and apply to the source of bleeding if it can be visualized. If the source cannot be seen, then use a vaporizer or atomizer to apply the vasoconstrictor before attempting to visualize again. By mixing the vasoconstrictor with a topical anesthetic, the patient will be able to better tolerate the application of nasal packing if needed. Make sure that the patient is leaning slightly forward and that the neck is flexed slightly to allow the blood to flow preferentially in an anterior direction, minimizing the trickle of blood posteriorly, which causes gagging. After applying the vasoconstrictor, it sometimes is not pos-

Table 11. Topical Agents Used for Epitaxis[79]

VASOCONSTRICTIVE AGENT	LOCAL ANESTHETIC
Phenylephrine 0.5 to1%	4% lidocaine
Epinephrine 1:1000, 0.25 mL	4% lidocaine, 20 mL
Oxymetolazine 0.05%	4% lidocaine

sible to visualize the bleeding source, since it may have stopped. If this is the case, the source is more likely to be anterior. If it is impossible to see the bleeding source with a headlight or nasal speculum, it may be necessary to use a nasopharyngoscope; this is especially true for posterior bleeding sources. With any patient suffering from persistent, repeated, or severe epistaxis, it is advisable to perform a complete physical exam looking for indications of systemic coagulopathies, and these patients also may warrant coagulation screening to rule these out. However, routine coagulation screening is not recommended for all emergency department patients complaining of epistaxis.[61]

Management. Treatment for epistaxis usually begins before the patient arrives to the emergency department. Indeed, it is the failure of these treatments that prompt the visit in the majority of patients.[53] However, once they arrive the emergency physician should institute noninvasive treatment before even performing a nasal exam by having the patient or assistant apply pressure to the anterior nasal septum with thumb and forefinger just distal to the anterior edge of the nasal bones. This should be continued for at least 10 minutes. If warranted, the patient's airway should be evaluated and secured and any patient in which hypovolemia or impending shock is observed or suspected should be attached to continuous cardiac and oxygen monitors and have at least one large bore intravenous line placed.[53-55] After an attempt has been made to visualize the bleeding source using vasoconstrictive agents, the offending vessel may be cauterized chemically if the bleeding has stopped, or pledgets soaked in the aforementioned vasoconstrictive solutions may be placed in the nose for 10-15 minutes if the bleeding continues. Some practitioners advocate inserting soaked pledgets as soon as the patient's airway, breathing, and circulation (ABCs) are secured and pressure is applied, especially for more heavy bleeding.[53-55] Chemical cautery should be attempted with silver nitrate sticks and only if the bleeding source has been identified and no longer is hemorrhaging as continuous flow will make this technique highly ineffectual. The septum should be cauterized only on one side at a time, as well, to minimize the likelihood of septal necrosis.[53,55,62] If these measures do not control hemorrhage and the bleeding source is thought to be anterior, then anterior nasal packing should be performed. Any technique used for packing the nose should be preceded by the application of local anesthetic agent to the involved nostril, which should accompany the vasoconstrictor application.

There are several commercial devices available to accomplish this, including the nasal tampon, Merocel sponge, and epistaxis balloons, as well as techniques for using ribbon gauze for packing. The nasal tampon is an absorptive material that is first coated with a water-soluble antibiotic ointment and then inserted into the nose and slid up against the nasal septum. The tampon then will expand to fill the nose and provide pressure to the bleeding vessel as blood or saline is absorbed. The Merocel sponge also can be inserted like a nasal tampon and it increases in size the same way. The epistaxis balloon can be inserted in the same fashion, but it becomes pressurized by adding normal saline or sterile water to the balloon interior.

The traditional method of using petroleum jelly-coated (Vaseline) gauze for anterior nasal packing is more time consuming and takes more careful technique than the rather simply inserted commercial devices, and the potential for incorrect insertion is much greater. However, when carefully performed, this technique can provide excellent hemostatic pressure. Using a nasal speculum inserted in the nose, grasp a long strip of half- or three-quarter-inch petroleum jelly-impregnated gauze with a forceps about 4 cm from the end. Making sure that the free end remains outside the nare, slowly insert the gauze along the floor of the nose in a posterior fashion until the gauze cannot go any farther, then grab the gauze with the forceps about 4 cm from the nare and insert the gauze in another section carefully on top of the first. The gauze should be placed as posteriorly as possible with each pass, and the end result should resemble the baffles of an accordion in a back-and-forth pattern, firmly filling the volume of the nasal vestibule.[52-54] Both ends of the ribbon gauze should remain outside the nose.

If the patient complains of a sensation of blood trickling down the posterior oropharynx, or if the source is too far into the nose to visualize, then the bleeding is posterior in location and the patient may need to have a posterior packing applied. Recurrent bleeding can be a sign of posterior epistaxis. A variety of devices are available for this purpose and should be found in any well-equipped emergency department, including the Nasostat balloon and the Storz catheter.[52] Also, a long epistaxis tampon may be used as with anterior epistaxis with the longer dimensions providing posterior hemostasis. After vasoconstricting the nose and applying the anesthetic agents, the device of choice is coated with 4% lidocaine jelly and inserted along the inferior surface of the nasal cavity and advanced until the tip may be seen in the patient's posterior pharynx. If one of the aforementioned commercial devices is used, the posterior chamber then is inflated with sterile water or normal saline until the bladder is firm, usually 4-8 cc, and the physician then pulls the device anteriorly to seat the posterior bladder firmly against the posterior nasal mucosa. Keeping traction on the device to ensure it does not become unseated, the larger anterior chamber then is filled with water, usually 10-25 cc. With both anterior and posterior chambers filled, the device provides pressure to both potential areas of bleeding. If neither of the aforementioned devices is available, then a Foley catheter also may be used to provide posterior hemostatic pressure. First cut the tip distal to the water bladder to avoid stimulating the posterior pharynx. Then lubricate the catheter with 4% lidocaine jelly. Insert the catheter until the tip can be seen in the mouth, like with the commercial devices, then inflate the ballon with the provided syringe and remove the syringe to avoid backflow out from the balloon. Pull the catheter anteriorly to place in the balloon into the posterior nasopharynx.

Since there is no anterior chamber with this technique, the Foley must be held in place with a clamp or hemostat placed across the catheter immediately anterior to the nasal ala while maintaining gentle anterior traction on the catheter. Unlike with the Storz and Nasostat devices, this technique for securing posterior hemostasis must be followed with anterior packing, and care must be taken to ensure minimal pressure on the ala by the clamp by placing gauze between them.[52] For patients with posterior bleeds who do not respond to tamponade packs, otolaryngology consultation should be obtained to consider more aggressive treatment, such as radiographic embolization or artery ligation.

Finally, with both anterior and posterior packing, there is the potential of infection, and the possibility of toxic shock syndrome should be recognized. All patients who receive nasal packing, either anterior or posterior, should be started on an anti-staphylococcal antibiotic, such as cephalexin 250 or 500 mg qid or amoxicillin/clavulanate 250 or 500 mg tid as a recommended antibiotic regimen, with clindamycin 150 or 300 mg qid or trimethoprim-sulfamethoxazole (Bactrim DS) bid reasonable second choices.[52-54] All patients with posterior packing should be admitted because of the risk of airway obstruction, aspiration, and the naso-vagal response.

Complications. Potential complications from epistaxis are similar to complications from any bleeding process, including hypovolemia and shock at the extreme, but these are very unlikely.[55,56] Patients may return to the emergency department or be seen in follow up with fever, rash, and nausea, and they should be suspected of developing staphylococcal toxic shock syndrome. Other complications include sinusitis, septal hematoma and perforation, vasovagal episodes, and mucosal pressure necrosis. Migration of the packing also has been described, especially with posterior packing.[54] Any anterior bleeding that does not stop in the emergency department should warrant an emergent ENT consultation as should every patient with a posterior bleed, as these patients often are admitted for surgical vessel ligation or embolization and observation.[53,54] After discharge, all patients who had packing applied must follow up with an otolaryngologist within 48 hours; good follow-up is essential for these patients to avoid possible pitfalls and complications.

Nasal Foreign Bodies. *Epidemiology.* Nasal foreign bodies are seen primarily in the pediatric population but also frequently are encountered in adults with mental disabilities.[63] Although the exact numbers have not been published recently, it is reasonable for any emergency physician to expect to see a patient with a retained nasal foreign body on a regular basis, and physicians should be adept at removing them without complication.

Pathophysiology. Typical objects include small beads and toys, pieces of food or candy, environmental objects such as rocks, objects found around the home such as paper clips and cat litter, and just about anything else that is small enough to fit into the nose of the patient.[64] If the object remains in the nose long enough, the tissue reaction around it can lead to the deposition of calcium, magnesium, and phosphates, leading to the creation of a rhinolith concretion.[63] Button batteries in the nose represent a true emergency due to the potential for leakage and significant tissue damage.[65]

Evaluation. Parents often will complain of foul-smelling breath emanating from their child or a mucopurulent, fetid nasal discharge that does not resolve over time.[63-68] Discharge from one nostril is classic for foreign body retention as well. Parents also may give a history of actually witnessing the child insert an object into his or her nose.[67] Parents who previously have experienced their child inserting an object into the nose may report that the symptoms are similar.

To examine a child with suspected nasal foreign body, the emergency physician must maintain a non-threatening and non-judgmental demeanor to allow for an exam that is both comprehensive and minimally traumatic for the patient.[63] Halitosis and foul-smelling nasal discharge may be noted. The presence of unilateral vestibulitis, which is an erosion of the skin around the nasal ala, is diagnostic of foreign body.[63] To visualize the foreign body, it often will be necessary to recruit the parent to assist in immobilizing the patient's arms if the child is very young. Often the object can be seen just with the naked eye and ambient light, but other measures may be taken, including shining a penlight into the vestibule or gently inserting an otoscope with ear speculum attached. The most common area of the nose to find a foreign body is the nasal floor immediately below the inferior turbinate, followed by the area just anterior to the middle turbinate. Most foreign bodies—especially vegetable matter—cause nasal mucosa to swell and become inflamed, which can be seen on exam, and this may obscure visualizing the object clearly.[63]

Management. Several methods have been described to remove nasal foreign bodies, including using curved nasal hooks, Magill forceps, alligator clips, towel clips, sponge forceps, magnets, nasal washing, Foley and Fogarty catheters, cyanoacrylate glue, and positive pressure ventilation.[63-70]

Before attempting to remove the object, it is best to reassure the patient and help keep them as calm as possible. An agitated patient not only can make removal difficult, but can lead to the disastrous complication of airway obstruction.[63-68] The patient's parents often are very helpful in keeping the patient calm during attempts to remove the foreign body; however, sometimes conscious sedation may be required. It also may be helpful to instill a nasal decongestant such as 0.5% phenylephrine into the nostril. Lubricating the nose with lidocaine jelly (Xylocaine) also can make removing a foreign body a less tenuous process.[63,71]

For solid objects such as peanuts, beads, or plastic toys it is usually possible to remove them using a curved nasal hook. First, pass the tip of the hook past the object, then turn the tip one quarter turn so that the hook is directly behind the object. Then use gentle traction to pull the object free from the nose. These objects and others that are firm and have irregular edges also can be removed with alligator clips or Magill forceps.[63-66] The removal of a spherical object likely will prove difficult to grasp and initial attempts to remove it should be with a nasal hook.[63]

The Foley and Fogarty catheter techniques may be employed if hooking or grasping the object directly fails or is unlikely to be successful. The Fogarty catheter is likely to yield better results because it is stiffer and more likely to cleanly pass the object upon insertion.[64] The technique is simple: Pass the inflatable bal-

loon past the foreign body, inflate the balloon with water from the provided syringe, and use gentle traction to pull the object free.

Removing metallic objects can be difficult, especially if the foreign body is a button battery and the edge is not freely available to be grasped. In these cases, it may be possible to use a magnet to remove it.[70] A small hobby magnet attached to the end of a cotton swab with cloth tape will suffice, although it is likely that the emergency physician would have to provide this device themselves.

A small dab of cyanoacrylate glue on the tip of a wooden or plastic applicator stick may be used to remove an object that cannot be grasped or hooked easily. Simply hold the end of the applicator flat against the object for 60 seconds and then gently pull the object free.[64]

If the foreign body is too far into the nose so that it cannot be grasped safely, the object has been pushed further posteriorly by attempts to remove it, or it is too fragile to tolerate forceps or hooks without breaking (i.e., bread or a sponge), then the nasal positive pressure technique may be useful. The pressure may be applied using a bag valve mask, wall oxygen, or the parent's or physician's own mouth.[63,64,66,68,71] Regardless of the source, the basic technique remains the same: Administer pressurized air into the mouth while occluding the uninvolved nostril. A variation involves having the patient close his or her mouth, inserting a male-male tubing adapter connected to oxygen tubing and attached to the wall into the uninvolved nostril and turning on the wall flow at 10-15 liters per minute.[68]

Complications and Disposition. Although most nasal foreign bodies are removed in the emergency department without complication,[63-71] it still is prudent to be aware of potential pitfalls. The most worrisome complication is the iatrogenic advancement of the foreign body into the patient's airway, requiring prompt removal using laryngoscopy or the establishment of a surgical airway. Carefully avoiding posterior pressure on the object should minimize this adverse event. Epistaxis from trauma due to attempts to remove the object by inserting an instrument into the nose has been encountered in retrospective studies[63,65,66,68] and usually can be avoided with very gentle technique. Barotrauma is a potential risk with the positive pressure techniques, but this has not been reported widely and, in comparison, physiologic sneezing subjects the nasopharynx to much higher pressures.[68]

More significant are the complications from not removing the foreign body. If left in place, these objects can cause significant morbidity, and any patient who cannot or will not tolerate their removal should be considered for removal under general anesthesia.[63,64,66] Nasal septal perforation and migration of the foreign body both have been described in the literature, as has facial cellulitis, especially with impacted button batteries.[65] To avoid these complications, every patient should be re-examined after the object is thought to have been removed to ensure that there are no retained fragments and that there is not another previously unknown object deep to the first one. For the emergency physician, all nasal foreign bodies need to come out, and no patient should be sent home until it is removed and the patient clinically is at or near his or her physiologic baseline.

Sialadenitis. Sialadenitis refers to inflammation of the salivary gland. This inflammation may be due to several pathogens, including several bacterial and viral agents. Although any salivary gland may develop this condition it is the parotid gland that is most often involved.[62,72-74]

Epidemiology. Although sialadenitis most commonly is seen in the debilitated adult population, it still is seen in the ambulatory emergency department patient with frequency. It is most common in the adult postoperative population; as many as 1 in 1000 postoperative patients develop postsurgical parotitis.

Pathophysiology. Although salivary gland inflammation may be caused by chronic inflammatory conditions, this article focuses on the acute condition. The development of a salivary gland infection may occur in one of three ways: through retrograde migration of bacteria in the excretory ducts of the gland, contiguous spread of a local infection in adjoining tissues, or as a suppurative complication of salivary stasis, and in many patients it is a combination of two or more of these that leads to the condition.[72,74] Bacterial migration and infection spread are self-explanatory, but salivary stasis most often is due to acute ductal obstruction from a salivary gland concretion, called a sialolith, or from dehydration.[74] Conditions that dehydrate patients reduce the flow of saliva through Stenson's duct, allowing bacteria to proliferate unchecked. It is because of this state of decreased salivary flow and relative dehydration that postoperative patients are predisposed to sialadenitis.[72] Sialoliths are more likely to be found in Wharton's duct-with its ostia lateral to the frenulum of the tongue, thanks to the more viscous secretions and the high concentrations of calcium phosphates found in the saliva produced in the submandibular glands, thus leading to obstructive sialadenitis.[72,74] However, the parotid gland is the most likely location to develop acute suppurative sialadenitis.[72] Patients who are immunosuppressed or have chronic medical conditions also are more likely to develop sialadenitis. Drugs that have anticholinergic and diuretic effects also are potential causes or contributors to developing sialadenitis.[62,72,74] In the past the most common cause of bacterial sialadenitis was *Staphylococcus aureus* as it was cultured in 50-90% of all cases.[72] However, recent evidence suggests that the true nature of the infection is polymicrobial, consisting of both aerobes and anaerobes, including *Streptococcus pyogenes*, *Haemophilus influenzae*, *Escherichia coli*, *Klebsiella pneumoniae*, and *Pseudomonas aeruginosa*.[72,73] It should be noted that recent evidence has shown that more than 70% of organisms cultured from these patients exhibit some degree of beta-lactamase or penicillinase activity, which should influence antibiotic selections.[72,73] Patients with mumps, actinomycosis, tuberculosis, syphilis, cytomega-lovirus, and cat-scratch disease also may have sialadenitis due to these agents.[74]

Diagnosis. Patients often complain of an enlarged and painful cheek or mouth, dryness of the mouth (xerostomia), and decreased salivary secretion (aptyalism).[74] Fever may or may not be present. Patients who have had sialadenitis or sialolithiasis in the past may identify their symptoms as the same as last time. Physical exam findings will reveal a tender parotid or submandibular gland, and it may be possible to express purulent

material from Stenson's or Wharton's duct by milking them with digital pressure.[62,72,74] It may be possible also to digitally palpate a sialolith in the involved duct if present.[72] Oral mucosa may appear dry and sticky. A tender, fluctuant salivary gland or excretory duct tract should raise concern for the presence of an abscess. If an abscess is suspected, the pus should be collected through needle aspiration and sent for culture and sensitivities. It is important to emphasize that sialadenitis is a diagnosis made primarily on clinical evidence and that imaging studies are to be reserved for patients who fail to improve with recommended therapy or are suspected of having an abscess. In addition to these indications, imaging of the salivary system with MRI, CT, and ultrasound typically is reserved for patients in preparation for invasive procedures to remove concretions or the gland (in chronic parotitis), and ultrasound guided abscess drainage may be useful if blind aspiration is unsuccessful.

Management. Conservative therapy is the mainstay of treatment for sialadenitis, consisting mainly of promoting salivary flow through adequate hydration, applying warm compresses to the overlying area, and administering sialogogues such as lemon drops in the mouth.[72,74] In addition to these measures, the patient should discontinue medications that can induce dehydration and have anticholinergic effects as aforementioned. As with all patients, underlying medical illnesses should be treated adequately as indicated. If the patient has evidence of bacterial sialadenitis, he or she should maintain excellent oral hygiene with antiseptic mouthwash and be placed on an antibiotic regimen that has specific efficacy against the gram-positive aerobic and anaerobic bacteria mentioned above. Primary antibiotic choices should have beta-lactamase inhibiting properties and include amoxicillin/clavulanate 500 mg tid or cefuroxime 250-500 mg bid; antistaphylococcal penicillins such as dicloxicillin are also useful.[72,73] Anaerobic coverage can be provided by adding clindamycin 450 mg qid or metronidazole 500 mg tid. Patients who fail to respond to conservative treatment after 48 hours are recommended to be admitted to the hospital for intravenous antibiotic therapy with ampicillin/sulbactam or a third-generation cephalosporin and for evaluation of another etiology such as viral agents, actinomycosis, cat-scratch disease, tuberculosis, Sjogren syndrome, lymphoma, and cervical lymphadenitis, with the appropriate treatment recommended as indicated.[62,72] Any patient with the condition of sialadenitis that is complicated by an abscess needs to be seen by an oral surgeon for definitive surgical drainage of the collection.[62,72,74]

All patients should be seen in follow-up 48 hours after initiating therapy to evaluate for response.

Sinusitis. Sinusitis is the common term used to describe the condition and symptoms caused by the inflammation of the mucosa that coats the sinuses of the paranasal anatomy. Rhinosinusitis has become a more prolific term in the medical community due to the associated involvement of the nasal mucosa in the vast majority of cases of sinusitis.[52,57-60] Sinusitis may be acute or chronic and may be due to viral agents, bacterial agents, or some combination of both.[52,57-60]

Epidemiology. In the United States adult population, sinusitis is one of the 10 most common diagnoses made by all medical practitioners and the fifth most common diagnosis for which an antibiotic is prescribed.[57-59] One in six adults are diagnosed with sinusitis every year, and in the United States sinusitis is responsible for more than $6 billion in direct medical costs. Adults in the Midwest and South are particularly more likely to have the diagnosis of sinusitis made, presumably because of the higher prevelance of environmental allergies in the region.[57]

Pathophysiology. The nasal and paranasal anatomies consist of mucosal and ciliated epithelium that lines the spaces in the facial bones and nose and include the maxillary, ethmoid, sphenoid, and frontal sinuses. These sinuses connect to the nasal cavity through small ostia that drains into the nares via openings beneath the superior and middle turbinates; specifically, the sphenoid sinus drain into the superior meatus, with the posterior ethmoid sinus below the superior turbinate, while the anterior ethmoid, frontal, and maxillary sinuses all share a common drain into the middle meatus as a structure known as the osteomeatal complex.[52] Mucus production and unidirectional ciliated transport of debris and contaminants in the sinuses normally prevent the accumulation of bacteria in these normally sterile areas. Factors that inhibit either of these physiologic mechanisms from working properly may lead to blockage of sinus drainage by either reducing the caliber of the ostia or obstructing it entirely, thus leading to the stagnant environment required for bacterial growth and proliferation.[57] The invasion of sinus mucosa by viral agents associated with upper respiratory tract infections thus leading to inflammation appears to be the most common precipitating cause of acute sinusitis, while allergic rhinitis has a larger role in the pathophysiology of chronic sinusitis.[57] Other conditions that lead to pro-sinusitis changes include ciliary dyskinesia, cystic fibrosis, inhalation of irritants, immunodeficiency, nasal polyps, deviated nasal septa, rhinolithiasis, trauma, and odontogenic infection—specifically the first, second, and third molars.[52,57,58] Upper respiratory viruses, including respiratory synsyctial viruses, influenza A, and parainfluenza, often are isolated from nasal cultures in up to 40% of cases.[57] There are three primary bacterial pathogens that are responsible for bacterial sinusitis: *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*, with the majority of nasal culture isolates made up of *S. pneumoniae* and *H. influenzae*. Cystic fibrosis patients with sinusitis have a greater likelihood of *Pseudomonas aeruginosa* presence in their sinuses.[52]

Diagnostic Evaluation. The diagnosis of sinusitis, either acute or chronic, most accurately is made when the overall clinical impression is evaluated, since no single indicator has been shown to reliably predict who will have confirmed disease by needle aspiration of the sinuses, the current gold standard of diagnosis.[57] Most patients will complain of a persistent upper respiratory tract infection that has lasted more than seven days. Purulent nasal discharge and sinus pressure or discomfort are complaints that should alert the emergency physician to the possibility of sinusitis, as are fever and headache.[52,57] Sinusitis-like symptoms that initially improve but then recur—known as biphasic illness—have been shown to be suggestive of bacterial sinusitis.[58] Although more common in the pediatric population,

halitosis occasionally is a related complaint in the history of present illness.[57,58] Physical exam may reveal fever, sinus tenderness, purulent nasal secretions, "allergic shiners," nasal edema on speculum exam, and posterior nasal drip in the oropharyngeal cavity.[52,57-60] Previous attempts have been made to assemble a list of signs, symptoms, and historical elements that could be applied to an accurate diagnostic algorithm and for determining who would best benefit from antibiotics, but current standard practice maintains that the clinician's overall impression remains the best tool for diagnosing sinusitis.[57-59] However, the diagnosis of acute bacterial sinusitis should be reserved for patients who have more severe symptoms regardless of duration or patients with symptoms lasting 7-10 days who also have maxillary tenderness in the face or teeth and purulent nasal secretions, especially if unilateral, as well as immunocompromised patients.[57,60]

Although there are numerous methods for imaging the paranasal sinuses, including CT, sinus radiography, and ultrasound, their usefulness is highly variable in practice and they have not been shown to have sufficient sensitivity, specificity, or cost effectiveness to justify their routine use in the emergency department.[57,58,60] Specifically, the mucosal inflammatory changes associated with sinusitis that are seen on imaging studies also are present in many patients with the common cold.[58] The absence of complete sinus opacification, air-fluid levels, and mucosal thickening on CT has been shown to have an estimated sensitivity of 90%, which may allow the emergency physician to use this negative test to help rule out bacterial sinusitis.[60] Otherwise, sinus radiography has limited value in the diagnosis of acute sinusitis.[57,58,60]

Management. Current debate about the management of sinusitis revolves around the usefulness, effectiveness, and consequences of treating the patient with suspected sinusitis with antibiotics vs. conservative treatment alone.[52,57-60] The increasingly prevalent patterns of antibiotic resistance are making the haphazard use of antibiotics more dangerous, and there has been a concerted effort by medical academics and practitioners to limit their use to cases where there is a definite indication lest they become even less effective. Indeed, several recent studies have shown that most cases of acute sinusitis diagnosed in the ambulatory setting are caused by upper respiratory tract infections due to viral agents, for which antibiotic medications are useless.[60] Therefore, only those patients who have a high probability of bacterial infection, including severe symptoms of any duration or symptoms of more than 7-10 days duration (especially unilateral) and a biphasic symptom pattern or immunocompromised patients, should be considered for instituting antibiotic therapy as an adjuvant to conservative treatments outlined below.[57] Although exceptions always exist, a patient with sinusitis symptoms of fewer than seven days duration is unlikely to have a bacterial cause and will benefit most from conservative treatment.[57,60] For all patients with symptoms of acute sinusitis, the relief of symptoms should be the directing goal of therapy and management should consist of steam inhalation to loosen nasal secretions, applying warm facial packs to the sinus areas for 10-15 minutes tid to qid to promote sinus drainage, consuming appropriate volumes of non-caffeinated liquids, avoiding cigarette smoke, applying saline nasal drops, using oral decongestants, sleeping with the head of the bed elevated to promote sinus drainage at night, and getting adequate rest.[57] If antibiotics are deemed necessary, then current recommendations state that first-line therapy should consist of amoxicillin, doxycycline, or trimethoprim-sulfamethoxazole, as these are the more narrow spectrum agents against the three most likely pathogens.[57,60] In most centers, amoxicillin is the most commonly prescribed agent as it has a superior coverage profile compared to other agents and resistance in *S. pneumoniae* still is fairly low.[57,59,60] Although some emergency physicians use newer and more expensive drugs to treat presumed bacterial sinusitis, such as oral cephalosporins, amoxicillin-clavulanate, or clindamycin, these agents have been shown to be no more effective than the aforementioned first-line agents and are significantly more expensive.[59] Thus, the use of alternative agents such as amoxicillin-clavulanate, cephalosporins, or clindamycin should be reserved for the patient who has failed primary treatment with amoxicillin, doxycycline, or trimethoprim-sulfamethoxazole.[57,59,60]

Complications. Potential suppurative complications from bacterial sinusitis include preseptal and orbital cellulitis, epidural abscess, meningitis, brain abscess, cavernous sinus thrombosis, subdural abscess, and, if present, should prompt prompt treatment and surgical consultation as needed.[52,58] Clearly septic patients or those patients with evidence of systemic infection should be admitted. Hospital admission should be considered in the patient with suspected bacterial sinusitis who has potential impediments to appropriate self care, does not have follow-up available, or cannot tolerate oral antibiotics. In the vast majority of emergency department-diagnosed sinusitis, the condition is uncomplicated and patients may be safely discharged home.

References

1. Shah RK, Blevins NH. Otalgia. *Otolaryn Clin North Am* 2003;36: 1137-1151.
2. Stewart MH, Siff JE, Cydulka RK. Evaluation of the patient with sore throat, earache and sinusitis: An evidence-based approach. *Emerg Med Clin North Am* 1999;17:153-178.
3. Sander R. Otitis externa: A practical guide to treatment and prevention. *Am Fam Phys* 2001;63:927-936. 941-942.
4. McCoy SI, Zell ER, Besser RE. Antimicrobial prescribing for otitis externa in children. *Pediatr Infect Dis J* 2004;23:181-183.
5. Boustred N. Practical guide to otitis externa. *Aust Fam Phys* 1999;28:217-221.
6. Dohar JE. Evolution of management approaches for otitis externa. *Pediatr Inf Dis J* 2003;22:299-305.
7. Miser WF. Otitis media and externa. In: Taylor RB, David AK, Fields SA, et al., eds. *Family Medicine: Principles and Practice*. 6th ed NY: Springer-Verlag, 2003;604-606.
8. Ress BD, Luntz M, Telischi FF, et al. Necrotizing external otitis in patients with AIDS. *Larynoscope* 1997;107:456-460.
9. Munoz A, Martinez-Chamorro E. Necrotizing external otitis caused by *Aspergillus fumigatus*: CT and high resolution MRI in an AIDS patient.*J Laryngol Otol* 1998;112:98-102.
10. Grandis JR, Branstetter BF, Yu VL. The changing face of malignant (necrotizing) external otitis: Clinical, radiological and anatomic cor-

relations. *Lancet Inf Dis* 2004;4:34-39.
11. Schwarz GA, Blumenkrantz MJ, Sundmaker WL. Neurologic complication of malignant external otitis. *Neurology* 1971;21: 1077-1084.
12. Rubin J, Stroeher G, Yu VL, et al. Efficacy of oral ciprofloxacin and rifampin for treatment of malignant otitis externa. *Arch Otolaryngol Head Neck Surg* 1989;115:1063-1069.
13. Berenholz L, Katzenell U, Harrell M. Evolving resistant Pseudomonas to Ciprofloxacin in malignant otitis externa. *Laryngoscope* 2002;112:1619-1622.
14. Bluestein C, Klein J. *Otitis Media in Infants and Children*, 3rd ed. New York, NY:WB Sauders 2001.
15. Rosenfield R, Bluestone C. *Evidence-Based Otitis Media*. St. Louis, Mo. BC Decker Inc; 1999.
16. Bluestone C, Klein J. Otitis Media in Infants and Children, 3rd ed. New York, NY: WB Saunders; 2001.
17. Steinbach WJ, Sectish TC. Pediatric resident training on the diagnosis and treatment of acute otitis media. *Pediatrics* 2002;109: 404-408.
18. Fried VM, Makuc DM, Rooks RN. Ambulatory health care visits by children: prinicipal diagnosis and place of visit. *Vital Health Stat 10* 1998;13:1-23.
19. Shappert SM. Office visits for otitis media: United States 1975-1990. Advance Data from Vital and Health Statistics, No 214, Hyattsville, MD. National Center for Health Statistics; 1992.
20. Dowell SF, Marcy SM, Phillips WR, et al. Otitis media-principles of judicious use of antimicrobial agents. *Pediatrics* 1998;101: 165-171.
21. Nyquist A-C, Gonzales R, Steiner JF, et al. Antibiotic prescribing for children with colds, upper respiratory tract infections and bronchiolitis. *JAMA* 1998;279:875-877.
22. Darrow DH, Dash N, Derkay CS. Otitis medial concepts and controversies. *Curr Opin Otolaryngol* 2003;11:416-423.
23. Casselbrandt ML, Mandel EM. Epidemiology. In: Rosenfeld RM, Bluestone CD, eds. *Evidence-Based Otitis Media*. Hamilton, Ontario: BC Decker; 1999:117-136.
24. Rothman R, Owens T, Simel DL. Does this child have otitis media? *JAMA* 2003;290:1633-1640.
25. McCracken GH. Diagnosis and management of acute otitis media in the urgent care setting. *Ann Emerg Med* 2002;39:413-421.
26. Ruuskanen O, Heikkinen T. Otitis media: etiology and diagnosis. *Pediatr Infect Dis J* 1994;13(1 Suppl 1)S:23-26.
27. Pichichero ME. Acute otitis media, II. Treatment in an era of increasing antibiotic resistance. *Am Fam Physician* 2000;61: 2410-2416.
28. Pichichero ME, Pichichero CL. Persistent acute otitis media: I. causative pathogens. *Pediatr Infect Dis* J 1995;14:178-183.
29. Darrow DH, Dash N, Derkay CS. Otitis media: concepts and controversies. *Curr Opin Otolaryn* 2003;11:416-423.
30. Dowell SF, Marcy M, Phillips WR, et al. Otitis media-prinicples of judicious use of antimicrobial agents. *Pediatrics* 1998;101:165-171.
31. Klein JO. Otitis media. *Clin Infect Dis* 1994;19:822-833.
32. Wald ER. Otitis media and sinusitis: A clinical update. *Clin Updates Pediatr Infect Dis* 1995;1:1-4.
33. Teele DW, Klein JO, Rosner BA. Epidemiology of otitis media in children. *Ann Otol Rhinol Laryngol* 1980;89 (suppl):5-6.
34. Rosenfeld RM, Kay D. Natural history of untreated otitis media. *Laryngoscope* 2003;113:1645-1657.
35. Little P, Gould C, Williamson I, et al. Pragmatic randomized controlled trial of two prescribing strategies for childhood acute otitis media. *BMJ* 2001: 322(7282):336-342.
36. Chan LS, Takata GS, Shekelle P, et al. Evidence assessment of management of acute otitis media, II: Research gaps and priorities for future research. *Pediatrics* 2001;108:248-24.
37. Arroll B, Kenealy T, Kerse N. Do delayed prescriptions reduce antibiotic usage in respiratory tract infections? A systematic review. *Br J Gen Pract* 2003;53(496):871-877.
38. Jacobs MR, Bajaksouzian S, Lin G, et al. Susceptibility of *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Moraxela catarrhalis* to oral agents: results of a 1998 US outpatient surveillance study. Presented at: 39th Interscience Conference on Antimicrobial Agents and Chemotherapy; Sept 26-29, 1999; San Fransisco, CA.
39. Blumer JL. Fundamental basis for rational therapeutics in acute otitis media. *Pediatr Infect Dis J* 1999;18:1130-1140.
40. Gudnason T, Gudbrandsson F, Barsanti F, et al. Penetration of ceftriaxone into the middle ear fluid of children. *Pediatr infect Dis J* 1998;17:258-260.
41. Whitney CG, Farley MM, Hadler J, et al. Increasing prevalence of multi-drug resistant Streptococcus pneumoniae in the United States. *N Engl J Med* 2000;343:1917-1924.
42. Dowell SF, Butler JC, Giebink GS, et al. Acute otitis media: Management and surveillance in an era of pneumococcal resistance—a report from the Drug-Resistant *Streptococcus pneumoniae* Therapeutic Working Group. *Pediatr Infect Dis J* 1999;18:1-9.
43. Palmu AA. Bacterial etiology of acute myrinigitis in children less than two years of age. *Pediatr Infec Dis J* 2001;20:607-611.
44. Van Zuijlen DA, Schilder AG, Van Balen FA, et al. National differences in incidence of acute mastoiditis: Relationship to prescribing patterns of antibiotics for acute otitis media? *Pediatr Inf Dis J* 2001;20:140-144.
45. Noble J, Green HL, Levinson W, et al, eds. *Textbook of Primary Care Medicine*, 3rd ed. St. Louis: Mosby; 2001.
46. Roberts JR, Hedges JR, eds. *Clinical Procedures in Emergency Medicine*, 4th ed. Elsevier, 2004.
47. Baloh RW. Vestibular neuronitis. *N Engl J Med* 2003;348: 1027-1032.
48. Paparella MM, Djalilian HR. Etiology, pathophysiology and pathogenesis of Meniere's disease. *Otolaryn Clin North Am* 2002;35:529-545.
49. Hain TG, Micco AG. Vestibular neuronitis. In: Goetz CG, ed. *Textbook of Clinical Neurology*, 2nd ed. Elsevier: Philadelphia 2003; 208-209
50. Hodges JM. Managing temorpormandibular joint syndrome. *Laryngoscope* 1990;100:60-66.
51. Isaacson JE, Vora NM. Differential diagnosis and treatment of hearing loss. *Amer Acad Fam Prac* 2003;15:68:1125-1132.
52. Waters TA, Peacock PE. Nasal emergencies and sinusitis. In: Tintinalli JA, et al, eds. *Emergency Medicine: A comprehensive study guide*. 5th ed. New York, NY: McGraw-Hill; 2000: 1532-1538.

53. Gluckman W. Epistaxis. *eMedicine* online medical journal. 2004 Jan 23: http://www.emedicine.com/ped/topic1618.htm.
54. Herkner H, Havel C, Mullner M, et al. Active epistaxis at ED presentation is associated with arterial hypertension. *Am J Emerg Med* 2002;20:92-95.
55. Evans AS, Young D, Adamson R. Is the nasal tampon a suitable treatment for epistaxis in accident and emergency? A comparison of outcomes for ENT and A&E packed patients. *J Laryngol Otol* 2004;118:12-14.
56. Viducich RA, Blanda MP, Gerson LW. Posterior epistaxis: Clinical features and acute complications. *Ann Emerg Med* 1995;25: 592-596.
57. Leggett JE. Acute sinusitis. When and when not to prescribe antibiotics. *Postgrad Med J* 2004 Jan;115:13-9.
58. Stewart MH, Siff JE, Cydulka RK. Evaluation of the patient with sore throat, earache and sinusitis: An evidence based approach. *Emerg Med Clin North Am* 1999;17:153-187.
59. Piccirillo JF, Mager DE, Frisse ME, et al. Impact of first-line vs second-line antibiotics for the treatment of acute uncomplicated sinusitis. *JAMA* 2001;286:1849-1856.
60. Gonzales R, Bartlett JG, Besser RE, et al. Principles of appropriate antibiotic use for treatment of acute respiratory tract infections in adults: Background, specific aims, and methods. *Ann Intern Med* 2001;134:479-486.
61. MA, Nilssen EL, Holland Thaha S, et al. Routine coagulation screening in the management of emergency admission for epistaxis-is it necessary? *J Laryngol Otol* 2000;114:38-40.
62. Peacock WF. Face and jaw emergencies. In: Tintinalli JA, et al, eds. *Emergency Medicine: A comprehensive study guide*, 5th ed. New York, NY: McGraw-Hill; 1530-1531.
63. Kalan A, Tariq M. Foreign bodies in the nasal cavities: A comprehensive review of the aetiology, diagnostic pointers, and therapeutic measures. *Postgrad Med J* 2000;76:484-487.
64. Cox RJ. Foreign bodies, nose. *eMedicine* online medical journal. 2001 May 17: http://www.emedicine.com/emerg/topic186.htm.
65. Loh WS, Leong Jl, Tan HK. Hazardous foreign bodies: Complications and management of button batteries in the nose. *Ann Otol Rhinol Laryngol* 2003;112:379-383.
66. Kadish HA, Corneli HM. Removal of nasal foreign bodies in the pediatric population. *Am J Emerg Med* 1997;15:54-56.
67. Francois M, Hamrioui R, Narcy P. Nasal foreign bodies in children. *Eur Arch Otorhinolaryngol* 1998;255:132-134.
68. Navitsky RC, Beamsley A, McLaughlin S. Nasal positive-pressure technique for nasal foreign body removal in children. *Am J Emerg Med* 2002;20:103-104.
69. Lichenstein R, Giuduce EL. Nasal wash technique for nasal foreign body removal. *Pediatr Emerg Care* 2000;16:59-60.
70. Douglas SA, Mirza S, Stafford FW. Magnetic removal of a nasal foreign body. *Int J Pediatr Otorhinolaryngol* 2002;62:165-167.
71. Sorrels WF. Simple noninvasive effective method for removal of nasal foreign bodies in infants and children. *Clin Pediatr* 2002; 41:133.
72. McQuone SJ. Acue viral and bacterial infections of the salivary glands. *Otolaryngol Clin North Am* 1999;32:793-811.
73. Brook I. Aerobic and anaerobic microbiology of suppurative sialadenitis. *J Med Microbiol* 2002;51:526-529.
74. Bhatty MA, Piggott TA, Soames JV, et al. Chronic non-specific parotid sialadenitis. *Br J Plast Surg* 1998;51:517-521.
75. [No authors listed.] *Tarascon Pocket Pharmacopoeia*. Lompoc, CA: Tarascon Publishing; 2004.
76. Uhari M, Mantysaari K, Meimala M. A meta-analytic review of the risk factors of acute otitis media. *Clin Inf Dis* 1996;22:1079-1083.
77. Sutton DV, Derkay CS, Darrow DH, et al. Resistant bacteria in middle ear fluid at the time of tympanostomy tube surgery. *Ann Otol Rhinol Laryngol* 2000;109:24-29.
78. Neff MJ. AAP, AAFP Release guidelines on diagnosis and management of acute otitis media. *Am Fam Phys* 2004;6:2713-2715.
79. Tintinalli JA, et al, eds. *Emergency Medicine: A comprehensive study guide*, 5th ed. New York, NY: McGraw-Hill.

Physician CME Questions

61. What is the most common bleeding source of anterior epistaxis?
    A. Sphenopalatine artery
    B. Kiesselbach's plexus
    C. Anterior ethmoid artery
    D. Olfactory veins

62. Recent evidence suggests that which of the following is the most common bacterial agent found in acute sialadenitis?
    A. *Staphylococcus aureus*
    B. *Klebsiella pneumoniae*

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

C. *Borrelia burgdorferi*
D. Multiple aerobic and anaerobic species

63. Which of the following techniques is recommended for removing fragile nasal foreign bodies, such as bread?
A. Magill forceps
B. Nasal washing
C. Storz catheter
D. Positive pressure

64. What is the most common bleeding source in posterior epistaxis?
A. Sphenopalatine artery
B. Kiesselbach's plexus
C. Anterior ethmoid artery
D. Posterior ethmoid artery

65. Which of the following is the most common initiating event that precedes acute sinusitis?
A. Trauma to the face
B. Exposure to someone with sinusitis symptoms
C. Viral upper respiratory infection
D. Smoke inhalation

66. Which of the following is a risk factor for pneumococcal resistance in otitis media?
A. Attendance in a large day-care setting
B. Recent exposure to antibiotics
C. Winter season
D. Low dose and prolonged treatment with beta-lactam antibiotics
E. All of the above

67. Which of the following syndromes is characterized by hearing loss and vertigo?
A. Multiple sclerosis
B. Basilar migraine
C. Meniere's disease
D. Vestibular neuronitis
E. Vertebrobasilar ischemia

68. Most cases of otitis externa can be treated with topical agents.
A. True
B. False

69. All of the following are vestibular suppressants *except*:
A. lorazepam
B. scopolamine
C. diphenhydramine.
D. pseudoephedrine.

70. Sensorial hearing loss may be associated with all of the following *except*:
A. viral infection.
B. trauma.
C. cholesteatoma.
D. autoimmune disease.

Emergency Medicine Reports
CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

In Future Issues:

Ear, Nose, and Throat Disorders, Part II

CME Answers

61. B
62. D
63. D
64. A
65. C
66. E
67. C
68. A
69. D
70. C