In even the most experienced hands, an adequate physical examination of the ears can be difficult to perform because of common problems such as cerumen blockage of the auditory canal, an uncooperative toddler or an exasperated parent.

The most common cause for a running ear in a child is acute purulent otitis media (OM) (persistent acute OM and recurrent acute OM). Fig. 1 gives the classification of OM.

Over-diagnosing acute OM, selecting inappropriate empiric therapy, or both, leads to overuse and misuse of antibiotics and causes increased drug resistance.

**The most common cause for a running ear in a child is acute purulent otitis media (OM).**

This article reviews purulent OM and discusses the diagnosis, the differential diagnoses and the current treatment recommendations proposed by the drug-resistant Streptococcus pneumoniae (DRSP) working group.1

**PURULENT OTITIS MEDIA**

Purulent OM is characterised by purulent fluid bathing the middle ear and is found in patients with acute OM (persistent acute OM and recurrent acute OM) and in patients with chronic suppurative OM.

**Definitions2**

Acute OM is an acute bacterial infection of the middle-ear cleft.

- Persistent acute OM is the persistence of symptoms and signs of middle-ear infection following 1 or 2 courses of antibiotic therapy.
- Recurrent acute OM is most commonly defined as 3 or more separate episodes of acute OM in a 6-month time span or 4 or more episodes in a 12-month time span.

**Epidemiology**

The peak incidence of acute OM is in children between 6 and 12 months of age.

**Pathogenesis2**

In the early stages of AOM there is erythema and oedema of the middle ear mucosa and tympanic membrane. As the infection progresses, purulent exudate accumulates in the middle ear and the tympanic membrane appears opaque, full or bulging with diminished mobility. The membrane may rupture, allowing the purulent effusion to drain causing a running ear.
Multiple, interrelated factors contribute to the development of acute OM, including the following:

- **Infection.** Acute OM often follows viral upper respiratory tract infection (URI), mostly in winter. The main bacterial causes of acute OM are *S. pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis* (90% β-lactamase producing), group A-B streptococci, *Staphylococcus aureus* and *Pseudomonas aeruginosa*. The bacterial flora isolated in chronic, suppurative OM is a mixture of aerobes and anaerobes. The common bacteria are *P. aeruginosa* (aerobe) and *Peptostreptococcus* sp. (anaerobe). Recent literature concludes that the role of adenoid tissue in the pathogenesis of OM is primarily as a focus of infection.

- **Dysfunction of the Eustachian tube.**

- **Allergy** has long been recognised as one of the causative factors of OM with effusion due to nasotubal mucosal congestion and obstruction of the Eustachian tube.

- **Immunological factors.** Recurrent acute OM is more prevalent among young children with an impaired ability to manage antigen exposure, e.g. IgA/IgG deficiencies, diabetes lymphoma, etc.

- **Barotrauma.** Barometric pressure changes can produce OM even in the presence of a normal Eustachian tube.

- **Nutritional deficiencies,** e.g. iron deficiency, protein/albumin abnormalities.

- **Inflammatory mediators.** Each of the causative factors mentioned stimulates the release of inflammatory mediators which increase vascular permeability and secretory activity of the middle ear.

**Other risk factors**¹
- An episode of acute OM during the first 6 months of life is a risk factor for recurrent OM.
- Siblings with a history of recurrent OM.
- Parental smoking is significantly (*p < 0.5*) associated with more than 1 episode of acute OM during the first year of life.
- A child born in autumn has a higher risk for recurrent OM.
- Children in day-care centres are at a higher risk.
- Breast-feeding less than 3 months.

Other conditions which may mimic acute purulent otitis media should be considered when evaluating a patient with a running ear. These are listed in Table I. To outrun the running ear all these facts should be kept in mind when evaluating a patient.

**HISTORY**
Some important questions to ask are:
- **Family history**
  - cystic fibrosis
  - allergies — nasal, chest and foods.
- **Environment**
  - Crèche — clean, low population, smoke free?
  - Smoke — at home, in car or at crèche?
  - Swimming and diving — nose clip or earplugs?
  - Industrial effluent, steel plant or cement factory?
- **Diet**¹²
  - Excessive quantities of milk, yellow cheese and chocolates?
  - Ingestion of poisons such as cheese puffs, multicolour sugar-coated chocolates, cold drink preparations and ice lollies?
  - Daily vitamin supplements?
  - Presence of iron deficiency?
  - Specific symptoms and signs of allergy
    - Allergic salute
    - Nasal crease
    - Blue shiners
    - Denne’s lines
  - Mouth breathing
  - Pale boggy inferior turbinates — eczema, allergic conjunctivitis, asthma.
  - Habits/holidays
    - Snoring?
    - Use of matchsticks, hairgrips, cotton buds to clean ears?
    - Water in ears: bath, shower, and swimming?
    - Holidays at the sea or at resorts with hot/warm water pools?
  - Other recurrent infections
    - Rhinosinusitis
    - Tonsillitis
    - Pharyngitis.
  - **Gastro-oesophageal reflux.**
  - **Teething.**

**EXAMINATION**

**Otoscopy**
Failing to remove earwax or debris, using an otoscope that provides inadequate light, or using an inappropriately sized speculum will impair visualisation of the tympanic membrane. It has also been shown that crying and screaming causes the tympanic membrane to become red. All these factors lead to over-diagnosis of OM.

Otorrhoea is a common physical finding of middle-ear infection, with drainage of pus through a non-intact tympanic membrane (through a perforation, ventilation tube, or both) or of outer ear infection. The discharge may be purulent, mucoid, cheesy, serous or serosanguinous. Clear otorrhoea is much less common and suggests a cerebrospinal fluid leak. Discharge coming from the middle ear always has a mucoid component.

Granulation tissue may surround a ventilation tube or perforation or may involve most of the membrane. There may be copious necrotic debris or changes of the skin in the external acoustic canal (otitis externa) related to the constant drainage. There may be tenderness on movement of the pinna,
intermittent discomfort or a dull earache. Small children and other patients who may have difficulty expressing themselves verbally may be irritable or ‘out of sorts’. People may complain of hearing loss or ‘squishy’ sounds in the ears or may note a foul odour coming from the ear. Finally, foreign bodies are a common cause of chronic otorrhoea in children, and should always be suspected.

**Nasal endoscopy**
Look for any pathology which interferes with the airflow through the nose and therefore interferes with the Eustachian tube function:
- septal deviation
- polyps
- foreign bodies
- chronic inflammation, e.g. allergies
- mucociliary abnormalities.

**Nasopharyngoscopy**
Exclusion of any pathology involving the Eustachian tube is mandatory, e.g. adenoid hypertrophy, tumours, adhesions, etc.

**SPECIAL INVESTIGATIONS**
- Microscopy, culture and sensitivity (MCS) for aerobes, anaerobes, fungi and mycobacterium. A pus swab for microscopy, culture and sensitivity, especially in a persistent, recurrent or chronically running ear, is mandatory and prevents the inappropriate empiric use of antibiotics.
- Granulation tissue should be submitted for both culture and histopathological evaluation.
- Blood tests:
• allergy tests (Fig. 2)
• full blood count
• CRP/ESR
• iron studies
• protein
• all the immunoglobulins plus IgG subfractions
• glucose
• HIV
• sweat test + Delta 508 gene — if cystic fibrosis is suspected, e.g. in a child with nasal polyposis.

• Imaging. In general, the diagnosis of the running ear is a clinical one. In certain cases, however, radiographic evaluation of the temporal bone may be helpful. Plain mastoid films, once the mainstay of radiographic evaluation of the temporal bone, have essentially been replaced by computed tomographic (CT) scanning. Plain X-rays of the sinuses and postnasal space can also be valuable.

• Histology. Biopsies of turbinates should be done for electron microscopic evaluation of the cilia if abnormal cilia are suspected.

MEDICAL TREATMENT

• Aural debridement with removal of granulations, topical agents on gauze ribbon or otowicks:
  • topical antibiotic (non-otoxic), e.g. ofloxacin, ciprofloxacin
  • topical cortisone
  • boric acid powder.
• Stimulate ciliary activity:
  • cortisone
  • nasal sprays, e.g. Vibrocil, Iliadin, Drixine.
• Decrease mucosal oedema of the nasal passages and Eustachian tubes:
  • pseudoephedrine
  • cortisone — short oral course; once daily in the mornings.
• Specific treatment for allergic rhinitis: see Table II.
• Control of infection with antibiotics: First take pus specimen for MCS. Antimicrobial therapy is initially directed toward the most common pathogens. Amoxicillin has been the empiric drug of choice for treating patients with acute OM because it is active against S. pneumoniae and most strains of H. influenzae, but in many areas the incidence of β-lactamase-producing organisms in patients with acute OM is increasing, and the efficacy of amoxicillin as the best initial therapy in these locations is being questioned.

The addition of a β-lactamase inhibitor extends the spectrum of amoxicillin against β-lactamase-producing H. influenzae and M. catarrhalis. Amoxicillin/clavulanate is a drug proven to be efficacious in acute OM. A higher dosage of amoxicillin/clavulanate (90 mg/kg/day of amoxicillin instead of 40 mg/kg/day as in current formulation), and a constant amount of clavulanate (6.4 mg/kg/day) is currently recommended. Until this formulation is registered in South Africa, 40 - 50 mg/kg/day of amoxicillin can be added to the amoxicillin/clavulanate regimen available. In patients allergic to penicillin and/or cephalosporins, azithromycin and clarithromycin (not erythromycin) have to be used (use fluoroquinolones in adults: ciprofloxacin for Pseudomonas and moxifloxacin or gatifloxacin for S. pneumoniae or H. influenzae).

The oral cephalosporin of choice selected by the DRSP therapeutic working group was a second-generation cephalosporin: cefuroxime axetil. It was considered ideal, because of the balance in Gram-positive (S. pneumoniae) and Gram-negative (H. influenzae and M. catarrhalis) bactericidal activity.

Cefprozil and cefpodoxime proxetil give promising clinical results but there is ‘limited evidence’ of efficacy against DRSP in clinical trials.

Clindamycin is ineffective against H. influenzae and M. catarrhalis and only becomes acceptable as possible choice for persistent and recurrent acute OM after tympanocentesis has confirmed that the pathogen is S. pneumoniae.

Intramuscular ceftriaxone sodium is used for patients with persistent
and recurrent acute OM. This drug should be restricted to third-line therapy for non-responsive acute OM. A 3-day 50 mg/kg ceftriaxone regimen is recommended.

Trimethoprim-sulfamethoxazole (TMP-SMX) should be precluded in South Africa owing to the high resistance rate of *S. pneumoniae* to TMP-SMX.

Ciprofloxacin is mainly used for *Pseudomonas* infections and can be used, with care, if culture shows a sensitivity to it.

Although 10 days of antimicrobial therapy for acute OM has traditionally been advocated, there is currently a growing interest in shorter courses of treatment. Shortened courses of antibiotics are likely to be successful for most patients older than 2 years of age, not in a day-care centre and with uncomplicated acute OM.

Many parents become frustrated when multiple courses of expensive antibiotics fail to alleviate OM. This frustration arises from unrealistic expectations of antimicrobial efficacy. The impact of antibiotics on OM is modest, though statistically significant. Treatment is successful in one of every four to five children treated. One must recall that OM is multifactorial — bacterial pathogens constitute only one of many causative factors. Whether the modest benefit of antibiotics justifies the cost and potential side-effects of treatment should be discussed with the patient’s family.

- Vaccination. A new conjugate heptavalent pneumococcal vaccine is another promising approach to reducing the magnitude of the problem of persistent and recurrent acute OM. Vaccinated children have significantly lower episodes of OM and tympanostomy tube placement.
- Diet and supplements
  - Control intake of dairy products and sugar
  - No colourants
  - Hypo-allergenic diet if there are severe chronic symptoms
  - Iron supplementation if necessary
- Environment
  - Smoke avoidance
  - Day-care centres — clean, smoke-free, low population density
  - Allergen avoidance.
- Keep ears dry and keep any ‘wax removing’ objects out of the ears.

### Table II. Antibiotic recommendation for drug-resistant pneumococcal AOM

<table>
<thead>
<tr>
<th>1st-line therapy*</th>
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<tbody>
<tr>
<td>• Amoxicillin 50 mg/kg/day for 10 days:</td>
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<tr>
<td>• Empiric treatment of first episode</td>
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<tr>
<td>• Empiric treatment in older children (&gt; 2 years)</td>
</tr>
<tr>
<td>• Empiric treatment in areas with low prevalence of penicillin-resistant pneumococci</td>
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</tr>
<tr>
<td>• Empiric treatment in young children (&lt; 2 years)</td>
</tr>
<tr>
<td>• Empiric treatment in patients who have received antibiotics during the month preceding the AOM episode</td>
</tr>
<tr>
<td>• Beta-lactamase stable antibiotics for 10 days:</td>
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<tr>
<td>• Empiric treatment in neonates</td>
</tr>
<tr>
<td>• Empiric treatment in immunocompromised patients</td>
</tr>
<tr>
<td>• Empiric treatment in areas with a high prevalence of beta-lactamase-producing bacteria</td>
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</tbody>
</table>

*In cases of clinical failure after full 3 days of therapy, referral for a diagnostic (and often therapeutic) tympanocentesis and MEF culture is recommended. This particularly applies to countries with a high prevalence of antibiotic resistant *S. pneumoniae* like many areas of South Africa.

<table>
<thead>
<tr>
<th>2nd-line therapy</th>
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<tbody>
<tr>
<td>• Amoxicillin/clavulanate in a high amoxicillin dose of 90 mg/kg/day for 10 days</td>
</tr>
<tr>
<td>• Cefuroxime axetil 30 mg/kg/day for 10 days</td>
</tr>
<tr>
<td>• Cefpodoxime proxetil 5 mg/kg/day for 10 days</td>
</tr>
<tr>
<td>• Azithromycin 10 mg/kg/day for 6 days†</td>
</tr>
<tr>
<td>• Clarithromycin 15 mg/kg/day for 10 days†</td>
</tr>
<tr>
<td>• Clindamycin 25 mg/kg/day‡</td>
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</table>

†Therapeutic option in areas with low prevalence of macrolide resistant pneumococci and for penicillin and/or cephalosporin allergic patients.

‡The use of this drug is restricted to confirmed pneumococcal AOM that is unresponsive to beta-lactam antibiotics.

<table>
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<th>3rd-line therapy</th>
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<tbody>
<tr>
<td>IM ceftriazone 50 mg/kg/day for 3 days</td>
</tr>
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1st-line therapy:
- Amoxicillin 50 mg/kg/day for 10 days:
  - Empiric treatment of first episode
  - Empiric treatment in older children (> 2 years)
  - Empiric treatment in areas with low prevalence of penicillin-resistant pneumococci
- Amoxicillin 90 mg/kg/day for 10 days:
  - Empiric treatment in areas with a high prevalence of penicillin-resistant pneumococci
  - Empiric treatment in young children (< 2 years)
  - Empiric treatment in patients who have received antibiotics during the month preceding the AOM episode
- Beta-lactamase stable antibiotics for 10 days:
  - Empiric treatment in neonates
  - Empiric treatment in immunocompromised patients
  - Empiric treatment in areas with a high prevalence of beta-lactamase-producing bacteria

2nd-line therapy:
- Amoxicillin/clavulanate in a high amoxicillin dose of 90 mg/kg/day for 10 days
- Cefuroxime axetil 30 mg/kg/day for 10 days
- Cefpodoxime proxetil 5 mg/kg/day for 10 days
- Azithromycin 10 mg/kg/day for 6 days†
- Clarithromycin 15 mg/kg/day for 10 days†
- Clindamycin 25 mg/kg/day‡

†Therapeutic option in areas with low prevalence of macrolide resistant pneumococci and for penicillin and/or cephalosporin allergic patients.

‡The use of this drug is restricted to confirmed pneumococcal AOM that is unresponsive to beta-lactam antibiotics.

3rd-line therapy:
- IM ceftriazone 50 mg/kg/day for 3 days
SURGICAL INTERVENTION

In the setting of acute OM surgery is necessary when specific knowledge of the causative organism requires tympanocentesis.

Indications for tympanocentesis with tympanostomy tubes include:
• severe otalgia
• severe illness or toxic appearance
• unsatisfactory response to antimicrobial therapy
• acute OM associated with confirmed or potential suppurative complications — intratemporal (e.g. mastoiditis, facial paralysis) and intracranial (e.g. meningitis, intracranial abscess)
• acute OM in a neonate or immunologically deficient patient
• persistent or recurrent OM in children with delayed speech development.

Mycingotomy with insertion of ventilation tubes improves health in the middle ear in most patients and reduces the frequency of recurrent infection.

Adenoidectomy will reduce recurrent acute OM, but is only recommended after antibiotic prophylaxis or myringotomy with insertion of tubes has been used. Tonsillectomy plays no role.

Tympanomastoid surgery is recommended in chronic suppurative OM when medical management fails or cholesteatoma is diagnosed.

References

IN A NUTSHELL

Every family physician is well aware of the diagnostic and therapeutic dilemma posed by a child with persistent or recurrent running ears.

The most common cause for a running ear in a child is acute purulent OM.

By their first birthday, 62.4% of infants have had 1 or more episodes of acute OM and 17.3% 3 or more episodes.

Acute OM often follows viral URI.

All patients with persistent, recurrent or chronic OM should have a basic medical history taken and an evaluation for underlying disease.

Most children with recurrent acute OM primarily have Eustachian tube dysfunction. If the disease is difficult to control despite maximum medical and/or surgical intervention, evaluation for underlying pathology should be considered.

Failing to remove ear wax or debris, using an otoscope that provides inadequate light, or using an inappropriately sized speculum will impair visualisation of the tympanic membrane. It has also been shown that crying and screaming causes the tympanic membrane to become red.

The goals of management of the running ear in particularly purulent OM are relief of symptoms, elimination of infection, ventilation of the middle ear and mastoid, avoidance of complications and prevention of further infection. These goals may require medical and/or surgical intervention.