Entire books have been written on the subject of canine ear disease. Rather than attempt to present all the available information here, I am providing an overview of how I diagnose and manage ear disease in dogs. It is critical to appreciate that ear disease is only a clinical problem (no more specific than pruritus) and that steps must be taken to prevent otitis externa from progressing to proliferative otitis. BOX 1 summarizes step-by-step approaches to specific clinical scenarios.

Identification of Primary Causes and Predisposing and Perpetuating Factors

Identification and treatment of the primary (underlying) cause(s) of ear disease are important to determining the appropriate management approach. The most common primary causes of otitis externa are:

- Hypersensitivities (atopy and cutaneous adverse food reaction); unilateral ear disease may be the only clinical sign of an underlying hypersensitivity
- Parasites (Otodectes and Demodex spp)
- Foreign bodies (e.g., grass awns)
- Autoimmune or immune-mediated diseases (e.g., pemphigus foliaceus)

If the primary cause of otitis externa is not addressed, the ear disease will likely recur; however, a discussion of the treatment recommendations for primary causes (e.g., parasiticide treatment, food trial) is beyond the scope of this article.

Predisposing factors must also be addressed if possible. Although predisposing factors do not cause ear disease, they increase the risk for its development and may make successful treatment more difficult. Anatomic factors (such as the long pendulous ears of basset hounds), excessive moisture in the ears (from swimming), and iatrogenic trauma (plucking hairs from the ear canals) are examples of predisposing factors.

Perpetuating factors do not cause otitis externa, but clinicians often consider them to be the reasons for disease when the problem is that the primary cause has not been identified or treated. Even if the primary cause is eliminated, perpetuating factors may cause the ear disease to continue. Examples of perpetuating factors include:

- Bacteria
- Yeast (Malassezia pachydermatis)
- Progressive pathologic changes
- Otitis media
- Undertreatment (dose or duration)—a very common problem

These factors must also be identified and treated or corrected. The treatment section of...
Specific Scenarios: Step by Step

Acute and/or Infrequent Otitis Externa

- Ascertain whether this is a first-time episode, a recurrence, or an unresolved previous infection (acute-on-chronic otitis). Unfortunately, differentiating between recurrence and unresolved infection is often difficult because of the lack of good follow-up, which emphasizes the importance of performing follow-up examinations on all otitis externa cases. The absence of clinical signs is not synonymous with resolution of disease. For dogs experiencing their first episode of otitis, educate the owners about the possible primary cause(s) and predisposing and perpetuating factors and outline the workups that may be necessary.

- Eliminate easily diagnosed primary causes (e.g., foreign bodies, parasites, masses) and perform a thorough otoscopic examination, including evaluation of the tympanic membrane.

- Diagnose and treat secondary infection(s) and inflammation. My first line of treatment for inflammation is a topical glucocorticoid ointment; I base other treatment choices on the patient factors outlined in Table 1.

- Recheck the patient after 7–14 days of treatment. This is critical in the proper management of ear disease (Table 2).

- Continue treatment for 7–14 days beyond resolution of clinical signs.

Chronic Otitis (Recurrent and/or Unresolved)

- Follow the steps outlined for acute otitis externa.

- Identify and treat the primary cause, any predisposing factors (if possible), and perpetuating factors.

- Treatment (Table 3) should last for a minimum of 30 days.

Chronic Pseudomonas Infections

These infections are especially challenging, and a discussion of the required therapy is beyond the scope of this article.

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### Table 1: Patient Factors and Therapy at Initial Presentation for Acute Otitis

<table>
<thead>
<tr>
<th>Patient Factor</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Only yeast present</td>
<td>Azole (for miconazole, add 3 mL of dexamethasone sodium phosphate [4 mg/mL concentration] to 30 mL of miconazole)</td>
</tr>
<tr>
<td>Cocci present ± rods</td>
<td>Neomycin or gentamicin</td>
</tr>
<tr>
<td>Only rods present</td>
<td>TrisEDTA + gentamicin</td>
</tr>
<tr>
<td>Dog is in pain</td>
<td>Add systemic glucocorticoids ± analgesics (tramadol, 2–5 mg/kg bid–tid and/or acetaminophen with codeine [codeine, 2 mg/kg bid–tid]); do not mix systemic glucocorticoids and NSAIDs.</td>
</tr>
<tr>
<td>Tympanic membrane not initially visualized (because of ear canal swelling)</td>
<td>Add topical fluocinolone acetonide + dimethyl sulfoxide + oral glucocorticoids (prednisone, 0.50–0.75 mg/kg PO q12h for 10–14 days); recheck dog in 10–14 days</td>
</tr>
</tbody>
</table>

### Table 2: Patient Factors and Therapy at Recheck for Acute Otitis

<table>
<thead>
<tr>
<th>Patient Factor</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tympanic membrane visible and swelling resolved</td>
<td>Discontinue prednisone; continue other therapy</td>
</tr>
<tr>
<td>Tympanic membrane not visible but swelling resolved</td>
<td>Perform ear lavage, using a standard otoscope or, preferably, a fiber-enhanced video otoscope with patient under general anesthesia</td>
</tr>
<tr>
<td>Tympanic membrane not visible and swelling not resolved</td>
<td>Continue prednisone for another 10–14 days and recheck; if the ear canals are still narrowed at recheck, consider referring patient to a dermatologist before contemplating ear ablation</td>
</tr>
</tbody>
</table>

### Table 3: Patient Factors and Therapy at Presentation for Chronic Otitis

<table>
<thead>
<tr>
<th>Patient Factor</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Only yeast present</td>
<td>Depending on what products have already been used, switch to 1% clotrimazole, 1% miconazole, or compounded 2% ketoconazole lotion; add topical steroids either by mixing dexamethasone sodium phosphate to a final concentration of 0.1% with current product or by concurrent use of a product containing fluocinolone acetonide</td>
</tr>
<tr>
<td>If drops are dispensed</td>
<td>Cleanse ear; in patients with severe ear inflammation, use a mild cleanser</td>
</tr>
<tr>
<td>Cocci ± rods</td>
<td>TrisEDTA (with or without 0.15% chlorhexidine) + either gentamicin or polymyxin B (depending on which antibiotics have already been used); Note: I rarely use fluoroquinolones in these cases unless the infection fails to respond to therapy. This means a follow-up examination is essential.</td>
</tr>
</tbody>
</table>
this article generally addresses perpetuating factors; however, if the primary cause is not identified and managed, treatment of the perpetuating cause will not eliminate ear disease.

**Diagnosis**

**History**

External ear disease is a form of skin disease, and, as with any skin problem, a detailed history must be gathered. History taking for an otic problem is similar to that for any general medicine problem. Two key points to discuss with the pet owner are the age of the dog when ear disease first appeared and whether the dog has been treated for skin or ear disease in the past.

Owners should be questioned about the first episode of ear disease in the dog’s life and not just when the current episode was first noticed. Determining the dog’s age at onset helps narrow the possible primary causes of otitis externa. For example, if the first episode developed when the dog was between 6 months and 5 years of age, atopic dermatitis or cutaneous adverse food reactions would be a likely cause; however, if the dog was older than 7 years when it first developed otitis externa, cutaneous adverse food reactions would still be a reasonable cause, but atopic dermatitis would be much less likely.

Another goal of the history taking is to identify whether ear disease is a seasonal problem; if so, atopic dermatitis is the more likely cause. It is also important to ask about any therapy used previously, the outcome of treatment, and how the outcome was determined. If the owner reports that otitis externa resolved, be sure to establish whether this was confirmed by a follow-up otoscopic examination or is merely the owner’s observation that clinical signs resolved (as opposed to resolution of infection). If resolution of previous ear disease was not confirmed by a veterinary examination, establishing seasonality becomes impossible.

**Clinical Examination**

After a thorough history has been obtained, a complete dermatologic examination should be performed. Dogs with ear disease frequently have concurrent skin disease that also needs to be identified and treated. The pattern of generalized skin disease can help identify an appropriate differential diagnosis for the ear disease.

To avoid missing abnormalities, the otic examination should be performed systematically, beginning with the pinna. Any alopecia, erythema, ulceration, crusting, scaling, or swelling should be noted and the ear canals palpated to identify pain, calcification, or thickening.

Because the presence of pain can limit proper evaluation of the ear canal, the dog should be evaluated for pain and sedated as needed before the otoscopic examination is performed. The presence, degree, and location of inflammation, ulceration, and proliferative changes of the ear canals should be noted. Likewise, it should be noted whether the tympanic membrane is visible and, if so, whether it appears normal. A ruptured or abnormal (bulging, necrotic, or thickened) tympanic membrane may indicate the presence of otitis media; however, the tympanic membrane may thicken in cases of chronic otitis externa without the presence of otitis media. If the tympanic membrane cannot be visualized, an attempt should be made to identify the reason. Potential causes include a ruptured membrane, swelling or debris in the ear canal, or a false middle ear (a condition in which the tympanic membrane is diseased and flaccid or partially forced into the tympanic cavity and bulla; the membrane is present but out of view).

The next step is to determine whether concurrent middle or inner ear disease is present. Because of the close association of sympathetic and parasympathetic nerves with the middle ear, Horner’s syndrome, keratoconjunctivitis sicca, and facial nerve paralysis may be present in cases of otitis media. Conduction (not neurologic) deafness may also be present. Inner ear disease may cause peripheral vestibular signs such as head tilt (to the affected side), circling (to the affected side), and nystagmus (with the fast phase away from the affected side).

As a time saver, some veterinarians have samples collected for ear cytology before they perform the clinical examination, but I believe that this practice makes it difficult to evaluate the true appearance of the ear canal. In addition, debris may be pushed and compacted into the horizontal canal, thereby limiting visualization of the tympanic membrane. I recommend collecting samples after completing the otoscopic examination.

**QuickNotes**

If the primary cause of ear disease is not identified and managed, treatment of the perpetuating cause will not eliminate the disease.
Laboratory Tests

Laboratory tests are a necessary component of the proper diagnostic workup for canine ear disease. Cytologic examination of a roll swab sample of any otic exudate—including quantification of the number and type of bacteria, yeast, and inflammatory cells present—is indicated. In my opinion, any organism seen on ear cytology in a dog with signs of otitis externa is potentially significant. A recent study reported that cytologic samples from normal dogs have very few yeast or cocci and no rods. Because small numbers of yeast or bacteria can cause a hypersensitivity reaction, I believe that treatment is warranted in such cases if the dog shows clinical signs.

I do not routinely conduct bacterial culture and susceptibility testing, but if such tests are used, they should always be done in conjunction with cytology. I submit samples for bacterial culture when proliferative changes are present along with neutrophils and rods on cytology and the dog has failed to respond to appropriate empirical antimicrobial therapy or in cases in which cocci bacteria persist despite appropriate therapy. Methicillin-resistant Staphylococcus spp exist in veterinary medicine and should be considered in resistant cases. Skin biopsies may be necessary depending on the differential diagnosis.

Topical Treatment

Topical therapy is the foundation of treating otitis externa. Most topical products contain a combination of glucocorticoids and antibacterial and/or antifungal agents. I prefer otic ointments because they usually contain mineral oil, which has ceruminolytic-like properties and seems to keep the ear clean during treatment without additional cleaning. Also, I think that the drugs get to the region of the tympanic membrane better with ointments than with liquid formulations (although this may be related more to volume than formulation).

I commonly use the following agents:

- **Aminoglycosides (neomycin or gentamicin):** These have good activity against gram-positive and gram-negative bacteria; gentamicin has better activity than neomycin against gram-negative organisms.
- **Enrofloxacin:** This agent is effective against gram-positive and gram-negative organisms. However, I reserve it for primarily culture-resistant gram-negative infections.
- **Polymyxin B:** This is one of the more effective antibiotics against gram-negative organisms, especially Pseudomonas spp.
- **Silver sulfadiazine:** This agent is effective against gram-negative organisms, especially Pseudomonas spp; it is a thick cream that must be mixed with equal parts of water or mineral oil to be instilled in the ear.
- **Antifungal agents:** Nystatin (mixed efficacy against Malassezia spp), clotrimazole 1%, miconazole 1%, and ketoconazole 0.1% are effective.
When gram-negative organisms are the only (or primary) organisms found on cytology, EDTA should be added to the treatment. Topical EDTA solution has a direct bactericidal action against gram-negative bacteria.\(^5\)\(^13\)\(^14\)

EDTA products that contain 0.1% ketoconazole are available and can be used if a Malassezia spp is concurrently present; however, I have two concerns about such combination products: (1) whether resistance to ketoconazole will develop with long-term use and (2) their alkalining effect on the ear when it is actually desirable to acidify the ear canal while treating Malassezia otitis.

Successful treatment of otitis externa frequently requires topical glucocorticoids, and I have seen cases resolve once topical glucocorticoids were added to the treatment regimen. Glucocorticoids are antipruritic and antiinflammatory, and they decrease glandular secretions (cerumen), pain, swelling, and glandular hyperplasia—all properties that can help restore the normal barrier function to the epithelium of the ear canal. When using topical glucocorticoids, it is best to begin with the most potent form; however, if long-term therapy is needed, a less potent agent should be used. The following topical steroids are listed in decreasing order of potency: mometasone, betamethasone, fluocinolone, triamcinolone, dexamethasone, prednisolone, and hydrocortisone. It is important to remember that topical steroids are systemically absorbed and may lower thyroid hormone concentrations,\(^13\)\(^14\) elevate liver enzymes, and even cause polyuria/polydipsia.\(^15\)

**Systemic Agents**

I use systemic antibiotics or antifungal agents only in patients that test positive for bacteria or Malassezia spp on cytology and that have (1) evidence of otitis media or (2) severe proliferative changes in the ear canals that failed to respond to topical treatment. Empirical choices for cocci include cephalosporins, amoxicillin–clavulanic acid, and clindamycin. Empirical choices for rods include cephalosporins, amoxicillin–clavulanic acid (dose tit versus bid for gram-positive organisms), and potentiated sulfonamides. I reserve fluoroquinolones for culture-proven resistant gram-negative rods because the overuse of fluoroquinolones has been associated with the emergence of antibiotic-resistant bacteria. The antifungal agents I use include ketoconazole (5 to 10 mg/kg qd, given with food to enhance absorption), fluconazole (10 mg/kg sid), and itraconazole (5 mg/kg sid).

I dispense systemic glucocorticoids (prednisone, 0.50 to 0.75 mg/kg q12h for 7–14 days) if the ear canals are edematous or stenotic. The appearance of the ear canals at the 7- to 14-day recheck dictates whether the prednisone is continued for another 7 to 14 days at the same or a decreased dose or is discontinued.

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**QuickNotes**

Successful treatment of otitis externa frequently requires topical glucocorticoids.

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**References**