Diagnosis and medical treatment of otitis externa in the dog and cat

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ABSTRACT

Otitis externa is no longer viewed as an isolated disease of the ear canal, but is a syndrome that is often a reflection of underlying dermatological disease. Causes are classified as predisposing (increase the risk of otitis); primary (directly induce otitis), secondary (contribute to otitis only in an abnormal ear or in conjunction with predisposing factors) and perpetuating (result from inflammation and pathology in ear, prevent resolution of otitis). Common primary causes include foreign bodies, hypersensitivity (particularly atopy and food allergy), keratinisation disorders (most commonly primary idiopathic seborrhoea and hypothyroidism) and earmites, particularly in cats. A systematic diagnostic procedure is required to identify causes and contributing factors. This should include history, clinical examination, otoscopy and cytology in all cases and culture and sensitivity as well as otitis media assessment and biopsy in severe and recurrent cases. Ancillary tests may be required depending on the underlying cause. Treatment consists of identifying and addressing predisposing and primary factors; cleaning the ear canal; topical therapy; systemic therapy where necessary; client education; follow-up; and preventive and maintenance therapy as required.

Key words: cat, diagnosis, dog, otitis externa, review, treatment.

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CAUSES, PATHOPHYSIOLOGY AND DIAGNOSTIC APPROACH

Classification and causes of otitis externa

Otitis externa is a syndrome, not a diagnosis[3,18]. The term refers to inflammation of the external ear canal, rather than to a specific disease process[29]. Clinically, otitis externa can be unilateral or bilateral, acute or chronic, mild to severe, nonrecurrent or recurrent and amenable or resistant to routine therapy. It has been classified according to the type of exudate as erythematoceruminous or suppurative, with the former subgrouped as parasitic or nonparasitic[9].

Otitis externa can be caused and perpetuated by many conditions and factors, frequently more than 1 at a time[29,53]. These have been classified as predisposing factors (increase the risk of otitis), primary causes (directly induce otitis) and perpetuating factors (result from inflammation and pathology in ear that prevents resolution of otitis)[3] and this has become standard usage. The classification was recently adapted to include secondary causes, which were previously included as perpetuating factors[53]. Secondary causes contribute to otitis only in an abnormal ear or in conjunction with predisposing factors. Table 1 lists and defines causes of otitis, and indicates which are most common. As illustrated in the table, primary causes can be local or generalised, while secondary causes and predisposing or perpetuating factors are more likely to be local. Most microbial infections of the ear are secondary to another disease or factor and are usually opportunistic[17].

Pathology and pathophysiology

The pathophysiology of otitis externa is not complex – in fact perhaps the opposite. Fig. 1 shows the self-perpetuating nature of the condition if untreated or inadequately treated.

The detailed pathology of otitis, particularly early on, differs to some extent according to the cause[51], but in general, changes are rather stereotyped. Acute inflammation and oedema, if not resolved, progresses over time to chronic inflammation, characterised by glandular changes, fibrosis and scarring, and, eventually, progressive stenosis and occlusion of the ear canal[29,53]. Permanent changes such as calcification and later ossification of cartilage can occur. Possible sequelae are otitis media and aural cholesteatoma (both also perpetuating factors)[29]. Chronic changes favour proliferation of bacteria and yeasts, further perpetuating pathology[53]. Ulceration of the ear canal can occur, usually in association with *Pseudomonas* infection[36]. The secondary lesions of chronic otitis are due to chronic irritation and microbial overgrowth[51].

Diagnostic approach

'Diagnosis and clinical management of otitis externa is often frustrating because there are numerous factors and diseases that predispose to otitis and numerous secondary pathogens that perpetuate the process' [35]

In the light of the widely divergent causes of otitis externa, a systematic diagnostic assessment is essential. The approach to the ear 2 decades ago was to examine and treat it in isolation[1]. The current approach differs substantially, as the ear canal has now been given its proper place as a specialised extension of the skin[8,29] and otitis externa is now recognised as a dermatological condition. Diagnosis of the *syndrome* is straightforward – it can be recognised by variable degrees of head-shaking, pruritus, pain, odour and exudation from the ear[50]. Othaemotoma may result from pruritus[53]. The diagnostic challenge in otitis is to determine the primary cause and identify secondary and perpetuating factors[59]. It is difficult to assess how often it is possible to make a specific primary diagnosis, as little data are available. A primary cause was identified in 8/12 cases of chronic, proliferative *Pseudomonas* otitis[40] and Griffin asserts that, 'In the majority of chronic ear cases I can find historical or physical evidence of the primary disease.'[18] It seems likely that the primary cause can be found in a reasonable number of cases and that predisposing, secondary and perpetuating causes can be identified and controlled in most. Identifying a primary cause is more important in chronic or recurrent otitis than acute otitis[49].

Routine diagnostic procedures

Table 2 shows recommended diagnostic procedures for otitis. The assessment in all cases should include a general and

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Table 1: **Causes of otitis externa in the dog and cat**[3,17,18,29,49,53]. **Common conditions are in boldface, and the most common primary causes are indicated with an asterisk.**

	PREDISPOSING FACTORS **Increase the risk of developing otitis externa**
Breed predisposition	Examples: Cocker spaniel, poodle, German shepherd
Conformation	Stenotic canals, hair in canals, pendulous pinnae, hairy concave pinnae
Excessive moisture	Swimmer's ear
Climate	High humidity
Excessive cerumen production	Idiopathic
Obstructive ear disease	Neoplasms, feline nasopharyngeal polyps
Systemic disease	Pyrexia, immune suppression, debilitation, catabolic states
Treatment effects	Trauma from cotton applicators, irritant topicals, superinfections by altered microflora, excessive cleaning
	PRIMARY CAUSES **Directly induce otitis externa**
Foreign bodies	Plant material (*e.g.* grass awns), hair, sand, dirt, hardened medications and secretions
Hypersensitivity diseases	**Atopy***, **food allergy***, flea allergy, contact hypersensitivity, drug reactions
Keratinisation disorders*	**Primary idiopathic seborrhoea**, **hypothyroidism**, sex hormone imbalance, abnormal cerumen production
Parasites	**'Classic' earmites (*Otodectes cynotis*)***, demodicosis, sarcoptic or notoedric mange, *Otobius megnini* ticks
Autoimmune diseases	Lupus erythematosus, pemphigus foliaceus, pemphigus erythematosus
Glandular disorders	Apocrine hyperplasia, sebaceous hyper- or hypoplasia, altered secretion rate, altered type of secretions
Microorganisms	Dermatophytes, *Sporothrix schenckii*
Miscellaneous conditions	Idiopathic inflammatory/hyperplastic otitis externa of the Cocker spaniel, juvenile cellulitis, IgA deficiency, pyoderma of the head
Viral diseases	Distemper
	SECONDARY CAUSES **Contribute to or cause pathology only in the abnormal ear or in combination with predisposing factors**
Bacteria	Numerous species, most commonly ***Staphylococcus* spp.**; ***Pseudomonas*** in chronic resistant otitis
Yeasts	***Malassezia pachydermatis***, *Candida albicans*
Foreign bodies	Small or microscopic, can include secretions
	PERPETUATING FACTORS **Prevent resolution of otitis; result from inflammation and pathologic response**
Progressive pathological changes	Hyperkeratosis, hyperplasia, skin folds, oedema, fibrosis, stenosis, calcification
Otitis media	Simple purulent, caseated/keratinous, choleasteatoma, proliferative, destructive osteomyelitis
Tympanic membrane changes	Opacity, dilation, diverticulum

dermatological history, physical and dermatological examination, otoscopy, and cytology[8,17,18,26,29,48,50,53]. A standard dermatological questionnaire can be used to ensure that important details are obtained in all cases[48].

Proper otoscopic examination is essential. Adequate visualisation depends on patient control (sedation or general anaesthesia are often required), a meticulously clean ear, and absence of severe inflammation and oedema. In some cases, local or systemic treatment might be required for a few days before otoscopy can be performed[17,53]. Otoscopy is used to assess the diameter of the ear canal, the amount and type of exudate, the presence of ulcers, foreign bodies, parasites, tumours and other space-occupying lesions as well as the integrity of the tympanic membrane[8]. In 1 study, otoscopic examination of the tympanic membrane was only considered adequate in 28 % of otitic ears (compared with 78 % of healthy ears)[28]. However, otoscopy was reasonably effective at diagnosing ruptured tympanic membranes, although the sensitivity and specificity were suboptimal – tympanometry had 100 % sensitivity and specificity, compared with 83 % and 93 % for otoscopy[28].

The odour and gross appearance of the exudate is somewhat helpful, but not very reliable[26,39,49]. Thus, although a particular kind of exudate can increase the index of suspicion for a particular kind of otitis (Table 3), gross examination alone is inadequate.

Cytology is *the* pre-eminent diagnostic tool in otitis externa[27,48,49,53] and is recommended for all cases where exudate or debris are present[36]. Sample collection and preparation has been covered in detail[12], but is essentially straightforward. A sample from the horizontal canal is collected onto a clean cotton-tipped swab, part of the sample is examined under oil and part is rolled onto a slide, dried, stained and examined for yeasts, bacteria, inflammatory and neoplastic cells. Cytology is more sensitive than culture[22], and culture (where indicated; see below) should never be performed without simultaneous cytology[53]. Cytology can demonstrate the number and morphology of bacteria, number of yeasts, presence of fungal hyphae, presence of parasites, number and type of leukocytes and whether they are phagocytosing organisms, the presence of excessive cerumen, keratinaceous debris and neoplastic cells[12].

Since microorganisms are present in normal ears, how does the clinician assess whether those seen on cytology are abnormal or not? The presence of inflam-

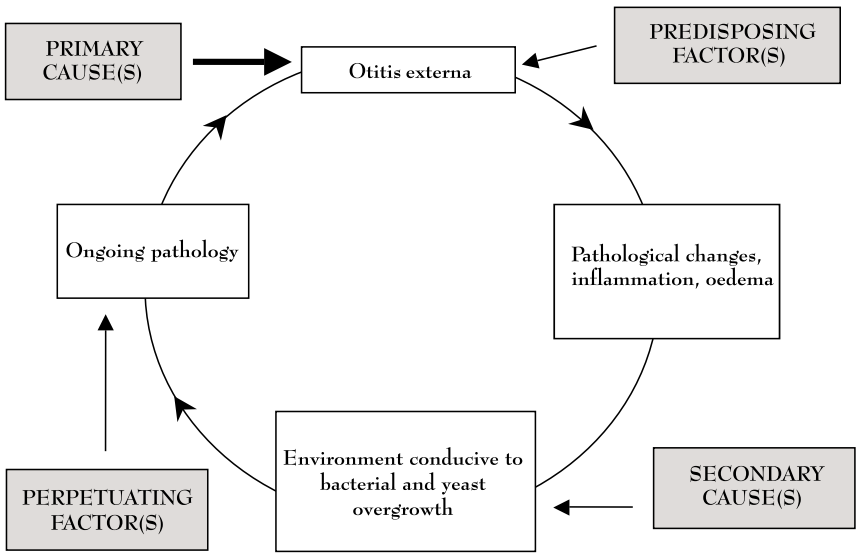

Fig. 1: **The self-perpetuating and stereotypical nature of otitis externa.**

matory cells, particularly if phagocytosing organisms are present, generally removes any doubt, but this is not always seen in otitis, particularly in *Malassezia* infections[39]. Therefore, numbers or relative numbers, of organisms are used as an index. Rausch[45] found that otitic ears had >10 *Malassezia* yeasts per high-power (10 × 40) field (>4 per 10 × 100 oil immersion field[13]), while healthy ears had <10 per high power field. This has since been used as the basis of recommendations. It has also been suggested that numbers of yeasts be evaluated in relation to numbers of bacteria[18] and the clinician should thereby assess which are the major organisms present. It is useful to grade numbers of organisms using a consistent scale, preferably on a pre-printed card, for follow-up purposes.

As a general guideline, healthy ears contain some keratinaceous cells, may contain low numbers of *Malassezia* and cocci, and have no, or extremely few, inflammatory cells. Large numbers of *Malassezia* and/or cocci should be considered abnormal, while any rods, fungal hyphae, ectoparasites, neoplastic cells and inflammatory cells (unless extremely sparse) are abnormal. If present in sufficient numbers, *Otodectes cynotis* mites are easily identified under oil. Unfortunately, mites are not always detectable, particularly in dogs[8,17,53]. This is at least in part because very few mites (2–3) can initiate pathology.

Additional procedures for chronic and recurrent otitis

Table 2 lists additional diagnostic techniques recommended for chronic and recurrent otitis. In these cases, a record should be kept, for assessment and follow-up purposes, of the grade and severity of oedema, the degree of canal stenosis, oedema or occlusion from chronic hyperplasia, the quality, character and colour of exudate, and cytological findings.

The overall usefulness of culture and sensitivity in otitis externa is limited. Many research groups have studied culture and sensitivity characteristics[4,13,20,25,33,34], but the results (and, indeed, results from individual cases) are quite difficult to translate into definite treatment recommendations. Agreement between cytology and culture is not always good. The sensitivity of culture is inferior to that of cytology[53], with the exception of *Pseudomonas* infections[19]. *In vitro* sensitivity is unlikely to be the same as *in vivo*, as drug concentrations are much higher in the ear than on sensitivity discs. In addition, most ears are treated in multiple ways. Cleaning agents, the mechanical act of flushing, antiseptics, multiple antibacterials in some preparations and vehicles will alter the microenvironment and affect bacteria in ways that cannot be predicted by testing a single drug in a laboratory. Many studies (and laboratories) include drugs such as unpotentiated penicillins and others which are rarely indicated for either topical or systemic use in otitis.

For the above reasons, culture and sensitivity are only recommended in the following circumstances, which usually occur in association with recurrent and/or chronic otitis[11,17,19,20,49,53]:

- Rods seen on cytology.
- Systemic antibiotics required.
- Failure to respond to initial treatment.
- Otitis media diagnosed or suspected.
- *Pseudomonas* infection suspected (even if rods not visualised on cytology).

If concurrent otitis media is present, the exudate in the middle ear should be cultured separately, as different organisms and/or sensitivity patterns often occur[13].

Unlike otitis externa, otitis media is difficult to diagnose[53]. It may be secondary to chronic otitis externa and may in turn perpetuate otitis externa[53]. The proportion of dogs with otitis externa that also have otitis media appears to vary regionally[49], but has been estimated to be as high as 50 %[36]. In chronic otitis, careful attention should be given to otoscopic examination of the tympanic membrane, but even under ideal circumstances the membrane cannot be adequately visualised in many cases[28] and it is intact in almost three-quarters of cases of otitis externa with concurrent otitis media[13]. Myringotomy is a useful diagnostic tool for otitis media[53].

Radiography is likely to be diagnostic of otitis media if the condition is very chronic, neurological signs are present, and/or the tympanic membrane is perforated[17,59] . However, normal radiographs do not rule out pathology in the middle ear[59]. Computerised tomography or magnetic resonance imaging are good diagnostic tools for acute otitis media[49].

In chronic cases, biopsy of the proximal vertical canal and/or proximal pinna can be performed[49]. This is an underused technique, and can provide useful diagnostic and prognostic information[48,49]. Any tumour or proliferative mass should be biopsied[36].

Any additional diagnostic procedures

Table 2: **Recommended diagnostic procedure for otitis externa.**

	Routine	Chronic/recurrent
General and dermatological history*	✓	✓
Physical and dermatological examination*	✓	✓
Gross assessment of exudate	✓	✓
Otoscopy	✓	✓
Cytology of exudate	✓	✓
Culture and sensitivity		✓
Otitis media assessment		✓
Biopsy		✓
Ancillary tests for primary cause	Variable	Variable

*More detailed information required for chronic/recurrent cases.

Table 3: **Possible causes of particular types of otitic discharge[12].**

Type of discharge	Suspicious for:
Copious dark-brown, waxy, sweet-smelling	Pure *M. pachydermatis* infection
Dark brown to black, crumbly exudate resembling coffee grounds	*O. cynotis* infestation
Dark yellow to pale brown, creamy	Gram-positive cocci
Heavy, sweet-smelling, oily, yellow to tan (ceruminous otitis)	Non-infectious causes such as seborrhoea, atopy, endocrinopathy
Pale yellow, thick, sweet-smelling, often caseous	Gram-negative rods

depend on the suspected or known primary problems, but might include haematology, serum chemistry profile, urinalysis, endocrine tests, allergy testing, and evaluation of the immune system[48].

MEDICAL TREATMENT OF OTITIS EXTERNA

'One of the most significant advances in the management of chronic otitis over the past 20 years is that we no longer expect that taping the ears over the head and applying a topical ointment for 7 to 10 days will take care of the problem.' [59].

Treatment of otitis is tailored to each individual case[50]. Therapeutic agents and products should be targeted at known causes and problems, the choice being based largely on a combination of diagnostic findings and personal experience. The number of commercially available products used in the ear, the array of extralabel treatments recommended, plus the combination of types of otitis and the variety of contributing factors have precluded the establishment of a solid, objective body of literature detailing which specific treatments are most appropriate in which specific circumstances. The general approach to treatment is as follows[11,17,18,36,49,53]: identify and address predisposing and primary factors; clean the ear canal; institute topical therapy; institute systemic therapy (where needed); client education; follow-up; preventive and maintenance therapy (as required). Aggressive surgical management might be indicated when intractable proliferation and stenosis of the ear canal are present[35,49,53]. One of the aims of medical therapy in dogs with known risk factors for chronic, severe, intractable otitis externa is to prevent the condition deteriorating to the point where surgery is the only option.

Treatment of predisposing and primary factors

Management of predisposing and primary factors varies widely according to the cause(s), and is beyond the scope of this review. Recent texts and reviews should be consulted for specific information[18,19,50,53,59].

Cleaning the ear canal

Cleaning and drying the ear canal is an essential part of assessment and treatment[27,50,53]. Cleaning allows optimal visualisation; removes debris; reduces the microbial population; removes microbial by-products such as toxins and enzymes; allows topical drugs to reach their site of action; increases the effectiveness of topical medications (some of which can be inactivated by exudate) and has a soothing effect. Unremoved debris can function as small foreign bodies and act as the nidus for reinfection[35].

In mild cases, home cleaning with a ceruminolytic is sufficient, but many cases require flushing under sedation or general anaesthesia[30]. In very severe otitis, systemic and/or topical medication must be administered for up to 2 weeks before the canal is sufficiently open to allow adequate cleaning[30]. Ear cleaning and drying products, and their uses, are listed in Table 4. Cleaning usually involves a ceruminolytic, a flushing agent and in some cases a drying agent. Ceruminolytics soften and emulsify waxy debris, and are usually detergents or surfactants[50]. Examples, in decreasing order of efficacy, are dioctyl sodium sulphosuccinate, propylene glycol, glycerine and mineral oil[50]. All ceruminolytics are potentially ototoxic and should not be used if the tympanum is known or suspected to be ruptured. Flushing solutions include saline, water, acetic acid, chlorhexidine and povidone-iodine[50] (Table 4). Saline does not damage the middle ear even under extreme circumstances[32] and can thus be recommended for routine use.

Ear flushing is approached as follows[30,53,59]: the integrity of the tympanic membrane is assessed, using history, severity and clinical signs in addition to otoscopy. If the membrane is intact, the canal is filled with a ceruminolytic, massaged and left for 5–10 minutes. Omit this step if the tympanum is known or suspected to be ruptured. The canal is gently flushed with warm flushing solution, using a rubber bulb syringe or soft tube (urinary catheter or feeding tube) with a 10 mℓ syringe. The latter apparatus is considered safest and is very effective. Use of a 3-way stopcock (attached to an infusion set leading to the saline bag, the flushing tube and an outlet tube) streamlines the process[27]. A vacuum system can

Table 4: **Selected cleaning and drying agents for otitis externa[30,50].**

Product	Trade names	Type	Indications	Dilution
Acetic acid (white vinegar)		Flushing and drying	Flushing; drying; maintenance for most types of otitis	1:1 to 1:3 in water
Chlorhexidine (5 %)	Hibitane (Astra Zeneca)	Flushing, some drying effect	Flushing; bacterial, CP otitis	1:100 in water for flushing
Dioctyl sodium sulphosuccinate (DSS)	Docusol (Kyron), Surfactol (Centaur)	Ceruminolytic	Ear cleaning; maintenance for yeast, ceruminous, CP otitis	
Glacial acetic acid, isopropyl alcohol	Swimmer's Solution (Kyron)	Drying	Drying; maintenance for yeast and exudative otitis	
Lactic acid, salicylic acid, DSS, propylene glycol, malic acid, benzoic acid	Epi-Otic (Virbac)	Ceruminolytic/ drying, mild antibacterial and antifungal	As for DSS above	
Povidone-iodine (10 %)	Betadine (Adcock Ingram)	Flushing	Flushing, bacterial otitis	1:10 to 1:50 in water
Saline (0.9 %)		Flushing	Flushing	

CP = chronic proliferative.

Table 5: **Topical otitis medications currently on the market in South Africa.**

Product	Glucocorticoid	Antibiotic	Antifungal	Antiparasitic	Other	Type
Auroto (Kyron)		Neomycin	Thiabendazole	Thiabendazole	Amethocaine	Drops
Betsolan (Janssen)	Betamethasone	Neomycin				Drops
Oridermyl (Centaur)	Triamcinolone	Neomycin, chloramphenicol	Nystatin	Lindane	Lignocaine	Ointment
Otomax (Schering-Plough)	Betamethasone	Gentamicin	Clotrimazole			Suspension
Otospectrine (Phenix)	Dexamethasone	Neomycin, polymixin B	Monosulfiram	Monosulfiram	Lignocaine	Drops
Panalog (Novartis)	Triamcinolone	Neomycin, thiostrepton	Nystatin			Ointment
Surfacticide (Centaur)		Nitrofurazone		Lindane	Amethocaine, DSS	Drops
Surolan (Janssen)	Prednisolone	Polymixin B	Miconazole			Drops
Terra-Cortril (Pfizer)	Hydrocortisone	Oxytetracycline				Suspension

DSS = dioctyl sodium sulphosuccinate.

be used but is not essential. The flushing tube must be sterile, narrow enough to ensure that there is a space between it and the canal to avoid pressure build-up, and atraumatic. The tube is inserted through an otoscope cone and the flushing process visualised through the otoscope. Debris is removed by gentle flushing and suction. Large particles and hairs can be removed using alligator forceps. After the first flush, excess liquid is removed from the ear by gentle suction, and the canal and eardrum reassessed. Any obstinate debris should be carefully removed using a curette or loop inserted through the otoscope head, and the canal flushed until it is clean. Cottonwool swabs should be avoided, as they are traumatic and can compact debris in the canal.

If the tympanum is ruptured, flushing fluid may enter the mouth or nasal cavity, and swallowing or fluid leakage from the nose may be seen[53]. Intubation of anaesthetised dogs should be routine to prevent aspiration pneumonia. If the tympanum is only discovered to be ruptured after the initial flushing, or has is ruptured during the procedure, the middle ear is gently and thoroughly flushed with saline or water to remove any traces of ceruminolytic and/or debris. Once flushing is complete, the canal is dried using gentle suction. Topical medication and/or a drying agent are instilled if required.

The main danger of ear flushing is inadvertent rupture of the tympanic membrane; this is most likely if the membrane is already compromised[30]. Introduction of ototoxic substances into the middle ear through a ruptured membrane is a related hazard. Contact irritation or allergy can result from ear flushing with more caustic substances[30]. To minimise ototoxicity and irritation, the mildest possible products should be used and if more caustic products are needed, they should be rinsed out afterwards with warm saline or water[30]. Iatrogenic damage to the ear canal and tympanic membrane are further minimised by avoiding 'blind' introduction of catheters or instruments; these should always be introduced through an otoscope cone and the procedure visualised as it is being performed. Resistant pathogens can be transmitted from one animal's ears to another through inadequately sterilised equipment; this can be avoided by proper sterilisation and by discarding equipment that cannot be properly sterilised, such as rubber tubes[30]. Auditory or vestibular dysfunction may rarely follow ear flushing even if no ototoxic substances are used; this is more common in the cat than the dog[30].

Owners can carry out maintenance or preventive cleaning at home, using products suited to the particular case (Table 4). A squeeze bottle or bulb syringe can be used; the latter should be cleaned with 50:50 vinegar:alcohol after each use, and should be changed at least every 2–5 weeks[30]. In very severe otitis, or with very fractious dogs, a temporary cleaning device (see reference[30]) can be inserted in the ear and left in place for 5–10 days. Although frequent home cleaning might be required initially[30], it is generally recommended that owners do not clean ears more often than once every 2 days[59]. Frequent home-cleaning can result in continual moisture in the ear with secondary infection[53], and/or irritation of the ear[30].

Topical therapy

Topical therapy is an important part of the treatment of otitis externa[17,18,27,49,53]. Combination or multipurpose products are frequently indicated, particularly initially, because of the mix of microorganisms, inflammation and sometimes, parasites that are present in most ears at the time of diagnosis[50]. Although symptomatic topical treatment is effective[27] and can be curative alone[49], the short-term effectiveness of such treatment can lull practitioners and owners into a false sense of security and lead them to bypass attempts to identify factors contributing to the disease[3,59]. This is considered by some to be a perpetuating factor of otitis[11].

Topical therapy should be selected on the basis of clinical findings, cytology, underlying causes and personal experience[8,17,27,36]. Treatment requirements may change as the case progresses[18,53]. Most routine topical otitis preparations contain a glucocorticoid, antibiotic, antifungal, and sometimes an antiparasitic agent, in an oily or aqueous vehicle[17]. Commercial products currently available in South Africa are listed in Table 5. Disinfectants, ophthalmic and self-formulated preparations are also effective in certain types of otitis[53,59]. The array of products highlights the fact that there is no 'magic bullet' for otitis externa. There is little scientific data to show that 1 combination treatment is better than another and personal preference plays an important role[11].

Components of topical otic medications

Glucocorticoids

Topical glucocorticoids are considered beneficial in most cases of otitis externa, regardless of the underlying cause of inflammation[8,29,36,53] and most otic prepa-

Table 6: **Suggested empirical treatment for different types of bacterial otitis[17].**

Type of infection	Appropriate drugs and disinfectants
Acute otitis, Gram-positive cocci on cytology, staphylococci or streptococci on culture	Neomycin, chloramphenicol Povidone-iodine, chlorhexidine, acetic acid
Acute otitis, Gram-negative bacilli on cytology, *Proteus* or *Escherichia coli* on culture	Neomycin, polymyxins, gentamicin Acetic acid, povidone-iodine
Chronic/resistant otitis, gram-negatives (usually *Pseudomonas*)	Gentamicin, polymyxin B, polymyxin E, colistin Polyhydroxidine iodine, *Systemic* ormethoprim-sulfadimethoxine, trimethoprim-sulfonamide, first-generation cephalosporin
Culture *Pseudomonas*	Ticarcillin, tobramycin, enrofloxacin, amikacin Silver sulfadiazine, Tris-EDTA-gentamicin solution *Systemic* enrofloxacin, marbofloxacin, orbofloxacin, gentamicin

rations contain a glucocorticoid[17]. Benefits include[17,27,29,50,53]:

- Potent antipruritic/anti-inflammatory action.
- Break the 'itch-scratch-itch' cycle.
- By reducing pain and pruritus, making it easier to medicate the animal.
- Reduce exudation and swelling, thus improving ventilation and drainage.
- In severe cases, part of pretreatment to allow visualisation of the ear canal.
- Reduce scarring and fibrosis, thus reducing hyperplastic and proliferative changes.
- Counter-intuitively, have some beneficial effects against secondary infection – allow antibiotics to reach the deep canal, reduce discharge that might inactivate antibiotics.

Systemic absorption of topical glucocorticoids may suppress the pituitary-adrenal axis. In a randomised study of 2 ear preparations, 1 containing triamcinolone, the other dexamethasone, 4 mg glucocorticoid daily in the ear caused significant laboratory suppression of the axis after 7 days in 7/8 dogs; and in 5/7 dogs, ACTH stimulation was still inadequate 14 days after cessation of treatment (the treatment period was 21 days)[38]. However, the clinical significance of these findings was uncertain[38]. Potentiation of ear infections by topical glucocorticoids is theoretically possible, but there is little evidence that this is a real problem[27]. In fact, human studies have shown that secondary infections such as those that occur in otitis externa are often better controlled by combined antibiotic/corticosteroid preparations than by antibiotics or corticosteroids alone[27]. A syndrome of acquired folding of the pinna, apparently due to loss of cartilage, has been identified in adult cats[53]. All these cats had been treated daily for 8 months to 2 years with topical glucocorticoid-containing otic preparations.

Despite the above and other theoretical disadvantages, topical glucocorticoids are relatively safe in practice[29]. However, as with any glucocorticoids used for any condition, those used in the ear should be administered judiciously. The choice depends on the nature, severity and chronicity of the condition. The general rule is to use the least potent and shortest-acting preparation possible, for the shortest period possible[29]. Selection is particularly important if long-term treatment (>3 months) is required[17]. More potent glucocorticoids may be needed for acute or acutely exacerbated otitis, but once the inflammation is controlled, short-acting, low-potency drugs are preferred[53]. The potency of the glucocorticoids is expressed relative to hydrocortisone (cortisol). The exact numbers differ in different reports, but the following is reasonably representative: hydrocortisone 1, prednisolone and triamcinolone 5, betamethasone and dexamethasone 25, fluocinolone 100[49]. However, triamcinolone has also been considered twice as potent as prednisolone (10 vs 4)[27].

Antibacterials

Bacterial infection is likely to be present in most cases of otitis when seen initially, and can easily be confirmed by cytology. Antibacterials are thus required in most cases initially, though they may be unnecessary in maintenance and preventive treatment. Neomycin, chloramphenicol, polymixin B and gentamicin are frequently included in topical otic medications, but a number of other drugs can be used to treat bacterial otitis[53]. Antibacterial drugs are not the only option for treating infection, and disinfectants such as povidone-iodine, chlorhexidine, dimethylsulfoxide and Tris-EDTA can be extremely effective[18,35]. These are especially recommended, usually in conjunction with antibacterial drugs, for the treatment of resistant *Pseudomonas* otitis[19,49]. Non-otic preparations are often used for chronic, resistant infections, and include ophthalmic antibacterials as well as self-formulated compounds[53].

Empirical choice of antimicrobials, based on cytological findings, is recommended except in chronic, recurrent cases, and/or if otitis media is present. (In these cases culture and sensitivity testing are indicated – see above.) The major distinction that must be made on cytology is whether cocci or rods (or both) are present. Choice of treatment is made accordingly (Table 6). Especially initially, topical drugs should be those unlikely to be needed systemically, so as not to limit the choices of systemic antibiotics for resistant cases of otitis externa, or subsequent otitis media[53]. Some authors suggest using 'first-line' drugs such as neomycin or polymyxin B initially, while 'second-line' choices would include drugs like gentamicin or chloramphenicol[49,53]. Resistant Gram-negative infections, particularly of *Pseudomonas,* can be a therapeutic challenge. Table 7 lists treatments that have been found to be valuable in these cases.

Many commonly used antibacterials are potentially ototoxic if used in the presence of a ruptured tympanum and/or otitis media, particularly if use is prolonged[53,60]. These include the aminoglycosides gentamicin, neomycin and amikacin, as well as chloramphenicol and polymyxin B[36]. In practice, ototoxicity is rare in small animals and the risk is probably somewhat overstated[17,19,27,35,55]. However, a non-otoxotic drug must be used if the tympanum is known to be ruptured. The impairment caused by aminoglycosides is likely to be auditory rather than vestibular and might remain undiagnosed in many cases, particularly if unilateral[35,42]. Inappropriate and/or long-term use of antibacterial agents can cause bacterial resistance; some authors therefore recommend that more potent and broad-spectrum antibiotics such as gentamicin and chloramphenicol should not be used as first-choice treatments[18].

Table 7: **Topical 'extra-label' treatments for refractory Gram-negative** (usually *Pseudomonas*) **otitis**[50,53,59].

Product	Preparation	Frequency
Acetic acid 5 % (white vinegar)	Dilute 1:1 to 1:3 in water	OID to BID[a]
Amikacin 50 mg/mℓ	Undiluted (or dilute up to 1:30 in saline)	BID
Chlorhexidine 5 %	Dilute to 1.5 % in PG	BID
Enrofloxacin 50 mg/mℓ	1 mℓ plus 9 mℓ saline, water, injectable dexamethasone, PG or EpiOtic	BID
Silver sulfadiazine	Dilute cream 1.5 mℓ in 13.5 mℓ water or 0.1 g powder in 100 mℓ water	0.5 mℓ per ear BID for 14 days
Tris-EDTA ± gentamicin	1.2 g EDTA, 6.05 g Tris and 25 mℓ white vinegar; make up to 1 ℓ in distilled water; adjust pH to 8.0, autoclave. Can add gentamicin to 3 mg/mℓ	5–10 min soak before antibiotic; or 2–12 drops BID (with genta); for 14 days

[a]OID = once daily, BID = twice daily, PG = propylene glycol.

Chronic topical antibiotics can also predispose to yeast infection[17]. Follow-up examinations are important to assess efficacy of treatment and minimise the development of resistant organisms.

Antifungals

Antifungal agents are indicated in most cases where yeast infection is present and probably in all fungal (as opposed to yeast) infections of the ear. In mild cases of yeast infection, glucocorticoids and flushing alone can clear the infection by normalising the environment[29]. By far the most common fungal infection in the ear is the yeast *Malassezia pachydermatis,* but many otic antifungals are also effective against dermatophytes, *Candida* and *Aspergillus* spp. Antifungals effective against *Malassezia* are ketoconazole, econazole, miconazole, nystatin, pimaricin, clotrimazole, cuprimixin and amphoterecin B[25,36,58]. Ketoconazole is considered the most effective of these. Nystatin may cause local hypersensitivity reactions. Griseofulvin, thiabendazole, tolcyclate and tolnaftate are ineffective *in vitro*[31], but thiabendazole appears to be clinically effective[11,27,53]. Povidone-iodine, chlorhexidine and 2.5 % acetic acid are also effective[53].

Antiparasitic agents

By far the most common parasite in the ear is the earmite, *Otodectes cynotis.* Earmites are the major single cause of feline otitis externa. To deal effectively with earmites, the entire animal should be treated with a standard acaricide, because the parasites can survive on other areas of the body. All in-contact animals should be treated[17,49,53]. The minimum duration of treatment is 3 weeks, to break the parasite's life-cycle[49,53].

Lindane (the γ isomer of BHC or γ BHC) was traditionally the acaricide used to treat *O. cynotis*[27]. The use of lindane in cats is controversial, with some authors advocating it[37] but others maintaining that all chlorinated hydrocarbons are contra-indicated in this species[2,23,47]. Concentrations over 0.1 % may cause toxic reactions in cats[5]. One of the reasons cats are susceptible to poisoning by chlorinated hydrocarbons is their fastidious habit of licking products off their coats[6,54]. Care should thus be taken to wipe away any overflow medication. No side-effects were reported in studies of lindane-containing otic preparations in cats[15,43]. Dogs and other mammals are quite resistant to the toxic effects of lindane[6,44]. In summary, lindane should be used with circumspection in cats, and should be used with care in any animal that is young, emaciated or systemically ill[21].

Otic preparations containing thiabendazole, rotenone, pyrethrins and carbaryl are effective against *O. cynotis*[14,16,18,35,53]. All have low toxicity to mammals and are highly unlikely to cause detrimental effects at the doses used for otitis[5,60]. Interestingly, a number of products without a miticide performed very well against *O. cynotis*[15,41,43,52,57], presumably due to unknown antiparasitic properties of the components or the effect of the oil base. In 1 of these studies, lindane performed substantially worse than a non-acaricide product[43]; in another, an otherwise identical product performed equally well with or without a miticide[15].

Systemic or topical ivermectin is effective against ear mites[16,46]. Systemic ivermectin can cause mydriasis, tremors and blindness in cats, is not recommended in dogs younger than 3 months and caused fatal toxicity in a 4-month-old kitten[16]. It can cause discomfort and pain after subcutaneous injection in cats[16]. Ivermectin is contra-indicated in Collies and Collie crosses[18,35]. Fipronil spray is effective against earmites and can be administered as a single treatment[53]; it should also be considered for otic tick infestation. Thiabendazole can be used for *Demodex* otitis in cats[49]. Topical amitraz or systemic ivermectin or milbemycin are effective against otic *Demodex* and tick infestations in dogs[36].

Topical anaesthetics

Topical anaesthetics are used in some otic preparations to decrease pain and pruritus[8,60]. They cause superficial anaesthesia only[10], and their efficacy in otitis is considered doubtful[27].

Vehicle

The vehicle is a significant component of any topical preparation. Unfortunately, in many instances little is stated about the vehicle in the product information. The specific formulation of the vehicle is important, as is the question of whether it is oil-based or aqueous.

Water-miscible bases are often easier to apply and less messy than oil-based products. They are usually better solvents for the active agent[24]. Ointments, creams and gels soften, hydrate, facilitate removal of scales and crusts, lubricate, protect, and facilitate penetration of the skin by the active agent[24]. In dermatology in general, it is recommended that exudative conditions (usually acute) should be treated with a product formulated with a minimally occlusive vehicle, while chronic, usually thickened, lesions, need occlusive vehicles to rehydrate the dry, thickened surface[24]. Most authors therefore recommend an aqueous vehicle (solutions, lotions, tinctures) in 'wet' ears and an occlusive vehicle (ointment/oil-based /creams) in 'dry' ears[7,11,17,18,27,53,59]. However, choice of vehicle might be more dependent on factors such as active ingredients, experience and owner convenience[17,49,53]. The type of vehicle might need to be changed as the healing process proceeds[11].

Many vehicles are potentially ototoxic if the tympanic membrane is ruptured. This applies particularly to oil-based preparations. Propylene glycol, which is quite commonly used in otic preparations, can be associated with hypersensitivity reactions[36].

Systemic therapy

Systemic therapy is required if[50,53]:

- Otitis externa is severe.
- There is concurrent otitis media.
- Owners are unable to administer topical treatment.
- Marked proliferative changes are present.

- Adverse reactions to topical treatments are suspected.

Systemic glucocorticoids are used for severe pain and inflammation[17] as well as chronic otitis with proliferative changes and allergic otitis[50]. Where systemic antibiotics are needed, appropriate empirical choices are trimethoprim-sulfas, clindamycin, cephalexin and enrofloxacin (for otitis media)[53], but where possible, selection should be based on sensitivity testing. (See also section on topical antibacterials, and Table 6.)

Client education

The major areas of importance in client education are:

- The nature of the syndrome – first, and crucially, the fact that what seems to be a local problem is often a manifestation of a generalised condition; second, that the underlying problem cannot always be cured; and third, that the local (secondary) consequences of otitis have to be addressed as well. The client must be informed about the possibility of chronic, proliferative otitis and the need to avoid this. Proper education will allow the client to understand the need for an in-depth assessment in some cases, and the need for follow-up examinations[36].
- Correct methods of applying topical medication and cleaners for use at home[59].

Follow-up

Follow-up checks should include progress reports from the owner and otoscopic and cytological examination. Initially, visits should be scheduled every 2 weeks[49], to monitor therapeutic response. Treatment often needs to change over time – initial response may not be adequate or initial therapeutic intervention may differ from long-term preventive or maintenance management. Owners and veterinarians should be aware that recurrence may be long delayed, and that a short-term improvement does not necessarily mean that the otitis is cured. In 1 study, the average time to recurrence was 3.6 months[56].

Preventive and maintenance therapy

Ongoing management is critically dependent on identifying the underlying cause(s) and on proper owner education, as well as on repeated evaluation. Cleaning and drying agents are often part of maintenance/prevention therapy (see Table 4). Long-term interventions are dependent on underlying causes[17,30,53,59].

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