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CLINICAL REVIEW

HEARING DISORDERS IN CATS
Classification, pathology and diagnosis

George M Strain

Practical relevance: Auditory function is a sense that is central to life for cats – being important in situational awareness of potential predators, pursuit of prey, and for communication with conspecifics, humans and other species. Deafness in cats is most frequently the result of a genetic disorder, strongly associated with white fur and blue eyes, but may also result from acquired causes such as advancing age, ototoxic drugs, infection, environmental noise and physical trauma. Deafness can be sensorineural, where there is loss of cochlear hair cells, or conductive, where sound is muffled on its way to the inner ear.

Clinical challenges: Establishing whether a cat is deaf can be difficult as behavioral testing of hearing is subjective and does not reliably detect unilateral deafness. Brainstem auditory evoked response testing is an objective measure but is limited in its availability. Currently, sensorineural deafness is irreversible because no treatments are available to restore lost hair cells. Conductive hearing loss can usually be treated, although full hearing recovery following otitis media may take weeks as the body clears the middle ear of debris.

Evidence base: The author draws on the published literature and his extensive research on clinical aspects and molecular genetics of deafness, principally in companion animals, to review types and forms of deafness in cats. He also discusses current diagnostic approaches and provides brief advice for managing cats with hearing loss.

Auditory function, along with vision, is a sense that is central and critical to life for cats. It is important in situational awareness of potential predators, for pursuit of prey, and in communication with conspecifics, humans, and other species, among numerous other activities. Impairment or loss of auditory function is at best an inconvenience, but it can be life-threatening due to undetected dangers such as motor vehicles and predators.

Anatomy and physiology of hearing

In the normal ear, vibrations of sound in the ear canal – alternating compressions and rarefactions – produce vibrations of the tympanic membrane. These vibrations are transmitted by the three ossicles of the middle ear (malleus, incus and stapes) to the oval window of the inner ear (Figure 1), becoming amplified in the process. The vibrations are in turn transmitted into the inner ear as a standing wave going up the fluid-filled scala tympani to the apex and back down the scala vestibuli (which is continuous with the scala tympani) to the round window, which deflects 180° out of phase with the oval window. The

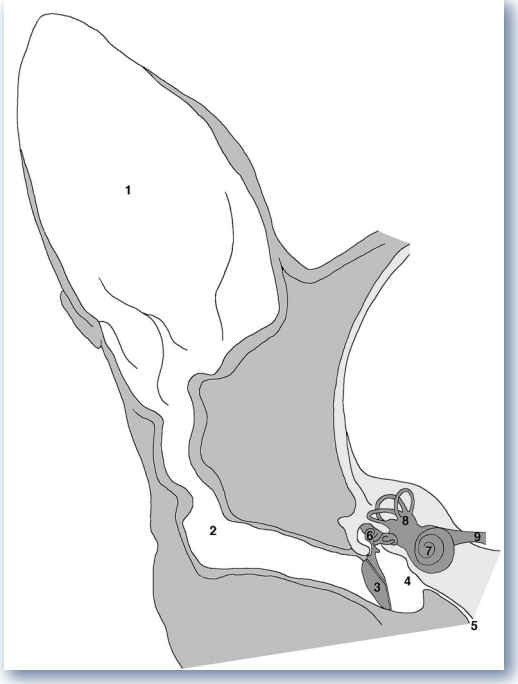

Figure 1 **Structures of the feline ear. 1 = pinna; 2 = external ear canal, consisting of a vertical canal and a horizontal canal; 3 = tympanic membrane; 4 = bulla/middle ear space; 5 = auditory tube; 6 = middle ear ossicles (malleus, incus and stapes); 7 = cochlea; 8 = vestibular structures; 9 = cranial nerve VIII.** *Reproduced from Ryugo and Menotti-Raymond,[1] with permission*

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Audible frequencies

The frequency range of hearing in cats, determined from behavioral testing, spans from 45 Hz to 65 kHz (Figure 2),[2–7] based on the criterion of the lowest and highest frequencies detected at a maximum stimulus intensity of 60 dB sound pressure level (SPL). For comparative purposes, the hearing range for dogs is 67 Hz to 45 kHz, and for humans is 64 Hz to 23 kHz.

The frequency specificity of the hair cells on the basilar membrane is mapped out along its length, with high frequency sounds detected near the base of the cochlea, near the oval window, and low frequency sounds detected at the apex of the cochlea. While hair cells can stimulate the axons of the spiral ganglion cells that they synapse upon to directly transmit low frequency sound information to the brain by one-to-one action potentials, the cells are unable to generate action potentials quickly enough to faithfully follow high frequency sounds. The concept that sound frequency content information is primarily conveyed based on where on the basilar membrane the hair cells were stimulated is known as the place theory of sound perception.

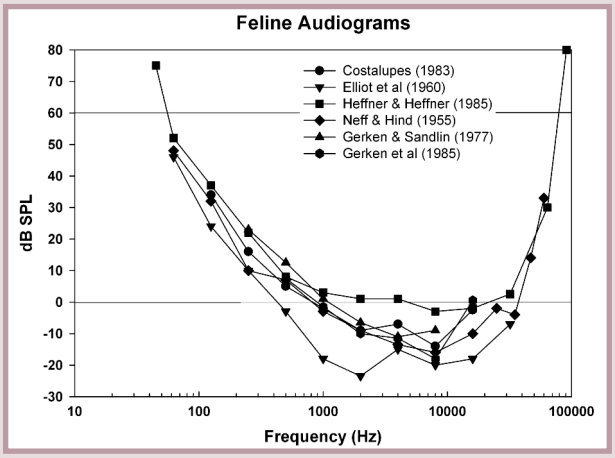

Figure 2 Feline audiograms compiled from published sources.[2–7] Hearing range is usually based on the frequency range for responses below 60 dB SPL, and is typically cited as 45 Hz to 65 kHz

The frequency range of hearing in cats spans from 45 Hz to 65 kHz (compared with 67 Hz to 45 kHz for dogs, and 64 Hz to 23 kHz for humans).

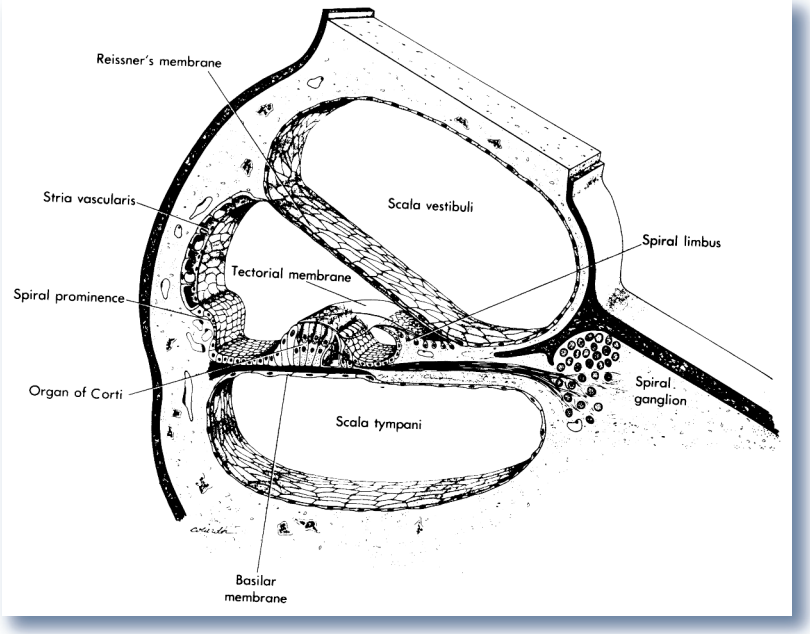

Figure 3 Structures of the cochlea in cross-section. Inner hair cells in the organ of Corti are the primary sensory transduction cells, while the outer hair cells utilize active processes to increase or decrease sensitivity. Pigment cells in the stria vascularis play a major role in maintaining high potassium levels in the cochlear duct to support hair cell viability. Hair cells synapse on neurons of the spiral ganglion, which in turn become components of cranial nerve VIII. *Reproduced from Bloom and Fawcett,[8] with permission*

pressures in the scala tympani and scala vestibuli cause deflections of the basilar membrane in the scala media (cochlear duct), upon which sits the organ of Corti that contains the sensory hair cells that ultimately transmit information about the sound into the brain via cranial nerve VIII (Figure 3).

The organ of Corti consists of one row of inner hair cells and three rows of outer hair cells that map out along the basilar membrane as it coils around in the snail-shell shaped cochlea. The inner hair cells are primarily sensory in function, while the outer hair cells primarily serve as an active system that can increase or decrease sensitivity. In addition to the outer hair cells providing a means to decrease hearing sensitivity, the brain also has a mechanism to protect the cochlea against loud sounds through the actions of two muscles in the middle ear: the tensor tympani and stapedius muscles. These muscles contract as a reflex response to loud sounds (and prior to vocalizations or in response to touch) and, in so doing, dampen the transmission of sound through the ossicles. This protective reflex is not fast enough, however, to guard against percussive sounds like gunfire.

The organ of Corti sensory cells – the hair cells – have stereocilia on their top surface that project upward into a gelatinous structure in the cochlear duct known as the tectorial membrane, and are surrounded by a potassium-rich fluid called endolymph (Figure 3). The stereocilia on each hair cell are arranged in a chevron pattern, pointing from the shortest on one edge of the top surface to the tallest at the opposite edge (Figure 4). Each stereocilium is attached to adjacent stereocilia by fine microfilaments that are in turn attached to potassium ion channels. When the basilar membrane is deflected upward by a sound wave, the hair cell stereocilia are pressed against the tectorial membrane, bending them and putting tension on the microfilaments. The tension opens mechanically gated potassium channels, allowing a large influx of potassium ions from the endolymph; this influx depolarizes the inside of the hair cell.

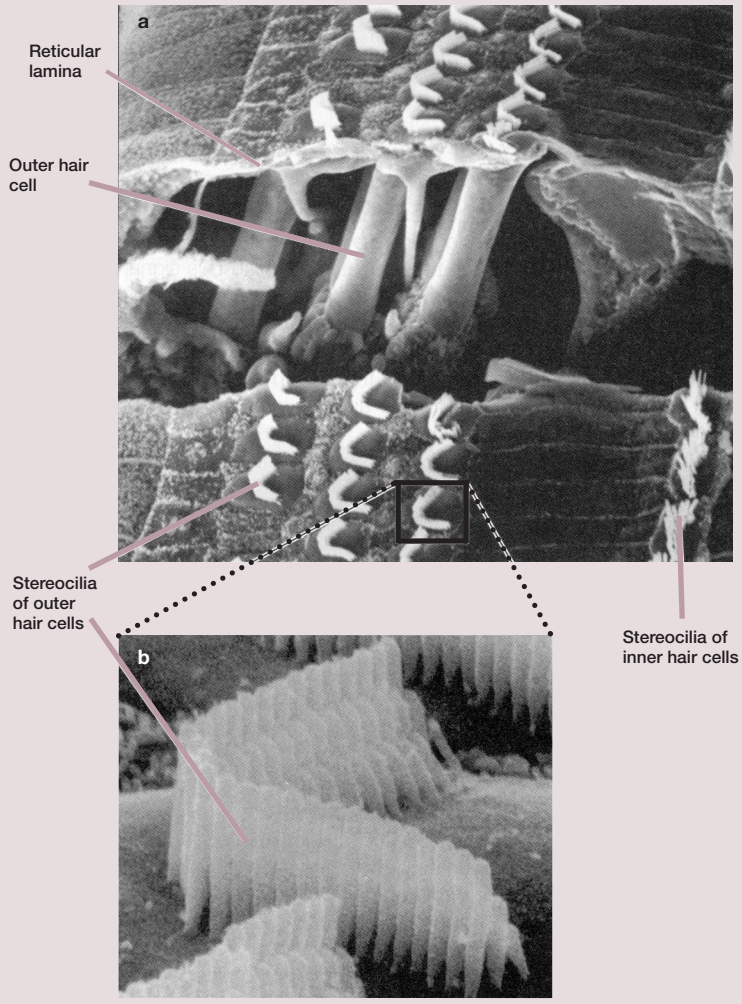

Figure 4 Scanning electron micrographs of cochlear hair cells. (a) Hair cells and their stereocilia. (b) Higher magnification view of the stereocilia on an outer hair cell. The stereocilia are approximately 5 μm in length. *Reproduced from Bear et al,[9] with permission*

The potassium-mediated depolarization in turn opens voltage-gated calcium ion channels, and the resultant calcium influx further depolarizes the hair cell and triggers the release of the excitatory neurotransmitter glutamate onto afferent spiral ganglion neurons that ultimately join to become cranial nerve VIII.

The high potassium levels in the endolymph fluid surrounding the hair cells are maintained by a modified vascular structure called the stria vascularis, located on the lateral wall of the cochlear duct. Important cellular components of the stria vascularis are melanocytes that migrated embryologically from the neural crest, and which play a vital role in maintaining the elevated potassium levels in the endolymph. Damage to hair cells can produce deafness as a primary event, but damage to the stria vascularis can also produce deafness, with hair cell death occurring secondarily to the strial degeneration and loss of potassium in the endolymph.

Forms of deafness

Numerous authors have discussed or examined the condition of deafness in white cats, dating back to as early as 1829[10] and including Darwin in 'The origin of species'.[1,10–33] However, many forms of deafness are possible, which creates the need for a classification system using a variety of criteria.

Hearing loss can be variously classified as:[34]

- **Unilateral** or **bilateral**, based on whether one or both ears are affected;
- **Partial** or **total**, based on the extent of hearing loss for a given ear;
- **Syndromic** or **non-syndromic**, based on whether other system diseases or phenotype abnormalities accompany the hearing loss;
- **Peripheral**, involving the outer, middle or inner ear; or **central**, where the pathology is retrocochlear.

Peripheral deafness can in turn be classified based on three pairs of criteria:

- **Hereditary** or **acquired**;
- **Congenital** or **late-onset**;
- **Sensorineural**, where cochlear hair cells die, or **conductive**, where loss results from reduced sound transmission to the cochlea.

These last three criteria combine into eight possible combinations including: hereditary congenital sensorineural deafness (the most common type in cats); acquired late-onset conductive deafness (probably the second-most common type); acquired late-onset sensorineural deafness; and others (Table 1).

Not all of these types of peripheral deafness have been reported in cats. Hereditary congenital sensorineural deafness – the only type of hereditary deafness reported in cats to date – is most commonly associated with white fur, especially in cats with blue eyes. Acquired late-onset conductive deafness is most commonly associated with otitis media and/or externa. Acquired late-onset sensorineural deafness is most commonly associated with drug ototoxicity (eg, gentamicin) or age-related hearing loss (presbycusis).

Table 1 Types of peripheral hearing loss

Deafness type	Possible cause(s)	Frequency
Hereditary congenital sensorineural	White pigmentation gene (*W*)	Common
Hereditary congenital conductive	Not reported in cats	–
Hereditary late-onset sensorineural	Not reported in cats	–
Hereditary late-onset conductive	Not reported in cats	–
Acquired congenital sensorineural	Prenatal ototoxic drug exposure	Uncommon
Acquired congenital conductive	Cartilage defect, canal atresia	Uncommon
Acquired late-onset sensorineural	Otitis interna, drug toxicity, presbycusis, environmental noise	Moderate
Acquired late-onset conductive	Otitis externa/media, canal stenosis	Moderate

Why are white cats deaf?

Figure 5 Deafness in blue-eyed white cats is perhaps the most recognized form of deafness in animals. ©iStock.com/Pley

White cats, especially those with blue eyes (Figure 5), are deaf as a consequence of the actions of the dominant pigment gene *W*, which acts by suppressing melanocytes. In the absence of melanin from melanocytes, the skin and hair are white. If the *W* gene acts strongly, it also suppresses melanocytes in the iris, producing an iris that looks blue because of the absence of melanin. Strong action by *W* also suppresses melanocytes in the stria vascularis, the modified vascular structure on the outer wall of the cochlea. This causes degeneration of the stria, followed by degeneration of the hair cells of the cochlea, and deafness within the first weeks of life.

Hereditary deafness

Pathology of hereditary deafness

The forms of hereditary deafness can be separated based on the type of pathology present. Steel[35–37] developed a classification system with three types of pathology that cover observed forms of deafness. These include a morphogenetic pathology and two types of degenerative pathology, as described below.

Morphogenetic

This category includes all structural abnormalities of the membranous and bony labyrinths, which at its extreme includes a complete absence of auditory and/or vestibular components. The effects of this pathology on hearing can vary from profound deafness to no hearing loss that nevertheless is accompanied by effects on vestibular structures. This rare condition is essentially a major birth defect.

Neuroepithelial

This pathology presents with a loss of cochlear hair cells as the primary event, beginning when normal histological development of the organ of Corti is finishing following birth. Outer hair cells degenerate before inner hair cells, beginning in the upper part of the basal turn and progressing to both the apex and the base.[35] Supporting cells of the organ of Corti collapse, leaving the basilar membrane and dedifferentiated cells (ie, those that have reverted back to their undifferentiated state). The stria vascularis and Reissner's membrane are initially unaffected and any or all vestibular structures can be affected. The expression of this pathology is typically bilateral. An endocochlear potential (voltage measured between the cochlear duct and the exterior of the cochlea, and produced by potassium secretion by the stria) may still be present in the endolymph of the cochlear duct because of continued potassium secretion by the stria vascularis.[38]

This pathology has been reported in mice,[39] guinea pigs,[40,41] dogs[42–44] and humans.[45] It may also occur in cats based on reports of the presence of deafness with coexisting vestibular pathology,[46,47] but this has not been documented histologically.

Cochleosaccular

This condition, also known as Scheibe type pathology, results from an initial degeneration of the stria vascularis on the outer wall of the cochlear duct, proceeding to degeneration of hair cells, and collapse of Reissner's membrane. It may occasionally also include collapse of the saccule, with collapse of the saccule wall and macular damage but without any effect on the remainder of the vestibular structures. The expression of this pathology is more often unilateral, but both ears can be affected. No endocochlear potential remains due to the strial degeneration. The initial strial degeneration is usually linked to the absence of functioning melanocytes, and as a result may be described as pigment-associated, often also linked to lack of pigmentation in the skin, iris and tapetum lucidum.

Cochleosaccular pathology has been reported in the white cat,[21,22,48] the Dalmatian[49–51] and other dog breeds,[52,53] Hedlund white mink,[54,55] several mouse mutants,[56,57] and as Waardenburg syndrome in humans.[58] As background reference, the normal postnatal maturation of the stria vascularis has been described for the dog[59] and the functional morphology of melanocyte types has been described for the mouse.[60] Unilateral congenital deafness has been shown to produce minor alterations in the ventral cochlear nucleus in rats[61] and cerebral cortex in cats,[62–64] but the changes are not sufficiently extensive to affect brain function except for localization of sound sources.

Hereditary congenital sensorineural deafness is the most common type of hearing loss in cats.

Genes associated with hereditary deafness

Along with deafness in Dalmatians, deafness in blue-eyed white cats is perhaps the most recognized form of deafness in animals. Most hereditary deafness in cats involves cochleosaccular pathology, although some neuroepithelial pathology associated with vestibular signs may also exist. Whether two genes or a single gene is responsible for white pigmentation in the cat is unsettled, as discussed on page 280.[65]

Genetics of white coloration in the cat

The cochlear pathology in white cats has been shown to be cochleosaccular in numerous studies,[17,19–26] but a more recent study has indicated that more than one type of cochlear pathology may exist.[1,29] It has also been suggested that the hearing loss in a given ear is not always complete,[25,66] but this has not been well documented and total deafness is the norm.

Gene loci

The Mendelian locus assignments for white color in cats are unsettled. Early publications described two dominant loci: *dominant white* (*W*) and *white spotting* (*S*).[32,67] *W* is autosomal dominant over color and is not related to albinism. Cats are all or mostly white, with some exhibiting a colored spot on the head that may fade or disappear with age. White animals can be *WW* or *Ww*, but the homozygous cats do not appear to develop other sensory disorders, in contrast to dogs homozygous with the merle gene, where ocular defects may coexist. Deafness can be unilateral or bilateral, and one or two blue eyes are frequently present, with the likelihood of deafness increasing in animals with two blue eyes.[32] Non-*W* cats with any hair and skin pigmentation color present can have blue eyes from the c^s Siamese dilution pigment locus,[34] but deafness does not appear to be associated with this locus. Most early deafness studies were of cats with the *W* locus.

The white spotting locus (*S*) presents with (1) homozygous recessive full solid non-white color (*ss*), (2) heterozygous bicolor or ventral white (*Ss*), which is dominant to solid non-white color, or (3) the homozygous dominant van color pattern (*SS*), named for cats from the Lake Van region in Turkey, where the only color is on the head and tail.[68] Blue eyes are not typically seen and deafness has not been reported to be associated with *S*.

Dominant white (*W*) may be allelic to *white spotting* (*S*), where different mutations at the same locus cause a similar phenotype, but possible epistasis (where the effect of one gene is determined by the presence of modifier genes) has complicated adoption of a consensus terminology. Recent thinking has combined the two loci into a single white spotting locus (*W*) that has pleiotropic effects (one gene affecting two or more phenotypic traits) of complete penetrance for a solid white coat and incomplete penetrance for deafness and blue iris color.[34] The locus has four alleles: three dominant and one recessive. The dominant alleles include solid white (*W*) > high degree of spotting white (W^h) > low degree of spotting white (W^l). The single wild-type recessive allele produces solid color (*ww*). W^h and W^l are equivalent to *SS* and *Ss*, respectively. This terminology structure is supported by recent genomic studies (below) and will probably become the accepted view.

Deafness prevalence

Three studies of non-purebreed white cats (n = 256) found a deafness prevalence of 50% (12.1% unilaterally deaf and 37.9% bilaterally deaf).[17,20,21,69] In white cats born from two white parents, the combined (unilateral and bilateral) prevalence was 52–96% across the studies. Combined deafness prevalence was 85% or 65% in cats with two blue eyes, 40% in cats with one blue eye, and 22% or 17% in cats with no blue eyes. The deafness in white cats can be partial or complete for a given ear.[21] There is little published prevalence data for purebreed cats, though there is some limited data available for three breeds,[66] including both white and colored cats, based on behavioral tests. Prevalence was reported as 18% in Norwegian Forest Cats (n = 329), 17% in Maine Coon cats (n = 134) and 11% in Turkish Angora cats (n = 474).[66] Iris color and coat color distributions were not reported, and percentages are likely underestimates because behavioral testing would not have identified unilaterally deaf cats. Another study of deafness prevalence in 84 white cats from 10 purebreeds found similar prevalence rates of 20.2% affected (9.5% unilaterally deaf and 10.7% bilaterally deaf) based on brainstem auditory evoked response (BAER) testing.[70] Deaf cats were seen in six of the 10 breeds: Turkish Angora, British Shorthair, Maine Coon, Norwegian Forest Cat, Persian and Foreign White. No sex differences were seen, but blue-eyed cats were more likely to be deaf than non-blue-eyed cats.

Whether two genes or a single gene is responsible for white pigmentation in the cat is unsettled.

A study of a cat colony with mostly white cats (n = 104) attempted to determine deafness inheritance using the statistical technique of complex segregation analysis.[66] The authors concluded that deafness was inherited as a pleiotropic gene segregating for deafness and blue irises with other polygenic effects.

Molecular genetics

A microsatellite marker study identified a locus for *S* near the *KIT* gene on feline chromosome B1 and ruled out the gene *EDNRB* from playing a role in white coloration.[68] Subsequent studies[71–74] confirmed *KIT* as the responsible gene and demonstrated that both *dominant white* and *white spotting* resulted from insertions of *feline endogenous retrovirus 1* (*FEV1*) in intron 1 of *KIT*. Endogenous retroviruses are copies of exogenous retroviral genomes inserted into the host genome from ancestral infections and integrated into the genome.[73] The white spotting pattern resulted from insertion of the full-length 7125 bp *FEV1*, while the dominant white pattern resulted from insertion of a 623 bp fragment of *FEV1* at the same position. The retrotransposon insertion responsible for the merle locus in dogs is a similar example of a retroviral insertion affecting pigmentation.[75,76] A specific gene mutation responsible for deafness in white cats has nevertheless not yet been identified in *S* or modifier genes.

Neuroepithelial deafness

Cases of combined deafness and vestibular disease have been reported in Siamese and Burmese kittens,[46,47] but little is known about neuroepithelial deafness in cats. Vestibular signs appear at or shortly after birth, are non-progressive, and may disappear with time. In some cases, deafness was determined based on behavioral tests; BAER testing was not routinely performed and histological studies were not reported.

Acquired deafness

Acquired hearing loss is much less common than hereditary deafness in cats. The most frequently encountered form is due to ototoxicity; other forms include presbycusis, and deafness due to infection, anesthesia, environmental noise or physical trauma.[77]

Ototoxicity

A variety of drugs and other chemicals have been demonstrated to have toxic effects on the inner ear, affecting auditory and/or vestibular function.[78–83] The most prominent of these are the aminoglycoside antibiotics,[84–88] antineoplastic agents,[89] diuretics and antiseptics.

Aminoglycosides

Aminoglycosides are the most frequently used antibiotics worldwide for treating gram-negative infections in humans, and are likewise heavily used in veterinary medicine. This drug class includes gentamicin, tobramycin, neomycin, kanamycin, amikacin and streptomycin. Gentamicin is one of the most commonly used antibiotics for topical treatment of otitis externa.

The side effects of aminoglycosides are nephrotoxicity (often reversible) and ototoxicity (both cochlear and vestibular, usually irreversible), and can result from systemic or topical application. Toxicity usually develops after long-term, high-dose administration, but is unpredictable and may occur after a single high dose[90] or long after cessation of treatment. Three weeks of twice daily topical otic treatment in dogs produced no observable BAER changes with either intact or excised tympanic membranes.[91] Perinatal cats are less susceptible to aminoglycoside toxicity than older animals, but still may be affected.[92]

Studies in recent years have revealed the sequential mechanisms of ototoxicity to be iron chelation, followed by free radical formation, and thereafter caspase-dependent apoptosis.[93] Co-administration of aspirin[94] or other antioxidants such as D-methionine or N-acetylcysteine with gentamicin has been shown to reduce or prevent gentamicin ototoxicity, but the clinical utility of such treatments in veterinary practice has not been demonstrated. Other possibly protective antioxidants include deferoxamine, dihydroxybenzoic acid, glutathione and α-lipoic acid.[83]

Different aminoglycoside antibiotics vary in their effects on the cochlea and vestibular structures; as a result, vestibular signs may be more prominent than auditory deficits. The auditory deficits, which are progressive, are frequently not noticed by owners or clinicians until significant damage has occurred. In addition to gentamicin, streptomycin, neomycin and others in this drug class may cause hearing loss.

Aminoglycoside toxicity usually results after long-term, high-dose administration, but is unpredictable and may occur after a single high dose or long after cessation of treatment.

Antineoplastic agents

Chemotherapeutic agents such as cisplatin and carboplatin produce irreversible hearing loss by their actions on hair cells and on the stria vascularis, but the vestibular system is not affected. The mechanism is thought to be through production of reactive oxygen species. Co-administration of antioxidants may provide some protective effect when drug use is deemed necessary.

Diuretics and other drugs

Loop diuretics, especially furosemide and etacrynic acid, can produce hearing loss, but the loss is usually temporary. However, diuretics can potentiate the ototoxicity of aminoglycosides and antineoplastics.[83] The mechanism is probably related to effects on ion transport by the stria vascularis. Salicylates and quinine can also produce hearing loss, which is often accompanied by tinnitus, but the effects are usually reversible with discontinuation of the drug.

Diuretics can potentiate the ototoxicity of aminoglycosides and antineoplastics.

Antiseptics

The antiseptic chlorhexidine has demonstrated permanent ototoxicity. This was especially prevalent when chlorhexidine was marketed in an otic preparation, but as with other ototoxic products the toxicity was not consistent.[95] Removal of the otic indication from the product has reduced the prevalence of this toxicity, but some practitioners still apply dilutions of other chlorhexidine formulations for otic use as a misguided economy measure.

Presbycusis

Presbycusis refers to reduced auditory perception with advancing age.[96–98] The mechanisms are primarily sensorineural, although conductive middle ear effects may contribute, and are progressive.[96] The mechanisms are also primarily peripheral, although there may be central changes as a component.

Little research has been published on presbycusis in animals,[99–103] and no feline clinical studies appear to have been reported. One long-term study of dogs using tone-evoked BAER demonstrated a progressive loss of hearing in aging dogs, with middle and high frequencies affected first.[100]

It is highly likely that presbycusis develops in a similar pattern in geriatric cats, but more study is required. Cats are considered to be geriatric based on when they are most likely to start having diseases associated with aging: >11.88 + 1.94 years, according to one source.[104] Another source classifies ⩾15 years of age as being geriatric and 11–14 years as being senior.[105] Based on what is known in dogs, the onset of losses associated with presbycusis might be expected to occur before the cat reaches geriatric age. However, recent unpublished data from the author's laboratory show that presbycusis is uncommon in cats in their teen years, and, if present after that age, is much less severe than in dogs.

The presence of presbycusis may contribute to problem behaviors in older animals.[106,107]

Other forms

A variety of other, less common forms of hearing loss exist, although the cause of acute-onset hearing loss is often never identified.

✣ **Infection** Otitis interna, often accompanied by signs of vestibular disease, can result in sensorineural deafness that is irreversible. Appropriate treatment should be pursued to forestall hearing loss.[108]

✣ **Anesthesia** On rare occasions, cats (and dogs) awaken from general anesthesia with bilateral deafness, typically following dental procedures.[109] The etiology is unclear and may involve multiple factors. In some cases manipulation of the jaw may result in inflammation and occlusion of the auditory tube, leading to middle ear effusion of mucus secretions from the lining of the middle ear, which becomes dessicated and retained to produce conductive hearing loss. This can often be addressed surgically. It has also been suggested that distraction of the cat's lower jaw with a mouth gag might restrict maxillary artery blood flow, resulting in cochlear hypoxia. A study in anesthetized cats found some indicators of reduced maxillary blood flow, but no cases of deafness were observed.[110]

✣ **Environmental noise** Noise trauma, or noise-induced hearing loss, increasingly affects humans and animals. As discussed earlier, reflex contractions of the tensor tympani and stapedius muscles are protective mechanims to dampen noise transmission in the middle ear, but provide no protection against percussive sounds such as gunfire. The effect of the noise may be a temporary threshold shift, with hearing recovering in hours to days, or a permanent threshold shift that is irreversible, in which case the hearing loss is cumulative, primarily affecting the hair cells. It is not clear that noise trauma is an issue for cats, but guidelines established by the US Occupational Safety and Health Administration for permissible human exposure provide a reasonable standard where environmental noise levels may be a concern, such as in animal shelters.[77,111,112]

✣ **Trauma** Physical trauma can result in hearing loss, which is usually conductive. Clinical localization of the site of trauma can indicate this source.

✣ **Other disease processes and anatomic defects** Conductive deafness can also result from inflammatory changes associated with chronic otitis externa, polyps or other masses in the ear canal or bulla,[113] or palatine defects.[114] Surgical correction can often restore hearing; even partial or total resection of the ear canal can result in restoration of partial hearing if cochlear function was intact prior to the surgery.

Diagnosis of hearing loss

A cat presented with suspected hearing loss should first have otitis externa and otitis media ruled out by otoscopic examination, and otitis interna ruled out to the extent possible by testing for sensitivity to palpation of the ear and the presence of vestibular signs. Factors that can suggest other etiologies are age (presbycusis), skin/hair/iris color (white/white/blue, hereditary), and a history of recent treatment with potentially ototoxic drugs (especially gentamicin), noise exposure or trauma.

A cat with suspected hearing loss should first have otitis (externa, media or interna) ruled out.

Limitations of behavioral testing of hearing

Behavioral testing of hearing is subjective, can be unreliable and will typically not detect unilateral deafness. The testing can be performed by producing sounds outside the visual fields of the animal and observing for a Preyer's reflex (startle response or ear movement) or head movement. Unfortunately, deaf animals may detect test stimuli through other sensory modalities, and hearing animals stressed in the exam room may fail to respond, or may quickly cease responding after recognizing that the stimulus is of no consequence. A deaf animal will fail to waken in response to a loud noise that doesn't activate other senses, but this will not identify unilaterally deaf animals, unless sleeping with the good ear to the ground. Animals with unilateral deafness will initially always turn to the hearing side in response to sound, but many adapt and this distinction then fails to be reliable.

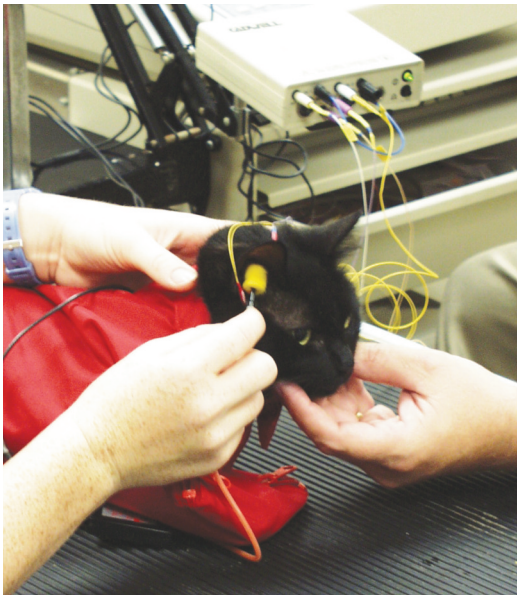

Figure 6 Cat gently restrained, with electrodes in place, and BAER testing of the right ear being performed

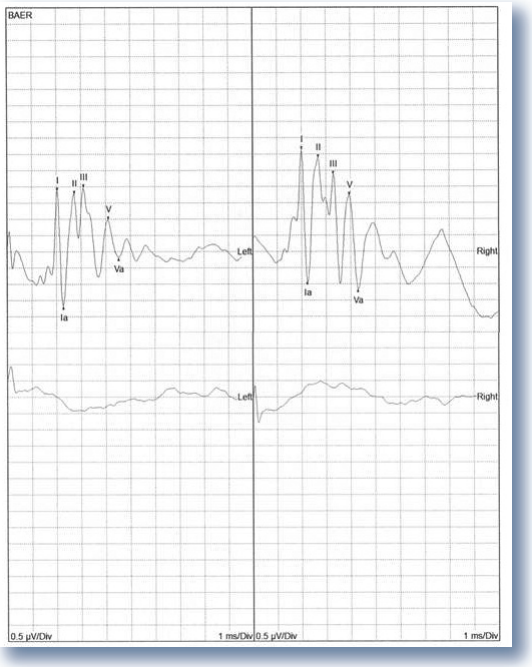

Figure 7 (top) BAER recording from a 2-year-old normal cat, with peaks I, II, III and V labeled on the tracing. Peak I is generated by cranial nerve VIII entering the brain, while the later peaks are generated in the brainstem. (bottom) By comparison, this essentially flat line is a recording from a bilaterally deaf cat. 0.5 µv/div and 1 ms/div

BAER testing

Objective testing of hearing is performed using the BAER (brainstem auditory evoked response), also known as the auditory brainstem response and several other variants.[115–118] This test can be performed both in awake and unconscious cats and is unaffected by drugs (except ototoxic drugs). An auditory stimulus is inserted into an ear and the response of the auditory pathway is detected by subdermal needle electrodes placed on the cranial vertex and outside the ear canal (Figure 6). The response is a series of peaks that occur within about 5 ms. The amplitude of the response is small enough (microvolts) that responses to multiple stimuli must be averaged to remove the confounding effects of physiologic (electromyogram and electroencephalogram) and other unrelated biologic and external signals that can mask the response. The stimulus typically used is a click sound, which contains all but the highest of the audible frequencies in the cat. Pure tone stimuli may also be used if attempting to obtain an audiogram of thresholds at different frequencies, but anesthesia is required and the process is time-consuming.

In the clinical setting, the BAER is used most commonly to screen for hearing or deaf ears, since pigment-associated hereditary congenital deafness nearly always presents as either total deafness in an ear or a normal response. The BAER recording of a deaf ear is essentially a flat line (Figure 7). The extent of partial hearing loss, when present, is very difficult to assess objectively with the BAER test. Response peak latencies increase and peak amplitudes decrease with increasingly softer stimuli or with progressively greater hearing loss, but due to test–retest variability and subject-to-subject variability it is unsafe to attempt to quantify partial hearing loss in the manner that is done in human subjects.

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The availability of BAER testing is limited because of equipment costs. A partial listing of available test sites, both within and outside of North America, is available online.[119]

Other tests of auditory function

Other tests of auditory function are available, but are less commonly utilized.[120] One of these involves otoacoustic emissions (OAEs), where the ear generates sounds, either spontaneously or in response to introduced test stimuli. OAEs are thought to reflect the function of cochlear outer hair cells, which contain contractile proteins that allow the cells to shorten or lengthen at a rapid frequency. Spontaneous OAEs are present in most normal ears at very low levels, and can occasionally be loud enough to be detected without amplification by the listener.[121] Evoked OAEs provide a sensitive early indicator of loss of auditory function, but only of the outer hair cells by definition.

The evoked OAE that has proven most useful in humans is the distortion product OAE (DPOAE). Two pure tones (f_1 and f_2, $f_2 > f_1$) are introduced into the ear canal and the resulting pure tone – the distortion product – is recorded at a frequency of $2f_1–f_2$. The frequency span of the cochlea is tested by varying f_1 and constraining the frequency

ratio to $f_2/f_1 = 1.21$, a ratio proven to produce the strongest response.[122] A response to a given test frequency pair that falls below the noise threshold may indicate loss of function. DPOAEs are increasingly being used in the veterinary setting, especially when it is desired to know what audible frequencies may be affected.[103,123,124]

An alternate test is the transient evoked OAE (TEOAE), where a transient evoked response to a series of wideband clicks or chirps is recorded. DPOAEs evaluate a wider frequency range (above 10 kHz) than do TEOAEs, and might be preferred in species like the cat (or dog) with a higher frequency hearing range, albeit offer less sensitivity to minor and subclinical conditions. A failed OAE test typically mandates referral for BAER testing, so a lower instrument cost and briefer testing time may be offset by the need for further tests.

Middle ear and auditory tube function can be assessed using tympanometry (or impedance audiometry), which is a widely accepted and used technique in human medicine. This non-invasive method of examining the function of the middle ear involves varying the atmospheric pressure in the external ear canal and inferring the amount of sound energy that is transmitted through the tympanum by measuring the reflected sound energy.[120,125,126] Pressure is typically varied from –400 to +200 daPa (decaPascals or mmH_2O) while a continuous 226 Hz tone is presented, and the intensity of reflected sound is measured and plotted as a function of ear canal pressure. The resulting graph displays the compliance (or static acoustic impedance or admittance) of the tympanum. Variations from the normal response graph can indicate ossicle disarticulation, tympanum scarring or otosclerosis, middle ear fluid, tympanum rupture or auditory tube dysfunction.

Treatment approaches and limitations

The treatment approach is dictated by the form of deafness or mechanism responsible for the hearing loss.

Conductive hearing loss can usually be treated by mechanical (ear canal cleaning), surgical or antibiotic approaches. Full hearing recovery following otitis media may take a few weeks as the body clears the middle ear of debris.

Sensorineural deafness, whether due to genetics, aging, ototoxicity or noise, cannot be treated because no treatment modalities are available at present to restore lost hair cells. Current research suggests that this may be possible in the future.

Cats with suspected hereditary deafness should not be used for breeding.

Management of cats with hearing loss

Cats with unilateral deafness or partial hearing loss require no specific management, except that those with suspected hereditary deafness should not be used for breeding and those with acquired hearing loss should have the causative source removed or eliminated where possible.

With bilaterally deaf cats, the primary concern is protection from undetected dangers such as motor vehicles or predators.[127,128] Preferably these cats should be kept as indoor pets since it can be difficult (although not impossible)[129] to constrain cats with yard fencing and hence to protect them from threats. Some deaf cats with outdoor privileges have been trained to return in response to flashing porch lights, but behavioral training is notoriously challenging in cats. The danger to infants and toddlers from reflex bites or scratches inflicted when a deaf cat is startled should be kept in mind.

The use of gesture communication, including canine obedience training signals, American Sign Language signs, and others, has been found to be effective in deaf dogs.[127] Deaf animals are naturally more visually attentive to humans, making them amenable to training in this form of communication. Owners of deaf cats may, with patience, be able to train their pets with this system.

KEY POINTS

- The most common form of deafness in cats is congenital and hereditary, and associated with white pigmentation, especially in white cats with blue eyes.
- Ototoxicity, frequently from aminoglycoside antibiotics like gentamicin, can be unpredictable and often results in total and irreversible deafness before the owner or veterinarian is aware of hearing loss.
- Behavioral testing of hearing is often unreliable. The brainstem auditory evoked response (BAER) test is the most reliable assessment tool, but access to testing facilities is limited.
- Sensorineural deafness is not currently treatable. Conduction deafness from obstructions of the outer or middle ear may be reversed.

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