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Anatomy & Physiology

The middle ear consists of the tympanic membrane, which terminates the ear canal, and the three small ossicles, the malleus, the incus and the stapes. The tympanic cavity is air filled and lined with respiratory epithelium. The Eustachian tube connects the middle ear cavity to the pharynx. Sympathetic nerves form a plexus on the promontory in cats and are vulnerable for trauma during middle ear surgery. The facial nerve travels through the incomplete facial canal before exiting the stylomastoid foramen and is therefore exposed to the middle ear cavity. The major function of the middle ear is to match relatively low impedance airborne sounds to the higher impedance fluid of the inner ear.

The inner ear is located within the osseous labyrinth of the petrous part of the temporal bone. The membranous labyrinth consists of three parts: the cochlea, vestibule and the semicircular canals. The vestibule is divided into an utricle and a saccule, called the otolith organs. The sensory transduction occurs in the organ of Corti, which is situated in the scala media en separated from the scala vestibuli and the scala tympani by Reissner’s membrane and the basilar membrane respectively.

It is here where the hair cells interact with supporting elements to convert fluid waves into the bending of hair bundles and resultant ion influxes.

The release of neurotransmitter from the basal portions of stimulated hair cells leads to neural impulses, action potentials. Once the nerve impulse is generated in the cochlea, the signal travels along the acoustic nerve to the cochlear nuclei. From here, many projections lead to the olivary nuclei at the same level. The axons of the olivary neurons project via the lateral lemniscus to the inferior colliculi, where they synapse on neurons that project to the primary auditory cortex.

The vestibular system functions to maintain the position of the eyes, trunk, and limbs relative to the position of the head, responding to linear and rotational acceleration and tilting.

Clinical signs of middle- and inner ear diseases and clinical examination

Clinical signs associated with middle ear disease often reflects concurrent otitis externa, especially in dogs. Otalgia (otic pain), lethargy, inappetence and pain on opening of the mouth are more suggestive of middle ear involvement. Neurologic signs like facial nerve paresis or paralysis or Horner’s syndrome may be present. Peripheral vestibular ataxia (head tilt, horizontal or rotary nystagmus, circling or falling toward the side of the lesion) is usually the most obvious sign of inner ear disease. Clinical signs of cochlear damage usually go unnoticed until complete deafness is recognized.

The diagnosis of middle- and inner ear disease is based on a thorough history and physical, neurologic, and otoscopic examinations. Increased opacity and hyperemia of the tympanic
membrane may be present with otitis media. A paracentesis can be performed in dogs with an intact tympanic membrane to obtain samples for culture and susceptibility testing and cytologic examination. Radiographs of the bullae may be useful although not very sensitive; ventrodorsal, lateral oblique and open-mouth views are most helpful. Anesthesia is necessary for proper positioning. Abnormalities of the bulla include increased opacity, sclerosis and lysis. However, fluid cannot be differentiated from tissue (polyp, neoplasia) and absence of radiographic changes does not rule out otitis media. Advanced imaging with CT and MRI is necessary in most cases for proper evaluation of the middle ear (and inner ear to some extent). CT is considered superior to MRI for bony changes, whereas MRI is better for detection of soft tissue abnormalities.

Deafness

Deafness is classified as inherited or acquired, conductive or sensorineural and congenital or late onset. Conductive deafness results from a lack of presentation of sound to the inner ear, usually secondary to otitis externa or media and therefore amenable to therapy. Sensorineural deafness occurs with abnormalities of the cochlear system, cranial nerve VIII or auditory pathways and higher brain centers.

Several methods have been employed to test hearing ability in dogs, ranging from behavioural studies to measurement of electrical responses after auditory stimulation. The last two decades, brainstem auditory evoked responses have been used increasingly to test hearing ability in veterinary medicine. The acoustic signal usually consisted of a click stimulus, which stimulates a large part of the cochlea.

Brainstem evoked response audiometry using clicks will suffice for differentiating neurologic from conduction deafness and is of use in assessing some brainstem pathologic changes. Frequency-specific information, however is needed in assessing the extent of neurologic (sensorineural) deafness, e.g. noise-induced deafness, deafness caused by ototoxicity and presbycusis (age-related deafness), which can all be partial and frequency-specific. There is no cure for sensorineural hearing loss, but ongoing research in human and veterinary medicine regarding cochlear implants, neurotrophic factors and stem cell research is yielding promising results.

Otitis media and interna

Otitis media generally develops as an extension of otitis externa through a perforated tympanum. Pharyngeal infections may, in rare instances, extend to the middle ear through the auditory tube. Cats may develop otitis media through this route as a sequel to upper respiratory tract disease. Involvement of the middle ear through hematogenous spread is only rarely encountered.

Organisms cultured most frequently from affected middle ears, in decreasing order of frequency, include Pseudomonas species, Staphylococcus intermedius, beta-hemolytic streptococcus, Malassezia, Corynebacterium species, Enterococcus species, Proteus species, E. Coli and anaerobes. Infections with these agents are usually associated with perforating foreign bodies or they occur as a sequela to chronic, usually proliferative or severe otitis externa. Bacteria can directly infect the middle and inner ear, or the bacteria can produce toxins that inflame the labyrinth.

Other causes of otitis media include fungal infections (Aspergillus, Candida), neoplasia, inflammatory polyps, trauma and primary tumors.

The therapy of otitis media and/or interna consists of systemically delivered broad-spectrum antibiotics. Amoxicillin potentiated with clavulonic acid or enrofloxacine are first choice antibiotics. Perforated tympanic membranes should close in 4 weeks, when the infection is cured. No ototoxic topical medications should be used when the tympanic membrane is not intact to avoid ototoxicity.

When the tympanic membrane is intact, but the pars flaccida is bulging, a paracentesis could be performed under general anesthesia. When the tympanic membrane is intact, a concurrent otitis externa can be treated with topical ointments containing antibiotics and corticosteroids.

Chronic unresponsive or recurrent otitis media warrants surgical interventional. Total ear canal ablation with lateral bulla osteotomy should be considered in cases with severe secondary changes of the external ear canal and concurrent otitis media. If the external ear canal is not affected, a ventral bulla osteotomy may be performed to remove gross exsudate and establish drainage from the middle ear.
Tumors of the middle- and inner ear

Tumors of the bullae or bony labyrinth may damage or involve the peripheral vestibular structures and result in peripheral vestibular signs. Likewise, tumors within the ear canal like squamous cell carcinoma, ceruminous gland adenocarcinoma, may spread locally and result in vestibular disease. Less commonly, neurofibroma, or neurofibrosarcoma of the peripheral nerve (N VIII) may result in slowly progressive peripheral vestibular signs.

In addition to the signs of peripheral vestibular disease, facial nerve paralysis or Horner’s syndrome is common when tumors are located in the middle and inner ear. These tumors are usually evident on skull radiographs as a soft tissue density within the bulla or a region of associated bone lysis. Diagnosis can be confirmed by biopsy findings. Because of the invasive nature of most of these tumors, total resection is difficult but radiotherapy or chemotherapy may be beneficial in some animals.

Otoxicity

Over 180 compounds and classes of compounds have been identified as ototoxic. Not all of them are equally toxic and some effects are reversible, but in most instances the deficit is permanent. In human medicine, the aminoglycoside antibiotics, the antineoplastic drugs cisplatin and carboplatin, loop diuretics, salicylates, quinine, deferoxamine, and various toxic substances are recognized for their propensity to cause ototoxicity. The best recognized, and perhaps most frequent, agents of ototoxicity in veterinary medicine are the aminoglycoside antibiotics, especially gentamicin, but polymyxins, chloramphenicol, and clindamycin are ototoxic as well. The importance of disinfectant-based (cloquinol, chlorhexidine, cetrimide, iodine, povidon-iodine and 70% ethanol) ototoxicity, for instance used for ear surgery, should not be underestimated however.

In order for a drug to exert ototoxicity, it must reach the inner ear. This may be the result of hematogenous spread following oral or parenteral dosage. The severity of ototoxicity depends on the concentration of the drug in the blood, the period of time the drug has been used, individual susceptibility (probably determined primarily by heredity), whether other ototoxic drugs are also being used, whether renal function is unimpaired, and whether there is concurrent noise exposure. However, more commonly, ototoxicity follows topical application of ototoxic agents into the external ear canal and their subsequent passage into the middle ear via a ruptured tympanum. Subsequent diffusion into the middle ear is enhanced by the presence of otitis media, which induces increased permeability through the round window membrane, which is an important portal for the passage of inflammatory mediators, toxins and drugs from the middle ear to the inner ear. The agent passes through the membrane of the round window and enters the perilymph in the tunnel of Corti. It thereby comes in contact with the hair cells of the organ of Corti and causes degeneration of the perceptive cells. This route of entry was demonstrated for gentamicin in the guinea pig, but similar structures in the vestibule make it likely that perilymph also reaches the sensory cells of the vestibular labyrinth.

The mechanism of toxicity is unclear, but the pathology includes hair cell loss with a progression from basal coil outer hair cells to more apical outer hair cells, followed by inner hair cells.

Drugs that primarily affect the cochlea, resulting in hearing impairment, are cochleotoxic whereas drugs that affect the vestibular system, resulting in vestibular dysfunction, are vestibulotoxic. Clinical signs of vestibular damage may be reflected very early (as soon as 10 minutes!) after the insult has been effected. Within three days central compensation results in diminishing and eventually disappearance of the nystagmus, gradual attempts to stand, and beginning efforts to eat and drink, but the head tilt is unchanged. Within three weeks the situation improves, but jumping and walking down stairs often still results in falling. The compensation is optimal after about three months. The head tilt however may still be obvious and permanent.

In general, ototoxic effects are dose related, therefore avoiding ototoxic chemicals or reducing the dose and frequency of administration is important for. Careful observation and regular follow-up examinations of the patient may allow detection of vestibular signs early enough to allow the clinician to suspend therapy. It is difficult, however, to detect early cochlear damage without sophisticated investigatory tools, such as Brainstem Auditory Evoked Response audiometry (BAER).

Geriatric canine and idiopathic feline vestibular disease

Geriatric canine vestibular disease, an idiopathic syndrome, is the most common cause of unilateral peripheral vestibular disease in old dogs. The mean age of onset is 12.5 years, and the disorder is characterized by the very sudden onset of unilateral peripheral vestibular signs. No
other neurologic abnormalities are observed. Approximately 30% of affected dogs also have transient nausea, vomiting and anorexia. The diagnosis is made on exclusion of other causes and on the alleviation of clinical signs with time. The prognosis for recovery is excellent. Occasionally vomiting is severe, and H1 histaminergic receptor antagonists, M1 cholinergic receptor antagonists or vestibulosedative drugs are administered for 2-3 days to alleviate the emesis associated with motion sickness.

Feline idiopathic vestibular syndrome is an acute, nonprogressive disorder similar to the previous syndrome in dogs and affects cats of any age. This disease is characterized by the peracute onset of peripheral vestibular signs with no abnormalities of proprioception or in other cranial nerves. The diagnosis is based on the clinical signs and the absence of ear problems or other disease. The prognosis is excellent; spontaneous improvement is usually seen within 2-3 days, with a complete return to normal within 2-3 weeks.

**Congenital vestibular syndromes**

Peripheral vestibular signs before 3 months of age in purebred dogs or cats are likely the result of a congenital vestibular disorder. Congenital unilateral peripheral vestibular syndromes have been recognized in the German Shepherd, Doberman Pinscher, Akita, English Cocker spaniel, Beagle, Smooth Fox terrier and Tibetan terrier, as well as in Siamese, Burmese and Tonkinese cats. Clinical signs may be present at birth or develop during the first few months of life. Head tilt, circling and ataxia may initially be severe; however with time, compensation is common and many affected animals make acceptable pets. The diagnosis is based on the early onset of signs. If ancillary tests such as radiography and CSF analysis are performed, findings are normal. Deafness may accompany the vestibular signs, particularly in the Doberman Pinscher, the Akita and the Siamese cat.

**Ventral bulla osteotomy**

An incision is made parallel with the midline, centered 2-3 cm toward the affected side from halfway the mandible to the level of the atlas. The platysma muscle is incised and linguofacial vein is retracted. The incision is deepened by blunt dissection between digastricus muscle and hypoglossal and styloglossal muscles until the bulla can be palpated. A Steinmann pin can be used to make a hole on the ventral aspect, the opening can be enlarged with a small rongeur. In cats both compartments should be opened. Material is collected for culture, sensitivity testing, cytology and histopathology. The cavity is flushed and drained with a Penrose drain. Closure is routine.