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Feline vestibular disorders. Part II: diagnostic approach and differential diagnosis

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Results of a neurological examination usually permit localisation of a vestibular disorder to either the central or peripheral parts of the vestibular system. Many different disorders located in the same part of the vestibular system will produce similar clinical signs. Therefore, additional diagnostic tests beyond a neurological examination are required in order to make an accurate diagnosis. The objectives of this review are to outline a diagnostic approach for disorders affecting the feline vestibular system, and to summarise the clinically important features of frequently diagnosed diseases affecting the vestibular system of cats.

A complete history and a thorough physical and neurological examination, including ophthalmoscopic and otoscopic examinations, are essential considerations in the management of a cat exhibiting clinical signs of vestibular dysfunction. A minimum data base, (including a complete blood count, serum biochemistry profile, urinalysis, thoracic radiographs, and abdominal ultrasound or radiography) should be obtained. Results of these examinations may provide evidence of multisystemic or concurrent disease.

Results of a neurological examination usually permit localisation of a vestibular disorder to either the peripheral or central parts of the vestibular system (LeCouteur & Vernau 1999). Since many different disorders located in the same area of the vestibular system will produce similar clinical signs, additional diagnostic testing is required in order to make a diagnosis. Generally, the less invasive diagnostic tests are done before the more invasive tests.

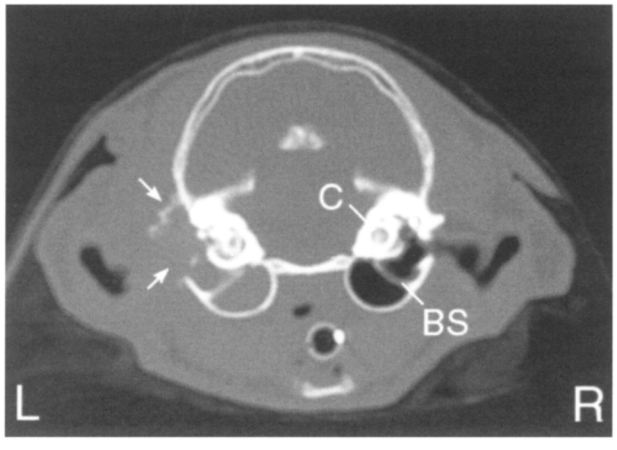

Fig 1. Transverse (precontrast) CT image of the head of a 12-year-old spayed female domestic long-haired cat, at the level of the tympanic cavities. Note the cochlea (C), and the bony septum (BS) that separates the dorsolateral and ventromedial compartments of the (normal) right tympanic cavity. There is a soft tissue/fluid density within the left tympanic cavity, associated with a productive/destructive process affecting the squamous portion of the temporal bone and the lateral aspect of the tympanic bulla (arrows). Squamous cell carcinoma caused these bony and soft tissue abnormalities.

Diagnostic approach for peripheral vestibular disorders

General anaesthesia is recommended in order to complete a thorough examination of the pharynx and ears, and to obtain diagnostic skull radiographs. Skull radiographs (lateral, dorsoventral, open mouth and left and right oblique projections) and, when available, computed tomography (CT), aid in the evaluation of the tympanic cavity and bulla, and petrosal portion of the temporal bone (Fig 1). Myringotomy (needle aspiration through the ventrocaudal part of the tympanic membrane) enables collection of fluid from the tympanic cavity (Fig 2), that may be submitted for cytological examination and

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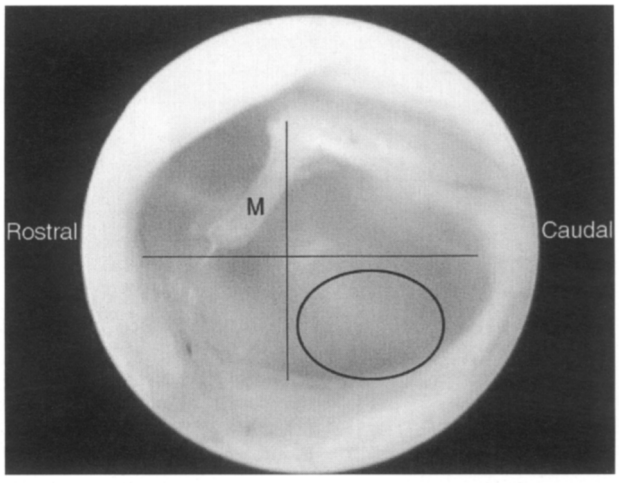

Fig 2. Left tympanic membrane of a normal cat. Note the area in the caudoventral quadrant of the tympanic membrane (circle) that should be used for myringotomy in order to avoid damage to the auditory ossicles and round or oval windows of the middle ear. (M=manubrium of the malleus).

microbial culture and sensitivity testing (Rose 1977). Abnormal tissue within the external auditory canal or tympanic cavity may be biopsied for cytological or histopathological examination.

Brain stem auditory-evoked potential (BAEP) testing may be used to estimate peripheral and central auditory function (Fig 3) (Holliday & TeSelle 1985). Since the auditory and vestibular parts of the vestibulocochlear nerve are in close proximity, BAEP testing may provide useful information regarding the functional status of the peripheral portions (and central pathways) of the VIIIth cranial nerve.

Diagnostic approach for central vestibular disorders

Magnetic resonance imaging (MRI) is the preferred modality for imaging the central vestibular system (Fig 4), although skull radiographs and CT may be useful in demonstrating bony abnormalities or signs of acute trauma such as haemorrhage. MRI is more sensitive than CT for imaging brain stem structures, and is not susceptible to the artifacts (such as beam hardening artifact) that may occur with CT examination of the caudal fossa and brain stem. Intravenous contrast medium should always be administered, as some lesions are not apparent without it.

Cerebrospinal fluid (CSF) analysis may be helpful in making a diagnosis of a cause of central vestibular system dysfunction. Results of CSF analysis usually are supportive, rather than diagnostic, of a disease process, unless organisms (eg, *Cryptococcus* spp) or tumour cells (eg, malignant lymphocytes) are present. CSF may also be submitted for serological assessment (eg, *Toxoplasma gondii* titre) or anaerobic and aerobic microbial culture and sensitivity testing.

Further diagnostic tests, such as BAEP testing, may be done to evaluate the integrity of the brain stem. In cats with mass lesions, biopsies (either open or CT-guided) should be considered, and the tissue submitted for cytological examination (conventional or crush preparation) and/or histopathology. As with lesions located elsewhere in the body, a definitive diagnosis may only be possible following biopsy.

Disorders of the peripheral vestibular system

Congenital peripheral vestibular disease

A congenital vestibular disorder has been

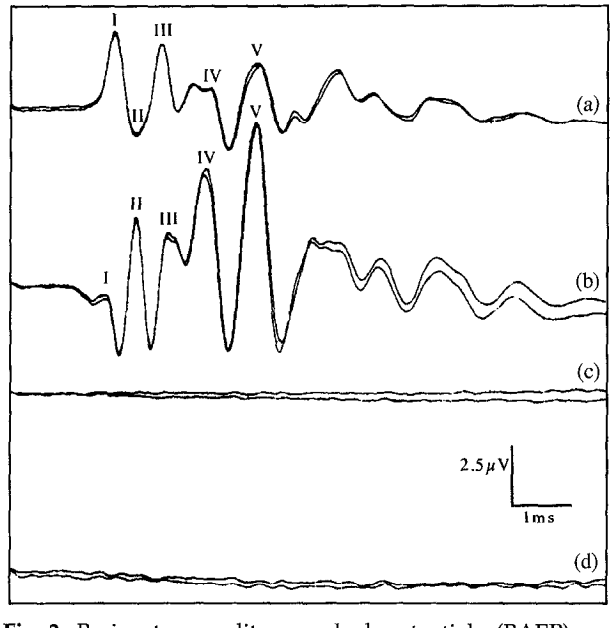

Fig 3. Brain stem auditory-evoked potentials (BAEP) recorded from a 4-month-old Maine coon cat with a right-sided nasopharyngeal polyp. Recordings (a) (vertex to mastoid) and (b) (vertex to T1) were recorded following stimulation of the left ear. Recordings (c) (vertex to mastoid) and (d) (vertex to T1) were recorded following stimulation of the abnormal right ear. Note that there is a lack of BAEP from the right ear, consistent with complete loss of auditory function in this ear. The five waves labelled I through V in (a) and (b) arise from the cochlear nerve and from brain stem structures in the auditory pathways. The influence of reference electrode position on the wave form is evident when recordings (a) and (b) are compared.

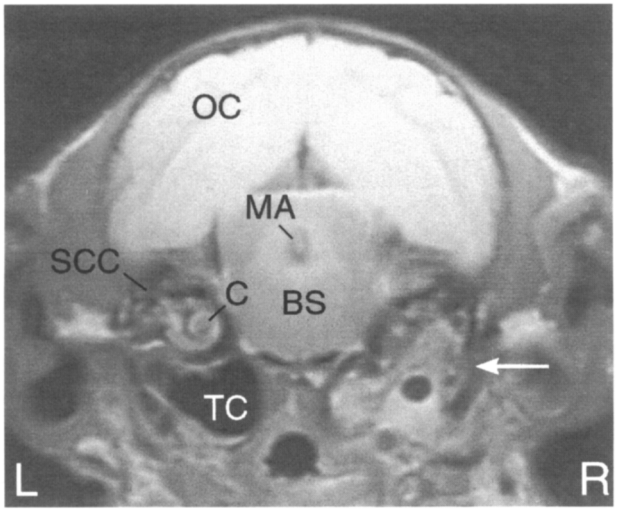

Fig 4. Transverse (proton-weighted) MR image of the head of a 7-month-old male Persian cat, at the level of the tympanic cavities. Normal structures evident on the left side include the ventromedial compartment of tympanic cavity (TC), cochlea (C), brain stem (BS), mesencephalic aqueduct (MA), semicircular canals (SCC), and occipital lobe of cerebrum (OC). Note the complete obliteration of the middle and inner ear on the right side secondary to a bacterial otitis media/interna (arrow). (Photograph courtesy of Dr Gregg D Kortz).

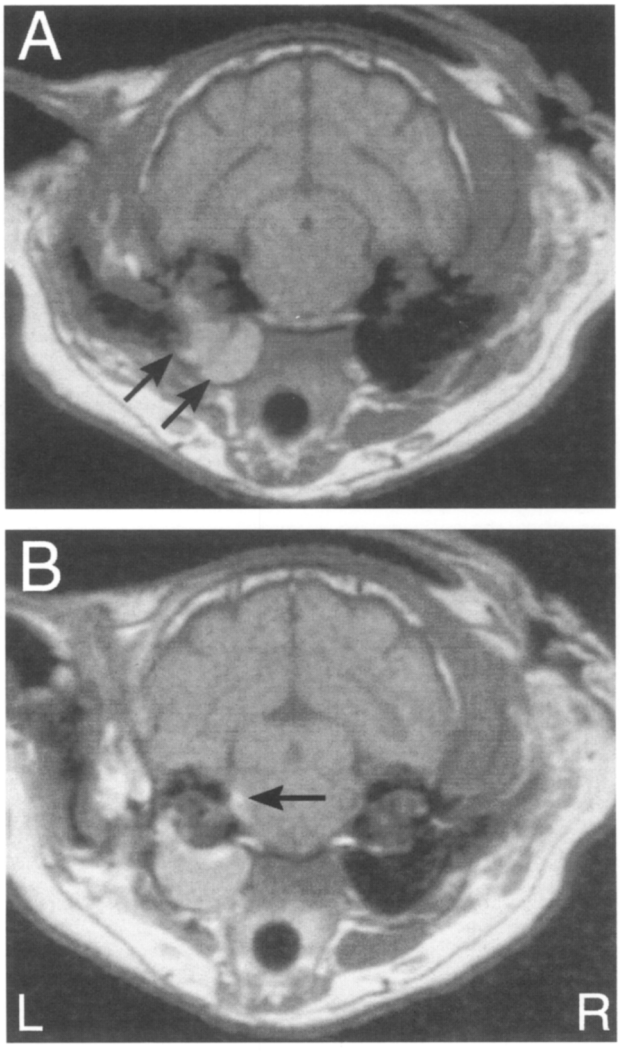

Fig 5. Transverse MR images of the head of a 14-year-old spayed female domestic longhaired cat, at the level of the tympanic cavities. In the precontrast images (A) the left tympanic cavity is filled with a soft tissue density (arrows) that occupies both the dorsolateral and ventromedial compartments of the tympanic cavity. Biopsy confirmed squamous cell carcinoma. In post-contrast (post-gadolinium) images (B) enhancement of the previously noted tumour is evident, and extension of the mass medially through the internal auditory meatus is apparent (arrow).

reported in Siamese and Burmese kittens (de Lahunta 1983). In affected Siamese kittens, clinical signs developed at 3–4 weeks of age. Usually kittens may demonstrate clinical improvement by 3–4 months of age. Clinical signs of vestibular dysfunction may be accompanied by deafness. Clinical signs in Burmese kittens developed at or shortly after birth, and were non-progressive. Lesions were not apparent on pathological examination. Although a hereditary problem is suspected in both breeds, it has not been proven (de Lahunta 1983). Congenital deafness has been reported in white cats (Bergsma & Brown 1971). Diagnosis of congenital peripheral vestibular disease is based on history, and results of serial neurological examinations and BAEP testing.

Neoplasia

Neoplasms involving the ear canal may cause peripheral vestibular disease when the temporal bone is affected or when secondary otitis media/interna is associated with the neoplasm. Affected animals may appear to be in pain on palpation of the tympanic bulla, or when the mouth is opened. Benign tumours reported to occur include polyps, papillomas and ceruminous gland adenomas. A 42-month median disease-free interval has been reported in cats following ear canal ablation and lateral bulla osteotomy for ceruminous gland adenocarcinoma (Marino et al 1994). Malignant tumours reported to occur include fibrosarcoma, osteosarcoma, chondrosarcoma, squamous cell carcinoma, ceruminous gland adenocarcinoma, lymphoma and sebaceous gland adenocarcinoma (London et al 1996, deLahunta 1983). Squamous cell carcinoma is the most common malignant tumour of the middle ear of cats (Lane 1992). Rarely, these middle ear tumours may extend medially to involve the brain stem (Fig 5) (Lane 1992, Rendano 1980).

Diagnosis is based on the finding of destructive bony lesions on skull radiographs or CT, and on biopsy results. Prognosis varies with the invasiveness of the neoplasm, however, it is generally considered poor (Kornegay 1991).

Otitis media and otitis interna

Otitis interna or labyrinthitis, is a common cause of peripheral vestibular dysfunction in cats. Usually otitis interna is bacterial in origin, and secondary to otitis media, which may occur secondary to otitis externa. Otitis media may also be caused by extension of infection from the pharynx to the tympanic cavity via the auditory tube, or by haematogenous dissemination. Other causes of otitis media/interna include yeast, fungi (eg, *Cryptococcus* spp), parasites, foreign bodies (eg, grass awns), and inflammatory polyps or neoplasms. Extension of infection to involve the brain stem by means of the internal auditory meatus may occur.

Ipsilateral facial nerve paralysis and Horner's syndrome may occur in association with otitis media/interna. Since the facial nerve contains parasympathetic preganglionic neurons that modulate lacrimal gland secretion, animals with labyrinthitis may have ipsilateral decreased tear production and may develop keratoconjunctivitis sicca.

Diagnosis of otitis media/interna is based on otoscopic examination and imaging. Otitis externa may be evident and/or there may be bulging and discolouration of the tympanic membrane if fluid or exudate is present in the tympanic cavity (Kornegay 1991). Imaging (skull radiographs, CT or MRI) may reveal a fluid or soft tissue density in the tympanic cavity, or sclerosis and lysis of tympanic bulla and adjacent bones. Skull radiographs may be normal in acute infections. If fluid is present in the middle ear, it should be collected by means of myringotomy for cytological examination and anaerobic and aerobic culture and sensitivity testing (Kornegay 1991, Rose 1977). Cultures from the external ear canal may also be submitted.

Prognosis is usually good when long-term oral antibiotic therapy is initiated on the basis of results of culture and sensitivity testing. Although clinical signs may improve within 1–2 weeks, antibiotic therapy should be continued for at least 6 weeks. In more chronic cases, unresponsive to medical therapy, surgical drainage of the tympanic cavity may be necessary by means of a lateral or ventral bulla osteotomy (Trevor & Martin 1993). It must be kept in mind that the tympanic bulla in cats is divided into two compartments by an incomplete bony septum (LeCouteur & Vernau 1999). The communication between the dorsolateral and ventromedial compartments may be obstructed by exudate, and both compartments should be surgically drained.

Feline nasopharyngeal polyps

Feline nasopharyngeal polyps are well vascularised soft tissue growths lined by epithelium that have been identified within the auditory tube, middle ear, external ear canal, or nasopharynx of cats (Kornegay 1991). Definitive aetiology for the polyps is not known, however, chronic inflammation, infection with calicivirus, and congenital or familial factors have been hypothesised as causes. Nasopharyngeal polyps usually affect cats less than 3 years of age, however, they have been reported in older cats. Polyps may grow into the middle ear, nasopharynx or external ear canal, and clinical signs occur secondary to obstruction of the nasopharynx or ear canal. Associated clinical signs include: dysphagia, voice change, inspiratory stridor, sneezing, rhinitis, otitis externa, and otitis media/interna, with or without associated peripheral vestibular disease. Diagnosis is made on the basis of an otoscopic examination, nasopharyngeal examination, and imaging (skull radiographs, CT or MRI). Treatment is surgical removal of the polyp by means of ventral bulla osteotomy (Fig 6). Prognosis following surgical removal is excellent, however, the polyp may return (Seitz et al 1996).

Feline idiopathic vestibular disease

This syndrome occurs in cats of all ages, particularly in the summer months (Burke et al 1985). Cats develop an acute onset of severe peripheral vestibular dysfunction in the absence of facial paralysis, Horner's syndrome or CNS involvement. Clinical signs are often preceded by, or occur concurrently with, upper respiratory tract disease. Within 72 h of onset, clinical signs stabilise, however, recovery may require at least 2–4 weeks. Residual deficits, such as a head tilt, may persist, and blindfolding or darkness will cause reoccurrence of signs well after recovery apparently has occurred. It is important to differentiate this idiopathic benign disorder, which resolves spontaneously without therapy, from otitis media/interna, which requires vigorous therapy.

Although the cause of this syndrome remains undetermined, it has been speculated that migration of *Cuterebra* larvae through the inner ear may be a cause in some cats (Williams et al 1998, Glass et al 1998).

Diagnosis is based on the exclusion of other causes of vestibular disease. Recommended therapy is supportive care, and prognosis for recovery is excellent. Since most affected cats recover with supportive care, and, therefore, necropsy usually is not done, treatment directed against *Cuterebra* spp is not recommended until more information about the association between cuterebriasis and feline idiopathic vestibular disease is available.

Toxicity

Therapy with ototoxic agents may result in degeneration of the vestibular and/or auditory receptors, usually resulting in permanent dysfunction (Pickrell et al 1993). Bilateral vestibular dysfunction may result from ototoxic drug administration. Ototoxicity may occur as a result of oral, parenteral, or topical drug therapy. Although ototoxicity from topical drug therapy to the external ear occurs more commonly if the tympanic membrane is ruptured, it may occur in the presence of an intact tympanic membrane.

All aminoglycoside antibiotics may have a toxic effect on either the peripheral vestibular or auditory system, or both (Winston et al 1953). Gentamicin and streptomycin are reported to affect primarily the vestibular system, whereas neomycin, kanamycin, tobramycin and amikacin affect primarily the auditory system.

Loop diuretics such as ethacrynic acid, bumetanide and furosemide are ototoxic. Furosemide is the least ototoxic and its effects are reported to be reversible if used for a short period of time.

Ear canal cleansers and vehicles in otic preparations such as propylene glycol, chlorhexidine and cetrimide also are ototoxic, particularly if the tympanic membrane is ruptured (Gallé & Venker-van Haagen 1986).

In the southeastern USA, cats may develop acute peripheral vestibular disease from ingestion of the tail of the blue-tailed lizard (Adair 1953).

Diagnosis is based on history, and results of ototoscopic examination and BAEP testing. Treatment consists of cessation of the drug therapy and initiation of supportive care. Prognosis for recovery from the vestibular dysfunction is good, particularly when the cause is recognised early, however, some permanent deficits may remain and deafness may be permanent.

Trauma

Trauma to the head may result in peripheral vestibular disease due to fracture of the petrosal part of the temporal bone or tympanic bulla. Facial nerve paralysis may accompany bony injury. Vestibular dysfunction has also been reported in cats secondary to a gunshot injury (Podell et al 1992). Fractures of the petrosal portion of the temporal bone may predispose cats to infection from organisms ascending from the tympanic cavity or ear canal. Diagnosis is based on history and imaging (skull radiographs or CT). Treatment consists of supportive care. Broad spectrum antibiotic administration should be considered.

Disorders of the central vestibular system

Thiamine deficiency

Thiamine deficiency may occur in cats fed raw fish exclusively, following chronic anorexia without vitamin therapy, and in cats fed highly processed foods without thiamine added (Jubb et al 1956, Loew et al 1970, Munday & King 1972). Thiamine is a dietary nutrient necessary for energy pathways. Because nervous tissue has a high metabolic rate, central nervous system (CNS) signs occur secondary to thiamine deficiency. The initial clinical signs of thiamine deficiency include anorexia, followed by vestibular ataxia. These signs progress rapidly to cervical ventroflexion, mydriasis, recumbency, seizures and coma. Pathological findings in thiamine deficiency are bilaterally symmetrical haemorrhagic necrosis of the brain stem periventricular grey matter.

Diagnosis is based on history, clinical signs, and reduced activity of plasma thiamine pyrophosphate. Treatment with parenteral thiamine at 100–250 mg twice daily is effective in most cats. Cats with severe neurological deficits may not recover.

Neoplasia

Neoplasms located at the cerebellomedullary angle may affect the central vestibular system

(Fig 5). Neoplasms may be primary (those that arise within the CNS) or secondary (those that metastasise or spread from local structures to invade the brain). In cats the most commonly reported primary neoplasm in this location is meningioma. Lymphosarcoma is the most commonly reported secondary neoplasm. Other less frequently reported neoplasms include glial tumours (eg, oligodendroglioma) and extension of middle ear tumours.

Diagnosis is based on the results of imaging (CT or MRI), CSF cytology, and biopsy procedures. Prognosis and treatment vary with the specific neoplasm. Surgical removal and/or radiation therapy of neoplasms in this location should be considered.

Infection

Any cause of meningoencephalitis may result in involvement of the central vestibular system. Causes include feline infectious peritonitis (FIP), toxoplasmosis, cryptococcosis, aberrant parasite migration (*Cuterebra* spp), bacterial meningoencephalitis secondary to extension of otitis media/interna or migration of a foreign body, and rabies.

Neurological disease, particularly central vestibular disease, occurs frequently in cats with FIP. The non-effusive form of FIP is most frequently involved. Although FIP may occur in cats of any age, it is most common in young cats. Systemic signs may or may not be seen in association with focal or multifocal neurological signs. A diagnosis of FIP is based on history, and physical and neurological examination findings (including ophthalmoscopic examination). Hyperglobulinaemia, and abnormalities of echogenicity in the liver and kidneys on abdominal ultrasound, may be detected. At present there is no single diagnostic test for FIP (Addie & Jarrett 1998, Foley et al 1998). Serum FIP titres are of limited assistance in the diagnosis of this disease since they only reflect exposure to a coronavirus (Foley et al 1998). FIP infection results in a severe pyogranulomatous leptomeningitis, choroiditis, ependymitis, and encephalomyelitis. MRI of the brain of some cats with FIP may reveal ventricular dilation (due to ventricular obstruction), and ependymal enhancement (Fig 7) (Foley et al 1998). Ependymitis and inflammatory ventricular exudate frequently obstruct the aqueduct and prevent CSF flow. CSF usually has a markedly elevated protein and white blood cell count (the majority of cells are neutrophils). FIP serology may be done on CSF (Foley et al 1998). Prognosis for affected cats is poor and effective therapy does not exist.

Toxoplasmosis is caused by the intracellular protozoan parasite *Toxoplasma gondii*. Focal or multifocal neurological signs may be present in cats with CNS infection. Diagnosis of toxoplasmosis is based on history (outdoor cat that hunts or is fed undercooked meat), results of physical, neurological and ophthalmoscopic examinations, demonstration of serologic evidence of disease, exclusion of other diseases, and response to therapy (Dubey & Lappin 1998). Cats should be tested for infection with feline immunodeficiency virus (FIV), since concurrent infection with FIV and toxoplasmosis, results in a worse prognosis. Serologic evidence of disease is a four-fold increase in the IgG toxoplasma titre, between acute and convalescent samples, and demonstration of IgM and IgG antibodies in CSF. Diagnosis of clinical toxoplasmosis cannot be based on serology alone. Treatment with clindamycin at 25 mg/kg, divided two or three times a day for 4 weeks, has been effective in treating a limited number of cats with suspected CNS toxoplasmosis. Trimethoprim-sulfa, at 15 mg/kg orally twice daily, may also be used to treat affected cats.

Cryptococcus neoformans is a saprophytic yeast, with a worldwide distribution. Infection is acquired from the environment, rather than from other animals (Jacobs & Medleau 1998). Primary infection occurs in the upper respiratory tract, sinuses and oropharynx, with secondary systemic spread and extension to the CNS. Although the major site of involvement in the CNS is the meninges, extension to the ependyma of the ventricles and brain parenchyma also occurs. In cats, unlike dogs, the *C neoformans* organism provokes only a mild inflammatory response. Diagnosis is primarily based on history, physical, neurological and ophthalmoscopic examinations. Definitive diagnosis is based on the finding of cryptococcal organisms in CSF (Fig 8). In cases where the organism is not visualised, the diagnosis may be based on the finding of cryptococcal capsular antigen in the serum or CSF, or by culturing the organism from the CSF. Although prognosis for cats with CNS cryptococcosis is poor, treatment may become more effective with the availability of fluconazole and itraconazole. Fluconazole has been effective in treating cats with nasal cryptococcosis. Although fluconazole attains therapeutic concentrations in the CNS, and itraconazole does not, itraconazole

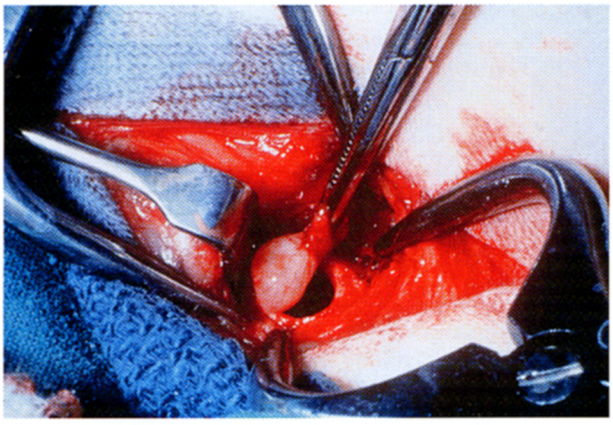

Fig 6. Intraoperative photograph of a nasopharyngeal polyp being removed from the tympanic cavity of a cat via ventral bulla osteotomy. (Photograph courtesy of Dr Stephen J Withrow).

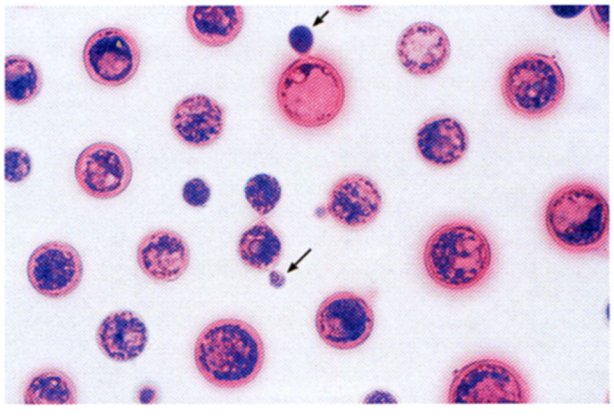

Fig 8. *Cryptococcus neoformans* in cerebrospinal fluid (CSF). Note that several organisms are budding (arrows). In cats cryptococcosis is often associated with minimal inflammatory reaction in the CSF, as is seen here, where white blood cells are absent. (New methylene blue, original magnification ×250). (Photograph courtesy of Dr Mary M Christopher).

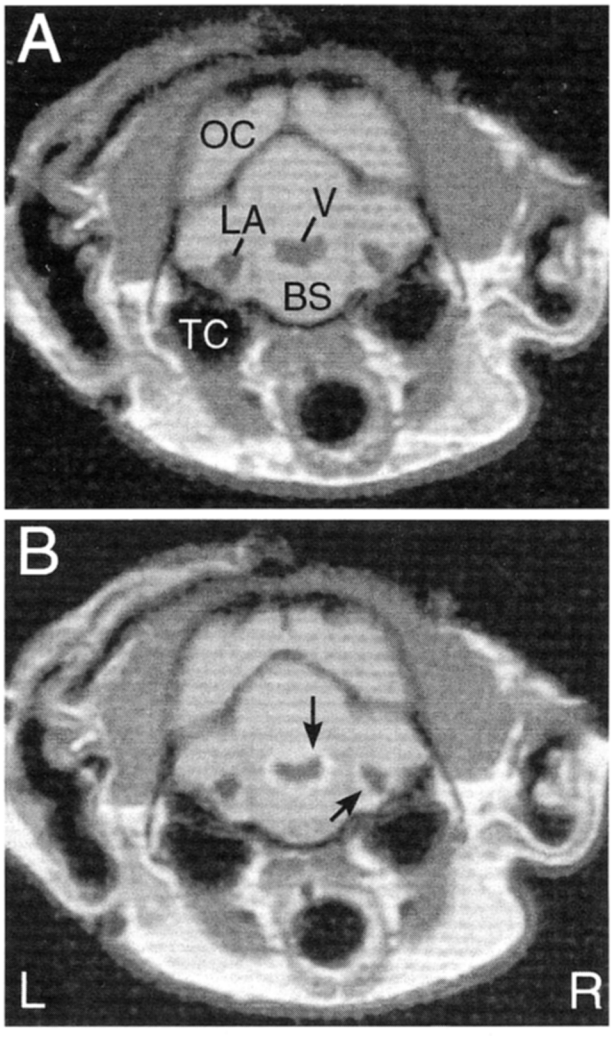

Fig 7. Transverse MR images of the head of a 1-year-old female Burmese, at the level of the tympanic cavities (TC). Normal structures evident in precontrast images (A) include brain stem (BS), and occipital lobes of the cerebrum (OC). Note the symmetrical enlargement of the IVth ventricle (V) and lateral apertures (LA). In the post-contrast (post-gadolinium) image (B) there is diffuse homogeneous contrast enhancement of the ependyma and subependymal white matter of the IVth ventricle and lateral apertures (arrows). This pattern of contrast enhancement is typically seen in MR images of cats with periventricular inflammation resulting from feline infectious peritonitis (FIP).

is highly lipid soluble, and may penetrate the CNS and become tissue bound. Although fluconazole and itraconazole have not been adequately evaluated in the treatment of CNS cryptococcosis, these drugs offer the potential of a more effective treatment for this disease.

Toxicity

Metronidazole toxicity results in central vestibular and cerebellar dysfunction in dogs and people. Metronidazole toxicity, resulting in clinical signs of acute CNS dysfunction, has been reported in three cats (Saxon & Magne 1993). The cats were treated with metronidazole at doses ranging from 48 to 62.5 mg/kg/day for 5 days to 10 months prior to presentation. Although diffuse neurological abnormalities were present in these cats (two of three cats also had seizures), signs of ataxia and disorientation were reported in all three cats. Vertical nystagmus is a consistent finding in dogs with metronidazole toxicity (Dow et al 1989), and could potentially occur in cats. History and clinical signs are the key to diagnosis. Treatment consists of supportive care, and withdrawal of the drug. Prognosis for recovery is good unless the clinical signs are severe.

Trauma

Cats with head trauma may have clinical signs of vestibular system dysfunction. Diagnosis is based on history, and the results of advanced imaging such as CT (in acute trauma) or MRI. Treatment involves aggressive supportive care plus surgical decompression in some cases. Prognosis is variable depending on the severity of the neurological injury, however, many cats will survive and return to being acceptable pets.

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