Neuro-ophthalmology: A review

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Summary

Evaluation of horses for eye problems and for suspected impairment of visual acuity often requires a thorough neuro-ophthalmic examination to be conducted. Along with a full ophthalmic evaluation, the results of such an examination should indicate the likely site of any lesion(s) accounting for blindness, miosis, mydriasis, anisocoria, Horner’s syndrome, strabismus, facial paralysis and facial spasm, nystagmus and other signs of vestibular disease, and cerebellar disorders, that may be present. Such a thorough examination with guidance for correct interpretation of findings is given here.

Introduction

During evaluation of a horse suspected of having an ophthalmic disorder, most busy clinicians include evaluation of several components of visual and eye function during the general physical examination (Palmer 1976; Adams and Mayhew 1985; Oliver et al. 1987; Blythe and Engel 1999; Ronéus and Gustafsson 2000; Reed et al. 2003, 2004; de Lahunta and Glass 2009; Mayhew 2009). These include observation of behaviour, mental status, head posture, vision, pupillary light reflexes, structures of the eye, facial sweating and inspection for facial symmetry. Usually, this examination allows the clinician to decide whether or not more detailed neuro-ophthalmic examinations are required.

Procedure for the neuro-ophthalmic examination

The primary aim of such an examination is to confirm whether or not a neurological explanation exists for any disorder involving the eye and its associated structures, and their central nervous system connections. Readers not reasonably well versed in performing neurological and ophthalmic examinations are to be encouraged to practice on a friendly, neighbourhood, mid-sized dog. One should be familiar with the visual, pupillary, cranial sympathetic, vestibular, cerebellar and general cranial nerve functional pathways (de Lahunta and Cummings 1967; Jenkins 1978; Mayhew 1980, 1989, 2004, 2009; de Lahunta 1983; Wolfe et al. 2008; de Lahunta and Glass 2009 [Figs 1–5]).

Firstly, evaluation begins from a distance, preferably before the patient is disturbed, for observations of behaviour, mentation and head, neck, trunk and limb position and movement. Head and neck deviations need to be assessed closely by straightening the neck along the midline to determine what asymmetry of eye position may be present. The patient may be allowed to smell the examiner’s hand for introduction and facial features, particularly eyes and associated structures, are observed for an expressional response. Examination of the parts of the head and the eyes then is undertaken to evaluate cranial nerve function (Table 1).

At this stage, the individual’s attention can be attracted by tapping lightly above the eyes with the finger tips to induce a combined visual and facial response of palpebral closure. The menace responses (Fig 6) from nasal and temporal fields are then assessed, while gently covering vision from the untested eye. This is followed by observation of eye position and pupil size and symmetry using a bright, noiseless, focused pen torch from 20–40 cm. The light is directed quickly from the fundus of one eye to the other, pausing for ~3 s at each pupillary aperture as the light source is sequentially brought closer in front of each eye. This allows the immediate pupillary response to be observed, uninterrupted by blinking. Any asymmetry or suspected deficit means that a dazzle response must be performed and the tests need to be redone in dim and in bright light, but not in direct sunlight. With practice, the central fundus and optic disc can be directly inspected by looking along the shaft of light from a pen torch; otherwise an ophthalmoscope should be used. Evidence of optic atrophy, peripapillary retinal lesions, globe position and trembling and ataxic eye movements and nystagmus can all be detected and recorded.

Eyeball position in the bony orbit, along with the size of the palpebral aperture and angle of the dorsal eyelashes, then are determined. Following this, both ventral movement and induced, normal, horizontal nystagmus of the globe are evaluated by first slowly raising the mandible to above horizontal and then rotating the head to both sides through an arc of 60–90°. Facial symmetry, facial reflexes, facial expression and especially eyelid, ear and lip muscle tone, all can then be determined. The bulk of the temporalis, masseter and pterygoid muscles is then assessed. Sensation in the form of cerebral perception and resentment is evaluated from the nasal septum on each side during facial reflex testing using blunt needle holders. Any increased temperature and presence of sweat at the base of an ear is noted.

At this stage one can document results of the examination, consult appropriate tables and figures and determine all possible neuroanatomical lesion locations that might explain any abnormalities detected.

Clinical neuro-ophthalmic syndromes

Blindness

Forebrain (cerebrum and thalamus) lesions result in horses that are blind with depressed menace responses in one or both eyes. Such a central blindness (amaurosis) with intact pupillary reflexes occurs in an eye contralateral to a lesion involving an optic tract, dorsal thalamus (lateral geniculate nucleus), optic radiation (part of
the internal capsule) or visual cortex (predominantly in the occipital lobe of the cerebral hemisphere). These structures constitute the central visual pathways. Each optic tract receives ~90% of all fibres, visual and pupilomotor, from the contralateral eye. It is therefore expected that with optic tract lesions, as well as blindness, there should be suppressed pupillary motor function evident when light is shone in the blind eye. However, clinically this is very difficult to discern. On the other hand, occasionally it is possible to detect visual field deficits with some selective and focal lesions involving these central visual pathways when performing visual field (nasal and temporal) testing.

A prominent lesion in one eye or optic nerve results in blindness and a suppressed menace response as well as a dilated pupil in that eye and poor pupillary constriction in both eyes when a bright light is shone in the blind eye. However, an individual can be clinically blind with absent menace response and a dilated pupil in one eye due to an eye or optic nerve lesion and still have some pupillary constriction in response to a very bright strobe light shone in that eye. This discrepancy comes down to, firstly, when is an animal blind? And secondly, visual path fibres probably are damaged more readily with various eyeball and optic nerve lesions than pupilomotor fibres are. With respect to visual perception, possibly the most sensitive test of crude visual pathway input is to place a blind patient having no menace responses in a dark enclosed area with a brightly lighted exit available to see if the patient can discern the escape route.

Swinging light tests described above also can be useful to help sort out difficult visual deficits, particularly as performing and interpreting consensual or indirect pupillary light reflexes in horses is problematic. Consider an animal that has no (poor) menace

<table>
<thead>
<tr>
<th>TABLE 1: Assessment of cranial nerves pertaining to vision and to other function of the eyes and their associated structures</th>
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<td>Cranial nerve</td>
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<tr>
<td>II Optic</td>
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<td>III Oculomotor</td>
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<td>IV Trochlear</td>
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<td>VI Abduens</td>
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<td>VII Facial</td>
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<td>VIII Vestibular</td>
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<td>Cochlear</td>
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![Visual pathway](Fig 1: Visual pathway. Figure reproduced from Mayhew (2009) with permission.)

![Pupillary light pathway](Fig 2: Pupillary light pathway. Figure reproduced from Mayhew (2009) with permission.)
response in the right eye, a normal menace response in the left eye, no discernable anisocoria and the left pupil responds directly to light shone in the left eye and the right pupil does respond to light shone in the right eye. Where is the lesion? Assuming facial and cerebellar functions are normal, going by this information the lesion should be in the left central visual pathways. However, now note the responses to the swinging light tests. Light shone in the left eye results in pupillary constriction in that eye. The light is quickly redirected into the right eye and although the right pupil initially is constricted it dilates back to its resting size as light reaches that eye! When the light then is redirected quickly into the left eye again the left pupil may or may not initially appear somewhat dilated, but it responds by constricting very quickly. This can be repeated as the light is shone back and forth. Also, when the left eye is covered the right pupil dilates to its full extent. The lesion is in the right eye or right optic nerve. Two points are of note: firstly, we are not discerning enough to detect minor degrees of anisocoria visually (without photographic evidence) and secondly, there is enough ambient light entering the normal eye to maintain some pupillary constriction in the blind eye.

The dazzle response or blinking in response to a noiseless flash of bright light, does not involve the visual pathways from the midbrain to the visual cortex. Also, its presence does not equate with vision but does indicate that light is stimulating the light pathways into the midbrain and thence to the facial nucleus, but it should not replace testing the true pupillary light responses as outlined. A dazzle response can be present in a centrally blind patient that also has no oculomotor nerve function and no pupillary light reflexes.

The prognosis for acquired peripheral blindness is poor compared to central visual pathway lesions for which patients appear to be able to compensate quite well, at times to regain quite amazing degrees of apparent visual acuity.

The postictal period may be associated with temporary central blindness presumably due to neuronal exhaustion in central visual pathways. Also foals, although they can see, may have poor menace responses in the first weeks post partum (Enzerink 1998), reminiscent of cerebellar dysfunction.

Fig 3: Ocular sympathetic pathway. Figure reproduced from Mayhew (2009) with permission.

Fig 4: Visual and light pathways. Figure reproduced from Mayhew (2009) with permission.

Fig 5: Vestibular system. Figure reproduced from Mayhew (2009) with permission.
Miosis, mydriasis, anisocoria and Horner’s syndrome

Miosis (constricted pupil), mydriasis (dilated pupil) and anisocoria (asymmetric pupil size) of varying degrees occur in many ocular diseases, often accompanied by degrees of visual impairment, although equine patients are not very frequently presented because of these individual problems alone.

The pupillary constriction (CN III) and dilation (ocular sympathetic) pathways should be reviewed (Figs 1–4). Normal resting pupil size will depend on the emotional state of the patient and the amount of ambient light reaching the retinas. Usually a calm horse has brisk initial pupillary responses. However, one failure during examination is to allow the horse to blink when a light is directed in to one eye, the dazzle response, thus obscuring the initial fast phase of pupillary constriction. This can be averted by allowing the horse to acclimatize to the light by starting at a distance of say 30–40 cm, swinging it from each eye in turn while moving closer to the eyes. Results of pupillary responses to the swinging light test are useful here as in evaluation of vision.

Consider a bright and alert patient that has no menace response but has a normal palpebral response, and is blind with a widely dilated, unresponsive pupil, all in the left eye. Light shone in the right eye results in the right pupil constricting and in a positive dazzle response and when the light then is swung briskly to enter the left eye the pupil remains dilated and there is no dazzle response in the left eye. When the light is then quickly retransferred to enter the right eye, the right pupil constricts strongly again. The lesion has to involve the left retina or optic nerve and the left oculomotor parasympathetic fibres. The presence of a retrobulbar abscess, cellulitis or neoplasm can readily explain these findings. With such lesions behind the globe the sympathetic fibres to the dilator muscles of the pupil can also be variably involved, confounding this clinical picture further.

In the presence of anisocoria it can be difficult to determine which pupil is abnormal. As a general rule an abnormally small pupil will not dilate fully in darkness but it will respond to light directed into that eye. In comparison, an abnormally dilated pupil will be most evident in bright light and will not constrict fully in response to light shone into that eye. Remember that daylight, and especially direct sunlight, are much more powerful than any portable light source.

Asymmetric infectious, traumatic and vascular brain diseases can result in midbrain oculomotor involvement and anisocoria. A mydriatic pupil, normal vision and usually no eye deviation are seen with parasympathetic oculomotor nerve involvement in horses, although this is uncommon to be found in isolation. With this syndrome and no other neurological abnormalities, an inability of the iris to respond that can result from previous atropine therapy must be a consideration. Anisocoria with poorly responsive pupils, with or without blindness, more often accompanies space occupying and other forebrain lesions, associated with brain swelling and subsequent ventral pressure on the midbrain and oculomotor nerves.

Horner’s syndrome due to sympathetic denervation of the eyeball consists of miosis, enophthalmos and protrusion of the nictitating membrane. This can be seen with no other neurological signs due to retrobulbar lesions involving postganglionic sympathetic fibres. The degree of miosis seen with Horner’s syndrome in horses is not dramatic (Fig 7). With sympathetic lesions more proximal, degrees of hyperaemic mucous membranes of the head (Fig 8), hyperthermia of the face and sweating of the face and cranial neck usually are clearly evident (Fig 9). This is

Fig 6: A true positive menace response is that the patient blinks its eyelids in response to a minimally threatening hand gesture as shown. This is assumed to equate to the presence of visual perception. The opposite is not true as there are several reasons why a patient that appears to see perfectly well and does not bump into strange, nonodiferous objects in its visual field may not have this positive menace response. Attracting the patient’s attention using a light or strong tap to the side of the face is useful to precede the test. If the visual threat is too strong the patient often will pull the head away in a startle response and even though being visual, may not blink; this response, akin to the dazzle (blink-to-bright-light) response, likely is sub-cortical and involves the midbrain and other brainstem structures without reaching consciousness. Temporal and nasal visual fields can be tested to evaluate uncrossed (at the optic chiasm) and crossed visual pathway fibres respectively. This does, however, take a co-operative patient and considerable experience to be repeatable and reliable. Figure reproduced from Mayhew (2009) with permission.

Fig 7: Left Horner’s syndrome present in this horse (A) is evident as moderate ptosis, lowering of the upper eyelashes and miosis (C) compared with the unaffected side (B). Note that there still is an angle present at the margin of the upper lid that usually is not present with the ptosis seen with facial paralysis. Figure reproduced from Mayhew (2009) with permission.
Fig 8: In this case of acute, temporary experimental Horner’s syndrome induced by local anaesthetic blockade of the cervical sympathetic trunk, a slightly constricted pupil is evident (A) compared with the normal eye (B). In horses, loss of sympathetic tone to superficial blood vessels induces vasodilation and this can be seen on any affected mucous membrane such as the bulbar conjunctiva in this case (A). Note that ptosis is not evident as the eyelids are being held open. Figure reproduced from Mayhew (2009) with permission.

caused by interruption of sympathetic fibres to the blood vessels and sweat glands of the head and neck (Smith and Mayhew 1977). With cutaneous vasodilation more circulating adrenaline bathes the sweat glands which has powerful sweat-producing effects in horses (Jenkinson et al. 2006).

If the sympathetic fibres are affected at or distal to the cranial cervical ganglion in the wall of the guttural pouch, sweating over the head extends caudally only to the level of the atlas. Preganglionic lesions proximal to this, as with neck lesions, result in sweating further down the neck to C2 to C3 (Usenik 1957). Cranial thoracic lesions can affect the sympathetic fibres in the origin of the cervical sympathetic trunk but also those innervating the skin of the remainder of the neck travelling with the vertebral nerve and segmental dorsal spinal nerve roots. There then is sweating over the whole neck and head (Fig 10). A first order sympathetic neuronal lesion in the descending, tectotegmentospinal white matter tract in the brainstem or cervical spinal cord results in sweating on the whole side of the trunk, neck and head as well as there being eye signs of Horner’s syndrome (Mayhew 1980). At least in horses with acute sympathetic lesions and resulting patterns of sweating, administration of α2 agonist sedative drugs results in expected sweating of normal skin but often reverses the vasodilation and sweating over the sympathetically denervated skin such that it becomes dry!

Horses affected by equine dysautonomia (grass sickness, Mal Sevo) often show bilateral ptosis (Pirie 2006). Specific muscles of the upper eyelid (and their innervation) that, when paralysed, may result in ptosis of the upper eyelids are: (a) the levator palpabrae superioris (CN III); (b) the levator anguli oculi medialis (CN VII); (c) Müller’s tarsal smooth muscle (sympathetic) (Hahn and Mayhew 2000b). In grass sickness cases there is no evidence for the ptosis being due to somatic facial (CN VII) or oculomotor (CN III) dysfunction suggesting that this is due to sympathetic dysfunction. Indeed, the ptosis can be readily reversed (Fig 11) using a low dose of topical α1 adrenergic agonist (0.5 ml of 0.5% phenylephrine) eye drops. Not only does the upper eyelid ptosis resolve within 5–20 min but the lowered angle of the upper eyelashes (i.e. pointing towards the ground) that is so characteristic of grass sickness cases, also resolves and often quite impressively so compared to the untreated side.

This lower eye lash angle present in grass sickness cases is likely due to paralysis of the smooth muscle innervating the eyelashes themselves, the arectores ciliarium, present in horses and cattle but not in man and dogs. This phenylephrine eye drop test is therefore very useful to assist in diagnosis of grass sickness and other causes of Horner’s syndrome in horses (Hahn and Mayhew 2000a) as long as other antagonist or agonist drugs such as xylazine have not recently been administered.

Third order sympathetic neuronal fibres do not pass through the petrosal bone in horses therefore Horner’s syndrome usually is not recognised with oitis media or with petrous temporal bone fractures. In horses, the sympathetic fibres innervating the eye are more often damaged in and around the guttural pouches. Finally, many systemic toxins, such as those mediated by atropine-like alkaloids and those acting with anticholinesterase activity, cause degrees of mydriasis and miosis, respectively.

Strabismus

An abnormal deviation of the axis of the eyeball is referred to as strabismus. Loss of function of the extraocular muscles innervated by the oculomotor nerve (CN III) should result in a lateral and slightly ventral strabismus. Medial strabismus should result from abducens nerve (CN VI) lesions. Rotation of the globe such that the medial aspect of the pupil moves dorsomedially should result with trochlear nerve (CN IV) involvement. In each of these cases the eyeball cannot be moved out of the deviated position so this can be considered as examples of fixed strabismus. These are rarely seen alone as acquired syndromes in horses. However, an eyeball deviation, particularly when it is ventral or dorsal and the eye is able to return to its normal position with induced head movement,
is frequently seen. Usually this is because of diseases of the vestibular system, and is referred to as vestibular strabismus (Fig 12). The nuclei of CNs III, IV and VI that control eye movement are connected with the vestibular system by way of the medial longitudinal fasciculus (MLF) (Fig 5).

Coordinated eye movements are orchestrated via the MLF in response to changes in positioning and movement of the head. The MLF conducts impulses to maintain ipsilateral antigravity tone to the eyeballs keeping them from lowering in the bony orbits. Thus unilateral vestibular disease often results in lowering of the eye in the bony orbit on the same side as the lesion due to loss of antigravity tone; a ventral strabismus. This may not be very evident with the head in a normal posture but usually is exaggerated when the tip of the nose is raised. Performing a fundic examination to identify the exact position of the optic disc also can assist in defining asymmetric positioning of the pair of eyeballs.

Congenital blindness can be associated with abnormal eyeball positions. Also, Appaloosa horses affected severely with night blindness can have dorsomedial strabismus, which may be noticed when they attempt to visually fix on an object (Rebhun et al. 1984).

Although many inflammatory, physical, metabolic, toxic and nutritional disorders may affect the regions of the brainstem where the oculomotor, trochlear and abducens nuclei are located, prominent signs of fixed strabismus rarely are seen. However, many severe asymmetric forebrain diseases result in turning of the head and neck and drifting towards one side, usually the side of the lesion. This also can involve the eyes being drawn towards one side, particularly with thalamic involvement, and these signs collectively comprise the adversion syndrome. The relative dorsomedial rotation of the eyeball (Fig 13) seen in bacterial meningitis and hypoxic and ischaemic encephalopathy in neonatal foals may not be a specific trochlear nerve paralysis. The eye can be moved in all four directions and out of the abnormal position so that it might reflect severe forebrain disease involving the upper motor pathways controlling eyeball posture. A similar dorsomedial eyeball rotation also can be seen with congenital cerebellar hypoplasia and with cerebellar atrophy in several breeds likely due to alteration in vestibulocerebellar tonic control of extraocular muscles.

Finally, with cases of congenital peripheral blindness there can be associated abnormal eyeball positions, with the globe directed dorsally and sometimes with wavering, searching eyeball movements (Boydell 1997; Barnett et al. 2004; Mayhew 2004, 2009; Gold et al. 2008; Wolfe et al. 2008).
Facial paresis and facial spasm

Facial paresis to paralysis is seen as degrees of weakness of the muscles of facial expression, including ptosis of the upper eyelid, and is a very common problem in horses with neurologic disorders (Fig 12). Dysfunction of the facial nerve produces decreased spontaneous and reflex movements of the eye, eyelids, lips and external nares. The eye and lips on the side of the lesion droop and the muzzle tends to be pulled to the opposite side with a unilateral lesion. Ptosis of the upper eyelid is a prominent feature of facial paralysis, this being somewhat unexpected as the facial nerve and orbicularis oculi muscle act to close the eyelids i.e. lower the upper eyelid. The ptosis is however due to paralysis of the strong levator anguli oculi medialis muscle in horses that is innervated by the facial nerve. It is possible that the bulk of atomic supraorbital muscles and paralysis of the frontalis muscle contribute to the ptosis also. Unfortunately, when upper eyelid ptosis is seen in horses the immediate thought is one of it being due to Horner’s syndrome (Fig 9). However, facial paralysis is certainly much more common than sympathetic denervation of the eyelids in horses and always must be considered the most likely cause.

Lesions involving the upper motor pathways controlling the facial nucleus and nerve, and which are in the frontal cortex, medulla oblongata and brainstem, can result in abnormal facial expression. This occurs without flaccid facial paralysis. There is still tone in the muscles of facial expression and facial reflexes (CN V sensory → CN VII motor) are present, but the face may be without expression or may be grimacing on one or both sides. Needle electromyographic examination of the facial muscles does not reveal denervation because lower motor neuron disease has not occurred. Large, focal cerebral lesions such as haematomas, S. neurona encephalitis and abscess have produced such signs of supranuclear facial motor dysfunction that appear to be ipsilesional, at least to the muzzle and lips of the distal face.

With distal, peripheral, facial nerve lesions usually one or two branches of the nerve, not all three nerve branches (auricular, palpebral and buccal) are involved. Pressure on the side of the face as a result of a tight halter or recumbency damages buccal branches, paralysing just the nares and lips; however, the ear and upper eyelid may droop because of additional direct trauma to the auricular and palpebral nerve branches. Brainstem lesions, particularly those caused by equine protozoal myeloencephalitis can selectively involve the facial nucleus in the brainstem and can mimic a peripheral lesion by producing a selective, partial, facial nuclear lesion and paresis.

Horses accommodate to unilateral facial paralysis very well but bilateral facial paralysis results in difficulty prehending and chewing food and they sequester food in the flaccid cheek pouches and drop a lot while eating. Permanent facial paralysis may necessitate enucleation of the eyeball because of keratitis sicca and exposure opthalmitis. Exercising horses may require false nostril surgery as a result of an obstruction to inspiratory airflow (Torre 2003). Chronic paralysis with muscle atrophy and fibrous contracture of the face can cause twisting of the muzzle and nares back across the midline towards the paralysed side.

In the early stage of irritative lesions, such as meningitis, neuritis and focal trauma involving the facial nerve, facial muscles can twitch and even remain in spasm prior to paresis or paralysis that often ensues. True, permanent, hemifacial spasm with constant contraction of facial muscles that only relaxes following facial nerve anaesthesia and general anaesthesia does not yet appear to be recorded in horses. This might be expected to occur following recovery from bacterial otitis media. As occurs in many other regional muscle groups (Beech 1982), facial muscles occasionally are seen to undergo repetitive contraction described as a facial tic. Some of these syndromes wax and wane and one facial tic in a horse has been seen to become profound when concurrent but unrelated hypocalcaemia occurred and to quieten with calcium treatment. The underlying cause for most tic-syndromes is notoriously not found (Beech 1982; de Lahunta et al. 2006; Gold et al. 2008).

Nystagmus and vestibular disease

Any oscillations of the eyeballs can be regarded as nystagmus and are almost always conjugal in nature. Movements can be of equal force and range in 2 directions when it is termed pendular or oscillatory nystagmus. If there is a slower (active) and a faster (rebound) phase to movement then the direction is traditionally defined by the fast phase. Normal (physiological) vestibular nystagmus can be induced by moving the head (and the vestibular labyrinth) back and forth in 2 directions. Most other types of nystagmus occur with the head at rest and are abnormal. The majority of these cases have some form of vestibular system disease.

The vestibular system is a special proprioceptive system that helps the animal maintain orientation within its environment with respect to gravity. The system helps to maintain the position of the eyes, trunk and limbs in relationship to movements and positioning of the head. The receptor end organ is located in the inner ear, consisting of the three semicircular canals, utricle and saccule. Movement of endolymph in the semicircular canals induced by movement of the head and by the effects of gravity stimulates receptors that transmit impulses to the vestibular nuclei in the medulla oblongata by way of the vestibular nerve and via its ganglion in the lateral wall of the fourth ventricle. There is a direct afferent connection to small parts of the cerebellum, the flocculonodular lobes and fastigial (medial) cerebellar nuclei (Fig 5).

Signs of vestibular disease vary depending on whether there is unilateral or bilateral involvement, and whether the disease...
involves peripheral or central components of the system. General signs of symmetric vestibular system dysfunction are staggering with the limbs, truncal sway, leaning, falling, drifting sideways when walking and a wide base stance and gait. Various changes in eye position (strabismus) and movement (nystagmus) occur.

In the case of unilateral and asymmetric vestibular system involvement, asymmetric posturing and staggering movements occur. The animal has a tendency to drift, lean and roll toward the side of the lesion. A head tilt is a relatively constant sign with asymmetric vestibular disease, along with nystagmus. The nystagmus tends to regress with time, but changes in head position may re-activate it. With unilateral, peripheral vestibular disease, nystagmus is horizontal or partly rotary with the fast phase directed away from the side of the lesion. With bilateral peripheral vestibular disease, vertical nystagmus has rarely been recognised rarely and it is temporary (Peele 1977). In central vestibular disturbances the nystagmus may be horizontal, rotary or vertical. Head elevation also exaggerates any tendency for strabismus and nystagmus, at least seen as a vertical deviation of the eye on the same side as the lesion.

Bilateral vestibular lesions are characterised by more symmetric signs, which strongly resemble generalised cerebellar disease with wide swaying movements of the head and sometimes the trunk. Significantly, no physiological vestibular nystagmus can be induced with lateral head motion in cases of dense, bilateral vestibular disease.

Bacterial otitis media/internal and head trauma with bleeding into the middle and inner ear cavities, would be 2 of the commonest causes of peripheral vestibular disease in foals and horses respectively. Both can result in a head tilt toward the lesion, facial paralysis on the same side as the lesion, and ipsilesional ventral strabismus and horizontal or otory nystagmus, with the fast phase of nystagmus directed away from the side of the lesion.

Temporohyoid osteoarthropathy with degenerative joint disease involving the temporohyoid joint occurs in mature horses and can lead by extension into the inner ear to peracute to chronically progressive, asymmetric and usually unilateral signs of vestibular disease. The cause of the condition is not certain but may well involve singular and repeated episodes of temporohyoid joint trauma associated with external injury, vigorous head movement, dental malocclusion and possibly even vigorous dental procedures, ending up with temporohyoid degenerative osteoarthropathy.

An acute, temporary, unilateral vestibular disorder with no other signs occurs sporadically in adult equids. The cause is unknown, although a viral vestibular neuritis or labyrinthitis is hypothesised. These animals appear to recover irrespective of medication. Some cases may be undiagnosed examples of other diseases.

Horses can present with asymmetric central vestibular signs caused by equine protozoal myeloencephalitis and by migrating metazoan parasites such as Strongylus vulgaris, Draschia megastoma and Hypoderma spp. larvae. Evidence of multifocal disease would make equine protozoal myeloencephalitis more likely than larval migrans (Anderson et al. 1990).

Prominent symmetric, central vestibular signs often dominate the diffuse spinovestibulocerebellar signs of perennial eye grass and Dallis grass mycotoxicoses. These vestibular signs include eye deviations and rapid, variable eyeball tremor or nystagmus, seen best on direct fundic examination. Central vestibular signs also can accompany many diffuse brain diseases such as rabies, Eastern, Western and West Nile equine viral encephalomyelitis and hepatoencephalopathy. Space-occupying lesions such as neoplasms, abscesses and cholesterol granulomata involving the choroid plexus of the fourth ventricle, may also affect the vestibular system.

Excessive ventral eye deviation seen when the head is extended on the neck, and a slight head tilt when the patient is blindfolded often are the last remaining signs to be seen following convalescence from vestibular disease. Vision is a major input modality to the vestibular system and vision is required to fully accommodate for vestibular deficits. Therefore, blind animals do not accommodate well.

The membranous labyrinth in the inner ear, the vestibular branch of CN VIII and the vestibular nuclei in the medulla oblongata run in parallel with the first components of the auditory system. Therefore, lesions affecting one system are very likely to affect the other. By this assumption, brainstem auditory evoked potential (BAEP) recordings can be used as an indirect assessment of vestibular pathway function. One horse with mild vestibular signs due to temporohyoid osteoarthropathy was confirmed by BAEP recordings to be totally deaf in the affected ear (Marshall et al. 1981). Although not predictive, it has been noted that horses with a flat BAEP tracing at 100 dB testing level, have tended to remain with residual vestibular signs compared to horses with acquired vestibular disease of comparable magnitude but with some appreciable and repeatable BAEP waveforms, even if that has only been a residual delay of and suppression of wave V of the BAEP waveform.

### Cerebellar disease

Cerebellar ataxia (Holliday 1979/80) is characteristically hypermetric (high stepping), hypometric (stiff legged), or a combination of these signs (dysmetric). In addition, head signs are present to include ataxia of the head and neck with wide, swinging, head excursions, jerky head bobbing, an intention tremour involving the head but not the body and limbs, and an abnormal menace response. Such a case is able to see and to blink its eyelids, but may not blink well in response to a menacing gesture directed toward each eye, although it may withdraw its head from the threatening gesture - a visual avoidance response. This apparent blindness (amaurosis) has been seen ipsilateral to unilateral cerebellar lesions. It is probable that the cerebellum positively influences the classic menace or eye preservation reflex. Thus, cerebellar lesions might result in suppression of the reflex. Newborn foals frequently have a slightly hypermetric gait reminiscent of cerebellar dysfunction and this is accompanied by an apparent anaurosis with lack of menace reflexes for the first week of life with no evidence of blindness or facial weakness.

### References


