Neurologic exam in Large Animals - Careful he might fall on you
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References
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Neurologic assessment in larger animals (horses, ruminants, pigs, camelids and probably any quadruped) is essentially the same as in dogs with obvious adjustments for size and temperament and RISK. Rabies is a potential transmissible infection to humans from all mammalian species and may present with any neurologic signs in ruminants and horses (including colic and apparent lameness). Hendra virus infection can also cause rapidly progressive neurologic signs and is a very significant risk to in contact humans.

Aim of any neurologic exam is to determine:

Whether neurologic deficits are present (or not) and if possible localize lesions to a particular part of the nervous system to enable formulation of (in conjunction with historical and physical exam findings) both a list of possible causes (differential diagnosis) and a diagnostic or treatment plan.

Essentially to determine whether forebrain (cerebrum), midbrain, caudal fossa (including caudal brainstem, cranial nerve, vestibular, cerebellar), spinal cord and/or peripheral nerve (including NMJ or muscle disease) is likely.

Neurologic abnormalities are relatively common - neurologic disorders of sheep and cattle in Australia were rated in top 5 reported diseases in 2009 (Finnie et al AVJ)

Essentially a neurologic exam consists of an assessment of

1. Level of consciousness, mentation and behavior
2. Posture and gait
3. Postural reactions
4. Spinal nerve function
5. Cranial nerve function
The first three of these can be made by observation only if necessary and give the most information. Smaller animals (sheep, goats, pigs, foals) be easier to handle and many of the tests used in dogs can also be applied to them but can be uncooperative. Horses and cattle can fall and injure examiners. Animals with altered mentation and those at risk of falling should be approached and moved carefully.

Assessment in a paddock - freely moving initially - is ideal. Animals with significant paresis or ataxia will be worse on concrete or bitumen -use grassed area or yard to examine.

1. **Behaviour /mentation/ level of consciousness**

History is important – behaviour in paddock or stall

Abnormal behavior and/or mentation is seen with forebrain disease but a decreased level of consciousness or arousal is also seen with brainstem disease due to involvement of the ascending reticular activating system of neurones (ARAS) and abnormalities in level of consciousness may be described as lethargic, dull obtunded, stupor. Coma is primarily seen with severe brainstem lesions (midbrain predominantly) rather than cerebrocortical disease.

Aimless or compulsive wandering, head pressing, decreased responsiveness, abnormal vocalization or hyperexcitability and mania may be seen. Circling may be seen with abnormalities anywhere in the brain including the forebrain and tends to be towards side of abnormality. Other abnormal behaviours may be seen with brain disease for example repeated yawning, adoption of abnormal postures, compulsive licking or aggression. Seizures (partial or generalized) may be seen in animals with forebrain disease.

Behavior can be very abnormal associated with pain or fear -eg animals that become acutely recumbent for any reason may thrash and become distressed and/or disoriented.

An assessment of visual acuity should also be made. Does an animal bump into things or be reluctant to move around. However poor responsiveness, postural deficits or imbalance in animals with forebrain disease or vestibular disturbance may be misinterpreted as a vision abnormality.

2. **Posture and Gait**

Abnormal postures may be the result of pain (eg cervical pain seen as a fixed, stiff or rigid neck position) or weakness or other neurologic abnormality. Head position is maintained by the vestibular system. Unilateral vestibular disease causes head tilt (evaluate with head in a sagittal plane) and possibly head turn. Cerebral and midbrain disease may also cause a head turn. In bilateral vestibular disease there may be no head tilt and wide sweeping movements of head.

In diseases causing generalized LMN weakness (eg botulism) cervical weakness is often evident with low head and neck carriage and inability to raise the head.

An extended head and neck position (star gazing) is seen in severe diffuse cerebral disease in PEM. Opisthotonus and extension of limbs is seen in decerebrate posturing (animals will be unconscious), decerebellate (acute severe cerebellar disease) posturing and diseases causing increased muscle tone such as tetanus.

Tremor, trembling or other abnormal movements may be seen associated with weakness, cerebellar disease (head and neck only and usually coarse tremor), diffuse CNS disease
(intoxications, neurodegenerative diseases) and basal nuclear disorders (nigropalladial encephalomalacia associated with centaurea sp intoxication)

Animals that are generally weak may adopt an abnormal stance – base wide or narrow in an effort to maintain a standing position.

Note: head position in foals normally more flexed than an adult horse.

Gait

Which limbs are abnormal? - lame or is a neurologic abnormality present?

Gait alterations can occur in diseases of the brainstem, cerebellum, spinal cord and peripheral nerves (including neuromuscular junction or muscle). Limbs affected and other abnormalities found on exam will help determine most likely location. Gait abnormalities (and postural deficits seen with brainstem, cerebellar, spinal cord and peripheral nerve lesions are ipsilateral to the lesion/s.

Cerebral disease rarely causes gait abnormality however may cause postural deficits on testing – these are contralateral to the cerebral lesion.

Weakness and/or ataxia

Weakness may be due to reduction in muscle strength (LMN) or reduction in voluntary motor movement (UMN). Ataxia (incoordination) is primarily the result of a sensory abnormality however often is difficult to distinguish between paresis and ataxia as both likely to occur in brainstem or spinal cord disease.

Ataxia is a feature of diseases that affect the cerebellum or vestibular system or spinocerebellar tracts in the spinal cord. Hypermetria or increased range of movement (seen as overereaching) is seen in cerebellar and cervical spinal cord disease (spinocerebellar tracts affected). Ataxia may also be seen as a reduced range of movement with a stiff or spastic movement with reduced flexion especially carpal and tarsal flexion – this may be seen with vestibular disease or cerebellar disease.

Weakness only is a feature of lower motor neurone (LMN, NMJ and muscle) disease however clinically weak animals also at times appear uncoordinated.

Most deficits are evident at a slower pace- walk and may be exacerbated by TURNS.

3. Postural reaction tests both proprioception (sensory awareness of the body in space which involves both conscious and unconscious mechanisms) and motor function to enable the appropriate movements.

Subtle deficits (both proprioceptive and motor) may require more complex manoeuvres to appear.

Paresis (UMN and LMN) and postural deficits may be seen walking up and down a kerb (zig zag) or up and down a slope (can be made worse by elevation of the head whilst walking). May show a tendency to float or overreach with TL limbs, scuff and buckle at onset in cervical lesions or misplace or scuff hind digits in TL or cervical lesions.
Length of stride, delay in protraction (swing phase) or overreaching, swinging of limb in or out (adduction or abduction), swaying of trunk all indicate proprioceptive deficit. Decreased ability to bear weight dragging of digit (wear on hoof?) are also indicators of paresis.

Note there are breed differences in normal gait. (Pasos, Tennessee walkers)

Other postural reactions eg hopping can be tested as in small animals in small ruminants and foals if cooperative.

Circling (8-10 times deLahunta) is most useful test in detecting subtle neurologic abnormalities – both ataxia and paresis (if you don’t get dizzy first) small tight circles with leading person in the middle. Normal animals will not pivot on a limb or step on itself (ie knows where its limbs are). Animals with spinal cord disease generally have both proprioceptive and motor (UMN) abnormalities and will pivot on the inside limb. Outside (pelvic limbs) may abduct excessively with a wider swinging motion (cervical or TL lesions) Forelimbs may cross or step on opposite limb (with cervical lesions).

Sway – is a test primarily of paresis (weakness/voluntary motor movement). This is done by trying to pull an animal (primarily horses) towards you by the tail both when stationary and when walking.

Normally a walking or stationary animal can’t be pulled by the tail to either side (towards person pulling). If they can it is indicative of paresis. It can be subjective some horses will move towards the puller (hoof placement however is appropriate). Try pulling both head and tail towards operator – a normal horse should not be able to do.

Weak animals will stumble on a weak limb and may knuckle over on fetlock. Animals with LMN lesions are easy to pull to one side. Those with UMN disease may be able to extend limb to resist pull when standing (spastic-exaggerated extensor reflex) but not when walking.

Note vestibular disturbance may cause a decrease in muscle tone on affected side and increased tone on the contralateral (opposite) side (hence tendency to fall to the affected side).

Ataxia may be more evident when freely moving – especially when stopping at a canter. Stopping an animal when moving or circling is also a good test of proprioception to see how long it takes to adopt a normal stance.

Back ing – animals with significant proprioceptive deficit and weakness will have difficulty backing (cooperative animals required here). Animals may be slow to move a limb when backed or in severe abnormality will sit or collapse backwards.

Paretic animal may sink, buckle digits or collapse if palpated firmly along the dorsal trunk

In recumbent animals an assessment of muscle tone and observation of any voluntary movement made.

4. Spinal nerves

Assessed by muscle mass and tone and spinal reflexes in recumbent animals.

If an animal can stand and walk spinal reflexes are going to be intact. In recumbent smaller and some large animals patellar reflexes and flexion reflexes can be assessed. These can be done in up limb but are not useful in down limbs in larger animals due to weight on the limbs.
Muscle atrophy – may due to denervation / disuse or inadequate nutritional plane. Neurogenic atrophy rapid and may be confined to one muscle or group (if one nerve affected eg suprascapular nerve in sweeney or diffuse eg equine motor neurone disease.

Cutaneous reflexes

These reflexes are most useful in the horse and elicited by tapping the skin with pressure (pen or other blunt object along the side which will elicit a contraction of muscle under the skin. In the neck this stimulates contraction of the cutaneous colli muscle (innervated by branch of facial nerve –arises from the caudal brainstem) and in the trunk stimulates contraction of the cutaneous trunci muscle (innervated by the lateral thoracic nerve which arises from the C8T1 spinal cord segment). This muscle contraction is elicitable in most normal horses but is less reliable in cattle. This also tests nociception (perception of pain). Focal loss of sensitivity not usually detected due to the overlap of cutaneous areas of each spinal cutaneous nerve . Loss of the cutaneous trunci reflex may be seen with severe TL spinal cord lesions caudal to a lesion or absence of reflex with C8T1 lesions. If a cut off in cutaneous trunci reflex (will be both cutaneous sensitivity and possibly nociception )is seen the level of injury will be approx 2 vertebral cranial to cut off.

Loss of cervical reflex may be seen with severe cervical spinal cord lesions or facial nerve lesions.

Focal loss of sensation may be seen in the tail, anus and perineum in animals with cauda equina neuritis or sacral fractures or tumours/empyema within the spinal canal at this level(caudal lumbar/sacral).

Anal reflex (contraction of the anus with stimulation) and tail tone can be tested when taking temperature

The perineal reflex- closure of anus and flexion of the tail in response to stimulation of the perineum tests integrity of the sacral nerves (pudendal/perineal) and caudal nerves (tail) and spinal cord segments from which these nerves arise (S1-Cd segments).

Inability to urinate with urinary sphincter weakness and bladder overflow and/or large amount faeces in rectum due to lack of rectal tone and contraction can be seen with sacral spinal cord segment or sacral nerve root/spinal nerve disease.

A Patellar reflex can be elicited in recumbent animals and tests the integrity of the L4L5 spinal cord segments, spinal nerves and femoral nerve. The tested limb should be held in in partial flexion and relaxed

Flexion or withdrawal reflex can be assessed in hind or forelimb of a recumbent animal. The skin of coronary band or bulb of hoof is squeezed. In the hind limb this reflex tests primarily sciatic nerve function (L6, S1,S2). Flexion of hip is a function of the femoral nerve as well therefore flexion reflex involves all lumbar and sacral spinal cord segments spinal nerve roots, spinal nerves, sciatic and femoral nerves.

In the forelimb the afferent arm of the flexion reflex depends where the hoof is squeezed (median or ulnar nerve in the horse +/- radial nerve in other animals). Flexion involves all of nerves of brachial plexus. (arising from the C6-T2 spinal cord segments spinal nerve roots and spinal nerves)

5.Cranial nerve exam
Cranial nerves may be affected peripherally (outside the cranial vault) or intracranially (within the brainstem or nerve trunks within the cranial vault). Cranial nerves 111 to XII arise from the brainstem (CN111 and IV from midbrain and V-X11 from caudal brainstem).

**Menace response** – blink or movement of the head in response to a visual threat is a response not a reflex - animals have to be attentive. Behavioural evidence of blindness is more useful in assessing vision (eg bumping into things, ability to avoid obstacles). Menace may be lost with normal or reduced vision in forebrain disease. In this case response deficit is contralateral to lesion.

Ophthalmologic exam is important as loss of vision is most commonly associated with ocular disease. Cerebellar lesions can cause reduction in menace response without vision loss. Menace not developed in foals or calves for several days after birth but will blink to a bright light.

**Pupillary Light reflexes** do not indicate vision – PLR is a brainstem reflex (midbrain)

**Eye position** – the pupil should be oriented in middle of palpebral fissure and oriented horizontally. Mild ventromedial strabismus is normal in foals with rotation ventrally of the pupil.

**Strabismus** refers to an abnormal eyeball position, rotation of the pupil or abnormal position of the eye and is seen in all head positions (fixed) with abnormalities of CN111, 1V or VI or associated with abnormalities of the extraocular muscles. Strabismus is rarely recognized (possibly with midbrain lesions)

**Positional strabismus** is seen with vestibular disease as vestibular input is important in maintaining eye position. Strabismus is ventral (may be slightly medial) but the eyeball can move out of this position when head is moved and usually ipsilateral to lesion. Congenitally blind animals may have abnormal eye positioning.

**Normal physiologic nystagmus** is seen with movement of head side to side (vestibulo-ocular reflex) (requires intact CNs 111,1V,V1, vestibular system and brainstem connections). Fast phase is in the direction of head movement.

In large animals if the head is elevated the eyes stay horizontal and appear to deviate downwards.

**Corneal reflex** tests corneal sensation (ophthalmic branch, trigeminal nerve). Retraction of the globe may require intact CN111 and IV in addition to IV.

**Sympathetic nerve supply to eye** is not a cranial nerve however is considered here. An abnormality in sympathetic innervations to the eye causes Horner’s syndrome and is seen predominantly with lesions in region of middle ear/guttural pouch, cervical vagosympathetic trunk and thoracic inlet. Horner’s syndrome is characterized by ptosis, enophthalmus and protrusion of nictitating membrane. In horses, dilation of facial blood vessels, hyperemia of nasal mucosa and conjunctiva ipsilaterally and sweating on side of face (around ear) is also seen - to level of upper body with preganglionic lesions and to level of axis with preganglionic lesions. Cattle show dilated blood vessels of the pinna and lack of sweat production over muzzle. Only eye signs are seen in sheep and goats. In the cervical spinal cord – severe lateral funicular lesions or T1-T3 spinal cord/spinal nerve lesions – Horner’s syndrome may be seen with (ipsilateral)sympathetic paralysis on one side of body as well as the head causing ipsilateral whole body sweating. Focal lesions T3-L4 (affecting the intermediate grey column within the spinal cord) causes focal sweating in a discrete area of skin denervated.
**Trigeminal nerve**

Motor to muscles of mastication and sensory to head

Bilateral lesions cause jaw drop and often salivation, inability to chew and tongue may appear protruded.

Normal horses may have asymmetric temporalis muscle mass (not masticatory)

In stoic animals stimulation of the nares, nasal septum (ophthalmic branch) may be requires to elicit a response to sensory stimulation.

Note sensory loss may be seen without motor dysfunction –due to the separation of motor nuclei and sensory nucleus/tract in the medulla. Contralateral forebrain abnormalities (where perception of a sensory stimulus occurs) may cause apparent decreased sensation (may be seen as “unawareness” of food impacted in the mouth). In the case of forebrain disease cranial nerve reflexes (eg corneal reflex) will be normal.

**Facial nerve**  paralysis or paresis results in ptosis of the upper eyelid (different to dogs and cats) drooping lips, deviation of the nasal philtrum (horse) and a drooped ear. Abnormalities may be subtle in food animals especially if paresis rather than paralysis. **Horses have a well developed palpebral reflex.** Reduced tear production is rarely recognized in horded with facial nerve paralysis (per Jeff Smith).

Focal thalamic and cerebral lesions may cause increased facial muscle movement (hyperreactivity) in response to a stimulus and grimacing.

**Vestibular cochlear nerve**

Deafness difficult to assess unless bilateral (may be congenital paints)

Abnormalities of vestibular function -head tilt, positional strabismus, ataxia, disequilibrium and spontaneous or positional nystagmus are seen in both peripheral and central vestibular disturbance. Fast phase of nystagmus is usually away from the side of lesion and horizontal or rotatory in peripheral nerve /inner ear disease. Vertical nystagmus is most commonly associated with central lesions but can be seen in peripheral vestibular disease. A change in direction of nystagmus with head position is more common with central lesions. Peripheral disease may be more severe but improves more quickly ( animals compensate ). Blindfolding will cause decompensation.

To distinguish peripheral from central causes of vestibular disturbance look for other signs that would indicate brainstem disease (altered consciousness, paresis, other CN deficits).

**Cranial nerves X, X1 and X11** enable prehension of food, swallowing and laryngeal function (X1). The swallowing reflex can be initiated by passing stomach tube. Prehension and swallowing can be assessed by watching an animal eating and drinking. Laryngeal function is assessed by endoscopic inspection. The severity of signs depend if an abnormality is unilateral or bilateral. If pharyngeal paresis is present food and water is found in the nostrils of horses and nostrils and mouth of ruminants. Dysphagia can also be caused by forebrain abnormalities with normal swallowing reflexes. Cranial nerves may be affected in guttural pouch disease, peripheral neuropathy, diffuse LMN or muscle disease including botulism, rabies.
Abnormal respiratory noise is seen in laryngeal paralysis

The **SLAP test or laryngeal adduction test** requires integrity of all cervical and cranial thoracic spinal cord segments, nucleus ambiguus (medulla) vagal nerve and recurrent laryngeal n. The horse is slapped in saddle area of thorax behind the dorsal scapula whilst palpating the larynx. A normal response closure of the glottis with adduction of vocal folds (can be palpated or observed with an endoscope). With a slap on one side, sensory information is projected cranially in the cervical spinal cord contralaterally to the motor nucleus of the vagal n. Theoretically unilateral lesions of the cervical spinal cord cranial to thorax will cause a lack of this reflex if the contralateral thorax is slapped. Usefulness? Horses with recurrent laryngeal paralysis (L sided usually) will not have this reflex on the affected side. It is seen in foals as early as 2 weeks of age.

**Accessory nerve** – abnormalities rarely recognized.

**Hypoglossal nerve** is motor to the tongue.

Abnormalities in hypoglossal nerve function are seen as atrophy of one or both sides (bilateral disease uncommon) of the tongue, abnormalities of position of the tongue in the mouth and weakness in tongue movement or when tongue is grasped. Atrophy of the tongue may also be seen with muscle disease. The tongue may protrude in severe forebrain disease but normal movement is present.