Wildlife brain and spinal cord injuries

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Introduction

Neurologic abnormalities can be difficult to assess in animals and even more so in non-domestic mammals, birds and reptiles.

Trauma to the head and brain can result from motor vehicle accidents, falls, trampling injuries, penetrating injuries, injuries inflicted by other animals and malicious attacks. Acute spinal cord injury most commonly is seen associated with direct physical trauma which may be associated with vertebral fracture as a result of motor vehicle accidents, falls, missile injury, dog or other animal attacks. Severe spinal cord injury can occur after struggling without any obvious vertebral injury. Concussive injuries may also cause significant spinal cord injury without associated fracture. Vertebral fracture may be pathologic associated with vertebral infection or neoplasia or nutritional disorders causing severe osteopaenia. Peripheral nerve injuries in a limb may occur secondary to fractures of the pelvis or limbs as a result of motor vehicle accidents, trapping injuries, avulsion injuries, bite wounds, ligature type injuries etc.

Unfortunately, many animals may come into care with neurologic signs with trauma suspected but have other causes of neurologic abnormality which then causes them to be found due to poor mobility or abnormal behavioural responses. Parasitic diseases, especially *Angiostrongylus cantonensis* (rat lungworm) infection can cause progressive spinal cord and brain abnormality in mammals and birds. Other infections, including bacterial, fungal or viral disease, can be seen that affect the brain or spinal cord and may cause focal, multifocal or more diffuse neurologic signs. This includes wobbly possum disease and lyssavirus infection in bats/flying foxes. Metabolic diseases (liver/kidney/hypoglycaemia) can affect both the brain and general peripheral nerve function. Generalised peripheral neuropathy or neuromuscular disease may occur associated with toxins (eg botulism).

How do I know whether an animal has a neurologic problem?

The nervous system of all vertebrate animals has the same basic components - brain, spinal cord and peripheral nerves with some variation in anatomy especially with respect to vertebral number (and therefore number of spinal nerves), brain size and in relative importance of various regions of the brain. However, the same assessment that is used for domestic mammals can be adapted for assessment of other mammals, birds and reptiles

Observations that are important in making a neurologic assessment:

1. State of consciousness /awareness/ responsiveness

Mentation and behaviour

Any involuntary movements eg tremor or abnormal movements (circling)

Any abnormality is indicative of brain dysfunction.

An assessment should be made as to whether responsiveness is normal or abnormal (subdued, dull, unconscious). Is the animal's behaviour appropriate for the environment or not?

Assess vision (ability to negotiate an unfamiliar environment, tracking of objects) and hearing (responsiveness to noise)

2. Posture

Head tilt may be associated with injury to the inner ear structures or brain. Head turn or body turn is seen with some brain diseases. Sustained extension of all 4 limbs and unconsciousness is associated with decerebration and is a very poor prognostic sign.

3. Gait / Movement

Is the animal able to stand and walk, perch or climb? **Weakness (paresis or paralysis**) may be seen in animals with spinal cord injury, peripheral nerve abnormalities, muscle disease or metabolic disease.

Can the animal voluntarily move all four limbs and tail?

Which limbs are affected – this may help determine the cause – if only the hindlimbs then a spinal cord problem below the brachial enlargement is likely (thoracic or lumbar). If all 4 limbs are affected, then a cervical spinal cord or brainstem or cerebellar (intracranial) abnormality is likely or a more diffuse peripheral neuropathy causing generalised weakness.

If one limb only is affected, then abnormality of the peripheral nerves (outside the spinal cord) is more likely.

Ataxia – is incoordination with normal strength and is usually associated with brain abnormality.

4. Postural reactions

These are reactions not reflexes and depend on all parts of the nervous system from sensory receptors (muscle and joint receptors and vestibular system) to the level of the cerebral cortex as well as motor pathways to effect the motor response. Proprioception is the sense of where the body is in space and postural reactions test the correction of muscle tone, limb, trunk and head position to support weight and maintain an upright position. The postural reactions involve both conscious and "unconscious" (subcortical) mechanisms. Postural reaction tests are used to test proprioception (sensory) and to identify paresis (motor response) that may not be evident on gait assessment.

Abnormalities may not be of specific localizing value but are important when considered with the rest of the neurologic exam. These are conscious reactions and may not be present in animals that have signs of altered mentation. The most useful tests are

Reaching/ Placing to a tabletop (visual and tactile)

Extensor postural thrust (vestibular) - extending limbs when lowered to the floor

Righting (vestibular)

5. Spinal reflexes

Spinal reflex testing assesses the integrity of the nerves and the spinal cord segments from which these nerves arise that mediate the reflex. Reflex abnormalities may be seen with CNS (spinal cord) or peripheral nerve disease. Reflex testing is useful in determining whether an animal is more likely to have a focal or multifocal spinal cord abnormality and the likely level of spinal cord injury or more likely to have a more diffuse peripheral neuropathy or neuromuscular disease.

Reflexes occur independently of higher centres (and will occur even if the spinal cord is transected cranial to the spinal cord segments that mediate the reflex) but reflexes may be altered by a spinal cord injury more cranial to the reflex circuitry - for appearance of abnormal reflexes such as the crossed extensor reflex.

The flexion (withdrawal) reflex is the most important reflex assessed. Pinching (applying a noxious stimulus) to a toe/wingtip should cause a strong retraction of the limb.

Fore (thoracic) limb reflex is mediated by all nerves of the forelimb and the spinal cord segments (lower cervical and upper thoracic) from which they arise.

Hind (pelvic) limb reflex is mediated by the lumbar sacral nerves and spinal cord segments.

This is the time to assess **muscle tone** - normal or increased (hypertonic) or decreased (hypotonic/flaccid). Increased muscle tone (limb extension) is seen in spinal cord and brain abnormality. Reduced muscle tone is seen in spinal cord disease affecting the segments where the nerves to the limbs arise or due to peripheral nerve disease.

Also assess **muscle mass** – as normal, reduced, atrophic. Muscle abnormalities may be seen in a specific muscle or group of muscles or may be generalised. Abnormality may be due to malnutrition or systemic disease but may also be due to denervation, disuse or myopathy.

This is also the time to test **nociception** (**response to a painful stimulus - "pain" perception**) in animals with poor or no voluntary motor movement (in one or more limbs) or in animals with specific peripheral nerve lesions. **Nociception is the conscious response to a noxious stimulus not reflex withdrawal of a limb and this assessment is critical in determining the prognosis for possible recovery of function in a limb. If an animal shows a conscious perception of a noxious stimulus then the prognosis is better and recovery of function may occur. If there is no conscious response to a noxious stimulus then prognosis for recovery of function in that limb is very poor.**

Perineal reflex (anal reflex) assesses the function of the sacral nerves. Pinching the skin adjacent to the anus or vent in animals with a cloaca with a haemostat will cause contraction of the anus/vent and often flexion of the tail.

6. Cranial nerve examination

Cranial nerve abnormalities may be seen associated with brainstem disease or generalised peripheral nerve/ neuromuscular disease. These are the nerves arising from the brainstem and the sensory tracts projecting to the brain. Abnormality in cranial nerve function may be seen with brain disease or abnormality of specific nerves outside the cranial vault. These nerves may be also be affected by any disease causing generalised peripheral nerve or neuromuscular weakness.

Assess palpebral fissure size and pupil size - May be altered by abnormalities in the visual pathway (optic nerve to brain) or nerves of the iris, midbrain disease or ophthalmic disease.

Pupillary light reflex - Shining a light into one eye causes constriction of the pupil in the illuminated eye. A consensual reflex (constriction in the other eye) may not be seen in some animals and is not seen in birds. The pupillary light reflex requires a strong light source to elicit in many anxious and/or frightened animals. This is a reflex and will be normal in animals that are blind due to cerebrocortical disease. Birds can voluntarily alter pupil size and PLR may be seen only briefly in normal birds.

Menace response test- This is a response not a reflex and non-neurologic abnormalities may affect this response. It is not present in birds and may not be seen reliably in other animals.

Palpebral reflex (CN V and VII) - Stimulating the skin of the medial canthus of the eye (CN V) causes animal to blink.

Facial sensation (CN V) may be tested by touching the inside of the nares and noting an aversive response.

Facial muscle movement CN VII, CN V in birds

Masticatory muscle mass, jaw tone, (CN V mandibular branch)

Swallowing, gag - assessment of ability to eat and drink (CN IX and X). Abnormality can cause dysphagia, aspiration, change in voice.

Tongue movement (CN XII)

Vestibulo-ocular reflexes Physiologic nystagmus - eye movements are elicited when head is moving and eye position is coordinated by CN VIII, CN III, CN IV, CN VI and brainstem (especially midbrain) pathways. An absence of vestibulo-ocular reflexes (normal eye movements) is associated with midbrain disease and is a poor prognostic indicator.

Abnormal eye movements can be seen when the head is stationary. Nystagmus may be rotational, horizonal or vertical and is seen with any disturbance to vestibular mechanisms including peripheral components (inner ear) and central components (brain abnormality).

7. Palpation of the head and vertebral column and manipulation of the head and neck – any evidence of painful response or palpable abnormality?

Findings on neurologic exam enable anatomic localization of abnormality - brain, spinal cord (cervical or thoracolumbar or lumbosacral) or peripheral nerve (including neuromuscular junction, muscle). Abnormalities may indicate focal, multifocal or diffuse disease. Anatomic localization is important in determining possible causes and prognostic indicators.

Brain/head trauma

Most primary brain injuries are the result of parenchymal contusion associated with impact and may or may not be associated with fracture of the bones of the cranial vault. Skull fractures are more likely in smaller animals. Epidural, subdural or subarachnoid haemorrhage and cerebral laceration due to skull fracture or penetrating wounds may also occur. There may be associated mandibular or other skull fractures (palate, nasal turbinates, zygomatic bones) and damage to the soft tissues of the face, eyes and/or mouth which make maintaining nutritional requirements very difficult. Neurologic abnormalities associated with brain injury include alteration in level of consciousness, mentation changes, abnormal posture, respiratory pattern changes, alteration in heart rate, visual disturbance, hemi or tetra paresis (weakness) or paralysis, vestibular dysfunction and other cranial nerve deficits. Cranial nerves may be damaged intra-cranially or extra-cranially (especially retro bulbar nerves). Spinal fracture (especially C1-C3) may also be associated with severe head trauma. Scleral haemorrhage may be seen as a result of subarachnoid haemorrhage (evident 12-24 hours post injury)

Maintenance of a patent airway and adequate oxygenation and ventilation should be addressed first in acutely injured animals. Thorough physical examination is necessary to detect any thoracic injuries, abdominal injuries, fractures, puncture wounds and any source of haemorrhage. Neurologic assessment should be done carefully with minimal handling and prior to any analgesic drug administration. An animal's level of consciousness, posturing, respiratory pattern, pupillary size and light responses and eye movements (brainstem reflexes) are the most important neurologic parameters in determining prognosis in animals with acute brain injuries.

Assessment for any evidence of spinal cord injury should be made by assessing voluntary movement in all limbs and tail, spinal reflexes (primarily flexion reflexes and assessment of muscle strength and pain perception. Non-life-threatening injuries, such as fractures should be immobilised/stabilised and further diagnostics and treatment deferred until the animal's neurologic status is stable. Lacerations and eye injuries should be cleaned and topical treatment given.

The two most important extra-cranial causes of secondary brain injury are systemic hypotension and hypoxaemia which result in decreased O_2 and nutrient delivery to damaged neuronal tissue. Hypoglycaemia or hyperglycaemia and other electrolyte derangements may also play a role. Systemic immune responses may also be a factor in severely traumatised patients.

Regardless of the mechanism the major cause of secondary, and often progressive, brain injury after head trauma is deficient O_2 delivery to brain tissue.

Treatment The treatment of brain injury is primarily medical

Medical treatment of the brain-injured patient is directed at supporting cerebral perfusion without exacerbating secondary brain injury. In human medicine hypoxaemia and hypotension, which may be episodic, are associated with a poor neurologic outcome. Recognition of problems and early treatment may improve outcome.

Recumbent patients should be positioned such that the head is elevated approximately 30 degrees above the heart without any compression of the jugular veins.

Maintenance of normovolaemia is necessary to maintain cerebral perfusion. Hypovolaemia can result in hypotensive episodes. Hypervolaemia can increase cerebral oedema in damaged tissue and secondarily increase ICP. The amount of fluids given should be based on physiologic parameters and not on empiric standard shock rates of fluid administration. If possible, an estimation of blood loss and identification of any ongoing haemorrhage should be made.

The use of **corticosteriods** in brain injury is controversial and now not recommended and any effect has not been demonstrated in brain trauma. Corticosteriods also have a hyperglycaemic effect which may exacerbate neuronal damage in brain injury. Persistent hyperglycaemia has been associated with a poor outcome in humans with brain injury and there is some evidence this is also the case in animals. No objective evaluation has been done in veterinary patients to assess effects of corticosteroids and are not recommended.

All trauma patients should be given analgesic drugs in at least the first few days (and longer depending on extent of injuries). Pain may increase intracranial pressure. Remember any sedative drugs will alter neurologic examination.

Antibiotics should be given to all animals with concurrent nasal or oral trauma, those with bleeding from the external ear canals, and animals with penetrating wounds to the skull. A broad spectrum bactericidal antibiotic should be used. Although all antibiotics will cross a damaged blood brain barrier many antibiotics do not reach therapeutic concentrations in nervous tissue.

Fluoroquinolones (enrofloxacin) reach adequate concentrations in normal brain tissue

Prognosis

Neurologic assessment should be repeated frequently. Animals that are unconscious with no evidence of response to a noxious stimulus (pain perception) and fixed dilated pupils have a poor prognosis at any time (presentation or subsequently). Decerebrate posturing may also be seen in these animals. Centrally fixed eyes and absence of the eye movements normally seen with movement of the head (vestibulo- ocular reflexes /physiologic nystagmus) in animals that are stuporous or show severe derangement in consciousness or mentation is indicative of severe midbrain injury and is associated with a very guarded to poor prognosis especially if persistent after more than 24 hours.

Animals with severe vestibular dysfunction (falling, rolling ,marked head tilt) have a fair prognosis despite the apparent severity of their neurologic signs and recovery is dependent on the extent of concurrent brainstem injuries (especially midbrain injury) and degree of paresis or paralysis.

In less severely injured animals, improvement may be seen with time but recovery may take weeks or months. Unfortunately, an accurate prognosis is often not possible initially. Trends are important – if there is neurologic improvement in the first 48 hours then there is reason to continue nursing. It may take more than 2-3 months before improvement becomes less likely. Prognosis with respect to wildlife is complicated by the requirement that the animals will almost certainly have to be able to return to their normal environment and be able to do this with the mobility and cerebral function to enable them to survive.

Nursing care is the most important part of rehabilitation. Nutritional support which may include nasogastric tube feeding in animals with severe facial or oral trauma and those unable to swallow normally, and physiotherapy may be required for weeks or months. Seizures may occur days, months or even years after head injury (acquired epilepsy).

Spinal Cord Trauma

Ultimately the degree of spinal cord injury depends on the rate (impact injury), amount (severity of compression) and time (duration of compression). Compression may be the result of vertebral malalignment (fractures/luxation), IV disc extrusion or haematoma formation. Lack of obvious malalignment on radiographs does not rule out vertebral instability or significant spinal cord compression or contusion.

Neurologic examination is critical in determining whether neurologic injury is present and at what level spinal cord injury is likely to be. The absence of nociception (deep pain sensation) in all limbs caudal to the injury site is a poor prognostic sign. If nociception and voluntary motor movement is present prognosis for functional improvement is fair.

Remember however that peripheral nerve injury may also result in anaesthesia of a limb.

Any imaging is only useful if the causative lesion is within the field of view imaged.
Neuroanatomic localisation of neurologic deficits is therefore important. However precise localisation may be difficult and imaging of all areas that may cause the neurologic abnormalities should be performed. Neurologic abnormalities due to one abnormality may mask those of a more caudal abnormality. Multiple spinal fractures are not uncommon. Animals may have both peripheral nerve injury (eg sciatic nerve injury associated with a pelvic fracture or a brachial plexus avulsion injury) and a spinal cord injury associated with a vertebral fracture.

Survey radiographs of the whole vertebral column are indicated in animals presenting with cervical signs and radiographs of the vertebral column caudal to C5 are indicated in all animals with signs in the pelvic limbs only.

Treatment of traumatic spinal cord injuries

No medical treatment has been shown to improve outcome in cases of spinal cord trauma. The use of corticosteroids in the treatment of acute spinal cord trauma is controversial. High dose IV Methyl prednisolone sodium succinate has previously been reported to be of benefit if given within 8 hours of spinal cord trauma in people but the validity of the results of the human study on which the recommended treatment was based has been questioned. Treatment with MPSS is not current in humans. No change in outcome has been found in studies in animals treated with MPSS. Corticosteroid administration can be associated with significant complications including life threatening GI bleeding.

Polyethylene glycol (PEG) has also been suggested as a treatment for acute spinal cord injury. Experimental studies in dogs with acute paraplegia and associated with IV disc extrusion treated with PEG did not show any difference in outcome when compared to similar animals in other case series where PEG was not administered.

In animals with intact nociception present and voluntary movement, medical treatment is a consideration if imaging does not suggest gross vertebral instability; however, improvement may take weeks to months. **Cage confinement** 4-6 weeks is recommended treatment in cases of presumed stable fractures, and /or mild neurologic deficits.

As a general rule

The severity of neurologic deficits and the length of time deficits have been present are both critical regarding the likelihood of return of function.

Animals without nociception have a poor prognosis especially after 24 hours. Animals without pain perception with identified vertebral fractures or luxation have a very poor prognosis whatever the duration of clinical signs.

Peripheral nerve injuries - need to be distinguished from a mechanical cause of weakness. If there is absent pain sensation (nociception) in the sensory distribution of the affected nerve prognosis for recovery is poor. If a motor nerve is affected -if any movement is present recovery of function is possible.

For generalised neuromuscular weakness (eg botulism) recovery is possible if nutrition / fluid balance can be maintained and ventilation is not affected.

Glossary:

Osteopaenia: reduced protein and mineral content of bone, less severe than in osteoporosis

Neurologic: relating to the anatomy, functions and organic disorders of nerves and the nervous system

Hypoglycaemia: abnormally low circulating blood glucose

Botulism: disease caused by a toxin produced by the bacterium *Clostridium botulinum*

Mentation: mental activity

Decerebration: loss of cerebral brain function due to damage to the cerebrum

Paresis: muscular weakness caused by nerve damage or disease or spinal cord damage

Neuropathy: damage to or disease affecting nerves, which can impair sensation, movement, or gland or organ function

Denervation: loss of nerve supply

Vestibular disease: disturbance of balance

Crossed extensor reflex: a withdrawal reflex - the flexors in the withdrawing limb contract and the extensors relax, while in the other limb, the opposite occurs

Noxious stimulus: a stimulus actually or potentially damaging to tissue and liable to cause pain, but does not always do so

Atrophic: wasting or decrease in size of an organ or tissue due to disease, injury, or lack of use

Perineal reflex (anal reflex): reflexive contraction of the external anal sphincter due to touching the skin around the anus

Aversive: tending to avoid or causing avoidance of a noxious or punishing stimulus

Dysphagia: difficulty swallowing

Poor prognostic indicator: an ominous sign that usually is associated with poor patient outcome

Nystagmus: repetitive, uncontrolled movements of the eyes

Parenchymal contusion: bruising of the brain

Scleral haemorrhage: bleeding in the sclera, the white part of the eye ball

Subarachnoid haemorrhage: bleeding within the membranes surrounding the brain

Normovolaemia: normal blood volume

Hypervolaemia: increased (excessive) blood volume

Haematoma: a localised collection of blood outside the vessels, due to disease or trauma