Nursing the neurologic patient
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Considerations in the treatment of brain disease
Brain function can be altered by disease of neural or surrounding tissues, or by metabolic abnormalities or toxins without primary brain disease. Some clinical signs are very obvious, eg. seizures or vestibular signs, but others are more subtle and subjective - animals may have significant forebrain disease and have no demonstrable abnormalities on clinical exam.

The clinical abnormalities seen with brain dysfunction are
1. Alterations in mentation or levels of consciousness (varying from dull to coma)
2. Abnormal behaviour (head pressing, getting stuck in corners, pacing, restlessness), circling, abnormal vocalisation
3. Seizures
4. Postural reaction deficits (tripping, stumbling)
5. Gait abnormalities ataxia (incoordination) and/or paresis
6. Visual and less commonly other sensory abnormalities
7. Cranial nerve deficits
8. Respiratory pattern abnormalities including panting

Diffuse brain dysfunction is most characteristic of metabolic encephalopathy (for example hepatic encephalopathy, cerebral hypoxia, hypoglycemia), intoxications, and some encephalitides. Focal (asymmetric) cerebral dysfunction is more characteristic of focal disease - tumour, granuloma, focal inflammation, haemorrhage or ischaemia.

However, a focal lesion anywhere within the brain can cause an increase in intracranial pressure and signs of diffuse cortical dysfunction. Endocrine and/or autonomic disturbances - can be seen with hypothalamic involvement. These include polyuria and polydipsia, hyperphagia or anorexia, poor temperature regulation or altered sleep patterns.

Severe midbrain (which joins the cerebral hemispheres with the caudal brainstem) dysfunction results from disease of the midbrain itself or midbrain compression as a result of cerebral swelling or increased intracranial pressure and cerebral herniation under the tentorium cerebelli (which separates the 2 compartments of the skull). This may result from trauma, haemorrhage, meningoencephalitis, tumour, hypoxia and other metabolic causes of cerebral oedema. The clinical signs of decerebration are unconsciousness (coma) with no evidence of pain perception, fixed dilated pupils, poor to absent eye movements, abnormal
respiratory pattern and decerebrate posturing - hypertonus of all muscles with opisthotonus, and rigid extension of all limbs. Signs of decerebration indicate a very poor prognosis. Prior to becoming decerebrate animals may show episodic posturing and pupillary dilation. This is an indication that tentorial herniation is imminent and treatment needs to be instituted to reduce intracranial pressure.

The management of animals with brain disease depends on the underlying cause but there are general principals which apply- maintain cerebral perfusion, ventilation, oxygenation, normoglycemia, normotension and frequent observation for changes in neurologic status. Changes in intracranial pressure can occur suddenly in animals with head trauma, intracranial tumours, inflammatory brain diseases, vascular accidents and in animals with metabolic abnormalities especially hypercarbia. Hypoglycemia and hypoxia may also cause cerebral oedema and a sudden increase in intracranial pressure or loss of brain autoregulatory mechanisms. All of the above can be life threatening and cause a sudden deterioration in neurologic status/death and require treatment.

The most common cause of brain abnormality disease in practice is head trauma and nursing care plays the most important role in recovery. Improvement may take weeks rather than days and progress is often slow. Severe trauma to the skull, brain and face can result from motor vehicle accidents, falls, trampling injuries, accidental blows to the head and malicious attacks. Injury to the brain is life threatening and early, appropriate treatment critical. Primary brain injury can be followed within minutes or days by secondary brain injury as the result of disturbances in cerebral blood flow and metabolism. These secondary changes may occur in already damaged neuronal tissue or may cause damage to brain unaffected by initial injury resulting in deteriorating neurologic status and can be fatal. Although veterinarians have little control over the primary brain injury, treatment is aimed at supporting cerebral perfusion without exacerbating secondary brain injury. Previously recommended treatments including sustained hyperventilation, corticosteroids and hyperosmolar agents such as mannitol are controversial as all may increase the incidence of secondary brain damage. The prognosis for recovery ultimately depends on the extent of primary brain injury but recovery is possible in many cases if brain protective treatment is instituted.

**Initial Assessment**

Most primary brain injuries are the result of parenchymal contusion associated with impact and not associated with fracture of the bones of the cranial vault. 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. Spinal fracture (especially C1-C3) may also be associated with severe head trauma.

Initial assessment of a trauma patient is critical. Before any examination is made, maintenance of a patent airway and adequate oxygenation and ventilation should be addressed. Thorough physical examination is necessary to detect any thoracic injuries, abdominal injuries, fractures and haemorrhage. Neurologic assessment should be done carefully with minimal handling. Neurologic examination needs to be modified in
unconscious patients and in animals that may have spinal fractures. Animals should be immobilised (eg taped/belted to a board) if a spinal injury is suspected.

Remember an increased respiratory rate or irregular respiratory pattern may be neurogenic in origin and not due to thoracic injury. Bradycardia may be seen with severe or progressive brain injury. An animal’s level of consciousness, respiratory pattern, pupillary responses and eye movements are the most important neurologic parameters in determining prognosis. Treatment to prevent secondary brain injury should be started immediately and repeat neurologic assessment made frequently (at least hourly initially). Then an assessment of all injuries should be made and treatment prioritised. Spinal fractures should be stabilised and life threatening injuries such as pneumothorax and ongoing haemorrhage treated. Non life threatening injuries, such as limb fractures should be stabilised and further diagnostics and treatment deferred until the animal's neurologic status is stable. Urine output should be monitored. Clean lacerations and treat eye injuries.

**Treatment**

The treatment of brain injury is primarily medical. If anaesthesia and surgery are necessary immediately, techniques to limit hypotension, hypoxemia and increases in intracranial pressure (ICP) should be used.

Medical treatment of the brain injured patient is directed at supporting cerebral perfusion without exacerbating secondary brain injury.

1. **Prevention of hypercarbia and hypoxemia - Ventilation and Oxygen**
   Animals with even marginal hypoxemia due to airway or pulmonary injury should be given supplemental O2 by mask or nasal cannula. Animals whose hypoxemia is due to inadequate ventilation secondary to brain injury require intubation and controlled ventilation. Assisted ventilation is necessary in hypoventilating animals to prevent increase in ICP associated with increased CO2 levels but aggressive hyperventilation should be used only in animals with severe neurologic deficits or deteriorating neurologic status to decrease ICP.

   Recumbent patients should be positioned such that the head is elevated 15-30 degrees above the heart without any compression of the jugular veins.

2. **Maintenance of normotension - Fluid therapy**
   Maintenance of normovolemia is necessary to maintain cerebral perfusion. Hypovolemia can result in hypotensive episodes. Hypervolemia 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 on going haemorrhage should be made. Blood pressure measurement is the most reliable indicator of adequate or inadequate intra vascular volume (normotension 90-140mmHg). Non invasive techniques of BP measurement (Doppler) require practice and trends are as important as a single measurement in an individual patient. PCV and TPP measurement will also help in assessing fluid balance after the first 24-48 hours. Fluid rate should be calculated to replace fluid lost and to meet maintenance requirements. 0.9% NaCl is best choice for initial replacement of fluid as fluids containing free water have been associated with a poorer outcome experimentally. 0.9% NaCl or lactated Ringers solution can be used for...

**Sedatives, Anticonvulsants and Analgesics**

Barbiturates (phenobarbitone) are known to decrease cerebral metabolic rate. Barbiturates can be useful in animals with head injuries causing seizures and in animals with brain injuries that are extremely agitated. Benzodiazepines (not in animals with hepatic encephalopathy) and other sedatives may also be used however many drugs may result in cardiovascular and respiratory depression which may reduce cerebral perfusion and may also result in increased agitation or dysphoria rather than sedation. Analgesics should be given to animals with facial injuries other orthopaedic/soft tissue injuries. Drugs that cause hypoventilation and hypotension should be avoided. Opiates should be used with care and in low doses.

Drugs may also reduce the ability to swallow normally and aspiration is a potential risk. Acepromazine in low doses may be more effective than other sedatives and may be associated with less respiratory depression (maintenance of normotension is important).

**Prognosis**

Animals showing sustained decerebrate posturing have a hopeless prognosis. Animals that remain semi comatose or unconscious and are unable to adequately ventilate despite treatment after several hours also have a hopeless prognosis. In less severely injured animals recovery may take weeks or months. Prevention of secondary brain injury is of critical importance in the first 48-72 hours after brain injury.

Nursing care becomes the most important part of rehabilitation after the first few days. Nutritional support which may include nasogastric or gastrostomy (PEG) 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. Improvement in neurologic status may not occur for days after brain injury and forward progress may be slow, but improvement may still be seen 3 months or more post injury in many cases. Seizures may occur days, months or even years after head injury (acquired epilepsy).

Considerations in the treatment of animals with spinal cord disease: Animals with spinal cord diseases present with varying degrees of ataxia (incoordination), paresis (weakness) or plegia (no motor movements) in one or more limbs and spinal reflex abnormalities depending on the location of the lesion (cause) within the spinal cord.

Spinal cord abnormalities may be due to disease of the spinal cord itself, the meninges which cover the spinal cord or due to compression of the spinal cord by extradural lesions such as vertebral abnormalities (fracture, tumour), IV disc herniations and IV disc infection (discospondylitis).

The management of animals with suspected spinal cord disease depends on the severity of the neurologic deficits, the time of onset (sudden, less acute-days or chronic-slowly progressive over weeks) and possible causes (spinal trauma and acute IV disc protrusion are emergencies).
The neurologic deficits associated with spinal cord disease include:

- Paresis or paralysis of one or more limbs. Spinal reflex abnormalities depending on level of lesion.
- Abnormalities in perception of pain - lack of pain perception in one or more limbs is a poor prognostic sign.
- Spinal pain.
- Bladder abnormalities - urinary incontinence or inability to urinate.
- Severe injury to the spinal cord in the cervical spine C1-C5 may cause ventilatory failure.

The management of animals presented with suspected or known intervertebral disc protrusion or spinal trauma is critical as with head trauma in preventing further irreversible damage to neural tissues.

Affected animals should be moved as little as possible. Animals with suspected spinal fracture should be transported if possible in lateral recumbency, restrained on a flat board or stretcher. All examinations including neurologic exam should be made with minimal handling. The first priority is to treat life threatening injuries - and then prioritise treatment. Spinal injury is a priority. Spinal fracture and vertebral instability needs to be ruled in or out and appropriate treatment given. Stability may be provided by cage confinement, external splinting or surgical internal fixation.

Animals presented with acute IV disc protrusion do not generally have vertebral instability but are at risk of herniating further disc material resulting in further and possibly irreversible spinal cord injury. Affected animals should be confined to a small cage with absolute cage rest (for weeks if medical treatment only is planned) or until further diagnostics and or surgery are undertaken.

In animals (especially toy breeds) where a cervical lesion is suspected care should be taken not to flex the head or neck until atlanto axial (C1C2) instability is ruled out. In animals with IV disc disease further extrusion of disc material may occur during the manipulation required for positioning for radiographs and this should be considered prior to undertaking any diagnostic tests.

Peripheral nerve disease (such as tick paralysis) also causes weakness or paralysis and may also be associated with cranial nerve paralysis (aspiration, respiratory paralysis) and ventilatory failure.

Acute idiopathic polyneuropathy (coonhound paralysis) is not an uncommon cause of tetraparesis and recumbency for long periods (weeks to months). There is no specific treatment but prognosis is good for good recovery with nursing and physiotherapy.

Care of the down dog (or cat)

Severely paretic or paralysed patients whether due to brain, spinal cord or generalized peripheral nerve disease are a nursing challenge. Neurologic improvement is often slow and many neurologic conditions require intensive nursing for weeks to months. Many conditions may have an ultimately good prognosis but animals may be recumbent for extensive periods of time.

The most important considerations are
1. Prevention of decubital ulcers (bed sores)
Soft bedding-compressible foam – at least 10cm (as thick as possible in large dogs) is ideal, air bed or waterbed.

2. Frequent turning (2-4 hours)- note this depends on individual dogs. Animals with significant unilateral vestibular disturbance animals may become very distressed on one side. Propping animals in sternal recumbency without compromising thoracic excursions or causing jugular compression may improve ventilatory capacity.
3. Non retentive bedding to prevent pooling of urine and skin scalding. Keep animals clean and dry at all times.

4. Maintenance of comfortable ambient temperature. Paralysed animals and animals with disturbed pain perception are at risk of thermal burns on heating pads or hot water bottles. Animals with brain disease may have abnormalities in temperature regulation.

5. Bladder management. Recumbent animals may require bladder expression or urinary catheterization (intermittent or closed system indwelling) depending on their neurologic abnormality. Animals with caudal lumbar spinal lesions often have poor bladder tone and urine overflows when any pressure is applied to the bladder. Animals with higher spinal lesions and some with lower lumbar lesions have increased urethral sphincter tone and are unable to urinate voluntarily with urinary retention (large bladder) and dribbling of urine when sphincter tone is exceeded. Animals with increased urinary sphincter tone (most common) are very difficult to express and catheterization is necessary to prevent urinary retention and likelihood of cystitis and also to prevent over stretching of the bladder musculature.

Closed collection indwelling catheter systems are useful in patients that are difficult to express to maintain a low residual volume of urine. Foley catheters should be used, placed using aseptic technique and the collection bag always kept lower than the animal. Indwelling urinary catheters should be used for as short a time as possible.

6. Defecation is not a concern in most animals with spinal cord lesions as defecation will occur reflexly when rectum is full. However in animals with caudal lumbar or sacral injuries faecal retention and incontinence can occur. Increased fibre or laxatives (Metamucil/duphalac) may be useful but repeated enemas may be required.

7. Maintenance of hydration and nutrition is very important. Recumbent, weak or deranged (head injured) patients may find it difficult to drink and cannot get to waterbowls by themselves. Offering water frequently from a sternal position or by syringe is necessary in many cases. Animals that are unable to swallow normally should be given IV fluids to meet maintenance requirements with careful monitoring of electrolytes. Animals at risk of aspiration may require pharyngeal and oral suctioning.

In animals that are able to eat highly palatable, high nutritional value food should be offered. Small amounts offered frequently may be needed in animals that tire easily. In animals that are inactive for long periods lower calorie diet or less food may be necessary. Oesophagostomy tubes or gastrostomy tubes may be required in animals that are unable to swallow normally, have major facial injuries, have megaesophagus or prolonged unconsciousness / sedation and calorie intake is calculated based on nutritional requirements.
Nutritional support should be considered in all animals that are unable to eat after 3-4 days.

8. **Physiotherapy is important to maintain joint and muscle mobility**, help reduce complications associated with recumbency (eg vascular), and improve animals mental attitude. Physiotherapy won’t speed neurologic improvement. Passive limb flexion and extension and limb massage is useful in recumbent animals. In animals that are able to support weight, walking supported with towel sling or harness or by tail base is good physiotherapy. Carts, especially in small paraplegic dogs, are also useful as many animals are much happier when up and about. Swimming (with support) is also excellent physiotherapy for many dogs.

One of the most important factors in recovery from neurologic injury or disease is time.