Neurological Emergencies

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Introduction
Neurological emergencies are common and require a cool head, careful patient assessment and prompt action! While there are many instances when an owner perceives their patient to be in a crisis when in fact they are not, any rapidly changing neurological dysfunction should be considered an emergency and the patient evaluated immediately. This presentation will give an overview of alterations in mentation, seizures and paralysis using case examples. An important general point is that you should have emergency protocols available as they improve outcomes in any emergency situation.

Altered Mentation: Stupor and Coma
Terms used to describe mental status include terms that describe level of consciousness (e.g. stupor, coma) and terms that describe behavior (e.g. dementia, hysteria). Level of consciousness is controlled by the ascending reticular activating system (ARAS). This mass of neurons extends through the brainstem to project to the thalamus and from there to the cortex. The ability to interact appropriately with the surrounding environment depends on the ability to process and integrate sensory information and to combine this information with learned information. The forebrain, and in particular the cerebrum and limbic system, is vital for normal behavior. Stupor is defined as decreased consciousness, but responsive to strong stimuli: these patients tend to be in sternal or lateral recumbency and are difficult to rouse. Coma is defined as unresponsive to stimuli. Stupor and coma are considered to be emergencies.

Causes of changes in mental status
There are numerous different causes of changes in mental status. These can be grouped into diseases that cause diffuse dysfunction of the forebrain (these are usually extracranial disorders that affect neuronal function but also include neurodegenerative diseases), focal forebrain diseases and brainstem diseases. Transient changes in mental status can be cause by seizures, narcolepsy, cataplexy and syncope.

Extracranial diseases
Neurons are dependent on adequate aerobic energy supply and appropriate concentrations of ions. They are extremely sensitive to toxins, and to changes in perfusion. The forebrain tends to show signs of metabolic disorders and toxicities sooner than the rest of the central nervous system (CNS).

a) Metabolic. Important metabolic causes of changes in mental status include hypoglycemia, hepatic encephalopathy, hyper and hyponatraemia, and hypothyroidism. Renal disease, pancreatic disease, hypo- and hypercalcaemia and kernicterus (bilirubinaemia) can also cause changes in mental status.

b) Toxic There are numerous potential toxins that can alter mental status, but one important group to consider are drugs including those that may have been prescribed to the patient (e.g. anti-epileptic drugs such as potassium bromide, ivermectin) or that may belong to the owner (recreational or prescribed). As a general rule, if an animal looks ‘drugged’ it might well be under the influence. By drugged I mean diffuse signs in an otherwise completely healthy animal that can’t be specifically localized, with bizarre behaviours frequently exhibited.

c) Perfusion. The forebrain in particular is acutely sensitive to changes in perfusion, Perfusion is dependent on blood pressure, intracranial pressure and viscosity. Hypotension can cause syncope, but rarely causes inappropriate behaviour. However, changes in viscosity will produce diffuse changes in mental status. Causes of changes in viscosity include polycythaemia rubra
vera, leukaemia and hyperlipidaemia. Hypertension can also produce extreme anxiety and even more dramatic changes in mental status.

**Forebrain diseases (Table 1)**

Any disease of the forebrain can produce changes in mental status. These diseases can be focal or diffuse in nature, and this should be reflected in the neurological examination. It should be noted that because the brain is enclosed within the skull, diseases that increase the contents of the cranial cavity such as neoplasia, or encephalitis, can result in secondary compression of the brainstem and multifocal or diffuse signs can ensue.

**Brainstem diseases (Table 1)**

A very similar spectrum of diseases can affect the brainstem, but changes in consciousness will only occur when the disease is severe enough to affect the ARAS or change intracranial pressure enough to cause significant brainstem compression. Changes in consciousness in a patient that is exhibiting brainstem signs is always a cause of concern and a reason for an emergency diagnostic workup.

**Table 1: Intracranial diseases that can cause changes in mental status**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Forebrain (FB) or Brainstem (BS)</th>
<th>Diffuse or focal signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neoplasia – Meningiomas; gliomas; choroid plexus papillomas; round cell tumors; metastases</td>
<td>FB and BS</td>
<td>Focal</td>
</tr>
<tr>
<td>Encephalitis – Fungal; bacterial; protozoal; rickettsial; viral; parasitic; GME, necrotizing encephalitides</td>
<td>FB and BS</td>
<td>Multifocal or focal</td>
</tr>
<tr>
<td>Anomalous – hydrocephalus; lissencephaly</td>
<td>FB</td>
<td>Diffuse or focal</td>
</tr>
<tr>
<td>Degenerative – Canine cognitive disorder; multisystem atrophy; storage diseases</td>
<td>FB</td>
<td>Diffuse</td>
</tr>
<tr>
<td>Nutritional – thiamine deficiency</td>
<td>FB and BS</td>
<td>Multifocal</td>
</tr>
<tr>
<td>Trauma</td>
<td>FB and/or BS</td>
<td>Diffuse, focal or multifocal</td>
</tr>
<tr>
<td>Vascular – bleeding disorder; thromboembolic disease</td>
<td>FB and/or BS</td>
<td>Focal or multifocal</td>
</tr>
</tbody>
</table>

**Diagnostic approach**

The diagnostic approach should include taking a careful history. Detailed information on any medications the pet is receiving or that are in the home is important. The physical and neurological examination should be targeted to identify any systemic problems and to localize the neurological signs. A careful evaluation of the heart and a fundic examination should always be performed. Diagnostic testing should start by ruling out extracranial causes and should include a serum biochemistry panel, complete blood cell count, urinalysis, and blood pressure. Plasma levels of anti-epileptic drugs, and assessment of endocrine function may be indicated. Thoracic radiography should always be performed in older animals. Once extracranial causes have been ruled out, advanced imaging (MRI or CT scans) of the brain and CSF analysis should be performed.

**Assessment of stuporous and comatose animals**

Significant changes in level of consciousness are due to disease of the brainstem, and typically signal very severe disease and impending brain herniation. Transtentorial herniation causes compression of
the mid brain and will cause coma, loss of the pupillary light reflexes and meiosis followed by mydriasis, decerebrate rigidity, and hyperventilation. These signs can be reversed with prompt recognition and treatment. Herniation through the foramen magnum will cause central vestibular signs, coma, changes in respiratory rate and pattern, changes in heart rate, rhythm and blood pressure and can be very difficult to reverse. However, we do regularly recognize trans-foramen magnum herniation on MRI in patients that do survive. The following should be assessed and documented to generate a coma scale.

1. Level of consciousness: Response to painful and other stimuli:
This will establish whether truly comatose (no response to any stimuli), stuporous (responds to painful stimuli), delirious/demented (responds inappropriately to environmental stimuli), depressed (quiet but responsive) or normal. Localization: diffuse cerebral damage or mesencephalic/pontine ARAS damage.

2. Brain stem reflexes.
   a. Pupil size, symmetry and response to light: Fixed dilated pupils and extreme miosis are both concerning.
   b. Physiologic nystagmus: physiologic nystagmus is the coordinated movement of eyes in response to movement of the head. The sensory stimulus comes from the vestibular system and projects via the medial longitudinal fasciculus (MLF) to the nuclei of the cranial nerves of the extraocular muscles (3, 4 & 6). The MLF runs up the brainstem on its dorsal surface and as such is susceptible to compression. NOTE if unconscious, you will not be able to elicit physiologic nystagmus.
   c. Dazzle reflex: This is a brain stem reflex in which the eye is retracted and the animal blinks in response to a bright light shone directly into the eye.
   d. Palpebral reflex. This brainstem reflex is maintained in stupor and light coma.

3. Respiratory rate and pattern:
Abnormal respiratory patterns are a sign of severe brain stem compression/destruction and signal the need for immediate treatment for increased intracranial pressure and diagnostic work up to facilitate treatment of the specific cause.

   a) Unilateral injury below level of mesecephalon: ipsilateral hemiparesis.
   b) Unilateral injury above level of mesecephalon: contralateral hemiparesis.
   c) Unilateral injury of mesecephalon: variable hemiparesis.
   d) Posturing:
      (i) decerebrate rigidity: opisthotonus with thoracic and pelvic limbs in rigid extension. Animal is in a coma, with no plrs and either pinpoint or dilated pupils. Poor prognosis. This is the result of loss of descending input to the flexors from supratentorial structures (red nucleus and telencephalon).
      (ii) decerebellate rigidity: lesions of rostral lobe of cerebellum in particular. See opisthotonus and extensor rigidity of thoracic limbs. Animal should have plrs and is not usually comatose.
      (iii) intermittent increased extensor tone in thoracic and pelvic limbs and neck can occur when you move an unconscious patient (and also patients with cervical lesions). This is the result of unopposed stimulation of postural muscles (extensor) in response to impulses from the vestibular system. Remember that movement stimulates the vestibular apparatus.

5. Heart rate and rhythm: persistent unexplainable tachycardia or bradycardia may be the result of brain injury. Cushings reflex = elevated intracranial pressure leads to systemic hypertension (in an effort to maintain cerebral perfusion) and a reflex bradycardia.

At the end of the evaluation you should have an idea how severe the problem is and whether the coma is due to brain stem or cerebral disease. Animals should then be monitored closely with documentation of vitals every 30 minutes (a continuous ECG should be placed) until they start to stabilize and improve.

*Management Rules for animals with suspected increased ICP:*
1. Ensure adequate ventilation: ideal pCO2 in a patient with brain injury is 25 - 35 mmHg.
2. Do not occlude jugulars.
3. Place head at angle of 30° from the horizontal: this is a compromise between trying to ensure good venous return while maintaining adequate cranial blood flow.
4. Check blood pressure: aim for normotension. Hypotension is very bad as it worsens CPP, and hypertension may be an indicator of severe CNS damage (especially if accompanied by bradycardia).
5. Pull CBC, chemistry panel (plus CK), +/- bile acids/ ammonia, +/- phenobarbital, bromide levels if appropriate and aim to complete a full diagnostic work up as soon as possible.
6. Institute drug therapy of ICP if indicated:
   Mannitol or hypertonic saline?
   The brain trauma guidelines still recommend mannitol over hypertonic saline but judging from the literature, this is about to change. Hypertonic saline is typically given to resuscitate a trauma case or whenever vascular volume needs to be expanded. Mannitol is used once vascular volume has been restored.
   When to give mannitol or hypertonic saline?
   1. In the face of deteriorating neurologic status (e.g. a pupil goes from responsive to dilated).
   2. If present with very severe neurological signs, e.g. bilateral dilated pupils and abnormal respiratory pattern.
   How much?
   Mannitol: 0.5 – 2.2g/kg at the first dose over 5 - 15 minutes. Too rapid an infusion causes an initial increase in ICP. Many people recommend following this with 0.7mg/kg furosemide iv 15 minutes later to help prolong the osmotic gradient created by mannitol.
   Hypertonic saline: 4ml/kg (7.5%) and 5ml/kg (3%).
   Following administration of mannitol or hypertonic saline, there is controversy over what to do with the fluids. Personally I usually give low maintenance fluids. Others may discontinue fluids for 2 hours and then restart at about 80% maintenance rate and yet others may increase rate! Collids can be used if vascular volume is still depleted.
   How often?
   This is a difficult question to answer as prolonged or repeated treatment results in hyperosmolality, electrolyte imbalances and even renal failure. The problem is that we are not measuring ICP directly with monitors at the moment and so can only judge the effect of the drug we give very crudely (rather like giving anti arrhythmic drugs without an ECG). Try to reserve mannitol for deteriorating neurologic status cases.
   Should you give steroids?
   Steroids are not in favor for the treatment of traumatic or ischemic brain injury. However, there are a couple of situations where anti-inflammatory doses may be useful: these include cases in which brain neoplasia is the top differential and cases in which inflammatory brain disease is the top differential. In both cases it is preferable to complete a full workup first, but if the case is critical, it may need immediate steroids to survive.

**Acute Spinal Cord Injury**

Acute spinal cord injuries are unfortunately one of the most common groups of neurological disorders in veterinary patients. Diseases as diverse as acute intervertebral disc herniations, fibrocartilaginous embolism (FCE) and traumatic injuries all cause acute injury to the spinal cord and activate similar pathophysiological mechanisms.

**Pathophysiology and therapeutic principles**

Following a traumatic accident, the spinal cord or cauda equina (if the lesion is caudal to the fifth lumbar vertebra) is damaged by contusion, laceration, compression, and vertebral instability (that can
cause repeated spinal cord contusion, compression and even laceration). Vascular injuries such as FCE trigger a similar cascade of events as a contusive injury (see below).

**Contusion**

Contusive injuries cause both primary and secondary damage to the spinal cord. The primary damage is mechanical and is a direct result of the trauma. This primary injury damages blood vessels (decreasing the perfusion of the area), causes release of neurotransmitters and increases membrane permeability. As a consequence, a cascade of destructive biochemical and metabolic events is initiated that causes accumulation of intracellular calcium, free radical production and progressive destruction of the microvascular bed. The result is an expanding area of secondary cell death that increases the volume of the injury. Although apoptosis can continue for many weeks after an injury, the majority of the damage occurs within the first 24 – 48 hours and clinical deterioration due to this secondary tissue damage does not usually continue beyond 24 hours after the injury. Bearing the secondary damage in mind, treatment of contusive and vascular injuries is aimed at limiting the extent of secondary tissue damage by

a) **Maintenance of normal blood pressure**: treat hypotension with fluids.

b) **Maintenance of tissue oxygenation**: supplement oxygen if hypoxemic

*Corticosteroid and methylprednisolone sodium succinate (MPSS) use*

Gluocorticoids are contra-indicated in traumatic CNS injuries but 48 hour CRIs of high doses of MPSS have been advocated for its ability to scavenge free radicals. This recommendation is based on the results of the human clinical trials (the NASCIS trials). In these human trials, MPSS was only useful if treatment is started within 8 hours of injury and it is important to understand its beneficial effects are controversial in humans and unproven in dogs. Finally, its side effects on the gastrointestinal tract can be severe. A multicenter clinical trial of MPSS and the fusogen, polyethylene glycol is currently underway in dogs with disc herniations and should provide more insight into the best medical therapy.

At the time of writing, there is no objective evidence that MPSS or PEG are beneficial adjunctive medical therapies of acute spinal cord injury in dogs.

**Laceration**

Lacerations of the spinal cord are devastating because they cause disruption of the neuronal tissue. The poor regenerative response of the central nervous system means that the damage caused by such injuries cannot be reversed and recovery can only occur if there is spared tissue. Experimental therapies focusing on transplantation of a variety of different glial and neuronal cells are being developed and transplants of both olfactory nerve ensheathing cells and mesenchymal stem cells have been performed in dogs.

**Compression**

Compression of the spinal cord decreases blood flow and deforms axons and myelin. The resulting local ischemia causes cell death and the mechanical deformation of the white matter causes a potentially reversible failure of conduction. This type of injury is treated by surgical decompression, but the decision on when to decompress the spinal cord and whether it is necessary at all is based on:

- an evaluation of the animal’s general health (many trauma patients cannot undergo anesthesia due to their other injuries).
- The severity of neurological deficits: if the animal has good voluntary motor function, decompression is not necessary

**Instability**

The most common cause for neurological deterioration following a traumatic injury is movement of the unstable spine. Two and three compartment models of the vertebral column are proposed in order to predict whether a spinal fracture is unstable: the two compartment model describes dorsal (articular facets, dorsal and lateral laminae, dorsal spinous processes and muscles) and ventral (vertebral body, dorsal and ventral longitudinal ligaments, transverse processes and disc) compartments. Damage to
both compartments indicates an unstable fracture. In the three compartment model the compartments are: i) ventral longitudinal lig, most of vertebral body and lateral and ventral disc. (ii) dorsal annulus and vertebral body, dorsal longitudinal lig, (iii) pedicles, articular facets, laminae dorsal spinous process and associated muscles and ligaments (e.g. ligamentum flavum). Damage to any two of the three compartments indicates instability.

Stabilization can be achieved by strict cage rest, external splinting or internal (surgical) fixation. Cage rest has the advantage of being inexpensive, avoiding surgical risks, and it does not exacerbate other injuries (such as skin abrasions). The big disadvantage is that it is not an effective method of stabilizing grossly unstable fractures because it simply relies on the splinting effect of the muscles of the vertebral column. It is suitable for use in animals with relatively stable fractures, and animals with severe skin abrasions in which a splint cannot be placed. External splinting is an inexpensive method of stabilizing the spine that avoids the risks of anesthesia and surgery but correctly placed splints can be difficult to manage (the splints can cause pressure sores). While external splinting is commonly felt to provide good stabilization of bending forces (flexion/extension) and rotation, it provides little protection against axial loading and of course does not facilitate decompression of the spinal cord. Although many people do not believe that external splinting should be used in any fracture in which both dorsal and ventral compartments are damaged, published studies suggest that outcomes from internal and external splinting in very similar cases do not differ significantly. Internal fixation (i.e. surgical stabilization) is the most effective method of stabilizing and decompressing the spinal cord and post-operative management does not involve the complications of a splint. However, it is costly, success depends on the skill of the surgeon and anesthesia may be contraindicated in a trauma patient.

**Summary of approach to spinal cord injury patient.**
1. First do no harm by moving the patient with extreme care.
2. Assess the patient as for an emergency (airways, breathing and circulation).
3. Treat hypotension and hypoxemia
4. Perform a careful neurological examination and determine
   a) neurolocalization
   b) severity of injury (presence of motor function and presence of deep pain perception).
5. Take survey lateral spinal radiographs: if no evidence of a facture or luxation, obtain VD views as well.
6. Determine whether there are unstable fractures from the radiographs.
7. If an unstable fracture is present, either place an external splint, or refer for further imaging and surgical stabilization.
8. Treat all secondary problems (e.g. cardiac arrhythmias, ruptured bladder)
9. Start physical rehabilitation as soon as possible.

**Prognosis**

Prognosis is related to the severity of the neurological deficits:

<table>
<thead>
<tr>
<th>Location and severity of signs</th>
<th>Prognosis with appropriate treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>C1-T2:</strong></td>
<td></td>
</tr>
<tr>
<td>• Tetraparetic</td>
<td>• Excellent</td>
</tr>
<tr>
<td>• Tetraplegic</td>
<td>• Good if respiratory function adequate</td>
</tr>
<tr>
<td>• Tetraplegic with loss of deep pain perception</td>
<td>• Extremely grave: most die from respiratory compromise and arrhythmias</td>
</tr>
<tr>
<td><strong>T3-L3:</strong></td>
<td></td>
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</tbody>
</table>
• Paraparetic or plegic
• Paraplegic with loss of deep pain perception

| LS | Perineal and lateral digit pain perception present | Excellent |
| LS | Perineal, lateral digit and tail pain perception absent | Excellent |
| LS | Perineal and lateral digit pain perception present | Permanent incontinence likely |

Additional points about prognosis are given below:

a) **Cervical fractures**: Surgical treatment is associated with high (36%) perioperative mortality, but all those that survive this crucial period can make a good recovery. Predictors of a poor functional recovery are i) delayed interval between injury and referral and ii) severe neurological deficits (non-ambulatory dogs)

b) **Sacrocaudal fractures**

These injuries do not cause paraplegia but do cause incontinence and paralysis of the tail. Guidelines for prognosis are: If anal tone and perineal sensation is present there is a good prognosis for normal continence; If anal tone and perineal sensation are decreased there is a 75% chance of recovery of urination; If anal tone and perineal sensation are absent there is a 50% chance of recovery of urination; If the animal is not urinating normally at 1 month after injury its prognosis is very poor.

c) **FCE**

The prognosis of FCE depends on the amount of tissue damage caused and follows the same guidelines as for trauma if the lesion is not complete. However, unlike a fracture or luxation, an FCE does not cause laceration of the spinal cord and so the prognosis of sensation negative animals is not as guarded as that of animals that have suffered a traumatic injury. It is notable that improvement after an FCE can be extremely rapid and so an animal should not be condemned in the first 48 hours after a vascular injury. Ideally, the patient should be monitored for 2 weeks after injury and if there is any sign of improvement the patient is likely to recover with time.

**Status Epilepticus and Cluster Seizures**

Status epilepticus (SE) is defined as continuous seizure activity for more than 5 minutes, or a failure to recover normal mentation between 2 seizures. Cluster seizures are more than one seizure in 24 hours and they are an extremely common problem in refractory epileptics. In both SE and cluster seizures, it is important to ensure an adequate diagnostic work up has been performed before making a diagnosis of idiopathic epilepsy.

**Treatment of SE**

1. Establish intravenous access.
2. **Give a benzodiazepine IV**. The options are:
   - 0.5mg/kg diazepam (historical gold standard)
   - 0.25-0.5mg/kg midazolam (non irritant)
   - 0.2mg/kg lorazepam (longer half life)

If can’t give IV, give double dose per rectum or same dose intranasally (IN). Intranasal administration should be performed with care as injecting a large volume IN could result in aspiration. Drip the drug in slowly, with the nose elevated (approximately 30°), dividing the dose equally between nostrils.
3. Pull blood and check glucose and blood gas (for Ca in particular) immediately. Submit stat CBC, chem panel, UA +/- bile acids/ammonia. Check phenobarbital and/or bromide levels if appropriate.
4. Check body temperature. If hyperthermic, (>105°F) start cooling. From a prognostic point of view, DIC occurs with temps of >107F.

5. If glucose is low (60mg/dl or less) (note you would expect it to be high due to the sympathetic discharge that accompanies seizures), a rough guideline is that **200mg/kg glucose IV should increase blood glucose by 100mg/dl.** 50% dextrose is 500mg/ml, ie 0.4ml/kg: dilute to 10% before giving. Be sure to check glucose on serum if borderline low as it can be an artifact.

6. Be suspicious of hypocalcemia in certain instances: a bitch that has whelped; a dog that has had parathyroids removed surgically. Dose: **05-1.5ml/kg of 10% calcium gluconate IV slowly** while monitoring ECG.

7. If the benzodiazepine doesn’t work, repeat twice. You can start an IV infusion, (but be sure to try appropriate phenobarbital therapy first or in conjunction). There are many different ways to set up the CRI. Here are 2 options:
   - Use the dose that stops the seizures when given as a bolus, and give this dose per hour as an infusion.
   - Remember to coat the plastic (run some drug through line & discard) and to protect from light if using diazepam. IV benzodiazepines do not give long-term seizure control, so you will need phenobarbital or levetiracetam (keppra) if this is an epileptic case (e.g. seizures are not the result of a toxicity) and not already on an antiepileptic drug. The benefits of diazepam are reduced cost when compared to midazolam. The benefit of midazolam is that it can be given into a peripheral vein (diazepam CRIs need to go via a central line).

8. If still no luck, or if only works for a few minutes, move onto a longer acting AED:
   - **Phenobarbital: loading dose is 12 – 20mg/kg IV. You will not usually give this as one bolus although some neurologists do.** Start with 4 - 6 mg/kg boluses if the dog is not already on phenobarbital. If animal is **already on phenobarbital, give 2 – 4 mg/kg phenobarbital IV boluses.** This will increase blood levels by about **2 – 4 ug/ml.** NB IV phenobarbital takes **15 – 30 minutes** to take effect. The speed of loading Phenobarbital will depend on the case and the clinician – consult with the neurology resident on call. **In cats, give 2- 4mg/kg IV boluses.** Note: some clinicians do go ahead and give 12 to 20mg/kg phenobarbital as one bolus in dogs that are not already on phenobarbital: if you plan to do this be ready to ventilate the animal.
   - An IV form of **keppra** is now available although expensive. This can be used instead of Phenobarbital, especially if there are concerns about the liver or excessive sedation. An appropriate maintenance dose is 20mg/kg q8h, and a loading dose of 60mg/kg can be given.

9. If seizures stop continue maintenance dosing of phenobarbital: 2-3 mg/kg PO or IV BID (if not already on phenobarbital), or consider increasing current dose if already on phenobarbital. Maintenance of keppra is 20mg/kg q8h.

10. If seizures don’t stop
   - a) Make sure you haven’t missed a metabolic problem (glucose, calcium, liver failure).
   - b) Check for evidence of hemorrhage.
   - c) Check for evidence of inflammatory disease (CBC)
   - d) Move on to general anesthesia: there are several options:
   - (i) **Pentobarbital: 1 – 3mg/kg iv:** give slowly and watch for seizures to stop. Note that pentobarbital is an intermediate-acting barbiturate, and takes several minutes to reach full effect – proceed cautiously! Follow up with a blood gas to check the animal is not hypoventilating. Intubate and ventilate if necessary. You often end up needing a lot more than 3mg/kg, but this is a nice guideline for how much to bolus: if no response, do not be afraid of giving more (you often need 9-12mg/kg), but be ready to ventilate.
   - (ii) **Propofol: 3-6mg/kg bolus followed by infusion of 0.1 – 0.8mg/kg/minute.** Follow up with a blood gas to check the animal is not hypoventilating. Intubate and ventilate if necessary. In some animals
propofol is pro-convulsant, but in most it is anticonvulsant! The advantage over pentobarbital is that it should have less cardiovascular and respiratory depressive effects, and recovery is much faster than from a pentobarbital coma. BUT it is expensive and in the long term can cause hypotension and respiratory depression.

e) Consider the need for an EEG. If you are unsure whether seizures have truly stopped, or if the animal is doing anything unusual (eg high heart rate), we run an EEG.

Once you have got to the anesthesia phase you must monitor:

1) Blood gases to ensure not hypventilating. Also, many of these animals can develop aspiration pneumonia and/or non-cardiogenic pulmonary edema.

2) Body temperature.

3) Blood pressure, heart rate and rhythm.

4) Bladder: keep it empty. A full bladder can make the heart rate increase.

5) Make sure jugular veins are not occluded: preferably put head at 30o from the horizontal.

6) Monitor pupil size and responsiveness: any sign of deteriorating neurologic status (see coma guidelines), give mannitol, 0.5 – 1g/kg slow iv.

We like the animal to be seizure free for at least 12 hours before attempting to discontinue any CRIs they are receiving as the animal comes out of anesthesia, you must make sure that it has adequate blood levels of Phenobarbital or keppra to prevent seizure recurrence. Animals recovering from pentobarbital induced coma will often paddle, and it can be difficult to determine whether this is a seizure or just recovery. To differentiate, check the heart rate and try moving or holding the animal. If it isn't a seizure, the heart rate has not usually increased, and the paddling will slow as you move the animal. If unsure, try a dose of a benzodiazepine and see what happens and/or obtain an EEG.

Treatment of Cluster seizures

Although dogs that have clusters of seizures may only experience them every 3 - 12 months, this can be a devastating problem because it typically ends with the dog being hospitalized. Over time, the emotional and financial stress on the owners can result in them electing for euthanasia. When faced with a dog that clusters, the basic steps in achieving seizure control should be undertaken. If clusters are still a problem, owners can be advised to give their dog one full additional dose of phenobarbital (or zonisamide or keppra, if the dog is on these drugs) orally after a seizure once the dog can swallow to try to prevent a cluster from developing. This can be repeated up to once per hour, but if it has been repeated three times to no effect, the owner should seek veterinary help. Another strategy is for the owners to administer rectal (1mg/kg) or nasal (0.5mg/kg) diazepam or other benzodiazepine (see SE protocol) after a seizure. There is a report that this is an effective approach to such cases9, however, owners must be instructed carefully in how to draw up and administer the diazepam. Finally, there are anecdotal reports of using an add on drug such as keppra around the time that a dog clusters as a pulse therapy. Many of these dogs seizure at very regular intervals and the owner is able to predict when the next group of seizures will occur and can administer additional gabapentin for that period.

References


