Neurological Conditions and Seizure Management

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I. INTRODUCTION

A number of conditions in the neonate may lead to neurological dysfunction which prompts evaluation. An assessment of the overall systemic health and metabolic state of the neonate needs to be made, with examination making allowances for the relative lack of maturity of many body systems. The goals of the neonatal neurological examination are similar to those of the adult horse: neuroanatomical localization of any lesions present to suggest a diagnosis and thereby help formulate a rational inclusive treatment plan. Seizure activity adds urgency to establishing a diagnosis, with the goal of therapy to first control seizure activity to prevent secondary injury while a diagnosis is made.

II. NEUROLOGICAL EXAMINATION

The most common neurological problems of foals include depression, seizure activity, abnormal behavior, loss of affinity for the mare and loss of the suckle reflex. All these signs are referable to dysfunction of the brain. Expression of neurological lesions will be dependent on the stage of development which the inciting damage occurred.

Posture

Carriage and movement of the foal’s head is noticeably different from the adult. In comparison, the occipitotantal joint is consistently more flexed, and movements of the head resemble jerking motions consistent with cerebellar dysfunction in the adult.

Mentation and Behavior

By nature foals tend to be inquisitive about their surroundings. The degree of affinity for the mare is an important indicator of abnormal behavior, with loss of affinity for the mare or lack of vigorous suckling often early signs of developing hypoxic ischemic encephalopathy. Foals are much more sensitive to touch than adults, with exaggerated responses to all tactile stimuli. Foals tend to struggle vigorously when first restrained, however, become limp when compressed along their long axis, and often display submissive behavior.

Suckle Reflex

Loss of the suckle reflex may be the first indication of neurological dysfunction. Although an instinctive process, suckling demands adequate cerebral function and an intact motor supply to the tongue (intact hypoglossal nerve).

Cranial Nerve Examination

Cranial nerve (CN) responses are similar to the adult. Evaluation of CN responses follows the familiar pattern of the adult, with allowance for the diminished strength of any reflex arcs. Pupillary light reflexes are sluggish at birth. In one study, all foals had a positive pupillary light reflex on the first day post-partum.2 The pupillary light reflex arc is located in the brainstem, and does not require a normal cerebral visual cortex.3 Therefore, an appropriate pupillary light reflex does not indicate the absence of a visual deficit. The menace response may be learned,4 or is delayed due to an initial lack of maturity of the cerebellum.3 The response is incompletely developed up to 2 weeks of age. The globe has a ventromedial deviation in the normal neonate, assuming an adult positioning within one month.

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Gait Evaluation

Compared to adults, limb movements of the neonatal foal can appear dysmetric with a delayed onset of an appropriate adult-like gait following extended confinement; however, this rapidly improves with exercise.3

Spinal Reflexes

Reflexes are best assessed with the foal in lateral recumbency and are hyperactive when compared to adult responses. The crossed extensor reflex is brisk. Although extensor tone predominates, continued gentle pressure will allow complete flexion of the limbs. Spinal ataxia is less common than cerebral disease, and when diagnosed it is most likely the result of congenital malformation, vascular accident, or trauma during the birth process.3

III. SELECTED DISORDERS

Acquired

Hypoxic Ischemic Encephalopathy (HIE), Perinatal Asphyxia Syndrome (PAS), Neonatal Maladjustment Syndrome (NMS)

The result of cerebral hypoxic or ischemic insult to the neonate, HIE/PAS/NMS is arguably the most common seizure precipitating syndrome faced by the equine clinician. The diagnosis of HIE is dependent upon a compatible history and clinical examination findings while ruling out other conditions.5 Excitatory amino acids, calcium ions, free radicals, nitric oxide, pro-inflammatory cytokines and products of lipid peroxidation are all thought to contribute to the syndrome, with the neonatal brain having an increased susceptibility compared to adults.6-8

Infectious Agents

Pathogens, whether viral or bacterial, have the ability to cause substantial CNS inflammation by their own actions or by the initiation of an excitotoxic neurotransmitter cascade. The neonatal foal is susceptible to meningitis resulting from systemic bacteremia due to an increased permeability of the blood-brain barrier.9

Bacterial Meningitis

Organisms recovered from meningitis cases are the same as those present in sepsis and this condition may be the result of bacteremia.10 Meningitis may present indistinguishably from HIE. Inflammation of the meninges is consistent with reluctance to move the neck, opisthotonus, proprioceptive deficits and stupor. Progressive increase in inflammation decreases cerebral blood flow leading to ischemic damage precipitating further neurological dysfunction.

Metabolic Derangement

Glucose levels are frequently abnormal in the compromised neonate, with hypoglycemia potentially having effects on neurological function. Hypoxia, neonatal seizure and pathologically jaundice exacerbate hypoglycemic brain injuries when hypoglycemia is severe and prolonged.11

Derangements of sodium and calcium are the most relevant with respect to neonatal neurological disorders. Acute hyponatremia results in cerebral edema with seizures, blindness and depression resulting from compression of the brain against the bony calvarium. Chronic hyponatremia is less likely to produce neurologic symptoms as osmotic equilibrium has time to occur. The rapid correction of hyponatremia causes neurological signs as a result of cell shrinkage by the osmotic draw of water from cells.12 Acute hypernatremia may cause shrinkage of brain parenchyma, and is countered by neuronal production of idogenic osmoles (osmotically active metabolic byproducts) in an attempt to maintain osmotic balance. During correction of hypernatremia, edema may occur if the rate of water delivery is not carefully monitored. Seizure activity (focal or generalized) is the most common central neurologic manifestation of hypocalemia. Tetany, agitation, confusion and depression may also be seen.

Trauma

Trauma can lead to abnormal mentation, gait and posture. Mild cases present with subtle neurologic deficits, whereas in others spinal reflexes may be diminished. Severe cases can present in recumbency. There may be bony disruption or direct injury to the neural tissue causing hemorrhage and swelling.

Hyperbilirubinemia

Bilirubin may cause degeneration and necrosis of cerebral neurons. Prematurity/immaturing, hemolysis, asphyxia, acidosis, sepsis and increased bilirubin itself may promote bilirubin absorption by nervous tissue.13 In severe cases kernicterus (bilirubin encephalopathy) occurs.14 Elevated levels of unconjugated bilirubin have been demonstrated in neonatal liver disease, neonatal isoerythrolysis, glucose-6-phosphate dehydrogenase deficiency, and other causes of intravascular hemolysis.15

Botulism

Botulism is a progressive flaccid paralysis resulting from Clostridium botulinum toxin production. Acetylcholine release at neuromuscular junctions is blocked. Aspiration pneumonia, respiratory paralysis, and sepsis resulting from prolonged recumbency and decubital ulceration occur. Diagnosis is by elimination of other causes of neurologic disease. It may be possible to identify the toxin in gastrointestinal contents in some cases but this is unreliable. Gastric ulceration in the neonate is most commonly incriminated as this allows germination of Cl. botulinum spores, liberation and local absorption of toxin. Monovalent and polyvalent antitoxin preparations are available and administration should not be delayed when foals first display suggestive signs.
Developmental or Congenital Abnormalities

Hydrocephalus

Hydrocephalus may be clinically silent or precipitate seizure activity. In one retrospective study, hydrocephalus occurred in 3% of foals that died or were euthanased. Detection by magnetic resonance imaging (MRI) has been reported. Clinical signs were reported to be similar to HIE, with a depressed suckle reflex and failure to thrive. Marked dilation of the cerebral ventricles was present, with other intracranial structures reported as normal.

Occipitoatlantoaxial Malformation (OAAM)

This is predominantly a condition of newborn to juvenile Arabian foals although isolated cases occur in other breeds. Presenting signs include a progressive ataxia and tetraparesis, with stillbirth or sudden death also reported. A palpably abnormal atlas and axis, with reduced flexion and audible clicking upon manipulation of the head is present. Radiological evaluation reveals occipitoatlantal fusion, hypoplasia of the dens and inappropriate localized ossification.

Narcolepsy/Cataplexy

Narcolepsy is the sudden uncontrollable onset of deep sleep. Cataplexy is the sudden loss of muscle tone and strength often leading to collapse, and whereas consciousness is not affected with cataplexy itself this often occurs concurrently with narcolepsy.

Both narcolepsy and cataplexy may transiently occur in Thoroughbred and miniature foals. The application of compressive manual restraint over a wide area of a foal’s body may induce a cataplectic state, thought to be an adaptation to the restrictive in utero environment of late pregnancy. Diagnosis is made by differentiating from syncope, the result of transient cerebral hypoxia due to a cardiac abnormality, and seizure activity.

Congenital Epilepsy

This condition is reported in some breeds. A review of Egyptian Arabians showed seizure activity itself to be self-limiting with age. Complications in affected foals included head trauma and pneumonia.

IV. SEIZURE DISORDERS

Seizure activity is an indication of forebrain neurological dysfunction as the result of abnormal electrical activity. Involuntary, spontaneous muscle contractions are accompanied by varying degrees of loss of consciousness. Seizures may be divided into focal or partial (initially involving a small part of the brain with limited manifestations), or generalized (widespread involvement of the brain with recumbency and loss of consciousness possible). Partial seizures do not necessarily progress into general seizures. When of a cerebral origin seizures often remain predictable and non-progressive in severity and duration. However, when due to an underlying systemic problem, seizure activity often worsens with any deterioration in the precipitating disease process.

Three phases of a seizure episode are recognized. The pre-ictal period may involve depression, abnormal activity and staring. The ictal period (overt seizure) encompasses the recognized manifestations of seizure activity: paroxysmal muscle contraction and altered consciousness. The post-ictal phase is one of depression and possible transient loss of neurological function e.g. central blindness.

Seizure Recognition

Recognition of seizure activity in the neonatal foal is problematic; however, it is essential to the successful management of neurologically compromised neonates. Subtle seizure signs include abnormal eye movement, tremors, excessive stretching when recumbent, excessive extensor tone, hyperaesthesia to touch and manipulation and apneustic breathing. Overt seizure signs include rapid nystagmus, paddling, hyperextension and excessive mouth movements (‘chewing gum fits’). Unobserved episodes may result in signs of unexplained physical trauma.

Pathogenesis of Seizure Activity

Disruption of neural tissue, whether as a result of infection, ischemia or trauma, involves many processes including vascular and cellular disruption, free radical production, breakdown of cell membrane lipids, and the release of inflammatory mediators. Apoptosis of neurons then occurs as a result of aberrant Ca intakes and excitotoxic neurotransmitter release. Loss of astrocyte functions during cerebral ischemia also affects neuronal cell viability. Astrocytes provide structural, nutritional and metabolic support to neurons.

Excitatory amino acids (including glutamate, aspartate) are the final common pathway in many neurologic disorders. Activation of glutamate receptors allows an excessive influx of calcium into neurons through ionic channels, resulting in neuronal swelling, membrane damage and cell death. Glutamate is then released from intracellular storage vesicles initiating a self-sustaining cycle of cell damage and aberrant electrical activity.

Conditions Associated with Seizure or Seizure-Like Activity

Seizure disorders of the foal can result from compromise of the dam or placenta during the fetal period, or disease of the neonatal foal itself including hypoxic insult and failure of passive transfer predisposing to sepsis. Any underlying medical conditions (infectious, inflammatory, developmental, congenital) must be managed and any lesions amenable to surgical correction addressed concurrently with seizure control.
V. DIAGNOSTICS IN NEUROLOGICAL CONDITIONS

Complete Blood Count (CBC) and Serum Chemistry

A CBC aids in the diagnosis of systemic infection that may have spread to the CNS. Serum chemistry assays major organ system function, chiefly liver (removal of blood borne toxins) and kidney (assessment of electrolyte homeostasis). Immunoglobulin G (IgG) levels following colostrum are a direct measure of immune sufficiency in the previously hypogammaglobulinemic neonatal foal. Blood gas evaluation assesses pulmonary gas exchange and acid-base status of the foal.

Cerebrospinal Fluid (CSF) Analysis

Cytology and microbiological culture of the CSF aids diagnosis, antimicrobial selection and prognosis in suspected cases of meningitis. Total protein concentration, nucleated cell count and RBC count should be routinely performed. Where increased intracranial pressure is suspected aspiration of CSF should be avoided as herniation of the brain through the foramen magnum may occur.

Radiography, Computed Tomography (CT) and Magnetic Resonance Imaging (MRI)

Congenital malformations, disruption of bony structures and deviation of neural tissue by trauma can be visualized with radiography and CT. Both plain and contrast radiographic studies are possible. Soft tissue masses, foci of infection or vascular disruption are more readily imaged with MRI.

VI. TREATMENT GOALS

Once a diagnosis of a neurological disorder has been made, the treatment plan should seek to achieve the following clinical goals:

- Manage seizure episodes and any neurologic dysfunction
- Restore cerebral perfusion and oxygenation if compromised
- Control cerebral edema
- Control cerebral inflammation
- Provide metabolic requirements of the debilitated patient
- Address concurrent physical injuries and medical conditions if present
- Manage seizure episodes and any neurologic dysfunction

Drugs for seizure control readily accessible to the practicing equine veterinarian are limited. Therapeutic targets include glutamate receptors, free radical formation, lipid peroxidation, and generation of arachidonic acid metabolites. 23, 24

Benzodiazepines

Drugs of this class are widely used for their anxiolytic, sedative, narcotic, anticonvulsant and muscle relaxant effects. 25 The benzodiazepines are important in the management of seizure disorders associated with fever, hypoxic insult, and are the first choice therapy for status epilepticus. 25 They have a rapid onset of action and minimal toxicity to the patient.

Diazepam is best used in the emergency control of seizures. 26 Rapid onset of short-term control of acute seizures is achieved with minimal depression to the foal. Recurrent seizure activity is often managed with repeated administration of diazepam as required; however, phenobarbital administration to effect is recommended in cases where two or more seizures occur over a short period. Chronic oral administration of diazepam is unsuitable for long-term seizure management as it has a very short half-life, induces hepatic enzymes, and may create tolerance. 27

Midazolam is the only available water-soluble benzodiazepine. 28 Midazolam is fast acting, rapidly penetrates the blood-brain barrier, and has a relatively short duration of action. Administration is possible by both the intravenous and intramuscular route. Initial control of a seizure episode can be achieved by bolus intravenous administration with ongoing seizure control via an intravenous constant rate infusion. 28 Side effects include respiratory depression and hypotension.

Barbiturates

Phenobarbital is used in the management of both acute seizure episodes not responding to shorter acting agents and for long-term control of recurrent seizure activity. Onset of action is rapid. Depression may result during the initial administration period; however, induction of hepatic enzyme metabolism during chronic dosage overcomes this problem. Serum levels require monitoring to ensure the foal remains within the therapeutic range during long-term maintenance therapy. 29

Pentobarbital is reserved for the treatment of uncontrollable status epilepticus. A high level of caution should be observed with its administration, and accumulation may occur. Pentobarbital usage is associated with respiratory depression, myocardial depression, hypotension, and low cardiac output. 30

Phenytoin for neonatal seizure control has been reported. 29 However, little is known about the pharmacokinetics of phenytoin in the equine neonate, with erratic plasma concentrations and considerable depression of some foals reported.

Potassium Bromide (KBr)

Potassium bromide (KBr) has been used since the 19th century as a human anticonvulsant and sedative, and is currently widely used in the management of refractory canine epilepsy. Use in
horses has been reported chiefly in adults; however, control in idiopathic epilepsy of foals has been reported. The author has also found KBr useful adjunctively in the control of neonatal seizures incompletely controlled by other agents.

Seizure Control in Difficult Cases

**Ketamine**

The use of ketamine traditionally has been avoided in patients with neurological injury although use in the management of human refractory status epilepticus has been reported. N-methyl-D-aspartate (NMDA)-receptor antagonism is responsible for its purported neuroprotective action which includes regulation of intracellular calcium levels, attenuated induction of nitric oxide (NO), reduced neuronal degeneration and cell death. Ketamine is useful for the short-term management of seizure activity, being administered as a constant rate infusion following control of a seizure episode. Foals vary in their response.

**Propofol**

Propofol is a short-acting minimally depressive intravenous hypnotic anesthetic agent useful for control of refractory status epilepticus. Usage in the neonatal foal for refractory seizure activity is possible; however, financial considerations preclude widespread usage.

**Gabapentin**

Gabapentin is a structural analogue of GABA although the exact mode of action is unknown. It is used for neuropathic pain in the horse and pharmacokinetics has been reviewed. Use for seizure control in the horse has not been widely reported.

- Restore cerebral perfusion and oxygenation if compromised

**Fluid Therapy**

In addition to meeting the metabolic needs of the patient, appropriate fluid therapy is essential to ensure adequate cerebral perfusion which may have been compromised by a cerebral insult. The brain controls its own perfusion rate and pressure despite fluctuations in arterial blood delivery by autoregulation, the loss of which has been reported after cerebral ischemia and reperfusion or traumatic brain injury. Systemic dehydration has not been shown to decrease existing cerebral edema, and a negative fluid balance has been proven to be detrimental to overall patient outcome.

**Supplemental Oxygen**

An appropriate fluid balance maintains cerebral perfusion and oxygenation while avoiding increased intracranial pressure. In situations where cerebral insult has occurred, judicious use of supplemental oxygen may be of further benefit as the avoidance of hypoxemia improves neurological outcome following traumatic brain injury.

- Control cerebral edema

Edema of the brain decreases perfusion and oxygenation, along with causing cerebral compression against bony confinement and further trauma. Edema may be due to disruption of the blood brain barrier (BBB), cellular disruption causing intracellular water collection, or osmotic imbalances between blood and tissue. Iatrogenic over-hydration potentiates CNS edema following cranial trauma and exacerbates pulmonary edema in recumbent patients (affecting ventilation and oxygenation). Current information suggests elevation of the head during recumbency prevents cerebral edema and improves survival, although the mainstay of edema control has traditionally been the use of hyperosmolar agents.

**Osmotic Agents**

Hyperosmolar (osmotic) agents are central to the control of increased intracranial pressure although firm guidelines for their usage do not exist.

**Mannitol**

Mannitol establishes an osmotic gradient across the blood brain barrier, improves overall blood delivery to the brain, yet decreases intracranial pressure and cerebral blood volume. Plasma volume expansion with mannitol use decreases hematocrit and therefore blood viscosity improving perfusion.

**Hypertonic Saline**

The intact blood-brain barrier is less permeable to saline than to mannitol, therefore hypertonic saline is a more effective and long-lasting hyperosmolar. Hypertonic saline solutions decrease brain water and intracranial pressure while temporarily increasing systolic blood pressure and cardiac output. In situations of increased cranial pressure, hypertonic saline increased survival compared to mannitol.

**Loop Diuretics**

Diuretics such as furosemide have little effect on cerebral edema. Establishing an osmotic gradient across the blood-brain barrier does not require systemic dehydration, and furosemide is detrimental to outcome due to the resulting hypovolemia decreasing cerebral perfusion and oxygenation. In the absence of peripheral or pulmonary edema, loop diuretics are not indicated in the management of cerebral edema.

**Magnesium (Mg)**

Experimentally, cell membrane integrity and permeability are improved by Mg administration, with the magnesium ion antagonizing cell membrane calcium channels and improving function of the Na/K ATPase membrane pump which reduces cell edema. Controversy exists as to the utility of magnesium.
- Control cerebral inflammation

**Anti-Inflammatories**

*Non-steroidal anti-inflammatory drugs* (NSAIDs) attenuate arachidonic acid metabolites that promote platelet aggregation, and facilitate inflammatory and immune reactions. Experimentally, COX inhibitors improve cerebral blood flow, decrease edema, protect COX-2-expressing neurons, and attenuate microglial activation.

*Glucocorticoids* are immunosuppressive, diminish upregulation of pro-inflammatory cytokines and reduce monocyte infiltration following cerebral ischemia. Glucocorticoids downregulate cytokine-mediated COX-2 expression by monocytes and astrocytes and inhibit phospholipase A2, attenuating release of arachidonic acid from the cell membrane. The use of glucocorticoids in cases of cerebral insult is controversial; however, benefit in cases of acute bacterial meningitis has been shown.40

**Dimethyl Sulfoxide (DMSO)**

Recognized as an anti-inflammatory, DMSO is also credited as a free radical scavenger, antioxidant, diuretic, vasodilator and Ca channel blocker. Dimethyl sulfoxide experimentally rapidly reduces raised intracranial pressure and increases cerebral perfusion without an effect on systemic blood pressure.41 The clinical utility of DMSO is also the subject of controversy.

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<th>Seizure control</th>
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<tr>
<td><strong>Diazepam</strong></td>
<td>0.05-0.4 mg/kg IV</td>
<td>Short-acting.</td>
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<tr>
<td><strong>Midazolam</strong></td>
<td>0.2 mg/kg bolus IV</td>
<td>Maintain with 0.1-0.2 mg/kg/h constant rate infusion.</td>
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<tr>
<td><strong>Phenobarbital</strong></td>
<td>4-10 mg/kg IV over 15 min</td>
<td>Long-acting. Maintain long term 5-10 mg/kg PO bid</td>
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<tr>
<td><strong>Pentobarbital</strong></td>
<td>3-5 mg/kg IV</td>
<td>May induce severe respiratory depression. Use only when non-responsive to other agents.</td>
</tr>
<tr>
<td><strong>Propofol</strong></td>
<td>2 mg/kg IV induction</td>
<td>Maintain with 1 mg/kg/h constant rate infusion.</td>
</tr>
<tr>
<td><strong>Ketamine</strong></td>
<td>0.02mg/kg/min IV</td>
<td>Constant rate infusion. Induces a varying level of central depression with beneficial cardiovascular effects.</td>
</tr>
<tr>
<td><strong>Potassium bromide</strong></td>
<td>10 mg/kg PO q 8 h.</td>
<td>Useful adjunct therapy or may be used as the sole agent for long-term management.</td>
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<tr>
<th>Control cerebral edema</th>
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<tr>
<td><strong>Hypertonic saline</strong></td>
<td>Up to 7 ml/kg</td>
<td>IV as 3% solution</td>
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<tr>
<td><strong>Mannitol</strong></td>
<td>1 mg/kg</td>
<td>IV as 20% solution</td>
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<th>Miscellaneous</th>
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<tr>
<td><strong>Magnesium sulfate</strong></td>
<td>0.05 mg/kg IV over 30 min.</td>
<td>Control excitotoxic glutamate neurotransmitter cascade.</td>
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- Provide metabolic requirements of the debilitated patient

Protein and energy malnutrition has been shown to exacerbate brain injury and worsen outcome from periods of global ischemia due to increased oxidative stress. Metabolic requirements have been shown to be increased above maintenance levels with cerebral trauma.42 Seizure activity rapidly depletes energy stores. The affected foal may also experience a period of inappetence compromising the ability to meet high nutritional requirements for rapid growth and tissue repair. Adequate nutrition is therefore important in all cases of cerebral dysfunction regardless of the presence of seizure activity.

- Address concurrent physical injuries and medical conditions if present

Wounds, contusions and decubital ulceration require topical treatments and dressings, and if sufficiently extensive concurrent systemic antimicrobials to counter bacterial dissemination in the compromised neonate. Leg wraps will avoid distal limb edema and secondary limb injury. Head protection ‘bumpers’ minimize secondary neurologic injury from repeated cranial trauma in recumbent or seizing foals. Corneal ulceration secondary to abrasion or exposure keratitis can be prevented by regular application of ophthalmic lubricants. If recumbent, the foal requires a supportive bed and a means to cleanly eliminate body waste. Elevation of the head (up to 30 degrees) minimizes cerebral edema.
Sedation of the seizing patient may be required to minimize self-trauma. Options are limited; however, $\alpha_2$ adrenergic agonists and benzodiazepines possess favorable qualities including the added benefit of muscle relaxation which decreases overall energy requirements. Contraindicated drugs include butorphanol (may increase CSF pressure) and acepromazine (lowers the seizure threshold).

VII. SUMMARY

A thorough understanding of the underlying pathology of neurological dysfunction is required. Concurrent or precipitating medical conditions must be managed. Along with management of the seizure activity, re-establishment of appropriate cerebral blood flow, fluid balance and nutrition is essential to successful management of these cases.

REFERENCES


