Anaesthesia for the patient with neurological disease

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References and Acknowledgements

• Lumb and Jones Veterinary Anaesthesia and Analgesia (2007) 4th ed. Edited by Tranquilli W, Thurmon JC, Grimm KA
• The staff, patients & clients of Greencross South Tamworth Animal Hospital and referring Veterinarians
  Thank you
• Many images were copied from the internet, acknowledged when identification was possible.
Anaesthesia for the patient with neurological disease

- Intracranial disease
  - pathophysiology
  - anaesthesia and analgesia for
    - head trauma
    - intracranial disease
    - epileptics
    - seizure management
    - patients with vestibular disease
- Spinal disease
  - anaesthesia and analgesic considerations
  - anaesthesia for
    - wobblers
    - AA luxation
    - spinal trauma
    - Meningitis
    - Disc disease
- Neuropathies and neuromuscular disease
- Anaesthesia for diagnostic procedures
  - anaesthesia for
    - CSF sampling
    - Myelography
    - CT & MRI
- Artificial ventilation
- Brain death

Physiology

- Whole-brain $O_2$ consumption represents about 20% of total-body $O_2$ utilization in man

- Approximately 60% of the brain’s energy consumption is used to support electrophysiologic function. The depolarization-repolarization activity that occurs and that is reflected in the EEG requires energy expenditure for the maintenance and restoration of ionic gradients and for the synthesis, transport, and reuptake of neurotransmitters.
Physiology

• The remainder of the energy (40%) consumed by the brain is involved in cellular homeostatic activities, such as trans-membrane sodium pump maintaining trans-membrane resting potential and ATP energy production.

• Local Cerebral Blood Flow (CBF) and local Cerebral Metabolic Rate (CMR) within the brain are closely coupled but very heterogeneous depending on the location, for instance both are approximately four times greater in gray matter than in white matter.

• The cell population of the brain is also heterogeneous in its O2 requirements.

• Glial cells make up about half of the brain’s volume and require less energy than neurons. Besides providing a physically supportive latticework for the brain, the glial cells are important in the reuptake of neurotransmitters and in the delivery and removal of metabolic substrates and wastes.

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Brain Oxygen Requirement

60% Activation metabolism
40% Basal metabolism

NEURONAL ACTIVITY
CELLULAR INTEGRITY

Physiology

• The brain’s substantial demand for substrate must be met by adequate delivery of O2 and glucose.
• However, the space constraints imposed by the noncompliant cranium and meninges require that blood flow not be excessive.
• Not surprisingly, there are elaborate mechanisms for the regulation of CBF.
Factors Influencing Cerebral Blood Flow (CBF)

- Chemical/Metabolic /Humoral
  - Cerebral metabolic rate
  - Anaesthetics
  - Temperature
  - PaCO2
  - PaO2
  - Vasoactive drugs - Anaesthetics, Vasodilators, Vasopressors

- Myogenic / Autoregulation

- Rheologic - Blood viscosity

- Neurogenic

Flow- metabolism Coupling

Cerebral Blood Flow & Cerebral Metabolic Rate

- Increased neuronal activity results in increased local brain metabolism, and this increase in CMR is associated with a well-matched, proportional change in CBF.
- Regional CBF and CMR measurements performed during manoeuvres designed to activate specific brain regions provide evidence of the strict local “coupling” of CMR and CBF. Although it is clear that both neural control and local metabolic factors play a major role in these adjustments in CBF, the complete mechanism of flow-metabolism coupling remains undefined.
- CMR is influenced by several phenomena in the neurosurgical environment, including the functional state of the nervous system, anaesthetic agents, and temperature.
- CMR in normal functional states:
  - decreases during sleep
  - Increases during sensory stimulation, mental tasks, or arousal of any cause.
  - During epileptoid activity, CMR increases may be extreme,
  - CMR may be substantially reduced in coma.
Role of glial cells and the blood-brain barrier in flow-metabolism coupling

The brain is well organized into neurovascular units including neurons, glial cells and the cerebral micro-vasculature. Glial cells may be microglia, astrocytes and oligodendrocytes. The integration of neurovascular units is important for the maintenance of cerebral autoregulation and flow-metabolism coupling.

- The regulatory changes involved in flow-metabolism coupling that have a short latency (~1 second) are mediated by metabolic and neurogenic pathways.
- The metabolic control is exerted by increases in perivascular potassium (regulated and maintained by astrocytes) or adenosine concentrations that follow neuronal depolarization.
- Neuronal control is enforced by a rich supply of nerve fibres from the cranial and peripheral nerves. The bigger vessels have the greater innervation.
- The mediators thought to play an important part in neurogenic flow-metabolism coupling are acetylcholine and nitric oxide, although roles have also been proposed for 5-hydroxytryptamine, substance P and neuropeptide Y.

Role of glial cells and the blood-brain barrier in flow-metabolism coupling

Relationship of astrocytes to oxygen and energy metabolism in the brain.

- Glucose taken up by astrocytes undergoes glycolysis for generation of ATP to meet astrocytic energy requirements (for glutamate reuptake, predominantly).
- The lactate that this process generates is shuttled to neurons, which utilize it aerobically in the citric acid (Krebbs) cycle.
The interdependency of cerebral electrophysiologic function and cerebral metabolic rate for oxygen (CMRO2). Administration of various anaesthetic agents including barbiturates results in a dose-related reduction in CMRO2 and cerebral blood flow (CBF). The maximum reduction occurs with the dose that results in electrocerebral silence (corresponds to about 2-3x MAC for isoflurane). At this point, energy utilization associated with electrophysiologic activity has been reduced to zero, but energy utilization for cellular homeostasis persists unchanged. Additional barbiturate administration causes no further decrease in CBF or CMRO2. EEG, electroencephalogram.

The effect of temperature reduction on the cerebral metabolic rate for oxygen (CMRO2). Hypothermia reduces both the components of cerebral metabolic activity that associated with neuronal electrophysiologic activity (“function”) and that associated with the maintenance of cellular homeostasis (“integrity”). This is in contrast to anaesthetic agents, which alter only the function component. CMR decreases by 6 to 7% per degree Celsius of temperature reduction.
Cerebral Metabolic Rate: effect of temperature: Hyperthermia

- Hyperthermia has an opposite influence on cerebral physiology.
  - Between 37 and 42°C, CBF and CMR increase.
  - However, at temperatures higher than 42°C a dramatic reduction in cerebral O2 consumption occurs, an indication of a threshold for a toxic effect of hyperthermia that may occur as a result of protein (enzyme) degradation.

Cerebral Blood Flow: effect of PaCO$_2$

Relative effects of PaCO$_2$ on cerebral blood flow (CBF) and cerebral blood volume (CBV).
C changes 1 to 2 mL/100 g/min for each 1 mm Hg of change in PaCO$_2$ around normal PaCO$_2$ values. This response is attenuated below a PaCO$_2$ of 3.3 kPa (25 mm Hg). CO$_2$ responsiveness has been observed in normal brain during anaesthesia with all the numerous anaesthetic agents that have been studied. Hyperventilation is aimed at reducing CBV in patients with intracranial hypertension but may be detrimental because of its effects on CBF. Note that the slope of CBF reactivity to PaCO$_2$ is steeper than that for CBV (~25 vs. ~20% per kPa PaCO$_2$, respectively).
Cerebral Blood flow: effect of PaCO$_2$

Notes:
• Functional MRI scans
• Red = high cerebral blood flow
• Carbogen is a mixture of 95% oxygen and 5% carbon dioxide
• Atmospheric air contains 0.04% CO2 (currently!)

Cerebral Blood Flow: effect of PaO$_2$

- Changes in PaO2 from 60 to more than 300 mm Hg have little influence on CBF.
- When the PaO2 is less than 60 mm Hg, CBF increases rapidly.
- The mechanisms mediating the cerebral vasodilation during hypoxia are not fully understood, but they may include neurogenic effects initiated by peripheral and/or neuraxial chemoreceptors as well as local humoral influences.
- At high PaO2 values, CBF decreases modestly. At 1 atm O2, CBF is reduced by 12%
Cerebral Blood Flow: effects of Viscosity

- Blood viscosity can influence CBF. Haematocrit is the single most important determinant of blood viscosity.
- Haematocrit variation within the normal range (33–45%) probably results in only trivial alteration of CBF.
- Beyond this range, changes are more substantial.
- In anaemia, cerebral vascular resistance is reduced and CBF increases. However, this may result not only from reduction in viscosity, but also in response to reduced $O_2$-carrying capacity of blood.
- The effect of viscosity reduction on CBF is more obvious in the setting of focal cerebral ischemia when vasodilation in response to impaired $O_2$ delivery is probably already maximal. In this setting, viscosity reduction accomplished by haemodilution results in increases in CBF in the ischemic territory.
- The best available information suggests that, in the setting of focal cerebral ischemia, a haematocrit of 30 to 34% will result in optimal $O_2$ delivery.

Cerebral Blood Flow (CBF)

The physiological factors known to influence cerebral blood flow regulation. In addition to these factors, recent studies have emphasized the role of the sympathetic nervous system influencing cerebral blood flow. CBF = cerebral blood flow

The functional role of the alpha-1 adrenergic receptors in cerebral blood flow regulation Sushmita Purkayastha, Peter B Raven Indian J of pharmacology 2011 Vol: 43 Issue:5 Page: 502-506
Cerebral Blood Flow (CBF)

- Cerebral blood flow is related to cerebral perfusion pressure (CPP) and cerebral vascular resistance (CVR)
  \[ \text{CBF} = \frac{\text{CPP}}{\text{CVR}} \]
- Cerebral perfusion pressure CPP is determined by mean arterial blood pressure (MAP) opposed by intracranial pressure (ICP). For most other organs, perfusion pressure = arterial – venous pressure but because the brain lies in a closed vault the downside pressure is not jugular venous pressure, rather it’s the ICP, thus
  \[ \text{CPP} = \text{MAP} - \text{ICP} \]
  Note that normally, ICP ≈ CVP (central venous pressure).
- To maintain CPP must maintain mean BP
  - Monitor: MAP should be 70-80mm Hg to give a CPP of 60-70mm Hg
  - Minimise CVS depression
  - IV fluids to maintain blood volume, venous return & cardiac output (Starling’s Law)
  - Do not elevate head > 30° or higher MAP will be required
  - Head up tilt = reduction in cerebral perfusion pressure
    For each 2.5 cm above the level of the heart MAP falls by 2 mmHg

Cerebral Blood Flow (CBF)

- Cerebral blood flow
  - PCT normal dogs 1.378 ml/100gm/sec
  - C-arm CT normal dogs 1.429ml/100gm/sec
- Anatomically tortuous Internal carotid arteries in dog
- Grey matter receives ≈4x flow of white matter
- With the exception of oedema reduction by mannitol and draining CSF, the only intracranial constituent whose volume can readily be modified by physiological or pharmacological interventions is the parenchymal cerebral blood volume (CBV), whose volume is set by vasomotor tone. Although the CBV forms only a small part of the intracranial volume, and such interventions produce only small absolute changes (typically ~10 ml or less in man, with a total CBV = 200ml), they may result in marked reductions in ICP in the presence of intracranial hypertension.
- Similar to the rest of the body, most of the CBV is located within the venous system: the pial veins and venous sinuses

Ref: C-Arm CT Measurement of Cerebral Blood Volume in Ischemic Stroke: An Experimental Study in Canines AJNR 2010 31: 536-540 T. Bley, C.M. Strothera, K. Pulfer, K. Royalty
Cerebral Blood Flow (CBF)

Normal dog with unilateral Internal carotid catheter

C arm CT

Ref: C-arm CT Measurement of Cerebral Blood Volume in Ischemic Stroke: An Experimental Study in Canines AJNR 2010 31: 536-540 T. Bley, C.M. Strothera, K. Pulfer, K. Royalty

Mouth gags are a risk factor for cortical ischaemia in cats


Post-anesthetic cortical blindness in cats: Twenty cases.

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Abstract
The medical records of 20 cats with post-anesthetic cortical blindness were reviewed. Information collected included signalment and health status, reason for anesthesia, anesthetic protocols and adverse events, post-anesthetic visual and neurological abnormalities, clinical outcome, and risk factors. The vascular anatomy of the cat brain was reviewed by cadaver dissections. Thirteen cats were anesthetized for dentistry, four for endoscopy, two for neutering procedures and one for urethral obstruction. A mouth gag was used in 16/20 cats. Three cats had had cardiac arrest, whereas in the remaining 17 cases, no specific cause of blindness was identified. Seventeen cats (85%) had neurological deficits in addition to blindness. Fourteen of 20 cats (70%) had documented recovery of vision, whereas four (20%) remained blind. Two cats (10%) were lost to follow up while still blind. Ten of 17 cats (59%) with neurological deficits had full recovery from neurological disease, two (12%) had mild persistent deficits and one (6%) was euthanised as it failed to recover. Four cats (20%) without documented resolution of neurological signs were lost to follow up. Mouth gags were identified as a potential risk factor for cerebral ischaemia and blindness in cats.
Mouth gags are a risk factor for cortical ischaemia in cats

- Cristina de Miguel Garcia, Martin Whiting & Hatim Alibhai. Cerebral hypoxia in a cat following pharyngoscopy involving use of a mouth gag Veterinary Anaesthesia and Analgesia 2012, 40, 103–108

The maxillary artery is a branch of the external carotid, which arises from the common carotid artery at the level of the angle of the jaw where the latter divides into the internal and external carotid arteries. In dogs and humans, the Circle of Willis receives blood by both the internal carotid and the basilar artery. However, in sheep, pigs and cats, only the maxillary artery supplies the Circle of Willis; the basilar artery carries blood away from the arterial circle and does not supply the brain. (Fig. 1) The Circle of Willis also receives a small amount of blood from the ascending pharyngeal artery. The medulla oblongata receives blood from the vertebral artery. Stiles et al. 2012 suggests that spring held mouth gags reduce blood flow to the brain through the maxillary artery by stretching the vasculature and adjacent muscles causing vascular compromise.

We suggest that where the use of such a gag is essential for surgery, the mouth should be closed every few minutes to enable restoration of the blood supply.

![Figure 1](image)

Figure 1 (A) External carotid artery; (B) Facial artery; (C) Lingual artery; (D) Rete mirabile; (E) Bucal artery; (F) Circle of Willis; (G) Basilar artery; (H) Maxillary artery; (I) Palpebral artery; (J) Mental artery.

Ref:
Cristina de Miguel Garcia, Martin Whiting & Hatim Alibhai. Cerebral hypoxia in a cat following pharyngoscopy involving use of a mouth gag Veterinary Anaesthesia and Analgesia 2012, 40, 103–108
**Cerebral physiology: Autoregulation of CBF**

- Autoregulation refers to the capacity of the cerebral circulation to adjust its resistance in order to maintain CBF constant over a wide range of CPP (≈mean arterial pressure).
- The limits of autoregulation occur at MAPs of approximately 50 to 150 mm Hg.
- Above & below the autoregulatory plateau, CBF is pressure dependent (pressure passive) and varies linearly with CPP.
- Autoregulation is influenced by various pathologic processes and, in addition, by the time course over which CPP changes occur.
- Even within the range over which autoregulation normally occurs, a rapid change in arterial pressure will result in a transient (3 to 4 minutes) alteration of CBF.
- Autoregulation is progressively abolished by increasing depth of anaesthesia & PaCO₂.
- The precise mechanism by which autoregulation is accomplished is not known. Nitric oxide may participate in the vasodilation associated with hypotension in some species, but not, according to a single study, in primates.

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**Cerebral physiology: Autoregulation of CBF**

- Cerebrovascular resistance (CVR) changes in response to changes in the cerebral perfusion pressure (CPP) to maintain the cerebral blood flow (CBF).
- Cerebral vasodilation (= a decrease in CVR) maintains CBF with reductions in the CPP. This increases cerebral blood volume (CBV), which results in critical increases in intracranial pressure in patients with poor compliance (steep part of pressure-volume curve).
- MAP = mean arterial pressure.
Cerebral physiology: Autoregulation of CBF

• Cerebrovascular resistance (CVR) changes in response to changes in the cerebral perfusion pressure (CPP) to maintain the cerebral blood flow (CBF).
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• This increases cerebral blood volume (CBV), which results in critical increases in intracranial pressure in patients with poor compliance (steep part of pressure-volume curve).
• MAP = mean arterial pressure.

Putting it all together:
Effect of PaO₂, PaCO₂, and mean BP on CBF
Effect of anaesthesia on Autoregulation of CBF

- Schematic representation of the effect of increasing concentrations of a typical volatile anaesthetic agent on cerebral blood flow (CBF) autoregulation.
- Both the upper and lower thresholds are shifted to the left.

Effect of PaCO₂ on Autoregulation of BP

Fig. 2.3 The effect of different levels of PaCO₂ on the autoregulation curve. The normal autoregulation curve is illustrated at a PaCO₂ of 6 kPa. During hypercapnia autoregulation is lost because the vessels are widely dilated by the CO₂. Conversely, at hypocapnia CBF remains constant over a wide range of blood pressures.
Effect of chronic hypertension on autoregulation

Effect of chronic hypertension on cerebral blood flow (ml/100 gm/min) with varying cerebral perfusion pressure (mm Hg).

Mechanisms for autoregulation: Neurogenic regulation

- Cerebral vasculature is extensively innervated
- Density of innervation declines with vessel size i.e. greatest neurologic influence appears to be on cerebral arteries
- Hemorrhagic shock, a state of high sympathetic tone, results in lower CBF at a given MAP compared to hypotension produced with sympatholytic drugs

- Cholinergic
- Adrenergic
- Serotonergic and VIPergic system
- Dopaminergic
Best Estimates of the Influence of Pure Catecholamine Receptor Agonists and Specific Pressor Substances on Cerebral Blood Flow and Cerebral Metabolic Rate

<table>
<thead>
<tr>
<th>Agonist</th>
<th>Cerebral Blood Flow</th>
<th>Cerebral Metabolic Rate</th>
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<tbody>
<tr>
<td>Pure</td>
<td></td>
<td></td>
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<tr>
<td>$\alpha_1$</td>
<td>0/-</td>
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<td>$\alpha_2$</td>
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<td>$\beta$ (BBB open)</td>
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<tr>
<td>Dopamine</td>
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<td>Dopamine (high dose)</td>
<td>-</td>
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<td>Fenoldopam</td>
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<td>Mixed</td>
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<td>Norepinephrine</td>
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<td>Norepinephrine (BBB open)</td>
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<tr>
<td>Epinephrine</td>
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<tr>
<td>Epinephrine (BBB open)</td>
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Vasodilatory Cascade

- Hypovolemia
- Cardiogenic
- Pharmacologic
- \(\uparrow\) oedema
- \(\uparrow\) CSF
- \(\downarrow\) Mean BP
- \(\downarrow\) CPP
- \(\uparrow\) ICP
- \(\uparrow\) Vasodilation
- \(\uparrow\) CBV
- \(\uparrow\) CMR
- \(\uparrow\) Viscosity
- Hypoxia
- Hypercapnia
Changing your own CBF: Chocolate enhanced learning

• 1) Eat some chocolate
  – Hypothesis: Eat chocolate → ↑ CBF → ↑ cognitive function (if impaired) ref Neurology Aug 2013
  – Recall CBF = CPP/CVR and CPP = MAP - ICP
    • If CPP remains same, CVR must ↓ ie vasodilation (→ ↑ CBV)
    • However caffeine content → ↑ MAP → ↑ CPP (→ ↓ CBV)
      in part opposes the vasodilation
  
• 2) Now hyperventilate
  → ↓ PaCO2 → cerebral vasoconstriction
  → ↑ CVR and ↓ CBF and ↓ ICP

= Cure for chocolate headache

Intracranial pressure

• Normal ICP 0-10m Hg
• Clinical signs of raised ICP:
  – Depression
  – Pupillary changes
  – Alterations is respiration
  – Severe ↑ ICP → Cushing’s triad: hypertension, bradycardia & respiratory disturbances
    • Due to compression of vital medullary centres
    • The patient tries to maintain perfusion by ↑ BP
    • Thus hypertension with concomitant bradycardia is common
    • Ventricular arrhythmias commonly occur
  – As ICP ↑ (>35mm Hg) the patient tries to cope by ↑ BP
    but CPP ↓ and CBF ↓
(A) Cerebral blood flow (CBF) autoregulation. CBF is maintained at 50 mL/100 g/minute for mean arterial pressure (MAP)/cerebral perfusion pressure = 50 to 150 mm Hg. 
(B) Linear relationship between partial pressure of arterial carbon dioxide (Paco₂) and CBF for Paco₂ = 20 to 80 mm Hg. 
(C) Paco₂ and CBF. 
(D) Intracranial pressure (ICP) and CBF.

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The Monro Kellie Doctrine

- The Monro Kellie Doctrine describes the interrelation of the various volume compartments of the CNS:
  - Brain (white and gray matter)
  - Ventricles with CSF
  - Subarachnoid space (SAS) with CSF
  - Volume of the blood in vessels

- The Monro Kellie Doctrine suggests that when the volume of one compartment increases, there must be a corresponding and compensatory decrease in the volume of the other spaces.
The intracranial pressure-volume relationship.

- The horizontal portion of the curve indicates that there is initially some latitude for compensation in the face of an expanding intracranial lesion.
- That compensation is accomplished largely by displacement of cerebrospinal fluid (CSF) and venous blood from intracranial to extracranial spaces.
- Once the compensatory latitudes are exhausted, small volume increments result in large increases in intracranial pressure, with the associated hazards of either herniation or decreased cerebral perfusion pressure (CPP) resulting in ischemia.

Methods to control intracranial pressure

- Positioning (head elevated – no more than 30°)
- Avoid increases in CVP (avoid jugular occlusion, give pelvic support, ↓ peak Inspiratory pressure during IPPV, smooth intubation with no coughing or excitement)
- No suxamethonium. Nor Ketamine unless used only as adjunct drug & normocapnia
- Ventilation
- Fluid therapy
- Mannitol 0.2 to 1gm/kg IV over 10-20 mins
- Furosemide 0.5-1.0mg/kg & CRI 1mg/kg/hr
- Sedation and anaesthesia
- IV Lidocaine
- Enriched inspired O2
- Hypothermia
- Intentional hypotensive anaesthesia
- Barbiturate coma
Ventilation

- Carbon dioxide dilates the cerebral blood vessels, increasing the volume of blood in the intracranial vault and therefore increasing ICP. Patients should be ventilated to normocapnia (PaCO2 4.0 kPa / 30mmHg).
- Previously, hyperventilation was used routinely to maximally reduce PaCO2. No studies have shown this to improve outcome in these patients.
- In man transcranial doppler (TCD) assessment and positron emission tomography (PET) shows hyperventilation can induce significant constriction of cerebral vessels and this increase in cerebral vascular resistance may reduce cerebral blood flow to below the ischaemic threshold.

![Graph showing the relationship between cerebral blood flow and PaCO2 during hypotension.](image)

**Effect of Ventilation during hypotension**

![Graph showing the relationship between cerebral blood flow and PaCO2 during hypotension.](image)

*Fig. 2.2 The relationship between cerebral blood flow and arterial PaCO2 at normal blood pressure and during extreme hypotension. The complete relationship between CO2 and cerebral blood flow at normotension is sigmoid-shaped, but between PaCO2 values of 3 and 9 kPa it is virtually linear; at mean blood pressures below 50 mmHg there is no response to change in PaCO2 during hypotension induced by either haemorrhage or trimetaphan.*
Intravenous fluid therapy

- Patients with severe brain injury should be kept normovolaemic. Previous regimens recommending that patients be kept ‘dry’ have essentially been discarded as there is significant risk of both hypotensive episodes (leading to a fall in cerebral perfusion) and systemic inflammatory response syndrome (SIRS) or multiple organ failure (MOF) leading to failure of oxygenation and ventilation. Dehydration has little effect on cerebral oedema.
- Free water (as dextrose solutions) should NOT be administered. This will decrease plasma osmolality and so increase the water content of brain tissue (the blood brain barrier acting as a semipermeable membrane).
- Elevated blood sugar levels are associated with a worsening of neurologic injury after episodes of global cerebral ischaemia. Ischaemic brain metabolises glucose to lactic acid, lowering tissue pH and potentially exacerbating ischaemic injury.
- Hypertonic solutions and osmotic diuretics such as mannitol will have the opposite effect. This mechanism requires an intact blood brain barrier. If this is damaged, as may be the case following injury, low molecular weight, osmotically active particles may leak into the cerebral interstitium. In this case mannitol may have no effect in reducing brain water content.
- Maintenance of the colloid oncotic pressure in the vessels by administration of colloids, plasma proteins or other high molecular weight compounds may, theoretically, be of benefit. However in practice, colloids offer little benefit over crystalloid solutions, unless the patient is hypoproteinaemic.

Intravenous fluid therapy

- There has been considerable interest in the use of hypertonic crystalloid solutions for the treatment of hypovolaemia in the presence of intracranial hypertension.
- Animal studies have proven the efficacy of hypertonic solutions in reversing shock, and sometimes in controlling ICP.
- Clinical trials suggest that survival after severe brain injury (GCS<9) may be improved with hypertonic solutions.
- However those injuries leading to a breakdown in the blood brain barrier show little or worsened response to hypertonic fluid administration.
- There is no single best fluid for patients with traumatic brain injury, but isotonic crystalloids are widely used and have good scientific basis.
- Normal saline or lactated Ringer’s solution should be the standard resuscitation fluid until further studies show a clear benefit from other therapies. Regardless of the fluid type chosen, normovolema must be maintained and episodes of hypotension avoided.
Mannitol

- Mannitol, a 6-carbon sugar, is widely used in head injury management, though it has never been subjected to a randomised control trial against placebo in man and the methods and timing of administration vary widely.
- It is an osmotic diuretic and can have significant beneficial effects on ICP, cerebral blood flow and brain metabolism.
- Mannitol has two main mechanisms of action:
  - Immediately after bolus administration it expands circulating volume, decreases blood viscosity and therefore increases cerebral blood flow and cerebral oxygen delivery.
  - Its osmotic properties take effect in 15-30 minutes when it sets up an osmotic gradient and draws water out of neurons.
- However after prolonged administration (continuous infusion) mannitol molecules move across into the cerebral interstitial space and may exacerbate cerebral oedema and raise ICP. Mannitol itself directly contributes to this breakdown of the blood brain barrier.

Mannitol

- Mannitol is best used by bolus administration where an acute reduction in ICP is necessary.
- For example the patient with signs of impending herniation (decreasing level of consciousness, unilateral dilated pupil / extensor posturing) or with an expanding mass lesion may benefit from mannitol to acutely reduce ICP during the time necessary for CT scanning and/or operation.
- Mannitol is wholly excreted in the urine and causes a rise in serum urine and osmolality. Patients with poor renal perfusion (shock), sepsis, receiving nephrotoxic drugs or with a serum osmolality over 320mOsm are at risk of acute tubular necrosis.
- Hypovolaemia should be avoided with the infusion of isotonic fluids as necessary.
Sedation and anaesthesia

• All but the most severely brain injured patients (GCS 3) will require anaesthesia for intubation.
• The cardiovascular responses to intubation induce a rise in ICP which is exaggerated in those patients on the cusp of the pressure-volume curve.
• Rapid sequence intubation is probably the safest method of establishing an airway in these patients.
• Do not induce gagging, laryngospasm, coughing or vomiting (all elevate ICP)
• For patients receiving IPPV, continuing sedation will be necessary in most to allow adequate ventilation and to prevent coughing or fighting the ventilator. Ensuing vasa-valva-type manoeuvres cause sharp rises in intracranial pressure. Which agents are used to achieve sedation is probably less important. However short acting preparations will allow finer control of the depth of anaesthesia and faster recovery from sedation. Agents with a longer duration of action such as diazepam may be best administered by intravenous bolus as required rather than by constant infusion to avoid build-up of active metabolites.

Sedation and anaesthesia

• Sedation is not analgesia, and pain requirements must be addressed to provide a quiet, comfortable patient. Adequate analgesia will also reduce the requirements for sedation.
• The use of neuromuscular blocking agents is not routinely required for continued ventilation. However some patients whose high sedative requirements lead to adverse cardiovascular effects may benefit from pharmacologic paralysis.
• The actual sedative & anaesthetic drugs used to facilitate intubation and IPPV are discussed later in this lecture
Effect of anaesthesia on cerebral physiology

- Anaesthetic agents cause dose-related and reversible alterations in many aspects of cerebral physiology including cerebral blood flow (CBF), cerebral metabolic rate (CMR), intracranial pressure (ICP) and electrophysiologic function (EEG, evoked responses).
- The changes in CBF and CMR can be of clinical importance in patients with neurosurgical diseases.
- Certain anaesthetic agents and techniques have the potential to affect the diseased brain and the conduct of the neurosurgical procedure adversely.
- However, in certain instances, the effects of general anaesthesia on CBF and CMR can be manipulated to improve both the operative course and the clinical outcome of patients with neurologic disorders.

NB: Most anaesthetics are dose related respiratory depressants

Depression of the ventilatory response to CO2 by pentobarbital and thiopentone in the dog.
The slopes are normalised to the same starting point on the abscissa.
Estimated changes in cerebral blood flow (CBF) and cerebral metabolic rate for oxygen (CMRO2) caused by volatile agents. The CBF data for halothane, enflurane, and isoflurane were obtained during 1.1 MAC anaesthesia (with blood pressure support) in human patients, expressed as a percentage of change from awake control values. The CMRO2 data for halothane, enflurane, and isoflurane were obtained in the cat and are expressed as a percentage of change from nitrous oxide (N2O)–sedated control values. The data for sevoflurane were obtained during 1.1 MAC anaesthesia in the rabbit and are expressed as a percentage of change from a morphine/N2O–anesthetized control state.

Cerebral blood flow (CBF)-cerebral metabolic rate for oxygen consumption (CMRO2)-cerebral blood volume (CBV) during increasing doses of an intravenous (propofol) and volatile anaesthetics. Changes are noted as a percentage value from the awake state. Despite similar changes in CMRO2, changes in CBF and CBV are markedly different among intravenous and volatile agents and among sevoflurane, isoflurane, and desflurane above 1.5 minimum alveolar concentration (MAC). EC50, median effective concentration.
Effect of anaesthetic drugs & hypocapnia on cerebral metabolic rate (CMR) & cerebral blood flow (CBF)

- Note that hyperventilation & the resulting hypocapnia can lead to vasoconstriction (decreased CBF) & an increase in CMR, a particularly unfavourable situation.
- Note also that increasing the inhalational anaesthetic agent concentration (minimum alveolar concentration, MAC) does not decrease CMR below a lower limit. However, it does increase CBF, thereby increasing CBV and intracranial pressure. This is hazardous for patients with poor intracranial compliance.
Aim of neuroanaesthesia

- Preserve and assist restoration of neurological function
- Maintain cerebral blood flow (CBF) & cerebral perfusion pressure (CPP)
  - Maintain MAP
  - Maintain cardiac output
  - Minimise increases in CBV (eg many anaesthetics, hypercarbia, hypoxia → vasodilation
- Maintain autoregulation
- Maintain flow- metabolism coupling
- Reduce Intracranial pressure (ICP)
- Avoid sudden increases in ICP (or sudden decrease with CSF tap)
- Cerebral protection minimising primary and secondary damage
- Reduce cerebral metabolic demand (deepen anaesthesia, use hypothermia and treat epilepsy).

Good Anaesthesia Practice avoids:

- Hypoxia and hypoxaemia
- Hypercarbia
- Hypotension
- Dehydration
- Overdosage
- Hypothermia
- Fear, anxiety & pain
- Hypoglycaemia
- Inattention
- Reflux & Aspiration
- Injury
Additional goals in anaesthesia of the neurological patient

• Pre-anaesthesia
  – IV fluids
  – Treat hypoglycaemia.
    • Max. IV 50% glucose dose is 0.16 ml/kg
  – Treat hypo or hypernatraemia slowly or → Osmotic demyelination syndrome
    • < 0.5 to 1 mEq/L/hr or 10 mEq/L over 24 hours
  – Treat with Thiamine if any possibility of thiamine deficiency
  – Mannitol
  – Oxygen
  – Seizure control
• Preserve CBF & CPP
  – Recommended agents: opioids, benzodiazepines, propofol, thiopentone, isoflurane, Sevoflurane
  – Avoid rises in ICP:
    • vomiting, coughing, straining, N2O, high dose ketamine, high dose myelography
    • → Cushing’s triad: hypertension, bradycardia and respiratory disturbances
    • Jugular vein occlusion
    • Tx Lidocaine bolus 1 mg/kg and CRI
  – Bradycardia common but avoid anticholinergics unless ↓ MAP
  – CVP monitoring or preferably Mean Circulatory Filling Pressure

BP measurement is critical

Indirect oscillometric BP measurement with PetMap is very helpful
Most oscillometric machines tend to overestimate pressures in hypotensive states
Anaesthetic considerations for patients with raised intracranial pressure

- PreTx IV fluids, correct electrolyte abnormalities especially sodium (slowly) (< 0.5 to 1mequiv/kg/hr)
- Baseline bloods and urine s.g.
- Preoxygenate: nasal O2 catheter if delays, hypoventilation, hypoxia etc
- Premed:
  - Opioids: no direct effect on ICP or seizure threshold
    - not morphine (vomiting)
    - Methadone 0.2mg/kg IM or fentanyl 2-5ug/kg IV then CRI
  - Benzodiazepines: ↓ ICP and ↑ seizure threshold
    - May potentiate respiratory depression due to induction agents
    - Diazepam & midazolam 0.1 to 0.2mg/kg IV
  - ACP: no change in ICP & may actually ↑ seizure threshold *
    - Clinical doses (0.01 to 0.03mg/kg IM or IV)
    - does not affect incidence of seizures
  - Metomidine: ↓ ICP and little change in seizure threshold
    - Cerebral vascular affects not well understood
    - May cause vomiting (not good)
    - ↓ flow- metabolism coupling
    - Use with caution

Anaesthetic considerations for patients with raised intracranial pressure

- Induction
  - Avoid coughing
  - PreTx 1 min prior induction lidocaine 1mg/kg to ↓ ICP
  - Care with intubation patients with caudal fossa disease
  - Suitable induction agents (given slowly to effect) especially if combined with fentanyl (since ↓ induction dose)
    - Thiopentone 5-10mg/kg
    - Propofol 1-8mg/kg IV
    - Alfaxan 1-2mg/kg (following IV premed)
    - Aggressive cats:
      - ketamine 2-5mg/kg + midazolam 0.2mg/kg IM
      - Avoid ketamine if seizing.
      - Larger doses raise ICP
The tentorium cerebri is a piece of dura matter, which separates the cerebrum from the cerebellum.

Often, pathology within the brain will be described as supratentorial (above the tentorium) or infratentorial (below the tentorium).

Acknowledgement: Images from Veterinary Imaging Chesapeake Veterinary Referral Center
Head Injury & CBF

• Direct and indirect effects on CBF and metabolism
• If seizing, CBF may be high, normal or low soon after ictus but is typically reduced.
• In man 30% of patients undergoing CBF studies within 6-8 h of a head injury have significant cerebral ischaemia.
  – Global hypoperfusion studies → 100% mortality at 48 hours
  – Regional ischaemia results in significant deficits.
• Cerebral blood flow patterns also vary in relation to the time after injury.
  – Initial reductions are replaced, especially in patients who achieve good outcomes, by a period of relative increase in CBF, which towards the end of the first week post-ictus may be replaced (in some patients) by reductions in CBF that are the consequence of vasospasm associated with traumatic subarachnoid haemorrhage
• Changes in CBF are non-uniform in the injured brain.
  – Blood flow tends to be reduced in the immediate vicinity of intracranial contusions
  – Cerebral ischaemia associated with hyperventilation may be extremely regional and not reflected in global monitors of cerebrovascular adequacy.

Spectrum of cerebral blood flow (CBF) patterns following severe head injury.
• Following an initial period of ischaemia lasting <24 h, CBF begins to rise and may exceed normal values on days 2-4; CBF may fall to subnormal levels at later time points, chiefly due to the presence of vasospasm secondary to traumatic subarachnoid haemorrhage.
• The CBF levels may never rise in some patients, especially those who have a poor outcome.
• Elevations in ICP result in reductions in CPP and cerebral ischaemia, which lead to secondary neuronal injury.
• There is strong evidence than maintenance of a CPP above 60 mmHg improves outcome in patients with head injury and raised ICP.
Management of ICP in head injury

• Mannitol
  – Although the BBB is disrupted by ischaemia, this process takes hours to days rather than minutes, and much of the cerebral oedema seen in the initial period after ischaemic insults is cytotoxic rather than vasogenic. Consequently, mannitol retains its ability to reduce cerebral oedema in the early phases of acute brain.

• Barbiturate coma
  – Many clinical studies have demonstrated that barbiturate coma can effectively lower ICP
  – The main disadvantages are two-fold.
    • significant episodes of hypotension,
    • the prolonged half-life makes clinical assessment difficult after barbiturates are stopped.
  – There is no good evidence for improved outcome.

Management of ICP in head injury

• Hypothermia
  – Mild to moderate hypothermia (32-34 °C temp.) provide protection from cerebral ischemia.
  – Achieved by
    • allowing loss of body heat
    • administration of cold IV fluids
  – ↑ Risk of:
    • cardiac dysrhythmias
    • coagulation dysfunction
    • sepsis.
  – Need of rewarming prior to emergence from anaesthesia (to avoid shivering and hypertension)
Management of ICP in head injury

- Anaesthesia may be required for case management, to control seizuring, for ventilation, imaging, for concurrent injury management or for CNS surgery
- Thorough evaluation for other injuries
- Rapid resuscitation to maintain adequate perfusion. NB Last slides: hypotension and hypoxia are associated with poor outcomes
  - CPP > 70mm Hg preferably but always >60mm Hg
  - IV fluids to improve MAP&CVP and HR to normal
  - Initially small volumes hypertonic saline 1-4ml/kg or colloids, then crystalloids
  - Mild haemodilution (PCV 30-35%) to lower viscosity and improve cerebral O2 delivery
  - Hyperventilation is detrimental → ↓CBF (25% ↓ per kPa PaCO2↓) though mild hyperventilation in short term maybe beneficial (↓ICP)
  - NO corticosteroids or glucose (unless hypoglycemia)
- Severity of head trauma in Veterinary patients ≈ degree of hyperglycaemia
- Glucose is linked to brain injury
  - Avoid jugular vein obstruction
- Nutritional support for comatosed patients and those with facial fractures

Anaesthesia for epileptic patients

- Seizure activity should be carefully differentiated from syncope and extra-cranial causes such as hypoglycaemia and liver disease
- Tx produces biochemical changes
  - Phenobarb → ↑Alk Phos ± ↓ Ca ± liver disease (pre+post bile acid test)
  - KBr → false ↑ Cl
  - All → PDPU so only withhold water after premed given
- IV access to manage fluids and seizure Tx if required
- Premed
  - Low dose ACP (5-60ug/kg) OK (Tobias 2006 JAAHA 42:283-289)
  - Opioids do not alter seizure activity (not morphine as → vomiting)
  - Low dose alpha2 in excitable patients
  - Not benzodiazepines alone in non-seizuring patients as → dysphoria & excitement
- Induction
  - Preoxygenate
  - Propofol, thiopentone and Alfaxan all OK slowly to effect
  - No mask inductions
- Maintenance
  - Sevoflurane is agent of choice otherwise isoflurane.
  - TIVA propofol CRI is good. Monitor BP and ventilation
- Seizuring
  - Seizure → ↑ CMRO2 and goes unnoticed in anaesthetised patient.
  - If ↑CMRO2 exceeds cerebral blood flow → neuronal injury
  - Seizure activity can persist into post-op period
- Recovery: monitor carefully in a quiet area
Anaesthesia for seizure management

- Control of prolonged seizures (status epilepticus or cluster seizures) prevents further neurological damage, minimises hypoxia, hyperglycaemia & hyperthermia
- Tx
  - Initially diazepam 0.2-2mg/kg IV or per rectum or Midazolam 0.05-0.5mg/kg IV or IM
  - Phenobarbitone 2-15mg/kg IV
    - 20mg/kg IV, IM and PO safe and well tolerated. t1/2 = 180min.
  - Pentobarbital 2-15mg/kg then CRI 0.1-0.2mg/kg/min
    - Disadvantage is prolonged emergence delerium. On going seizuring or barbiturate recovery?
  - Propofol
    - For Status epilepticus refractory to phenobarb and or diazepam, propofol 2-8mg/kg boluses effective in 11/12 cases (Steffen & Grasmueck 2000 J of S.A. Practice 41:496-499
    - Can maintain sedation with CRI 0.1 to 0.4mg/kg/min. Expensive
    - If use CRI, respiratory monitoring required
  - Prolonged inhalation anaesthesia with sevoflurane or isoflurane can be used
    - Careful monitoring & care required: airway management, respiratory & BP monitoring, adequate bedding, temperature, turn every 4 hrs, bladder care (in dwelling catheter), eyes lubricated q6hrly, wash & swab mouth q4-q6hrs
- Recovery
  - Sometimes see neck tremors. Tremor or seizure recurrence? Differentiate by checking if patient responds to sensory input: stroking or speaking, if so, its not seizuring.
  - Dogs recovering from propofol can display intermittent forelimb extension
  - Many patients recovering from anaesthesia can show a period of excitement that usually only lasts a few minutes. The patient may need comforting and soothing during this period. If extended recovery then consider other causes: hypoxia, pain, (additional analgesia ± sedation required?), distended bladder. This is not seizure activity.
  - Monitor carefully
  - Check temperature & glucose prior to weaning of infusions

Anaesthesia for patients with vestibular disease

- Anaesthesia for patients suffering from vestibular disease will often create a degree of deterioration in clinical signs for 24-48 hours
- Use short acting drugs to encourage a rapid recovery that is free of excitement
- Low dose medetomidine and propofol induction has been recommended
- Nausea may be seen following anaesthesia
- Surgery for middle ear disease is particularly painful and a multimodal analgesic plan is required (opioids, NSAIs, bupivacaine splash blocks, Ketamine-lidocaine CRI)
Anaesthesia for Spinal Disease

- Often very painful requiring multimodal analgesia regime: Opioids, NSAIs, ketamine CRI, Lidocaine CRI, topical morphine (preservative free) applied to exposed spinal cord.
- Drugs with neuroprotective properties
  - NO corticosteroids!
  - Ketamine CRI
- Haemorrhage is common especially neck surgeries (ventral slots etc)
  - Wide bore catheter for rapid IV fluids
  - DEA -ive fresh blood available
  - Monitor BP
  - IPPV if hypercapnia, since causes vasodilation and sympathetic nervous system stimulation and may contribute to blood loss
- Hypothermia is common
  - Especially small patients
  - Monitor
  - Prevention and treatment
- Neuropathic pain
  - E.g. Traumatic injury to nerves, nerve root entrapment, neoplasia of brachial plexus, nerve root & nerve sheath tumours, discospondylitis, meningitis, phantom limb pain
  - Signs: depression, withdrawal from interaction, anorexia, aggression, hyperaesthesia, allodynia
  - Tx as above PLUS Gabapentine, low dose α2 agonists, local anaesthesia, tricyclic antidepressants

Analgesia for Spinal Disease

- Opioids
  - Good for inflammatory pain but not so useful in neuropathic pain
  - Severe pain requires mu agonist opioids not mixed agonist-antagonist that have a ceiling effect
  - Dose related respiratory depression but not when Tx painful patient, so not usually a clinical problem
  - MAC sparing
  - Vagotonic (bradycardia)
- Drugs
  - Methadone 0.2 to 0.3mg/kg IV 0.2 to 0.5mg/kg SC IM
    - Fat soluble, so accumulates if CRI or repeated doses
    - NMDA receptor antagonist
    - Does not release histamine
    - Racemic mix of “d” and “l” isomers in Australia
  - Morphine 0.2 to 0.5mg/kg SC, IM, CRI 0.1 to 0.2mg/kg/hr
    - Water soluble, good CRI, good for epidural use
    - Histamine release IV especially if dose > 1mg/kg
  - Fentanyl 5ug/kg IV and or CRI 0.02 to 0.2ug/kg/min
    - Respiratory arrest possible
    - Vagotonic, atropine may be required
  - Buphrenorphine 20ug/kg SC IM IV, trans-buccal
    - Mixed agonist-antagonist
  - Fentanyl patch 3-5ug/kg/hr
    - Onset 12-24hrs
    - Variable plasma levels
    - Duration 72 hours
Analgesia for Spinal Disease

• Ketamine
  – NMDA antagonist
  – Dose 50-100ug/kg IM or IV bolus then CRI 5-20ug/kg/min

• Lidocaine
  – MAC sparing
  – Analgesic when given IV, duration exceeds blood levels
  – Negative ionotrope
  – Dose 1mg/kg IV then CRI 20-50ug/kg/min
  – ↓ dose in hypovolaemia, not cats, not liver disease, tolerance develops with prolonged infusions

• Medetomidine
  – Alpha 2 agonist
    • Intense peripheral vasoconstriction, initial hypertension, reflex bradycardia and resultant decrease in cardiac output (often > 50%)
    • good analgesic
    • Not in cardiac disease, elderly, anaemic, pregnant patients
    • Dose 1ug/kg IV then CRI 1-2ug/kg/hr

• Gabapentine
  – Anticonvulsant and for Tx of Neuropathic pain
  – Dose 5-10mg/kg orally 2-3x daily
  – Adverse effects: mild sedation
  – Do not to discontinue abruptly (mimics alcohol withdrawal syndrome)
  – Increases synaptic conc of GABA, release of mono-amine neurotransmitters & other complex actions

• NSAIs
  – Should be used in conjunction with other analgesics
  – Adverse effects: Gastric and duodenal ulceration
  – Significant species differences in prostaglandin regulation of renal blood flow means toxicity studies are not necessary comparable between species
  – Do not give pre-anaesthesia as renal toxicity exacerbated by hypotension
  – 17th biggest killer of humans in USA mortality studies
  – Serious interaction with ACE inhibitors in man

• Amantadine
  – No dominant mechanism of action has been identified. It is a dopaminergic, noradrenergic and serotonergic substance, blocks monoamine oxidase A and NMDA receptors, and seems to raise beta-endorphin/beta-lipotropin levels
  – Older uses as anti-influenza drug & anti-Parkinsonian drug
  – 3-5mg/kg 1-2x daily. Slow onset of action – days to weeks

• Amitriptyline
  – Tricyclic anti-depressive used for the Tx of neuropathic pain
  – Adverse effects: nausea and depression
  – Dose cats 0.25 to 1.0mg/kg po q24hr, dogs 1-2mg/kg po q12hr
An anesthetic considerations for specific spinal surgeries

- **Cervical spondylopathy (Wobblers)**
  - Often Dobermanns so check buccal bleeding time and von Willebran’s factor test
  - If positive Tx: Desmopressin and plasma
  - Avoid drugs that affect platelet function (ACP, many NSAIs)
- **Surgery (dorsal or ventral)** often associated with haemorrhage so keep venous pressures low (minimum peak inspiratory pressures during IPPV and avoid excessive abdominal pressure (empty bladder & elevate pelvis with padding if ventral recumbent)
- **Ventral slot surgery**
  - Trachea and vagocarotid trunk are retracted →
  - Tracheal retraction may affect airway patency
  - Iatrogenic to recurrent laryngeal nerves affecting laryngeal function post-op
  - Vagal stimulation → bradycardia (Tx = reposition retractors, infrequently anticholinergics required)
  - Head and neck are extended
    - Do not lower head → haemorrhage
    - Do not raise head higher than body → ↑ risk of air embolism
- **Ventral venous sinus haemorrhage** is common and often serious
  - Monitor BP (direct and indirect)
  - Have appropriate fresh blood on hand to administer if required
- **Cervical cord surgery** can result in respiratory compromise
  - Monitor capnography and SpO2 ± arterial blood gases
  - Be prepared for IPPV if required (unusual)

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An anesthetic considerations for specific spinal surgeries

- **Atlanto-axial luxation**
  - Challenging anaesthetic
  - Toy breeds < 2 yr
  - Congenital instability of A-A joint causes chronic or acute cord compression
  - Respiratory compromise or failure especially at induction
  - Adequate sedation to enable IV catheter placement with minimal restraint
  - Struggling → rapid deterioration
  - If a support neck brace and bandage has already been applied, cut the bandage before induction to permit intubation
  - Rapid IV induction technique causing minimal respiratory depression (Alfaxan best or propofol, thiopentone)
  - No benzodiazepines as muscle relaxation further destabilises the joint
  - Pre-oxygenation by the “flow by” technique, avoid face mask struggling/avoidance
  - Support head and neck in neutral position during induction, then position in lateral recumbency for intubation
  - Use laryngoscope
  - Care when positioning for radiology
  - A neck brace must be applied if patient is too immature for surgery
    - Not to tight otherwise pharyngeal URT obstruction during recovery and/or difficulty swallowing leading to aspiration pneumonia
Anaesthetic considerations for specific spinal surgeries

- Spinal trauma
  - Stabilise on a board prior to transport
  - Extension of the spine rather than compression when moving patient
  - Check and treat for co-morbid life threatening conditions: haemorrhage, shock, pneumothorax, lung or myocardial contusions, ruptured bladder
  - IV access, IV fluids, IV opioid analgesia
  - Nasal oxygen where needed
  - Stabilise cardiopulmonary function where possible prior to anaesthesia

- Hemilaminectomy
  - Multimodal analgesia (opioid CRI, NSAIs, lidocaine & ketamine CRI, direct spinal application of morphine)
  - Post-op oxygen supplementation
  - Hypothermia is common
  - Post-op seizure is not uncommon if there has been spread of myelographic dye into the cerebral ventricular system.

- Meningitis
  - Often severe pain and hyperaesthesia making patients difficult to restrain
  - Low dose medetomidine (1-5ug/kg IM) plus and opioid e.g. fentanyl 10ug/kg IM
  - CRI opioid, lidocaine + ketamine

Anaesthesia for animals with neuropathies or neuromuscular disease

- Myasthenia gravis is often associated with megoesophagus, dysphagia and possible aspiration pneumonia
  - Dx: Chest radiography before anaesthesia:
    - megoesophagus? aspiration pneumonia?
  - No benzodiazepines or Alpha 2 agonists (worsen muscular weakness)
  - Fast IV induction-intubation sequence
    - Sternal recumbency keeping head elevated
    - Preoxygenate
    - ± Cricoid pressure during intubation
    - Suction and dry swabs available to retract tongue & clear oropharynx
  - Monitor ventilation (ETCO₂) & IPPV available
  - Variable signs of anaesthetic depth
    - Palpebral reflex, jaw tone etc maybe diminished because of disease NOT anaesthetic depth
Anaesthesia for animals with neuropathies or neuromuscular disease

• Myasthenia gravis is often associated with megoesophagus and dysphagia
  – Post-op support plans
    • PEG tube for nutrition
    • Permanent tracheostomy
    • No naso-oesophageal or nasogastric tubes
  – Recovery
    • Inspect and clear pharynx
    • Leave ET cuff inflated during extubation to draw out secretions
    • Support pharynx higher than nose after extubation to encourage secretions & regurgitation to drain through mouth rather than aspirated
    • Supplementary warmth. These patients may not be able to shiver
    • Turn regularly to minimise atelectasis
    • Nasal O2 or IPPV as under ventilation is common
  – Adequate analgesia
    • Severely affected neuropathy patients may not exhibit normal signs of pain

Anaesthesia for animals with neuropathies or neuromuscular disease

• Jasmine 6 yr old Fn Tenterfield terrier
  – over 2.5 years 4 recurrences of severe generalised paresis
  – Resolution 2-5 weeks
  – Dx Polyradiculoneuropathy
  – Severe partial URT obstruction and hypoventilation last admission

Tx Permanent tracheostomy
Anaesthesia for animals with neuropathies or neuromuscular disease

• Megoesophagus
  
  Intubate head up in sternal recumbency, dry swaps and/or suction to clear oropharynx

Conscious radiograph

Anaesthesia for animals with neuropathies or neuromuscular disease

• Tetanus
  – In dogs and cats, similar considerations to other neuromuscular diseases.
  – Admantos & Boag 2007 Vet Rec. 161, 298-303 “Thirteen Cases of tetanus in dogs”
    • If recumbent, turn every 4 hrs of sternal recumbency to prevent atelectasis
    • dysphagia & risk of aspiration pneumonia (3/13)
    • interestingly no hypoventilation but sometimes hypoxia requiring supplementary O2 (4/13)
    • tracheostomy for URT obstruction (1/13),
    • 2 episodes of respiratory arrest in one dog requiring intubation and IPPV for 5min
    • 1 dog developed seizures Tx phenobarb 1mg/kg bd
    • Sedatives & muscle relaxants combinations in most cases as IV bolus ± CRI:
      – ACP 9, Diazepam 7, Midazolam 1, Butorphanol 2, Dantrolene (oral) 3, Propofol 1,
        Pentobarbital 2, Giaphenesin 1
    • 12 survived 1/13 died following episode of hyperthermia
    • Nutritional support in 10 dogs showing dysphagia 7/13 PEG tubes 1/13 oesophagostomy tube all requiring a general anaesthetic
  – triad of rigidity, muscle spasms and autonomic dysfunction (massively elevated catecholamine levels)
## Anaesthesia for animals with neuropathies or neuromuscular disease

- **Snake bite envenomated patients**
  - Severely envenomated animals often require artificial ventilation
  - About half require sedation or anaesthesia to maintain endotracheal intubation and minimise struggling against the ventilator
  - There are several commonly used drug infusions for maintaining intubation of small animals on ventilators.
    - Morphin CRI 0.1-0.2 mg/kg/hr + Midazolam CRI 0.1 to 0.5mg/kg/hr
    - Fentanyl CRI 6ug/kg/hr
    - Propofol CRI 2mg/kg/hr
    - Pentobarbitone CRI 2-4mg/kg/hr
    - Alfaxan CD 6-7mg/kg/hr for dogs 7-8mg/kg/hr for cats
  - Inhalant ananesthesia using ¼ to ½ MAC isoflurane or sevoflurane offers the best method to maintain sufficient sedation for intubation in snake envenomated patients.
    - Common argument for using CRIs rather than gas is that gaseous anaesthesia is comparatively more cardiac depressive. However at the sub MAC doses required for envenomated patients, this argument is spurious.
    - During Tx, the ventilator is switched off every 10-30 mins to check if the patient has regained the ability to spontaneously ventilate.
    - When spontaneous ventilation commences, this usually indicates sufficient antivenom has been administered.
    - After another 1 to 30 mins the (highly insoluble) inhalant is switched off and because of their rapid elimination, return of consciousness occurs within 1-5 mins. The patient can then be extubated and assessed for ability to maintaining a patent airway and adequate ventilation.
    - Pre-place 2 nasal catheters, one for administering O2 the other for ETCO2 measurement.
Anaesthesia for neurological diagnostic procedures

- **CSF tap**
  - If raised ICP, then tonsilar herniation is possible
  - If suspicious of ↑ ICP then
    - Cisternal CSF sampling could be avoided or delayed
    - preTx Mannitol & IPPV
  - Avoid tube kinking
    - Armoured ET tubes
    - Observe respiratory effort and capnography to assess possible obstruction
  - Adequate depth of anaesthesia to prevent movement

Anaesthesia for neurological diagnostic procedures

- **Myelography** (intrathecal = subarachnoid administration)
  - During administration → rapid big ↑ ICP → ↓ CNS blood flow, bradycardia, hyper & hypotension, apnoea (the Cushing triad), arrhythmias, and cardiac arrest
  - PreTx with Atropine
  - Administer slowly
    - Try to obtain a CSF sample initially to lower CSF volume
    - CT myelography requires only 25-50% dye volume required for radiographic myelography
  - Dye may travel into brain basal subarachnoid space and ventricular system → meningitis & seizures
    - Elevate head
    - Slow injection
    - Assume seizure will occur and Tx with diazepam and/or phenobarb on recovery
Other uses for Ketamine in Neurological disease

- MAC sparing
- Neuroprotective
  - Provided eucapnia $\rightarrow$ ↓ ICP and ↑ CBF without ↓ BP or CPP
  - Review of 276 papers no evidence of harm as induction agent and preferred because of advantages in haemodynamically compromised patients
  - ↓ reduction of damaging spreading depolarisations in injured brains
  - ↓ neuronal injury after cerebral ischemia by blocking the excitotoxic effects of glutamate but high doses ↓ ischemia-induced increase in neurogenesis
  - Chronic use $\rightarrow$ schizophrenia in mice
- Treatment of depression
  - Increases glutamine levels (as NMDA receptors blocked)
  - $\rightarrow$ ↑ brain derived neutrotrophic factor
  - $\rightarrow$ ↑ dendritic sprouting & connections (permanent)
- Acute pain
- Chronic pain
- Status epilepticus (man)
- Epidural analgesia

Artificial ventilation

- For full presentation on artificial ventilation please go to www.stah.net.au and select the tab labelled “For Veterinarians”
Artificial ventilation

• A practical guide

adverse effects of IPPV

• reduction of blood flow to right atrium
• increase in pulmonary input impedance
• reduced capacitance of pulmonary bed
adverse effects of IPPV

Try not to exceed a maximum inspiratory pressure of 20cm H₂O

Unless the airway pressure is measured with a manometer, you don’t know!

adverse effects of IPPV

circulatory effects minimised by:

• increase in tone of capacitance vessels
  – minimise depression of the sympathetic nervous system
  – adequate circulating volume. IV fluids are essential.

• minimise mean airway pressure
  – Inspiratory to expiratory time ratio I:E ratio < 1:2
  – abolition of thoracic & abdominal m. tone
  – Maximum inspiratory pressure of 20cm H₂O

• Maximise inspiratory flow rate
  – “fast” & “slow” alveoli means this requires compromise

• eucapnia.
  – ETCO₂ 30-40mm Hg dogs
  – ETCO₂ 20-30mm Hg cats
adverse effects of IPPV

barotrauma

• High inspiratory pressures can lead to pneumothorax or pneumomediastinum

• closed chest: damage with inflation pressures > 70 cm H$_2$O

• open chest: damage with inflation pressures > 40 cm H$_2$O

Measure peak inspiratory pressure

Circle absorber

Bain
Choices of ventilator

1. Direct patient ventilator
2. Anaesthetic machine ventilator
3. Using a direct patient ventilator to run as an anaesthetic machine ventilator
   1. Bag in the bottle (2 separate circuits)
   2. Connect ventilator by a long 22mm corrugated tubing to the connection site of the rebreathing bag in the anaesthetic machine’s patient breathing circuit (circle, Bain or T piece).
      • Gas from the ventilator and gas from the anaesthetic circuit serge backwards and forwards in the connection tubing which needs to be long enough to contain an equivalent volume equal to 2-3x the tidal volume to prevent excessive amounts of ventilator gas entering the anaesthetic machine patient breathing system (PBS)
      • This ≈ 10cm of 22mm corrugated tubing per kg of patient’s weight

Direct patient ventilator
Anaesthetic machine ventilator

Remember to close the patient breathing circuit pop-off valve before starting ventilator

Using direct patient ventilator as an anaesthetic machine ventilator

Method 2: Connect ventilator’s breathing circuit to rebreathing bag port of the anaesthetic machine’s patient breathing system using 10cm corrugated 22mm tubing per kg
Artificial ventilation: Monitor!

Brain death
Defining brain death in small animals (Schedule used for kids > 1 year old)

- Absence of all brain and brainstem function
  - Comatose: no purposeful response to any stimulus
  - Brainstem function is absent when:
    - Pupils are mid-position and do not react to light
    - Eyes do not blink when touched (corneal reflex)
    - Eyes do not rotate in the socket when the head is moved from side to side (oculo-cephalic reflex).
    - Eyes do not move when ice water is placed in the ear canal (oculo-vestibular reflex)
    - Child does not cough or gag when a suction tube is placed deep into the breathing tube
    - Child does not breathe when taken off the ventilator
- Repeat in ~6 hours