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ANAEThESIA PROBLEM RECOGNITION AND MANAGEMENT: CAN WE LEARN FROM AIRLINE PILOTS?

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Anesthesia began in the mid 19th century from people experimenting with inhalation of nitrous oxide (N₂O) or ether at parties and public inhalations for "ladies and gentlemen". Veterinary anesthesia using N₂O was attempted (unsuccessfully) by Sir Humphrey Davy and his students conducting narcosis experiments on dogs and chickens 25 years before the discovery of human anesthesia. The first successful administration of general anesthesia was credited to Dr Crawford Long of Jefferson County, Georgia, USA in March 1842 using ether. Dr John Snow similarly used chloroform in the UK to provide pain relief during child birth including for Queen Victoria. The volatile anesthetics were administered via gauze soaked with the agent, applied to the patients' face. This crude equipment as well as lack of maintenance of an airway, no use of supplemental oxygen and minimal monitoring resulted in high levels of patient mortality. Currently mortality of anesthestia in healthy humans is over 1:100,000 cases.

The high risk of mortality in veterinary anesthesia

Historical changes to veterinary dog and cat anesthesia mortality data for the USA are shown in the table above. In 1955 the mortality rate was high, over one dog or cat per 100 anesthetics. Most animals were anesthetised with pentobarbitone and not intubated or given high inspired levels of oxygen. The mortality rate was halved by 1980, with the widespread use of inhalation anesthesia, in part because patients were intubated, administered 50% to 95% oxygen and could be ventilated. Problems that lead to anesthetic deaths were recognised with minimal monitoring equipment and included apnoea, airway or breathing problems, irregular pulse or heart rate (dysrhythmias), circulatory collapse and severe hypothermia.

Subsequent improvements to mortality data (1990’s – 1 death per 300 anesthetics) are of a smaller magnitude but are attributable to better, continuous monitoring, better problem recognition and better problem management. Most critical problems during anesthesia are either caused by, or lead to tissue hypoxia. Newer anesthetic agents and analgesics have resulted in better perfusion, oxygenation and anesthesia recovery. Recovery from anesthesia accounts for up to 50% of all pet animal anesthesia deaths. Causes of deaths in recovery are not well documented but hypothermia is considered to be one important factor.

Many risk factors underly the high rates of veterinary anesthesia mortality.

<table>
<thead>
<tr>
<th>Period</th>
<th>Dog mortality</th>
<th>Cat mortality</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>1955 – 1957</td>
<td>1.1%</td>
<td>1.8%</td>
<td>USA, Lumb &amp; Jones</td>
</tr>
<tr>
<td>1979 – 1981</td>
<td>0.43%</td>
<td>0.25%</td>
<td>USA, Lumb &amp; Jones</td>
</tr>
<tr>
<td>1990</td>
<td>0.23%</td>
<td>0.29%</td>
<td>UK, Clarke &amp; Hall</td>
</tr>
<tr>
<td>1994</td>
<td>0.23%</td>
<td>0.3%</td>
<td>USA, Gaynor &amp; Dunlop</td>
</tr>
</tbody>
</table>

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Lack of specific parameters and devices for assessment of pain and the response to analgesic therapy in animals

Pain assessment is subjective in animals and human infants. Current monitored parameters (e.g. heart rate, respiratory rate, body temperature) do not clearly indicate the presence or absence of pain.

Inadequate & inaccurate handwritten, graphical anaesthesia record keeping which has implications for patient care, staff and legal obligations.

Recording during anaesthesia is complex, time consuming, stressful and often performed by nurses with minimal training who have multiple tasks to perform.

Difficulty obtaining specialist advice, either in an emergency on a remote situation.

This is a particular problem in small or rural veterinary hospitals. Internet technology could permit remote patient assessment & conferencing.

Inability to provide remote (& continuous 24 hour) monitoring of critical patients

Internet technology and digital monitor communications could enable “off-site” patient monitoring.

Lack of veterinary anaesthesia morbidity & mortality data from general practice

In human anaesthesia, better identification and understanding of problems has lead to development of better monitors and systematic problem management.

Anaesthesia Preparation, Planning, & Set-up

Knowledge, equipment, training
Experience & teamwork
Patient assessment incl. medical problems
Considerations for anaesthesia
Specific conditions – e.g. geriatric, heart disease
Plan for anaesthesia and support/monitoring
Set up & check equipment

Anaesthetic induction & start inhalation GA.
Confidence & experience
An organized, planned process
Efficient – preparation, planning & organisation
Procedures: technical skills & knowledge
Checks, check lists & communications
Critical timing: induction/transition to maintenance
Half intra-anaesthetic deaths occur in 1st 15 min.

Anaesthesia Monitoring
Time
Accumulation of data: observation & use of equipment
Document (graphical)
Recognise changes / trends

Response to abnormal flight situations
Recognition / awareness
Communication
Importance vs Urgency
Agreement
Assessment & communications

Anaesthesia Problem Recognition & Problem Management
Methodical approach
Check lists
Communication
Assessment

Systematic Problem Management
Planning: expected & unexpected
Management
Training & simulation
Discipline
Responsive & upward communications

Airline pilot training in systematic approach and methodical problem recognition, management and communications has resulted in an impressive safety record. There are many similarities between both anaesthesia & aircraft flight and training of anaesthetists and pilots. Human anaesthesia has adopted many of the airline industries systematic approach and problem management methods as well as the use of simulators for training.

This presentation will conclude with a suggested basis for a systematic, methodical approach to veterinary anaesthesia problem recognition and management based on principles used in the airline industry.
ANAESTHETIA OF GERIATRIC PATIENTS WITH MITRAL INSUFFICIENCY
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Physiological changes with Age

Cardiovascular system function decreases with age due to a decline in cardiac response to sympathetic nervous system stimulation and a rise in peripheral vascular resistance due to thickening of elastic fibres in the walls of large arteries. The result is increased blood pressure, a reduction in cardiac output and a reduction in vascular volume. Stroke volume tends to become “fixed” so increasing cardiac output such as with exercise is dependent on increasing heart rate.

Respiratory function - There is loss of strength of the muscles of respiration and a decrease in elastic recoil of the chest in part due to ossification of rib cartilage. These changes increase the resting volume of the thoracic cage, which predisposes older (recumbent) animals to atelectasis.

Central nervous system - There is a reduction in brain weight with age due to a loss of individual cells and increased breakdown/decreased production of neurotransmitters. The anaesthetic drug requirement decreases with age = reduced sedative and anaesthetic drug doses.

Hepatic function - There is an age-related increase in BSP retention in part due to a decrease in liver blood flow. Drugs dependent on liver metabolism and biliary excretion for their elimination have a prolonged plasma half life in aged patients. Elevated liver enzymes don’t necessarily equate to liver disease (ALT, AST & GLDH – cellular leakage; ALP & GGT – cholestasis, ALP isoenzyme - steroid treatment). The magnitude of elevation is NOT related to the degree of liver cellular change (need bilirubin or bile acid levels). Clinical signs of liver disease include inappetance, weight loss, vomiting, diarrhoea, icterus, bleeding, ascites, PU/PD & abnormal behaviour. Check serum protein, albumen and blood glucose in cases of liver dysfunction pre-anaesthesia.

Renal function – Age is associated with reduction of cortical renal mass with up to 50% loss of glomeruli and tubular atrophy, particularly of juxta-glomerular nephrons. GFR decreases by up to 50% but this is also due to a reduction in renal blood flow. Clearance of creatinine, urea and inulin is prolonged with age, although plasma levels of creatine and urea may change imperceptibly as a result of proportional decreases in production. Therefore, geriatric patients have a reduced renal reserve, so are less tolerant of dehydration or fluid overload, and have prolonged drug elimination. Animals with “slight” elevations of urea and creatinine may have lost up to 2/3 of their glomerulo-tubular function.

Pharmacodynamic Changes with Age - Plasma drug levels are elevated and plasma half life is prolonged, due to reductions in blood volume, renal and hepatic function. Accompanying the decrease in blood volume is a reduction in albumin mass. Therefore, plasma protein binding of drugs is reduced, resulting in higher levels of unbound (active) drugs. Receptor numbers present in a given tissue (eg, alpha and beta adrenergic receptors) decline with age. These changes suggest that drug doses and dosage intervals may need to be reduced, especially with chronic therapy.

Cardiac Disease due to Depressed Myocardial Contractility

Many older dogs, particularly the smaller breeds, have low grade mitral murmurs commonly associated with mitral insufficiency. Such dogs who have no history or signs of cardiac disease can be administered sedation and general anaesthesia without significant risk, similar to other older patients. However dogs with mitral murmurs and evidence of cardiac disease should be carefully evaluated prior to GA.

Valvular heart disease, most commonly mitral insufficiency, is characterised by valvular regurgitation, ventricular hypertrophy and dilation, with pulmonary distension that can lead to right sided congestive heart failure. Avoid sedation or anaesthesia of patients with frequent bouts of syncope or those with pre-existing interstitial lung oedema who have respiratory distress and productive coughing with a conscious SpO2 below 88%. These patients require medical management to improve cardiac function and decrease the level of oedema prior to general anaesthesia.

Dilated Cardiomyopathy is commonly seen in giant breeds of dogs. Poor myocardial function reduces contractility and stroke volume, which leads to atrial and ventricular dilation, valvular regurgitation and dysrhythmias. Avoid sedation or anaesthesia of patients...
with marked ventricular dysrhythmias, an irregular pulse and/or pulse deficits or frequent bouts of syncope.

**Anaesthesia Management Techniques:**

1. Get the resting heart rate of the animal pre-anaesthesia and plan to keep the rate during anaesthesia +/- 10% of this value. Avoid causing tachycardia (reduces myocardial oxygenation) or bradycardia (decreases cardiac output).
2. Minimise stress or painful stimulation which leads to catecholamine release, increased afterload and decreased cardiac output. Sedation with opioid drugs, particularly morphine will reduce stress and may reduce pulmonary vascular resistance.
3. Administer pre-GA sedatives via subcutaneous (SC) or intramuscular (IM) routes rather than intravenously to reduce the “peak” cardiovascular effects and gain longer duration of sedation.
4. Provide oxygen by face mask for 3 to 5 minutes to any animal with signs of cardiac dysfunction prior to induction of general anaesthesia – this is the period where myocardial hypoxia will occur.
5. Sedative and anaesthetic induction drugs causing vasodilation are generally better than those causing vasoconstriction (increases afterload/decreases cardiac output)
6. Heart disease reduces blood flow, so use lower doses of IV anaesthetic induction drugs and administer them slowly (over 30 to 60 sec).
7. Limit the administration of intravenous crystalloid fluids during anaesthesia to 5 ml/kg/hr. Avoid administration of additional IV fluid boluses to treat hypotension during anaesthesia.
8. Do less, not more in patients with pre-existing cardiac distress - there is no emergency cure for cardiac failure!

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**BLOOD PRESSURE IN CATS AND DOGS: WHEN IS IT ACCURATE AND HOW DO YOU TROUBLE SHOOT SUSPECT MEASUREMENTS.**

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Systemic arterial blood pressure (BP) monitoring is widely used during anaesthesia, and for assessing conscious animals suspected of being hypertensive. In both cases accurate measurement of blood pressure is important to permit useful clinical application.

The blood pressure (BP) waveform is like a sine wave, having an upstroke (systolic or ejection phase, peak = SAP) and a downstroke (diastolic or filling phase, bottom = DAP). Mean arterial pressure (MAP) is calculated from the area under the BP curve. More time is spent in the diastolic phase so the area under the diastolic curve is approximately twice the area under the systolic curve. Therefore diastole has the greater influence on MAP.

\[
MAP = \frac{DAP}{1/3} (SAP – DAP) \\
MAP = \frac{(2xDAP + SAP)}{3}
\]

Blood flow is positively correlated with mucous membrane colour (high flow = pink) and is inversely related to arterial blood pressure (high BP = low flow = pale). Therefore we should assess mucous membrane colour when monitoring arterial blood pressure.

**Clinical BP monitors fall into 2 categories:**

1. **Invasive, direct monitors** that use an arterial catheter connected via a transducer to measure systolic, diastolic, and mean BP
2. **Non-invasive indirect blood pressure (NIBP) monitors** that use a pneumatic cuff placed around a limb or the tail to estimate pulse rate, systolic and in some cases, diastolic and mean BP. There are two commonly used types of blood pressure monitor:
   - Doppler ultrasonic devices (Doppler) that use a blood flow transducer placed on a distal limb artery to indicate return of blood flow when the pneumatic cuff is deflated manually. Dopplers are most accurate at detecting systolic BP and consistently work in conscious cats.
   - Automated oscillometric (Auto-NIBP) devices that typically detect pulsations in the pneumatic cuff when blood flow returns to the limb as the cuff is automatically deflated. These devices work best when applied to the upper arm of adult humans (cuff volume 100 to 250 mls) with a limb blood volume of 150 mls and an increase of 10 to 20 mls with systole. The typical error of measurement is 15%. Using neonatal cuffs (volume 10 to 20 mls) on the hair-covered, distal limbs of dogs
and cats the error of measurement increases up to 25%. In general, oscillometric devices must detect the pulse rate in order to accurately determine NIBP. Most technology detects mean pressure oscillations (mean BP is therefore the most accurate value) then derives systolic and diastolic pressure.

**Errors of measurement attributable to monitors**

- Doppler – inability to get a clear blood flow signal or variable signal volume as the pneumatic cuff is deflated causing subjectivity as to the correct reading point
- Doppler – feedback either from another “ultrasound” device or from the monitor being too close to the sensor (a microphone) causes “static” or reverberating noise
- Doppler – frequency response is “too slow” to detect the small amounts of blood flow at the peak of systole in small animals, thus “missing” the true systolic sound.
- Doppler – damage to the sensor either from moisture or formation of an air-bubble between the ultrasound crystal and its epoxy coating result in damping or failure to detect the signal
- Auto-NIBP cuffs and Doppler sensors should be positioned at the level of the heart (gravity).
- Auto-NIBP - small blood volume changes in the hair covered limb of small animals (e.g. cats) cause small changes to the cuff volume, therefore small changes to pneumatic cuff pressure, which may not be detected by the monitor.
- Auto-NIBP – the automatic monitor’s deflation rate will result in a steeper pneumatic cuff pressure-volume curve for neonatal cuffs, increasing the likelihood of missing the true mean pressure value
- Auto-NIBP – stepwise release of “fixed” amounts of pressure or volume will result in readings tending to be grouped at similar values, distorting the true range of variability of blood pressure in the population.
- Auto-NIBP – the algorhythm used to derive systolic and diastolic pressure use human direct BP data. Accurate calibration studies using direct BP data from a large sample of the dog or cat population don’t exist to validate the methodology.

**Errors of measurement attributable to pneumatic cuffs and their application sites**

- We use a cuff bladder width that is 40% of limb circumference, validated in adult humans. Similar studies have not been done in animals to prove this ratio is correct. If the cuff width is too narrow readings will be erroneously high and if the cuff width is too wide, readings will be erroneously low.
- Cuff position is classically on the thoracic limb, 1/3 above the carpus towards the elbow. More recent studies show that pneumatic cuffs placed at the base of the tail generally result in more repeatable and accurate measurements, followed by pneumatic cuffs placed above the hock.
- The occlusive part of the bladder should be placed directly over the artery to be occluded
- Clipping off some hair will help cuff contact and reduce signal damping in animals caused by long or thick hair coats
- Short, bent legs alter the geometric relationship between cuff and limb.
- Thick hair damps the transfer of pressure from the artery to the cuff.
- Motion at the measuring site will generally affect any NIBP results
- Leaks in pneumatic cuffs are caused by holes in the cuff, cuff tubing or cracks in the plastic, luer connectors. Neonatal cuffs are intended for single use!
- Severe hypotension (MAP<40 mmHg) is associated with increased measurement error
- Deflate the cuff between readings or it will cause pain & then BP will increase
- In anaesthetized cats, NIBP devices tend to underestimate SAP
- In conscious cats NIBP monitors tend to overestimate the BP
- Stress resulting in increased muscle tone will affect the ability to compress the artery, commonly resulting in over-estimation of BP. Hospitalising animals and making repeated readings during the day will reduce this cause of error.
- Hypertension is usually not constant so a number of readings taken over a period of time will result in more accurate measurement and clinical assessment.

**HEART RATE IN ANAESTHESIA – WHAT DOES IT REALLY MEAN?**

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Anaesthetic depth and cardiopulmonary function are difficult to assess based on physical signs. Heart rate
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CHAPTER 2

Scientific proceedings: companion animals programme

AnESTHESiology

during anaesthesia is easily measured, but commonly poorly interpreted. Inherently we want to believe that it will give an indication about anaesthetic depth, with high or rising heart rates suggesting light anaesthesia and low or slowing heart rates indicating deep anaesthesia. However absolute heart and respiratory rates don’t correlate with anaesthetic depth.

Heart rate may increase with increasing catecholamine levels (e.g. pain, moderate hypothermia) but may decrease with marked vagal stimulation (e.g. laryngeal stimulation, ocular pain, distention of the stomach, traction of the mesentery) and extreme hypothermia. Some sedative or anaesthetic drugs cause bradycardia (e.g. alpha-2 agonists, opioids and the inhalation agents) and some cause tachycardia (e.g. thiobarbiturates, ketamine).

The incidence of complications associated with general anaesthesia show that hypothermia occurs in up to 85% of small animals. Between 10 and 15% of anaesthetized animals have complications during anaesthesia that require intervention with bradycardia and hypotension separately accounting for between 15 and 20% of these. This study from Colorado State University also showed that bradycardia was more common in dogs compared to cats, and most commonly associated with abdominal or spinal surgery.

<table>
<thead>
<tr>
<th>COMPLICATIONS</th>
<th>DOGS</th>
<th>CATS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Hypotension SAP &lt; 80 mm Hg</td>
<td>179</td>
<td>58</td>
</tr>
<tr>
<td>2. Dysrhythmias</td>
<td>64</td>
<td>12</td>
</tr>
<tr>
<td>3. Blood loss &gt; 10% &amp; transfusion</td>
<td>16</td>
<td>-</td>
</tr>
<tr>
<td>4. Blood loss &gt; 10%, NO transfusion</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>5. Low PCV or Protein &amp; transfusion</td>
<td>15</td>
<td>-</td>
</tr>
<tr>
<td>6. Hypoventilation, CO2 &gt; 55 mm Hg</td>
<td>33</td>
<td>1</td>
</tr>
<tr>
<td>7. Hypoxemia, PaO2 &lt; 70 mm Hg</td>
<td>14</td>
<td>-</td>
</tr>
<tr>
<td>SaO2 &lt; 85%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. CPR (resulted in 8 deaths)</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td>10. Deaths in recovery</td>
<td>3</td>
<td>-</td>
</tr>
<tr>
<td>TOTAL COMPLICATIONS</td>
<td>12%</td>
<td>10.5%</td>
</tr>
</tbody>
</table>

Gaynor JS, Dunlop CI, Wagner AE: Complications and mortality associated with anaesthesia in dogs and cats. JAAHA 35:13-17;1999

In a retrospective study at a referral hospital in Sydney, of 60 dogs anaesthetized for acute spinal disc prolapsed requiring anaesthesia for imaging studies and surgery, more than half required treatment for bradycardia during anaesthesia. There was some suggestion that treatment was more likely to be required in animals that were more painful.

Problem Management: Bradycardia

Marked bradycardia results in decreased blood flow and perfusion (Flow = HRate x Stroke Vol.) Very slow heart rates can lead to myocardial hypoxia and asystole, although severe hypoxia initially causes tachycardia.

1. Define the problem where possible in measurable terms
   • In larger dogs, less than 50-60 beats per minute
   • In smaller dogs, less than 70-90 beats per minute
   • In cats, less than 80 to 100 beats per minute
   • A more useful method is to determine the heart rate of each animal, non-stressed, at rest, prior to anaesthesia. Heart rates 20 to 30% less than the normal resting rate would defined as bradycardia.

2. Recognize the problem
   • slow heart or pulse rate, irregular pulse rate +/- pulse deficits
   • pale or muddy mm’s with profound bradycardia & reduced blood flow

3. Best Clinical Assessment
   • continuous pulse monitors (eg pulse oximeter or Doppler monitor)
   • ECG (measures electrical activity, not mechanical heart beats)

4. What are the Causes
   • increased parasympathetic (vagal) activity with painful stimuli
   • inadequate anaesthetic depth (most usually inadequate anaesthesia)
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- specific anaesthetic drugs eg opioids, inhalation anaesthetics
- hypothermia

5. First Response – WHAT SHOULD YOU DO RIGHT NOW?
- check patients pulse rate
- check that the patient is breathing
- check the mucous membrane colour and administer oxygen if pale or muddy

6. Treatment
I. Cease stimuli causing vagal responses
II. Evaluate depth of anaesthesia
   - if painful stimuli are the underlying cause, then consider additional analgesia
   - consider administering a “test” dose of an analgesic drug e.g. IV opioid
III. Administer parasympatholytics such as atropine
   - atropine 0.01 to 0.02 mg/kg IV (often repeated)
   - followed by atropine 0.02 to 0.04 mg/kg SQ
   Note: administration of small doses of atropine IV occasionally can exacerbate bradycardia. In such cases the response should be to administer further doses of IV atropine, typically in 0.01 to 0.02 mg/kg increments until an increase in heart rate is observed. Usually 30 to 60 sec. should be allowed between atropine doses unless the bradycardia is severe.
IV. Administer chronotropic drugs
   - if non-responsive to atropine consider administration of catecholamines
   - reverse drug induced bradycardia

Hypothermia occurs in up to 85% of anaesthetized small animal patients, resulting in body temperatures commonly below 34°C. This causes prolonged and poor quality recoveries and may contribute to mortality. Hypothermia during anaesthesia can be prevented.

Greater heat loss occurs in smaller animals
Heat loss or gain occurs in an exponential manner (Body Weight 0.75) with its greatest impact on small patients in part because of their large body surface area relative to body mass. Hypothermia occurs in anaesthetized cats, small dogs and foals less than 6 weeks of age, even when positioned on circulating warm water heating pads unless the surrounding air is also warmed.

Thermoregulation & Anaesthesia
Anaesthesia depresses CNS thermoregulation and prevents usual methods of conserving heat such as seeking a warm environment, body positioning, hair coat erection, peripheral vasoconstriction or generating heat by shivering. Heat loss during surgery is exacerbated by clipping hair from surgery sites., using cold or evaporative skin prep solutions, wetting and flattening the hair coat and opening body cavities.

Problems with hypothermia include:
- CNS depression which reduces the requirement for anaesthetic drugs so patients appear “deeper”
- lower tissue blood flow alters drug distribution, metabolism and excretion, prolonging recovery
- peripheral vasoconstriction decreases surface heating efficiency

Preventing hypothermia during surgery traditionally relied on skin surface contact heating from hot water filled bottles, circulating warm water blankets and electric heating pads placed under the animal or under the surgery table top. IV fluid has been warmed by placing IV fluid lines in dishes of warm water.

Calories and warming IV fluids
A calorie (cal) is the amount of heat required to raise 1 ml (or 1 gm) of H₂O 1°C. The specific heat of animal tissue is 0.83 cal/gm. Therefore a 10 kg dog requires 8,300 cal (8.3 kcal) to raise its temperature 1°C.

Warming IV fluid administered during surgery:
A 10 kg dog administered IV fluid at 10 ml/kg/hr = 100 ml/hr.
If the fluid is warmed to 44°C and the dog is 34°C, then we can deliver: (44-34°C =) 10°C x 100 ml/hr = 1000 cal/hr

To Warm the 10 kg at 34 °C dog to 37 °C
The dog needs:

IS HEAT LOSS IN SMALL ANIMAL ANAESTHESIA REALLY A PROBLEM? WHY DOES IT OCCUR AND CAN WE PREVENT IT?
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Hypothermia occurs in up to 85% of anaesthetized small animal patients, resulting in body temperatures
(37-34 =) 3°C x 8,300 cal = 25,000 cal (approx) / 1000 cal/hr (from the IV fluid)
= 25 hours!

Warming IV fluid may prevent cold fluid exacerbating heat loss but is not effective for warming severely hypothermic animals.

Respiratory heat loss due to humidification is significant
During inspiration the nose and pharyngeal mucosa transfer heat and moisture to the air which is largely recovered during expiration, thus conserving heat. Air has a low heat capacity (0.24 cal/gm) and a low weight (1.3 gm/l). Saturated air holds 44 mg H₂O/L at 37°C which requires 24 calories. A 10 kg dog taking 20 x 100ml breaths/min ventilates 120 L/hr so requires (24 cal/L x 120 L/hr) = 2880 cal/hr for humidification. Intubation inhibits heat/moisture conservation via the nose, resulting in body temperature loss of about 1/3 °C/hr.

Thermal Burns
Thermal injury to skin is an exponential relationship between source temperature and contact time. Burns do occur at temperatures below 50°C and are still commonly seen. Hot tap water may reach 60°C and 10 seconds of skin contact would result in epidermal necrosis. In 2008 the UK Veterinary Defence Society reported a high incidence of burns caused by use of warmed wheat bags which can produce similar temperatures.

Electric heating pads usually have a low thermal mass and cycle on until the thermostat reaches it’s high point, then cycle off until cooling to the low point, then turn back on repeating the process. Simple controllers can be variable in performance or fail, causing higher temperature delivery and potentially burns. These devices should always be insulated from the animal’s skin surface.

New Techniques for Patient Warming
Ideal warming devices deliver constant heat of sufficient “mass” at physiologically safe temperatures using techniques that permit effective heat transfer to the patient.

IV fluid line heaters
Electric fluid warmers provide constant, controlled heating of IV fluids which is more reliable than placing IV fluid lines in dishes of warm water. The IV line simply fits into a thermal track and a hinged “door” closes to hold it in place. These IV line warmers are better at helping prevent further heat loss rather than “warming” hypothermic patients (see above).

Inspired air warming & humidification
Electric heated humidifiers can effectively warm animals but are large, costly and not well designed for non-rebreathing circuits. Artificial noses connected to the ET tube adapter are potentially useful but increase dead space. Miniature circle breathing systems designed for small animals can warm and humidify the re-circulated gas because removal of CO₂ by absorber is an exothermic reaction resulting in inspired air warming and humidification. Heated circle systems will further improve this method of patient warming.

Warm air heating blankets
A constant flow of warm air at controlled, physiologic temperatures is delivered to a blanket which distributes the heat widely to the animal’s surface. Blankets designed for people generally lie over the patient. Animals have a hair-coat which differs from people, so heating is more effective if applied from underneath so warm air rises into the hair coat where it is trapped, forming a warm layer. Typical human warm air blankets are manufactured by punching 1 to 2 mm holes in the surface layer. This can result in high air flow “jetting” through these holes, causing a wind tunnel effect which can cool small animals. Some blankets have a porous surface, resulting in minimal surface air flow, less of a wind tunnel effect and less likelihood of contaminating the air with micro-organisms.

Warming animals in recovery is slow, consumes nursing time and is a cause of thermal injuries. Some warm air systems are designed specifically to warm animals in cages, with adapters to duct warm air through metal door grills and blankets designed to fit cat or dog cages and still permit rapid access to the animal in an emergency.
for airway obstruction, potential for aspiration of muco-purulent nasal discharge or haemorrhage and to facilitate examination or biopsy.

- Prior to ending the procedure examine the pharynx and laryngeal opening with a laryngoscope for the presence of fluid, blood clots or tissue pieces which should be removed. The endo-tracheal tube cuff should remain inflated until the point of extubation. Obstruction of the nasal airway may cause the animal to mouth breathe which can be a problem early in anaesthesia recovery when the animal is not fully conscious. The patient should be placed in a recovery cage and be observed continuously once extubated, until sternal and breathing without distress.

Sedation:
- Consider using analgesics to reduce response to sensory stimulation caused by scoping or biopsy device entering the nasal passage. Opioid agonists such as morphine, fentanyl and buprenorphine can be administered subcutaneously 15 to 30 minutes prior to stimulation or anaesthesia induction. Topical 2% lignocaine at the time of stimulation will help.
- Acepromazine can be used in young healthy animals to increase sedation, but it should be avoided where a biopsy of a nasal tumour is anticipated because it increases haemorrhage which can be difficult to stop due to inability to access.
- If additional sedation is required in such cases, consider using a benzodiazepine such as diazepam or midazolam

Anaesthesia Induction & Maintenance:
- Generally a rapid induction is desirable, but more importantly a rapid and smooth recovery to minimise the period that the animal is extubated and unaware of its need to mouth breathe.
- Consider O2 by face mask if airway obstruction is severe or ensure that the animal can mouth breathe. Frequently a supplemental dose of the parenteral anaesthetic will be required to reduce the patient’s response to stimulation, typically dosed at about ¼ of the induction dose.

Monitoring:
Because these procedures are usually short, I prefer to use a pulse oximeter because it continuously monitors the pulse and also gives an indication of the adequacy of oxygenation. The probe can be placed on the ear, a foot or above the hock instead of the tongue. The pulse oximeter can also be used in recovery, particularly if the patient is not continuously observed.

Laryngeal Or Tracheal Obstruction
Includes animals with loss of laryngeal function (including ixodes holocyclus “tick” paralysis), brachycephalic syndrome, presence of foreign bodies and tumours.

Considerations:
- Maintain airflow during anaesthesia or anaesthesia recovery when the patient is not intubated. Animals cannot be intubated for a laryngeal function exam.
- Consider causes of this problem. If the cause is a poly-neuropathy, the animal may also have a mega-oesophagus which can result in aspiration pneumonia. A retrospective study of dogs with mega-oesophagus at Colorado State University showed a 50% incidence of aspiration pneumonia following general anaesthesia and approximately half these animals subsequently died. Avoid profound sedation or anaesthesia in animals with mega-oesophagus.
- Examination of laryngeal function can only be observed during light anaesthesia. Even for experienced veterinarians it can be difficult to be confident that an animal has a paralysed larynx. Lack of movement may be associated with the depth of anaesthesia or the anaesthetic drugs themselves, particularly propofol. During coughing, the arytenoid cartilages should abduct.
- Following sedation, these patients should be under observation because additional muscle relaxation can lead to complete airway obstruction. If this does occur, administer some parenteral anaesthetic and intubate the patient, then allow the animal to remain intubated for as long as possible. Place the animal in a recovery cage while it is still fully anaesthetised, “untie” the endotracheal tube and deflate the pilot balloon & cuff, then don’t disturb the patient.

Sedation:
Because many drugs can affect laryngeal function, it may be desirable to avoid administration of premedication. Keep premedicated animals under observation.

Anaesthesia Induction & Maintenance:
- Generally a rapid induction is desirable, but more importantly a rapid and smooth recovery to minimise the period that the animal is extubated and unaware of its need to mouth breathe.
- Thiopentone is the anaesthetic induction agent that will best allow assessment of laryngeal function. Use a low dose, sufficient to allow the mouth to be opened. Subsequent smaller doses (¼ of the induction dose) may be required. Once the examination is finished, intubate the patient, administer oxygen if the animal appears to be hypoxic, then place it in a
recovery cage and observe until extubated, sternal and breathing without distress.

Pulmonary Dysfunction

Considerations:
- Don’t anaesthetize animals with lung disease without very good reasons! These might include placement of chest drains, bronchoscopy or foreign body removal.
- Properly examine the patient for underlying heart disease or aspiration pneumonia.
- These animals will benefit by administration of oxygen by face mask for 5 minutes prior to induction of anaesthesia. For circle systems flow 30 to 100 ml O2/kg/min and for non-rebreathing systems flow 200 ml O2/kg/min. Animals that resist restraint or are distressed may be hypoxic.
- Mild sedation may help to relax these animals, which improves oxygenation.
- If tracheal collapse is suspected, a smaller diameter, long (new) endotracheal tube should be available. If airway obstruction occurs during anaesthesia, this tube can be carefully passed down to the thoracic inlet, and if needed, to the bronchial bifurcation. Once the obstructed airway is passed, airflow will occur.
- Bronchoscopy of smaller animals will necessitate that they are not intubated, which causes two problems. Firstly, administration of additional parenteral anaesthetic will be required and secondly the bronchoscope obstructs their airway making ventilation difficult. The scope should never fully occlude the airway and insufflate oxygen at approx. 60 – 100 ml O2/kg/min. If this is not practical, then pass the scope and work for a short period (3 to 5 minutes) then remove the scope and administer O2 either via face mask or endotracheal tube.

Recovery from anaesthesia can lead to hypoxia in animals with poor respiratory function, so continuous monitoring with a pulse oximeter, administration of O2 and positioning in sternal recumbency should be considered. A rapid recovery from anaesthesia is also desirable.