BREED CONSIDERATIONS AND MYTHS AND MISCONCEPTIONS IN SMALL ANIMAL ANESTHESIA
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Key Points

- There are few “true” breed sensitivities to anesthetics
- Breed idiosyncrasies to various anesthetic, sedative, and analgesic drugs should be considered when formulating an anesthetic plan
- Many myths in small animal anesthesia derive from mis-interpretation of data or singular experiences that evolve into “scientific fact”
- This lecture will attempt to dispel some common myths and misconceptions in small animal anesthesia as well as discuss breed considerations in anesthesia

Some material from this lecture is taken from a publication by Wagner et al in 2003, which is referenced at the end of these proceedings notes.

Misconception - Certain Dog Breeds are Sensitive to Anesthesia:

Although many dog breed publications will report anesthetic sensitivity to certain drugs (e.g. ketamine, acepromazine) or to anesthesia in general, there are very few scientifically documented breed sensitivities to anesthesia. When owners or dog breeders present their animal and express concern about perceived anesthetic risk based on the animal’s breed, it is important for the veterinarian to educate them to the general risks of anesthesia in all breeds/species and to reassure the client that the particular breed of the animal will be considered but that it is unlikely that there is a heightened risk based on the breed.

The one known and documented drug sensitivity based on breed is that of greyhounds having delayed recoveries after thiobarbiturates (thiopental). This delayed recovery is due in part to the typically lean body type of these dogs such that there is little adipose tissue to which the drug can redistribute; however, the primary reason for the delayed recovery is that this breed metabolizes the drug via a different hepatic pathway and drug metabolism occurs much more slowly. Most anesthesiologists extend this breed sensitivity to other sighthound breeds as well (whippets, Italian greyhounds, borzois, afgan hounds) even though these breeds have not been specifically studied. Since thiopental is no longer commercially available in the USA, this breed/drug sensitivity is of little clinical importance today.

Boxers with English genetic lines may be more sensitive to the hypotensive effects of acepromazine. In these individuals, acepromazine appears to dramatically vasodilate them. Boxers that come from American genetics do not appear to have this sensitivity. When presented with a boxer, if unsure of the genetic line, use acepromazine cautiously or at low doses, and monitor blood pressure throughout the procedure.

Truth - Certain dog breeds present unique anesthetic challenges:

There are certainly dog breeds that present unique anesthetic challenges or considerations. Brachycephalic breeds (bulldogs, boston terriers, Pekinese, etc) usually have the classic 4 anatomic features that can make intubation and airway management challenging: stenotic nares, elongated soft palate, everted laryngeal saccules, hypoplastic trachea. These breeds of dogs have an increased work of breathing due to increased upper airway resistance, and will be prone to hypoventilation and hypoxemia when sedated and breathing room air. These cases need to be monitored closely whenever they are sedated. Intubation can be challenging due to the long, floppy soft palate, large epiglottis,
everted laryngeal saccules and smaller-than-expected trachea. Preoxygenation prior to intubation is warranted, since an increased F\textsubscript{\text{\textsuperscript{o}}\text{\textsubscript{2}}} will improve P\textsubscript{\text{\textsuperscript{\text{a}}}\text{\textsubscript{O}}\text{\textsubscript{2}}}, buying an extra window of time during intubation before the animal becomes hypoxemic. Extubation should only be performed when the animal is able to actively and forcibly “spit out” the tube. Even after extubation, the animal should be monitored, especially if sedatives or opioid analgesics are on board. Brachycephalic breeds also often have increased vagal tone and can become quite bradycardic, so anti-cholingergics should be available if needed to increase heart rate.

Truth – Certain dog breeds respond differently to sedatives and analgesics:

Some general guidelines should be considered when making sedative/analgesic drug choices and calculating dosages in dogs. As a rule, giant breeds of dogs have a smaller body surface area to volume ratio, and, as such, require much lower doses of sedative/analgesic drugs on a mg/kg basis. Conversely, very small dog breeds, or pediatric animals, have a large body surface area to volume ratio, and will require higher doses of sedative/analgesic drugs on a mg/kg basis. Some dog breeds do not sedate well with opioids and benzodiazepines, the Northern Breeds of dogs being the best example. These dogs tend to get very dysphoric and vocal with opioids, so concurrent sedation with alpha-2 agonists may be necessary. Regional analgesic techniques, when they are an option, are a great alternative for pain management in these dogs.

Myth - Pre-anesthetic medication complicates anesthesia by adding too many drugs and will delay recovery:

There are many valid benefits to pre-anesthetic medication. These include sedation and anxiolysis, which decreases the animal’s stress response and the stress of those personnel that have to restrain it, preemptive analgesia prior to a surgical procedure, significant decrease in induction and inhalant dose requirements, a smoother and more consistent anesthetic plane, and a smoother and less painful recovery. Most dogs and cats should receive an IM or IV preanesthetic that includes an opioid +/- sedative. By incorporating premedication, induction dose requirements are reduced by 50% or more, depending on doses used and how sedate the animal becomes, and inhalant requirements similarly reduced by ≥50%. This means that blood pressure, heart rate, and respiratory drive are all better maintained during surgery, because it is high doses of inhalants, not premedications, which depress cardiovascular and respiratory function the most dramatically.

It is true that some premedications (e.g. acepromazine, opioids, alpha-2 agonists if not reversed) may delay recovery, but this is generally a desirable quality. Anesthesia and surgery are exhausting events and a restful recovery, where the animal is kept quiet, warm and comfortable, are necessary components of a successful surgical outcome. If the animal needs to be discharged to its owner at the end of the day, the premedication should be planned such that doses are kept low for drugs that are not reversible, or reversible drugs can be used. The clinician should be mindful, however, of analgesic needs as well and not limit analgesic therapy simply for the sake of a quick recovery.

Misconception - Acepromazine is a dangerous drug:

Acepromazine is commercially available as a 10 mg/ml concentration. Old literature cites acepromazine doses of up to 1.0 mg/kg for dogs and cats. In today’s anesthetic practice, doses 10 – 100 times lower are the “norm”. This means that the old literature citing excessive sedation, prolonged recovery, dangerous hypotension, extra pyramidal signs, seizures etc. are probably largely due to the high doses used in those studies.

Acepromazine does have side-effects, as do most drugs. Besides dose-dependent sedation (which is often desirable), acepromazine causes dose-dependent vasodilation and hypotension. Commercial acepromazine should be diluted into 1.0 mg/ml or 2.0 mg/ml concentrations such that
reasonable volumes of small doses can be drawn up into 1 ml syringes. Doses should be kept low, e.g. 0.005 – 0.05 mg/kg in dogs and cats; rarely does one need to go all the way up to 0.05 mg/kg, particularly if combining with an opioid. Combined with agonist opioids (i.e. not butorphanol or buprenorphine), acepromazine gives reasonable dose-dependent sedation in non-fractious patients. At these doses a significant problem with hypotension is not expected in healthy patients, but monitoring blood pressure in every patient and administration of IV crystalloids to every patient that has anesthesia for more than 30 minutes is recommended. Patients with significant dehydration or volume depletion, or underlying disease that increases their risk of anesthetic-related hypotension, should not receive acepromazine as a premedication. Acepromazine can be very useful in the recovery period for animals with emergence delirium or dysphoria. In these instances, administer a very low dose (0.005 mg/kg) IV and wait 5-10 minutes for the effect. This dose of acepromazine will not excessively sedate the animal but it will calm it down. Because sedation is not excessive, pain assessment can still be possible. Caution should be exercised in using high doses of acepromazine in recovery to “quiet” a patient, as the animal may be expressing significant pain and the sedation produced by acepromazine at these higher doses will make pain interpretation difficult.

Myth - Low doses of alpha-2 agonists have no effect on cardiovascular function:
Dexmedetomidine is the predominant alpha-2 agonist being marketed today in small animal medicine. Most animals will sedate very well with doses ranging from 3 - 10 mcg/kg IM, especially when this drug is combined with an opioid. Even at very low doses (~ 1 mcg/kg), cardiac output will decrease to < 40% of resting values, primarily because of bradycardia but also because of a decrease in contractility. This decrease can persist for up to an hour. Healthy young animals with cardiovascular and systemic organ “reserve” can probably tolerate this decrease in cardiac output, but care should be exercised when using this drug in older or debilitated patients.

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Misconception - Anticholinergics should always be used with general anesthesia:
The primary role of anticholinergics (atropine and glycopyrrolate) is to increase heart rate and dry up salivary secretions. Many drugs used for anesthesia, particularly premedications, reduce heart rate (opioids, alpha-2 agonists). However, in the case of opioids, bradycardia is not accompanied by a decrease in cardiac output because contractility is preserved, thus whole body perfusion is not compromised. In the case of alpha-2 agonists, bradycardia is a cardiovascular reflex response to the increase in afterload caused by peripheral vasoconstriction which is mediated by the alpha-2 agonist.

There are significant disadvantages and considerations when using anticholinergics in the peri-anesthetic period. They can often lead to tachycardia (especially atropine), which increases myocardial work and probably gives the patient the sensation of a “pounding” heart. They dry up mucous secretions, but they also slow mucociliary transport in the trachea and make the secretions viscous and more likely to occlude a small endotracheal tube. In animals which tend to salivate a lot (cats) or when ketamine is used (which increases salivation), clear the oropharynx with a cotton-tipped applicator (cats) or swab it out with 2x2 sponges on a hemostat (dogs) prior to intubation to clear the view for intubation.

Many anesthesiologists prefer to take a “wait and see” approach to bradycardia and treatment with anticholinergics. Many animals never get bradycardic during anesthesia, so may never need a dose of anticholinergic.

Myth - Opioids cause dangerous respiratory depression:
The only documented species in which respiratory depression from opioids is significant is non-human and human primates. Normal, healthy dogs and cats do not exhibit profound respiratory depression after opioids, and several studies have documented that arterial CO₂ tensions are only
slightly elevated and still within clinically acceptable limits even after relatively high doses of hydromorphone or other opioids. When opioids are used in the peri-anesthetic period they may contribute to the multifactorial respiratory depression that is seen with general anesthesia. Some of these factors include: profound respiratory depression from inhalants and induction drugs (especially propofol), hypothermia, increased resistance to breathing in an anesthetic circuit, anatomic work of breathing (especially in obese dogs or those that are thin and deep chested in dorsal recumbancy), and surgical positioning.

**Myth - Opioids cannot be used in cats:**

Non-painful cats given agonist opioids without concurrent sedation may display manic or hallucinatory behavior, with increased locomotor activity, dilated pupils, vocalization, etc. When a cat is painful, however, administration of an agonist opioid is usually associated with visible relaxation, increased sleeping behavior, and more normal posturing. When using opioids for premedication in cats, the addition of acepromazine or dexmedetomidine will modify the excitatory effect of the opioid.

There is some debate as to whether morphine is a useful opioid in cats. In people, some of the analgesic benefits of morphine are due to the glucuronide metabolites of the drug. Cats do not metabolize drugs into their glucuronide metabolites very effectively, so by extrapolation it may follow that morphine’s analgesic potency is reduced in cats. This also may be the reason why morphine may have a longer half-life in cats, but this has not been extensively studied.

It is also well-documented that opioids can cause hyperthermia post-operatively in cats. This is most often a problem when the cat has become cold during anesthesia and then hypothalamic set points seem to overshoot in the recovery period.

**Misconception - Butorphanol is an effective analgesic:**

Butorphanol has antagonist activity at the mu opioid receptor, which is the primary CNS receptor in the opioid group that mediates analgesia. Most clinical studies have shown that butorphanol, when used alone for pain, provides very little analgesia in dogs and cats and lasts only 45-60 minutes. It is now a DEA schedule IV drug, so licensing and record-keeping are the same as for effective analgesics such as morphine, hydromorphone etc.

**Misconception - Ketamine protects cardiovascular function:**

Ketamine increases heart rate and blood pressure, primarily as a result of indirect effects of catecholamine release from the adrenal medulla. The increase in heart rate and blood pressure results in an increase in myocardial work and oxygen demand. In animals with cardiac disease, particularly those with hypertrophic cardiomyopathy, this may result in ischemic areas of myocardium. Additionally, animals that have been chronically ill or stressed may not mount the adrenal response typical of ketamine, and then direct cardiovascular depression is possible.

**Misconception - Ketamine is a good analgesic:**

Besides its dissociative properties, ketamine acts in the spinal cord dorsal horn as an NMDA antagonist. The NMDA receptor is an important mediator of chronic pain and “wind up” states, where pain perception may be amplified over the actual sensory input. For this reason, ketamine has become popular as an analgesic adjuvant in the peri-operative period and its inclusion probably helps with post-surgical analgesia. One study suggested that dogs maintained on ketamine CRI’s (with fentanyl) did slightly better post-operatively with respect to pain (as reported by their owners) than dogs that received fentanyl alone. At this time, however, there are no studies that demonstrate significant analgesic properties of ketamine. Therefore, ketamine should never be considered a “stand alone” analgesic and when used for analgesia it should always be combined with an opioid.
Myth - ECG monitoring tells us about cardiac function:

The ECG is simply an electrical trace reflecting electrical pathways of conduction in the heart. An animal that has been recently euthanized will have a normal ECG for minutes after cardiac pumping has stopped. The ECG tells us nothing about blood pressure, perfusion of organs, or whether the heart is actually pumping at all! While useful for detection and identification of arrhythmias, the ECG should never be used as a substitute for pulse palpation, measurement of blood pressure, and evaluation of oxygenation by visual assessment or by pulse oximetry.

Misconception - Pulse oximeters tell us about perfusion:

Pulse oximeters are useful monitors of capillary blood flow and capillary hemoglobin saturation with oxygen. Many provide a pulse wave that indicates moment-to-moment pulsatile flow through the capillary bed being monitored. Globally, if one capillary bed is receiving pulsatile flow, then it follows that all capillary beds in the body are also receiving that flow. This may not be true depending on positioning of the animal, but that probably is more of an issue in large animals than in dogs and cats. Pulsatile flow does not necessarily correlate with perfusion pressure or oxygen delivery to tissues. An animal can be extremely hypotensive, where perfusion to vital organs such as the brain, lungs, liver, and kidneys is compromised, while the pulse oximeter beeps away at a steady rhythm and good % saturation. Thus, pulse oximetry is a very useful monitor of hemoglobin saturation, but to really monitor tissue oxygen delivery one must also employ methods to measure blood pressure.

There are probably many other anesthetic misconceptions or “rules that need to be broken”, so these proceedings are not intended to be all-inclusive. Also, as new drugs become available, or as we learn more about the available drugs in anesthesia, more myths and misconceptions will inevitably arise in the future.

References: