

## CVT / Veterinary Technician Feature Article

# Principles of Anesthesia

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*The following is a lecture outline discussing the basic principles of small animal anesthesia. As veterinary technicians, it is critical for you to understand basic anesthetic concepts- especially when you are given the responsibility of administering anesthesia and monitoring your veterinary patients during surgical procedures. The goal of... If you have any questions, please feel free to Email our hospital at [ce@neamc.com](mailto:ce@neamc.com).*

## Stages of Anesthesia

### Stage I - Stage of voluntary movement

- Encompasses period of time from initial administration of anesthesia to loss of consciousness
- Increased HR, pupil dilation, ataxia, recumbency, salivation, increased jaw tone, increased blood pressure
- **Eyeball position** normal, palpebral reflex present

### Stage II - Stage of delirium and involuntary movement

- From loss of consciousness to onset of regular pattern of breathing
- Increased HR, struggling, breath holding or tachypnea, increased blood pressure, increased jaw tone, laryngospasm in cats
- Dilated pupils, variable eye position

### Stage III - Stage of surgical anesthesia

#### **Unconsciousness with progressive depression of reflexes, divided into 3 planes**

- **Light Plane** - considered light until eye movement ceases
- **Medium Plane** - stable respiration and pulse, sluggish palpebral reflex, strong corneal reflex
- **Deep Plane** - decreased tidal volume, increased respiratory rate, central pupil, palpebral and corneal reflex absent, PLRs not present, progressive bradycardia and hypotension, as progresses closer to Stage IV patient loses diaphragmatic function and can become cyanotic

### Stage IV - Dangerous for patient- high risk of anesthetic death.

- CNS extremely depressed, respiration ceases, severely decreased blood pressure,

- anal and bladder sphincters relaxed
- Pupils dilated, palpebral and corneal reflexes absent

## Preanesthetic Medications

### Anticholinergics

#### Atropine

- **decreased secretions**- oral, pharyngeal and respiratory secretions
- **dilate bronchi** - causes increased respiratory dead space
- decreased motor and secretory activity in GI tract
- suppresses vagal influence on heart
- minimal effect on blood pressure at normal preanesthetic doses
- dilates pupils (relaxes sphincter muscle of iris)
- suppresses muscarinic action of anticholinesterase (used to reverse nondepolarizing muscle relaxants)
- if given IV may initially increase vagal tone
- **increases incidence of cardiac arrhythmias and sinus tachycardia** (most common arrhythmia prior to induction of anesthesia if 2nd degree AV block, most common arrhythmia after induction of anesthesia is VPCs and ventricular bigeminy)
- **contraindicated if preexisting tachycardia**
- cleared from blood quickly in dogs and excreted unchanged in urine, cats clear via atropine esterase in liver

#### Glycopyrrolate

- physiologic effects similar to atropine but longer duration of action
- vagal inhibition lasts 2-3 hours, antisialagogue effects can last up to 7 hours

### Tranquilizers

#### Acepromazine (Promace®)

- phenothiazine derivative - blocks post synaptic dopamine receptors in CNS, may inhibit release of and increased turnover rate of dopamine, thought to depress portions of the RAS in brain (responsible for control of temperature, basal metabolic rate, emesis, vasomotor tone, hormonal balance, and alertness)
- decreases arterial blood pressure
- hypothermia
- decreased seizure threshold?
- antiemetic
- prevents or decreases the severity of malignant hyperthermia

#### Diazepam (Valium®)

- **Benzodiazepine** - acts on benzodiazepine receptor in CNS - causes increase in inhibitory neurotransmitter glycine in spine (muscle relaxation effects), sedation and anticonvulsant effects mediated by GABA
- minimal respiratory and cardiac depression
- low toxicity
- overdose treated with flumazenil ( 1 part flumazenil to 13 parts diazepam)

### Midazolam

- also benzodiazepine- physiological effects similar to diazepam
- shorter duration of action, water soluble (nonirritating)

### Opioids- Receptors - mu, kappa, sigma, delta

- **Mu** - supraspinal analgesia, respiratory depression, euphoria, physical dependence
- **Kappa** - spinal analgesia, miosis, sedation, dysphoria
- **Sigma** - psychomimetic activity, hallucinations, respiratory and vasomotor stimulation
- **Delta** - modifies mu activity

	<b>Mu</b>	<b>Kappa</b>
<b>Morphine</b>	agonist	agonist
<b>Naloxone</b>	antagonist	antagonist
<b>Butorphanol</b>	partial agonist	partial agonist
<b>Buprenorphine</b>	partial agonist	antagonist?

### Morphine

- increased serotonin synthesis - causes analgesia
- decreases medullary respiratory, cough and vasomotor center activity
- stimulates medullary vomiting center
- decreases basal metabolic rate - decreases body temp 1° to 3° F
- respiratory depression - decreases respiratory minute volume, increases alveolar CO2
- no significant myocardial depression
- can cause histamine release, peripheral vasodilation, bradycardia, increase in ADH release (can decrease urine production up to 90%)
- stimulates sphincters of GI tract - can cause constipation but increased peristalsis combats this effect
- can cause excitation in cats
- miosis
- metabolized by liver

### Oxymorphone- mu opiate agonist

- 10x more potent than morphine
- induces more sedation and less hypnosis
- little respiratory and cough suppression

### Fentanyl- mu opiate agonist

- 250 x more potent than morphine
- analgesia, sedation, respiratory depression and exaggerated response to loud noises
- short duration of action but respiratory depression can last for several hours
- can have respiratory depression, apnea of panting
- usually does not cause vomiting
- causes vagal mediated bradycardia but has little effect on cardiac output or blood pressure unless given with barbiturates (can cause hypotension if given with barbiturates)
- **transdermal patches** - significant variability in time to achieve therapeutic levels and levels themselves, cats achieve therapeutic levels faster (usually in about 6 hours), duration of action persists for 72-104 hours (3-5 days), duration of action generally longer in cats than dogs

### Butorphanol (Torbugesic®)

- mixed agonist/antagonist
- less respiratory depressant effects
- no change in bile flow (morphine decreases bile flow)
- no change in histamine
- excreted in urine (small amt excreted in bile)

### Buprenorphine (Buprenex®)

- partial agonist/antagonist
- 30x more potent than morphine
- respiratory depression
- lasts 6-8 hours

## Alpha-Adrenergic Agonists

- Anxiolysis
- Sedation
- Sympatholytic
- Easily reversed by antagonists
- No profound respiratory depression
- Some analgesic effect
- Contraindicated if cardiac or pulmonary disease due to myocardial depression and

- pulmonary hypertension
- Duration of analgesia is about half the duration of sedation

### Xylazine (Rompum®)

- cardiac depressant effects
- arrhythmogenic (AV block)
- when given IV causes bradycardia followed by 5-10 minutes of hypertension followed by longer periods of decreased cardiac output and hypotension
- decreases heart rate by increasing vagal tone
- decreases respiratory rate but increases tidal volume
- duration of sedation approx 30-40 minutes, duration of analgesia 15-20 min
- frequently causes vomiting, decreases GI motility and prolongs gastric emptying time
- may inhibit platelet aggregation

### Medetomidine (Domitor®)

- more selective for alpha-2 receptors
- duration 2-3 times xylazine - sedation 60-90 min with analgesia for 30-45 min
- dose 10ug-30ug produces sedation and analgesia, cats often need larger dose for comparable sedation
- effects similar to xylazine, however hypertension often persists and may not be followed by hypotensive period

## Injectable Anesthetics

### Barbiturates

- **classified** according to duration of action
- **mechanism of action**- barbiturates act on CNS neurons in a manner similar to GABA - cause depression of the CNS by interference with passage of impulses to the cerebral cortex
- **metabolized** by kidney and biotransformation in the liver, the short acting barbiturates (pentobarbital, amobarbital and secobarbital) are primarily metabolized by the liver
- **all highly protein bound** - change in blood pH can affect anesthetic depth - acidosis causes increased depth, alkalosis causes dissociation and therefore decreases anesthetic depth (only undissociated form can penetrate cell membranes)

### Pentobarbital

- Subanesthetic doses can cause hyperexcitability
- **Cardiovascular effects** - decreases BP, CVP, PaO<sub>2</sub>, pH, body temp and stroke volume

- **Respiratory effects** - respiratory depression
- **Deep anesthesia** depresses renal blood flow and urine flow by circulatory depression and reflex vasoconstriction, stimulates release of ADH
- **Leukocyte counts** decrease by 20% with pentobarbital anesthesia
- **Red blood cell numbers and Hct** decrease - may be due to splenic dilation (oral administration does not affect blood values of RBCs or WBCs)
- Pentobarb **freely crosses the placental barrier** (high mortality of neonates if used for C-section)
- Complete recovery from anesthetic effects takes 6-18 hours and may be prolonged in cats (24-72 hours)

### Methohexital

- Ultrashort acting barbiturate - short action due to redistribution
- Overdose causes temporary apnea - lethal dose is 2.5 times median anesthetic dose

### Thiopental Sodium (Pentothal®)

- Main excretion via urine and liver, high fat levels in blood bind drug and cause decreased duration of action
- Causes **marked depression of respiratory centers**
- Five minutes after administration heart rate, aortic pressure, PVR and left ventricular systolic and end diastolic pressures increase - arrhythmias are common - bigeminy, extrasystole, V tach, V fib
- Prolonged anesthesia with thiopental causes pronounced hyperglycemia, lactic acid and amino acids in blood and decreased liver glycogen

## Nonbarbiturates

### Etomidate

- Imidazole derivative
- single injection produces brief anesthesia
- No depression of cardiovascular or respiratory systems, causes minimal respiratory depression in neonates in utero
- Decreases cerebral metabolic rate of oxygen consumption and has anticonvulsant properties (may be brain-protective)
- Causes depression in adrenal function for up to 3 hours following single injection can cause Addisonian crisis with prolonged infusion
- Stored in propylene glycol - can cause acute hemolysis
- Can cause nausea, vomiting with multiple doses

### Propofol

- **Nonbarbiturate** sedative/hypnotic

- Limited solubility in aqueous solution, formulated in emulsion
- Preparations support bacterial growth – should be discarded 6 hours after opening vial
- No excitement phase with induction and so can be used for sedation, induction or maintenance as a CRI
- **Metabolized** by redistribution, cytochrome P450 enzymes in liver and elsewhere and glucuronidation in liver, drugs affecting cyt P450 will prolong propofol anesthesia
- Minimal change in HR, causes vasodilation and can induce hypotension which does not trigger a reflex increase in HR, arrhythmias uncommon
- Respiratory depression and apnea most common adverse effect, caused by reduction in TV and RR with depression of hypoxic drive, rapid administration of drug more likely to cause apnea
- In people most common adverse event is pain on injection, not as commonly noted in veterinary patients
- Safe to use with increased intracranial pressure, liver disease, kidney disease

### Contraindications to propofol use

- moderate to severe cardiac disease
- hypovolemia
- hypotension
- cats that are anemic
- epilepsy or hx of seizures

### Adverse effects associated with propofol use

- **Excitatory effects** – occasionally see myoclonus, twitching, opisthotonus, unsure if this activity is a form of a seizure
- **Pancreatitis** – increase in FFA and TG levels after propofol administration, anecdotal reports of pancreatitis following propofol administration
- **Heinz body anemia in cats**, cats show prolonged recovery after propofol anesthesia if used daily for procedures lasting longer than 30 minutes (this effect was not seen with daily doses of propofol given only as an induction injection)

## Dissociatives

- Dissociative anesthetic agents induce an anesthetic state by interruption of ascending transmission from the unconscious to conscious parts of the brain rather than by generalized depression of all brain centers
- varying degrees of hypertonus and purposeful or reflexive movement of skeletal muscle occur unrelated to surgical stimulation
- eyes remain open with nystagmic gaze
- analgesia is intense but of short duration – greater analgesia for somatic pain than for visceral pain

## Ketamine

- **CNS effects** – significant increase in cerebral blood flow, intracranial pressure and cerebrospinal fluid as a result of cerebral vasodilation and elevated systemic blood pressure
- **Hallucinations** may occur during emergence from ketamine anesthesia
- **Premedication** with acepromazine, xylazine or benzodiazepine may decrease incidence of adverse reaction
- **CV effects** – sympathomimetic effects – increased HR and arterial BP, Mean arterial pressure, HR, cardiac output increase while peripheral vascular resistance remains unchanged – causes increased cardiac work and myocardial oxygen demand – effects blunted by premedication and concurrent gas anesthesia
- **Resp effects** – Unlike most anesthetics ketamine does not depress the ventilatory responses to hypoxia, with higher doses transient apnea and shallow breathing can be seen, - can cause increased salivatin and respiratory mucous production - can be prevented with anticholinergic
- Ketamine metabolized by the liver in dog and human, in cat mostly metabolized by kidney
- Mild increase in intraocular pressure

## Telazol

- Tiletamine with zolazepam – tiletamine has a longer duration of action and greater analgesic effect than ketamine
- effective in producing general anesthesia in cats and primates, inconsistent response in dogs and other species
- causes seizure activity at higher doses
- causes tachycardia and hypertension, minimal respiratory signs expect at high doses (apnea, shallow breathing)

## Inhalational Anesthetics

- **Isoflurane**- most popular anesthetic gas used
- **Sevoflurane**- newest agent, most expensive, rapid induction & recovery
- **Halothane**- older agent, used primarily in large animal practice

**MAC = minimal alveolar concentration of an anesthetic gas required to keep a dog from gross movement in response to painful stimuli**

- Increased catecholamines increase MAC, decreased catecholamines decrease MAC
- Hypothermia decreases MAC, hyperthermia increases MAC
- Premedication decreases MAC

Inhalational anesthetic induction and elimination

- Anesthesia is taken into the alveoli and then equilibrates with the blood in a manner similar to O<sub>2</sub> and CO<sub>2</sub>, anesthetic agent is then taken up by the tissues until equilibrium is reached between the blood and tissues, when venous blood returning to the lungs has the same amount of anesthetic as the alveoli the blood will not take up more anesthetic (at equilibrium)
- Induction of anesthesia depends on alveolar ventilation, cardiac output and solubility of the gas
- Elimination occurs via the same route, to recover from anesthesia, concentrations in the brain have to decrease, with no anesthesia entering the alveoli, anesthetic goes from the higher concentration in the blood to the lower concentration in the alveoli
- Duration of anesthesia affects recovery – longer duration prolongs recovery

## Effects of anesthetic gas on organ systems

### Cardiovascular

- inhalant anesthetics all decrease cardiac output due to a decrease in stroke volume from depression of myocardial contractility
- Isoflurane is least depressant to cardiac output
- Heart rate is variable but generally normal to increased
- Blood pressure is decreased in a dose dependent fashion
- Myocardium is sensitized to arrhythmogenic effects of catecholamines – halothane most arrhythmogenic, isoflurane and sevoflurane least arrhythmogenic

### Kidneys

- decrease renal blood flow and glomerular filtration in a dose dependent fashion
- reduction in renal function highly dependent on animal's state of hydration and hemodynamics during surgery

### Liver

- depression of hepatic function and hepatocellular damage may be caused by any of the inhalant anesthetics
- decreased clearance of drugs from the liver occurs during anesthesia due to decreased blood flow through the liver as well as decreased intrinsic clearance
- isoflurane least likely to cause liver damage due to less compromise of cardiac output and therefore better tissue oxygenation

### Skeletal Muscle

- Malignant Hyperthermia can occur in susceptible patients

## Anesthetic Machines

## Vaporizers-

- Convert liquid anesthetic to gas, picked up by carrier gas (oxygen) and delivered to patient, increased flow of carrier gas (oxygen) increases anesthetic delivery to patient
- Each agent has its own type of vaporizer- ie isoflurane can not be used in a vaporizer designed for halothane or sevoflurane.

## Breathing Systems

- **Rebreathing systems** – exhaled gas flows back to patient after removal of CO<sub>2</sub> – conserves oxygen, anesthetic gas, heat and moisture
- Tubing – pediatric if <15#, Adult if >15#
- Exhaled gas flows through one way valve- goes through reservoir bag and CO<sub>2</sub> absorbent canister then through inspiratory valve and tube, Pop off valve vents gas to scavenger system – prevents buildup of excess pressure
- **Semiclosed systems**– fresh gas inflow exceeds uptake of oxygen and anesthetic by patient \*\*gas flow is set at 2-4 times patient's tidal volume\*\*
- **Nonrebreathing systems** – animals <8#, incorrect terminology – some rebreathing of exhaled gases occurs especially at low flow rates, fresh gas goes to patient and is exhaled through the reservoir bag and not recycled

## Signs of Inhalational Anesthesia

### Respiratory

- increased RR +/- breath holding in stage I
- irregular respiration and breath holding in stage II
- regular breathing that depends on threshold of stimulation in stage III
- as anesthesia progresses through stage III intercostals muscles weaken and thoracic movement decreases, breathing becomes abdominal - abdomen bulges while thorax collapses during inspiration, reverse occurs during expiration, after abdominal breathing respiration will cease with further increase in anesthetic depth

### Circulation

- stage I and stage II – pulses are strong and accelerated
- as anesthesia increases blood pressure decreases and pulses weaken

### Ocular

- ocular signs can be variable – circulatory and respiratory signs more reliable
- during light and medium anesthesia eyeballs turn downwards and 3<sup>rd</sup> eyelids come up
- pupils become dilated during stage II then become constricted (this varies with

- premedication such as atropine or opiates)
- with deep anesthesia or overdose pupils become dilated and unresponsive
  - palpebral reflex is lost on transition from light to medium anesthesia, corneal reflex is lost shortly thereafter

### Pharyngeal Reflex

- increased muscle tone during Stage II then progressively declines
- lose resistance to opening mouth fully during medium anesthesia

## Factors affecting Anesthesia

### Age

- Very young animals have limited muscles and fat to which anesthesia can be redistributed
- Very old animals can have decreased cardiac, hepatic and renal function

### Breed

- **Brachycephalic dogs** – enlarged soft palate, restricted respiratory passages-avoid prolonged anesthetic recovery (use quick acting anesthetics), leave ET tube in as long as possible
- **Sighthounds** – greyhound, borzoi, afghan etc. – complications include:
  - hypothermia from low body fat
  - malignant hyperthermia
  - impaired biotransformation of drugs in liver causes prolonged recovery especially from thiobarbiturates
  - recommended drugs include propofol, ket/val, methohexital

## Changes in clearance of anesthetic

- **Liver and kidney** - has the most effect if liver or kidney extracts a high fraction of drug from the blood eg. lidocaine and morphine have high hepatic clearance
- **Lungs** – major pathway of elimination of inhalation anesthetics – drugs that decrease ventilation eg. opioids may delay pulmonary excretion of gas
- **Changes in distribution** – with IV induction agents concentration of drug leaving blood and going to tissues determines anesthetic plane – since concentration of drug in arterial blood is a function of cardiac output anything that decreases cardiac output will cause more drug to go to brain and myocardium where most of the output is being distributed – this can further compromise cardiac function

## Monitoring of Anesthesia

## Cardiovascular

- **EKG** – bradycardia, tachycardia or arrhythmias – does not measure mechanical performance and so can be normal with poor myocardial function and poor tissue perfusion
- **Blood Pressure** – product of cardiac output, vascular capacity and blood volume – vasodilation causes increased perfusion but can cause hypotension, agents to increase blood pressure cause vasoconstriction which can decrease peripheral perfusion, blood pressure needs to be maintained to perfuse the brain and heart
- **Indirect BP** – if cuff is too tight BP erroneously low, if too loose – falsely high
- **Doppler** – more accurate in smaller animals, all techniques are least accurate when BP is low and vessels constricted
- **Normal BP** 100/80 to 160/120, MAP 60-100
- At systolic pressure <80 or MAP <60 blood flow is inadequate to perfuse brain and heart
- **Causes of hypotension**
  - hypovolemia
  - peripheral vasodilation
  - decreased myocardial contractility

## Respiratory

- Rate is of limited value, change in breathing rate is a more sensitive indicator
- Tidal volume 10-20 ml/kg
- End tidal CO<sub>2</sub> (ETCO<sub>2</sub>) is a measurement of CO<sub>2</sub> in expired gas at the end of exhalation, since alveolar and capillary PCO<sub>2</sub> are equilibrated it is an estimate of PaCO<sub>2</sub>

## PaCO<sub>2</sub> as a parameter

- **PaCO<sub>2</sub>** 35-45 mmHg- normal, <35 = Hypoventilation, >35 = Hyperventilation
- **PaCO<sub>2</sub> >60** indicates severe respiratory acidosis requiring mechanical ventilation
- **PaCO<sub>2</sub> <20** indicates severe respiratory alkalosis and decreased cerebral blood flow causing impaired cerebral oxygenation
- Causes of increased PaCO<sub>2</sub>:
  - Hypoventilation – airway obstruction, thoracic or abdominal restrictive disease, pleural space disorder, pulmonary parenchymal disease, inappropriate ventilator settings
  - Dead space rebreathing
  - Hyperthermia or increased CO<sub>2</sub> production
  - Recent bicarbonate therapy
- Decreased PaCO<sub>2</sub> *caused by* hyperventilation
- Venous PCO<sub>2</sub> is 3-6mmHg higher than arterial

## PO<sub>2</sub> as a parameter

- PO<sub>2</sub> is the tension of oxygen dissolved in plasma, value is irrespective of hemoglobin concentration
- SaO<sub>2</sub> is a measurement of hemoglobin saturation of oxygen
- O<sub>2</sub> content = (Hb x 1.34 x % saturation) + (.003 x PO<sub>2</sub>)
- Pulse oximeter (SpO<sub>2</sub>) measures pulse rate and hemoglobin saturation of oxygen – measures absorption of infrared light through a blood sample- oxyhemoglobin, reduced hemoglobin, methemoglobin and carboxyhemoglobin all absorb red to infrared light differently
- Pulse ox is designed to measure oxyhemoglobin and reduced hemoglobin – if methemoglobin or carboxyhemoglobin are present they will affect measurement
  - Oxyhemoglobin absorbs at 660
  - Reduced hemoglobin absorbs at 940
  - Methemoglobin absorbs at both frequencies so SpO<sub>2</sub> reads at 85%
  - Carboxyhemoglobin absorbs at 660 so falsely increases reading
- Pulse ox accuracy is greatest at range of 80-95% hemoglobin saturation
- Poor performance of pulse ox – can be caused by peripheral vasoconstriction – can't pick up pulse, thickness of tissue can interfere with light transmission, electrical or optical interference

Blood oxygenation level	PaO <sub>2</sub>	SaO <sub>2</sub>
Normal	>80	>95
Serious hypoxemia	<60	<90
Very serious hypoxemia	<40	<75