ANESTHETIC EMERGENCIES & ACCIDENTS

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Human Errors

- Miscalculation of the drug dose; getting one decimal wrong can mean ten times of overdose that may induce severe toxic effect
- Mislabeling of the syringe, misfiling to a wrong vaporizer etc. may constitute severe hazard
- Equipment failure or misused devices; most notably delivery of hypoxic mixture to the patient
- Exercise precautions to avoid human induced medical mishaps

Bradycardia

- As a general rule, for heart rates < 60 beats per min in dogs, and < 25 beats per min in adult horses

Cause

- Deep anesthesia (inhalant anesthetic overdose)
- Increased vagal tone (in brachycephalic breed, opioid medication etc.)
- Parasympathetic stimulation (distend bladder, manipulation of the viscera, oculocardiac reflex etc.)

Problem

- Reduced cardiac output
- Arrhythmias (danger to progress to worse arrhythmias, e.g. complete cardiac arrest, if not corrected timely)

Treatment

- Identify cause and address it
- Dog and cat:
  - Atropine – 0.02 ~ 0.04 mg/kg IV
  - Glycopyrrolate – 0.005 ~ 0.01 mg/kg IV
  - If no response to above treatment, then consider giving isoproterenol 0.5 mcg/kg or epinephrine 0.02 mg/kg IV bolus (or CRI at a lower dose to effect)

Tachycardia

Cause

- Light anesthesia
- Drug induced/iatrogenic; atropine, glycopyrrolate
- Hypotension-reflex tachycardia
- Hypercapnia induced sympathetic drive

Problem

- Increases myocardial oxygen consumption; may lead to arrhythmias
- Less time for the blood volume to fill the heart, so lead to reduced stroke volume (cardiac output)

Treatment

- Deepen anesthesia if too light
- Usually none if drug-induced sinus tachycardia
- Treat hypotension (with fluids or inotropic agents)
- Treat respiratory acidosis
- For severe sinus tachycardia, consider beta-adrenergic blockers; propranolol (0.02~0.06 mg/kg), or esmolol (0.25~0.5 mg/kg) slow IV

Hypotension

Causes

- Anesthetic overdose
- Hypovolemia due to intra-operative bleeding or peri-operative fluid deficit

Problem

- Inadequate tissue perfusion, particularly important for vital organs such as brain, heart, kidneys, lungs and liver (and muscles for horses to prevent post-anesthetic myopathy)

Treatment

- Lighten anesthetic depth
- Fluid therapy
- Provide injectables (e.g., opoids, benzodiazpeines, ketamine, propofol, systemic local anesthetics etc.) to reduce volatile anesthetic requirements
- Positive inotropes (dopamine, dobutamine, ephedrine)

Premature Ventricular Contractions (PVC's or VPC's)

Cause

- Usually acidemia, hypoxia or hypercapnia
- Pain
- Myocardial contusion from trauma, usually peak at 24 hours following the accident
- Gastric dilitation/volvulus (GDV) syndrome in dogs - peak frequency occurs following surgery
- Sympathetic imbalance
- Drug induced – thiopental (bigeminy), halothane (decrease arrhythmogenic threshold)

Problem

- May lead to ventricular fibrillation, if not corrected timely

Treatment

- Correct causes
  - Respiratory acidosis - ventilate
  - Metabolic acidosis - $NaHCO_3$
  - Switch to isoflurane or sevoflurane from halothane
  - Hypoxia
    - Increase O_2 fraction
    - Decrease shunt fraction
    - Increase ventilation
    - Decrease dead space
    - Correct diffusion impairment
- Drug therapy
  - Lidocaine: 1~2 mg/kg IV bolus, up to 8 mg/kg (4 mg/kg in cats).
  - If not responsive, then Procaineamide: 5 ~ 10 mg/kg slow IV or beta-adrenergic blockers such as propranolol (0.02 ~ 0.06 mg/kg), or esmolol (0.25 ~ 0.5 mg/kg) slow IV

Perivascular Injection

Causes

- Displaced catheter, no use of catheter for drug administration, leaky vessels
- Use of irritant agents such as thiopental or guaifenesin (guaicol glycerine ether; GGE)

Problem

- Causes pain, abscesses, necrosis, thrombosis

Treatment

- Best to administer drugs always using catheters to avoid accidental perivascular injection
- Dilute with saline
- Subcutaneous lidocaine
- Subcutaneous steroid (water soluble)
- Pressure bandage application

Brachycephalic Problems

Anatomical handicaps

- Stenotic nares
- Elongated soft palate
- Everted laryngeal ventricles
- Hypoplastic trachea-select several sizes of endotracheal tubes, select smaller size ET tube than non-brachycephalic dogs
- Large thick tongue

Goals

- Rapid, smooth induction, i.e. Use propofol or thiopental etc.
- Pre-oxygenate using a face mask prior to anesthetic induction
- Quick, smooth recovery
- Avoid drugs with known vomiting reflex such as xylazine and morphine
- Avoid excessive premedication
- Leave endotracheal tube in as long as possible during recovery
- Observe recovery, pay attention to upper airway obstruction

Cardiac Arrest

Common Causes

- ***Deficiency of oxygen is the ultimate cause of all cardiac arrests.***
- Respiratory failure
- Acid-base disturbances
- Electrolyte imbalances
- Autonomic imbalances
- Hypothermia
- Air embolism
- Toxicity
- Anaphylactic reactions
- Drug overdose~
- Cardiac disease, arrhythmias

Signs of cardiac arrest

- Absence of a pulse or a palpable or audible heart beat
- Apnea
- Loss of consciousness
- Loss of corneal ocular reflexes
- Eyes are fixed, wide open
- Pupils are dilated and unresponsive to light

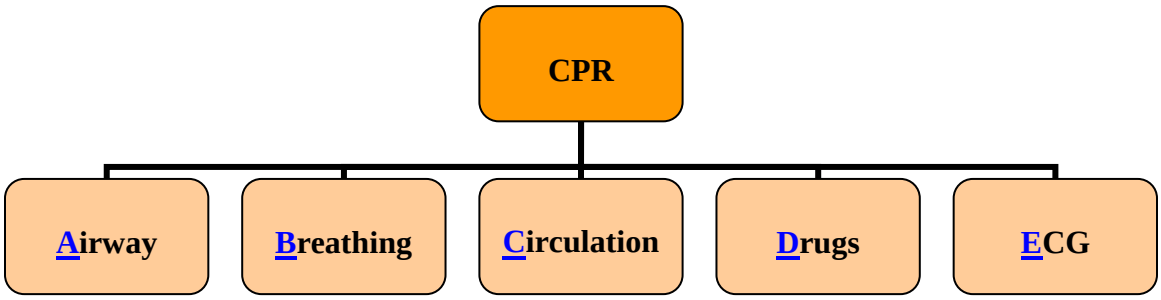
Basic cardiac life support (BCLS)

- Aim is to deliver oxygen to the lungs by artificial ventilation, and then transport the oxygen to body tissues by external cardiac compression

Advanced cardiac life support (ACLS)

- Restoring spontaneous breathing and circulation and sustaining them

Rules of ABCDE



Airway (A)

- Establish a patent (secure) airway as quickly as possible
- Clear the airway of any obstructions (excessive mucus, tongue, foreign objects).
- Perform endotracheal intubation with a cuffed endotracheal tube.
- If this is not an available option, consider transtracheal catheter ventilation (using a 14g needle or over-the-needle intravenous catheter with a 3mm endotracheal tube connector connected to O_2 line or a Bain Circuit) or complete tracheostomy using a tracheostomy tube set.

Breathing (B)

- Attach endotracheal tube to a source of 100% oxygen (preferable to room air, if possible).
- Administer 6 ~12 breaths/minute.
- Ratios of one breath per 5 chest compression are used when simultaneously performing chest compression.
- The amount of gas volume is 10 ~ 20 ml/kg at a peak inspiratory airway pressure of 20 ~ 25 cm H_2O
- Inspiratory time is approximately set at 1.5 seconds and I:E ratio approximately 1:2~3.
- A continuous flow of 100% oxygen at 50~150ml/kg/min administered though endotracheal tube or a cannula inserted transtracheally.
- Although not as efficient, mouth-to nose ventilation is performed when endotracheal tube and respiratory assist device is not available, and could be life-saving.

Circulation (C) - Cardiac compression

- Place the patient in lateral recumbency on a firm surface and compressing the chest at a rate of 80 ~ 120 compressions per minute, devoting equal time to compression and relaxation.
- Compress the heart in small animals (<10 kg) from both sides, taking advantage of the cardiac pump mechanism of establishing cardiac output. In larger animals (>10 kg), compress over the junction of the dorsal and middle third of the 5~7th intercostal space, relying on the thoracic pump mechanism for generating cardiac output.
- Compress the chest to depress the chest wall by 30%. The duration of compression (*cardiac systole*) should be at least 50% of the total compression-relaxation cycle to

produce maximal flow. Release the pressure completely during relaxation to allow cardiac filling.

- Blood flow generated by cardiac compression will temporarily sustain cerebral and myocardial viability only if oxygenation is adequate. However, cerebral blood flow is less than one-fifth normal and coronary flow even less during external compression.
- Complications, which may arise from external compression, include sternal and rib fractures, blunt traumatic damage to intrathoracic and intraabdominal viscera and pneumothorax.

Mechanisms of blood flow during external chest compression

- Controversial.
- Two theories predominate. They probably both operate.
  - *Cardiac pump theory*. External chest compression squeezes the heart between the thoracic walls, forcing blood into the aorta. Blood flow depends on the rate and pattern of compression.
  - *Thoracic pump theory*. Phasic changes in intrathoracic pressure result in venous inflow into the thorax and forward flow into the aorta. A prolonged compression time (50 -60% of the cycle) favors flow produced by changes in intrathoracic pressure.
- Application of an alternating counter pressure to the abdomen to increase caudal vascular resistance and divert blood to more vital tissues may be effective. This is accomplished by alternating abdominal and cardiac compressions.

Open-chest Cardiac Massage

- Opening the chest for direct cardiac massage is indicated if external compressions are ineffective in producing a palpable femoral pulse after a maximum of 5 to 10 minutes.
- This is often required in cases where it is difficult to generate significant intrathoracic pressure fluctuations. Such cases include patients with pneumothorax, hemothorax, flail chest, severe obesity, poor thoracic wall compliance (bulldogs), and diaphragmatic hernia.
- To perform a left-sided thoracotomy for open-chest CPR, make a left sided incision at approximately the fifth or sixth intercostal space, from just below the origin of the ribs dorsally to just above the sternum ventrally.
- The coat is clipped only in long-haired breeds, just enough to see the rib space.
- Use Mayo scissors to penetrate the pleura.
- Insert retractors to spread the ribs and expose the thoracic cavity.
- A pericardiotomy is usually recommended.
- The heart is grasped and massage begins.
- Massage is performed in a rhythmical manner, with compressions originating at the apex and extending to the base. The resuscitator should feel the heart enlarge as the chambers fill.
- Despite some clear theoretical advantages, there seems considerable reluctance of performing emergency thoracotomy so as to perform internal cardiac compression because of lack of supporting evidence of increased chance of survival.

Drugs (D) administration

Recognize and treat arrhythmias

Rationale for drug administration

- Epinephrine
  - Benefits are mainly due to its alpha-adrenergic actions.
  - It increases arterial wall tone and total peripheral resistance. This allows better intrathoracic arterial flow by reducing the tendency of vessels to collapse from the pressure induced by chest compressions.
  - Epinephrine also increases diastolic pressure which is important for myocardial perfusion, and renders the fibrillating heart more apt to defibrillate.
  - It also diverts blood flow away from non-vital, towards vital tissues.
  - Dose of epinephrine is between 0.02 mg/kg to 0.2 mg/kg. Repeat this dose each 3 to 5 minutes if needed.
- Sodium bicarbonate
  - Administered when a metabolic acidosis has developed or is strongly suspected (indicated by the patient's history) or if resuscitation has proceeded for longer than 20 minutes.
  - The dose is approximately 0.5~1 mEq/kg for each 10 minutes of arrest.
  - Venous blood gases are most indicative of bicarbonate needs.
  - Bicarbonate therapy is currently controversial, with concern that overzealous use may induce hyperosmolality, hypernatremia, paradoxical intracellular acidosis, and iatrogenic alkalosis. (see Acid Base Physiology and Anesthesia lecture). Alkalosis lowers the serum potassium levels and shifts the oxygen-hemoglobin dissociation curve, impairing oxygen delivery to the tissues.
  - The carbon dioxide generated may cause respiratory acidemia in the presence of inadequate ventilation and also intracellular acidosis. Therefore, bicarbonate therapy is recommended only when specifically indicated.
- Calcium salts
  - No longer recommended since their administration is associated with a poorer survival rate and accelerated development of irreversible neuronal damage. They may be given if specifically indicated e.g. hypocalcemia or hyperkalemia.
- Glucose containing fluid solutions
  - Hyperglycemia has also been associated with a poorer survival rate and higher incidence of neuronal dysfunction. The use of glucose-containing solutions is therefore relatively contraindicated unless specifically indicated.
- Intravenous fluids
  - Given at 10 to 20 ml/kg to offset peripheral vasodilation not corrected by alpha-adrenergic agents. Rapid rates of fluid administration are not indicated unless severe hypovolemia exists.

Routes of drug administration

- Central venous catheters (jugular) provide a more rapid onset of drug effect compared with drug administration through peripheral catheters.
- If peripheral sites are used, external cardiac massage must effectively establish circulation if the drug is to reach the heart.
- Remember the brain-arm circulating time is approximately 30 seconds with normal cardiac output. Cardiac compression less than this duration is less effective and will impede the delivery of the drug from injection site to the heart (target organ).
- Administration of drugs (epinephrine, atropine, lidocaine) into the tracheal tube lumen allows prompt absorption across the tracheal mucosa and serves as a useful alternate

route for drug administration. This is usually done using 2-3 ml of saline as diluent for the drug administered into the lumen of endotracheal tube, followed by 2-3 large breaths with artificial ventilation to promote dispersion of the drug within the pulmonary tree. Double or triple the IV dose when given in this route.

- Intracardiac administration of epinephrine is rarely indicated and in fact discouraged. It may be used when the chest is already open. Other routes which permit rapid uptake of drugs include the intraosseous and sublingual routes.

Electrocardiography (E)

Electrocardiographically, there are three forms of cardiac arrest

1. *Asystole* (the flat liner) is absence of any electrical activity.
   - Initial pharmacologic therapy of choice is epinephrine at a dose of 0.02 to 0.2 mg/kg.
2. *Ventricular fibrillation* (the hay wire)
   - Appears as completely chaotic, irregular, bizarre deflections.
   - There are two types of fibrillation:
     - Coarse (with large oscillations)
     - Fine (small oscillations).
   - There are no recognizable P or QRS waves.
   - The treatment is to defibrillate with DC current.
     - Apply adequate amount of electrode gel at the center of one paddle and rub the paddles together
     - Turn the defibrillator on
     - Select energy level:
       - External 50 to 100 wattsec (J) (<10kg), 100 to 400 wattsec (>10 kg)
       - Internal 5 to 15 wattsec (<10 kg), 20 to 80 wattsec (>10kg)
       - The internal dose is approximately 1/10th the external dose.
     - Charge the defibrillator
     - Position paddles: beware of burn and human personal injury
     - Fire the defibrillator
   - If ineffective, repeat defibrillation. If still no effect, particularly with fine fibrillation, epinephrine may be given to raise the fibrillation threshold. Lidocaine and sodium bicarbonate may then be tried if fibrillations are still present
   - Although not as effective, chemical cardioversion of ventricular fibrillation in the absence of mechanical defibrillator can be carried out with a mixture of 1.0 mEq potassium/kg and 6.0 mg acetylcholine/kg by intravenous injection.
3. *Electromechanical dissociation* (the normal wave form)
   - Is present when there is a recordable electrocardiogram but no effective cardiac output
   - This form of cardiac arrest carries a fairly poor prognosis.
   - Some success has been reported with administration of dopamine (5~10 mcg/kg/min) and also dexamethasone (2 mg/kg).

Postresucitation Life Support

- This is the most difficult part of the resuscitation. Post-resuscitation cardiac arrest frequently occurs. All patients resuscitated should be placed in an intensive care unit for monitoring and support therapy.
- Postresuscitation therapy
- Re-arrest frequently occurs and resuscitation will be necessary

- Inotropic support (e.g. dopamine at 5~10 mcg/kg min intravenous infusion) to maintain blood pressure
- Aim is to optimize cerebral perfusion and oxygen delivery (maintain normotension, moderate hypoventilation to PCO_2 of 30 ~ 35 mm Hg, maintain normal intracranial pressure with diuretics and corticosteroids, oxygen therapy) and control the cerebral metabolic demand for oxygen (suppress seizures). Barbiturates and hypothermia have not been shown to be useful under these circumstances. Neuroprotective drugs such as free radical scavengers (DMSO, desferoxamine) and calcium channel blockers are, at this time, of experimental value only and result in poor to variable responses.
- Close monitoring at least for further 24 hours is strongly recommended after successful CPR.