REassessment **C**ampaign **O**n **VE**terinary **R**esuscitation **Overview**

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CPA (cardiopulmonary arrest): Complete failure of both respiratory & circulatory systems

Common causes of veterinary CPA include:

- Hypotension – secondary to hypovolemia, sepsis, or drug administration
- Hypoxemia – secondary to hypoventilation or lung disease
- Metabolic derangements such as hyperkalemia
- Acidemia
- Vagal stimulation
- Trauma
- Cardiac arrhythmias

Potential signs of impending CPA:

- Dramatic changes to effort/rate/rhythm of breathing – agonal, decreased or increased RR
- Absence of pulse
- Significant hypotension with systolic BP < 50 mm Hg
- Irregular to inaudible heart sounds
- Dramatic changes in HR or rhythm
- Absence of surgical bleeding
- Changes in MM color – white/cyanotic
- Fixed & dilated pupils (within 30-45 seconds)
- Distressed vocalizations
- Patient collapse

Diagnosis of arrest:

- Absence of ventilation
- Cyanosis
- Absence of a palpable pulse (disappears at systolic < 60 mmHg)
- Absence of heart sounds (disappear at systolic < 50 mmHg)
- Dilation of the pupils

Stages of CPR:

- Goal of CPR: To establish & maintain cerebral perfusion & oxygenation
- Initial interventions: Evaluate patient responsiveness, breathing, pulse
- **BLS (Basic Life Support) – CAB**
    - **Enhancing artificial circulation via chest compressions**
        - Goal is to maximize myocardial & cerebral perfusion
            - Myocardial perfusion pressure = Aortic diastolic pressure – right atrial pressure
                - Drives coronary blood flow
                - Positively correlated to improved outcomes
            - Cerebral perfusion pressure + MAP – intracranial pressure
                - Drives cerebral blood flow
        - External cardiac massage (ECM) at rate of 100-120 compressions/minute
        - Compressions – hard, fast, full chest recoil, minimize interruptions
            - Compressed to 33-50% thoracic diameter
                - CO increases with higher compression rates
                - Properly performed = 20% of normal CO
            - 50:50 ratio compression to relaxation
        - Size of animal matters:
            - Patients < 15 lbs – **Cardiac pump theory**
                - "Blood flow is generated by pressure on the heart chambers directly"
                - Compressing the heart between the left & right chest wall
                - External compressions cause artificial systole, generating higher pressure in the ventricles resulting in closure of the AV valves
                - Chest relaxation is artificial diastole where intracardiac pressure falls and ventricular pressure decreases below atrial pressure, opening the AV valves
                - Arterial blood flow is a result of direct compression of the ventricles
                - Thin, compliable chest walls

- Patients > 15 lbs – **Thoracic pump theory**
    - "Squeezing pulmonary vasculature & increasing intrathoracic and airway pressure generates blood flow"
    - Not as efficient as cardiac pump theory
    - Boyle's Law – the more the thoracic volume is compressed, the more the intrathoracic pressure will increase
    - Compressions over widest point of chest
    - Too big to effectively compress ventricles
    - Forward blood flow in larger patients results from a generalized increase in intrathoracic pressure
    - Studies show that the majority of forward blood flow results from a generalized rise in intrathoracic pressure rather than from direct cardiac compression.
- Open-chest internal cardiac massage (ICM)
    - Guidelines recommend:
        - Immediate for all animals > 40 lbs
        - After 10 minutes CPCR regardless of size
        - All cases of trauma/hemorrhage
        - Pleural fluid of any kind
        - When the thorax or abdomen is already open during surgery
    - Associated with increased risk of spontaneous circulation (ROSC) & improved neurologic outcomes

- **Endotracheal intubation**
    - Establishing an open & clear airway
        - Extend neck
        - **Suction** as necessary
        - If obstructed:
            - Sharp abdominal thrusts & digital finger sweep
                - Emergency tracheostomy as needed - can insert needle or cath into trachea distal to obstruction while placing
            - Remove obstruction with long hemostats or Doyen intestinal clamps
    - If airway is obscured by swelling or mass, palpate the larynx and manually direct the tube into the glottis
    - Verify placement
        - Monitor ETCo2
            - ETCo2 > 15 mm Hg associated with higher survival rates
            - Tracheal expired gas will contain CO2 but esophageal gas will not
                - ETCo2 changes when blood flow to the lungs changes, and is an indirect indicator of SCV and systemic blood flow
            - When ventilation is controlled ETCo2 is linearly related to SV even during low blood flow rates
- **Providing assisted ventilation**
    - Breathe with 100% O2 at 8-10 B/min
        - Start with 2 long breaths
    - **Ambu-bag** (bag-valve mask) can be used
    - Peak airway pressure < 20 cmH2O
    - Detrimental effects of relative hyperventilation:
        - Vasoconstriction to cerebral bloodflow
        - Elevated mean intrathoracic pressures compared with lower ventilation rates
        - Decreases in myocardial pressures
        - Increased mortality
    - Acupuncture using Jen Chung GV26 – 25ga needle inserted to 10-20mm in the nasal philtrum
        - *Cheyne-Stokes* – respiratory pattern characterized by rhythmic waxing & waning of depth of respiration until breathing stops entirely
    - *Impedance threshold devices (ITD's)* – placed on proximal end of ET tube during resuscitation to create an increase in negative pressure during chest-recoil phase of chest compression
        - Increase in negative pressure creates a vacuum that results in more blood being pulled into the heart so more output in the next compression
    - *Simultaneous Compression-Ventilation CPR (SCV CPR)*
        - Application of external thoracic compression and simultaneous ventilation at high airway pressures (40-60 cm H2O) can increase intrathoracic pressure
        - Greater increases in arterial & aortic BP, carotid blood flow, and CO have been measured.
    - *Interposed abdominal compressions (IAC)*
        - At 70-90 compressions/min, alternated with each chest compression can improve CO
        - Result in increased venous return to the thorax in diastole, increasing forward flow
        - Conflicting results

- *Abdominal compression*
    - Placing a sandbag on the abdomen caudal to the ribs (watch pressure)
    - Binding the hind legs while separated by a rolled towel and along the ventral abdomen

- **ALS (Advanced Life Support) –**
    - Venous access
        - **Drug administration via peripheral vein should be followed by a 5-50 ml fluid bolus (depending on patient size), then elevate the extremity for 10-20 seconds**
        - IC drug injection is contraindicated – Risks include lacerating lung tissue, disrupting cardiac vasculature, or injecting drugs that may trigger arrhythmias or myocardial ischemia (epi)
        - Central venous access best because circulation times of drugs better
        - IO via femur, proximal tibia, or humerus
    - ECG interpretation **– No alcohol on leads in case defibrillation needed**
        - Most common arrhythmias:
            - Ventricular asystole – Flatline (most common)
                - Complete absence of electrical or mechanical activity from the ventricles
                - Drug treatment – Epinephrine every 3-5 minutes
                    - Single dose vasopressin
                - Follow up with Atropine (Glycopyrrolate in rabbits) once heart rate is established
            - PEA (Pulseless Electrical Activity)/electromechanical dissociation
                - A normal sinus-appearing ECG with no palpable pulse or audible heart beat
                - Lack of mechanical activity in the heart prevents effective CO and forward perfusion
                - Same drug treatment as above
            - Ventricular fibrillation
                - The rhythm most responsive to treatment
                - Total disorder of the ventricles of the heart, accompanied by incoordination of contraction
                - ECG waveform erratic & chaotic
                - Sharp precordial thump may convert in smaller if no defibrillator
                - Theory behind defibrillation is a massive electrical shock will cause the complete depolarization of all the individual myocardial fibers. A condition of electrical homogeneity is established which is inimical to re-entry.
                - Defibrillation threshold – the minimum amount of energy required to defibrillate the heart
                    - Lidocaine increases this threshold
                    - Beta agonists and aminophyline lower this threshold
                        - Epinephrine/albuterol - bronchodilators
                - Treatment is immediate electrical defibrillation @ 3-5 j/kg, then 5-7 j/kg, then 7-10 j/kg
                    - Repeat at 50% higher setting, 2 additional times, if unsuccessful
                    - Compressions/ventilation for 1-2 mins
                    - Drug Therapy: low-dose epinephrine & vasopressin (complement defibrillation)
                - Safety points:
                    - Excess alcohol is flammable
                    - Everyone CLEAR
                    - Defibrillate on expiration when possible
    - Drug administration
        - Fluids:
            - Bolus fluid therapy in euvolemic or hypervolemic patients may cause a significant rise in CVP and right atrial pressure, severe pulmonary edema, diminishing myocardial and cerebral perfusion pressure to detrimental results.
                - There is a positive correlation between coronary perfusion pressure and success of resuscitation (aortic diastolic pressure – right atrial diastolic pressure
            - Aggressive fluid blousing decreases the gradient between the two, and lowers coronary perfusion pressure
        - Types of drugs used:
            - *Atropine* – vagolytic (abolishes parasympathetic tone)
                - Indicated in PEA, asystole, symptomatic bradycardia, and in vagal arrests associated with elevated vagal tone
            - Alpha adrenergic agonists – cause peripheral vasoconstriction resulting in an increased diastolic pressure and increased coronary flow
                - *Epinephrine* – (both alpha & beta effects but alpha most effective) - indicated in all CPA
                    - Increases aortic diastolic pressure & improves myocardial perfusion
                    - Produces an intense vasoconstriction that prevents arterial collapse and maintains arterial pressure

            - Catecholamine with strong alpha (most helpful) and beta effects (both inotropic and chronotropic effects)
            - Used for: increased systemic vascular resistance, increased arterial BP, increased HR, increased coronary and cerebral blood flow, and increased myocardial oxygen requirements
            - Improve cerebral & coronary blood flow by preventing arterial lumen collapse and increasing peripheral vasoconstriction
        - Vasopressors – used to increase vascular resistance and increase aortic BP
            - Maximizes myocardial and cerebral pressures & blood flow to the heart and brain
            - *Vasopressin* – indicated in all CPA's (may be superior to epi)
                - One of the most potent endogenous vasoconstrictors via its interaction with receptors on vascular smooth muscle
                - Non-catecholamine vasopressor
                - Longer half-life than epi
                - Is effective in the presence of acidosis
        - Antiarrythmic drugs – used with V Fib or pulseless ventricular tachycardia not responsive to initial defibrillation
            - *Lidocaine* – Use with caution after ROSC
                - Suppresses ventricular arrhythmias by reducing automancy
            - *Amiodarone* – must be diluted
                - Slows HR and prolongs AV nodal refractoriness
                - Controls life-threatening arrhythmias & slows AV nodal conduction
        - Buffer therapy
            - *Sodium bicarbonate* indicated in animals with preexisting acidosis, severe hyperkalemia, or in prolonged arrests > 10 minutes
                - Results in the rapid generation of CO2 which is a potent negative inotrope that aggravates intracellular acidosis
        - Electrolyte therapy
            - Calcium administration not routinely recommended as may exacerbate ischemia/reperfusion injury
            - *Calcium glucanate* used for severe hyperkalemia and known hypocalcemia
            - *Magnesium sulfate* for hypomagnesemia
                - Sometimes described as a chemical defibrillator
    - Emergency drugs that can be administered via the ET tube:
        - Epinephrine, atropine, naloxone, vasopressin, lidocaine
        - Use long red rubber catheter and advance past the tip of the ET to the carina
            - Exposes the drug to the large surface area of the lung & pulmonary circulation, where it is absorbed and delivered to central circulation
        - Increase drug doses by 2 times
        - Follow with small amount saline
        - <u>Never</u> Sodium Bicarbonate (inactivates surfactant) or calcium
    - Some drugs NOT recommended:
        - *Doxapram* for respiratory arrest – increases cerebral & myocardial O2 demand
        - Sodium bicarbonate only is diagnosed severe metabolic acidosis before arrest (Never IT)
        - Routine use of calcium not advised
- Anesthesia-related arrests
    - Reversal agents:
        - *Flumazenil* - reversal agent for benzodiazepines (diazepam, alprazolam, and lorazepam)
        - *Naloxone* – reversal agent for opioids (morphine, meperidine, oxymorphone, fentanyl, hydrocodone)
        - *Atipamezole (Antisedan)* – reversal agent for alpha-2 agonists (medetomidine or dexmedetomidine)
        - *Yohimbine* – reversal agent for xylazine (Rompun)
- Ongoing post-resuscitative care
    - Supplemental O2 (50-150 mL/kg/min)
    - Support the heart to reduce pulmonary edema
        - Inotropic support – dobutamine or dopamine
        - Vasodilator & vasopressor drugs – nitroprusside
        - Antiarrythmic drugs – lidocaine
        - Furosemide
    - Mildly hypothermic patients (> 97 degrees) should not be aggressively rewarmed
    - Cerebral protection is usually indicated:
        - Mannitol & furosemide administration
        - Head elevation
        - Maintenance of normal BP and DO2

        - Ensuring normocapnia
            - Hypercapnia can lead to cerebral vasodilation and increased intracranial pressure
            - Hypocapnia can lead to hyperventilation which can cause excessive vasoconstriction & decreased cerebral blood flow
    - Cardiovascular support
        - Stable & perfusing ventricular escape rhythms should never be suppressed (lidocaine, etc.)
    - Ventilator support

CPCR considerations:

- Move the patient as necessary
- Well-stocked crash cart
- R lateral recumbency or dorsal recumbency for barrel chested animals
- Regularly assess effectiveness of CPR
    - Lubed Doppler transducer can be placed on eye & listen for "swoosh" to indicate blood flow to brain

Survival:

- Postresuscitation syndromes can occur
    - Cardiovascular instability & arrhythmias
    - Myocardial dysfunction
    - Hypotension secondary to loss of vasomotor tone
    - ARF or GI mucosal barrier compromise secondary to hypoperfusion
    - SIRS with ALI
    - Dysregulation of coagulation
    - MODS
    - Transient cerebral dysfunction
    - Iatrogenic injury – incisions, fractures
- Post ROSC hypotension associated with diminished functional status in survivors
- "You aren't dead until you're warmed and dead"
- Pre-arrest hyperglycemia associated with worse outcomes & administration during CPR not recommended
- Prognosis is typically poor with less than 4-9% surviving post CPR
    - 0-3% survival to discharge
- CPA secondary to an end-stage disease reduces chance for positive outcome

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