Cardiopulmonary Cerebral Resuscitation (CPCR)
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Cessation of blood flow and ventilation constitutes cardiopulmonary arrest (CPA) that leads to inadequate oxygen and nutrient delivery to tissue, shock and ultimately death. Cardiopulmonary arrest is typically associated with loss of consciousness, collapse, lack of a palpable pulse, pale or cyanotic mucous membranes, lack of effective respirations, and lack of measurable blood pressure. There are many predisposing causes for CPA, including sepsis, cardiac failure, pulmonary disease, neoplasia, coagulopathies, anaesthesia, toxicities, multisystem trauma, traumatic brain injury, and systemic inflammatory response syndrome. Additionally abnormally high vagal tone such as may accompany severe vomiting, tenesmus or upper-respiratory tract obstruction might initiate some CPA events.

Closed and careful monitoring for deterioration in critical patients is essential as the most successful CPCR is the one that is avoided. The role of recognizing signs of impending CPA is therefore very important. Before a possible episode of CPA the patient may become obtunded, hypothermic, bradycardic or hypotensive, develop dilated, unresponsive pupils and change his respiratory pattern progressing to gasping and agonal breaths at the time of CPA. Unfortunately, some cases that require CPCR present after CPA have already occurred.

Cardiopulmonary-cerebral resuscitation refers to re-establishing blood flow to the cerebral and coronary systems in the event of CPA by performing manual cardiac and thoracic compressions and manual ventilation until spontaneous circulation and ventilation occurs. The decision to begin CPCR has to be based on clinical signs, consideration on potential outcome and underlying disease process (CPCR should be attempted only in patients that have a possible treatable disease) and ideally on a previous agreement with the animal owners. The patient’s resuscitation code should ideally be discussed with the owner at time of hospital admission or in case of deterioration of the clinical condition.

There three phases of CPCR:
1) Basic Life Support (BLS)
2) Advanced life support (ALS)
3) Post- resuscitation care

**Basic life support (BLS):**
Basic life support should be started immediately and involves establishing and maintaining an airway (A), with supplemental oxygen initiating artificial ventilation (B) and starting manual cardiac and thoracic compression to re-establish circulation (C).

**Airway:**
An airway should be rapidly established by placing a well-fitting, cuffed endotracheal tube. When orotracheal intubation cannot be performed an emergency tracheostomy or mouth-to-nose respiration should be considered.
Breathing:
Manual ventilation should be instituted with 100% oxygen at a rate of 10-12 breaths per minute. All breaths should be given over 1 second followed by a pause to allow normal relaxation of the chest. High volumes and high pressures must be avoided to prevent iatrogenic trauma. An Ambu® bag or an anaesthetic breathing system delivering 100% oxygen should be used.

Circulation:
External chest compressions are intended to move blood from the chest to the vital organs and enhance venous return. External chest compressions should be performed at a rate of 80-100 compressions/minute with the patient on a firm surface in lateral recumbency. Compressions should be performed with a 1:1 ratio of compression to relaxation and the chest wall should be allowed to expand fully between compressions. External chest compressions employ either the thoracic pump (the compressor’s hand should be placed over the widest part of the chest wall) for animal heavier than 15 kg, or the cardiac pump (the compressor’s hand placed directly over the apex of the heart (4th - 6th intercostal spaces) for smaller animals. If the chest compressions are successful we should be able to detect the presence of a peripheral femoral pulse and the patient should have an end tidal CO2 > 10 mmHg on a capnograph.

Advanced Life Support (ALS):
Following BLS (if possible simultaneously), a venous access should be gained. Alternatively the endotracheal rout offers a simple and rapid approach to drug administration during CPCR. Epinephrine, atropine, vasopressin and most drugs can be administered, but with at least 2 to 3 times the intravenous dose, diluted in an appropriate volume (e.g. 5 mL/20 kg) of normal saline and injected via a catheter through the ET tube. Fluid therapy is necessary only if the patient was hypovolaemic before the CPA. If a patient was on any medication that is a potential cardiac or respiratory depressant, that drug must be immediately reversed. An electrocardiogram should be performed to determine the cardiac rhythm. Drugs should be administered based on a particular cardiac rhythm and timing during CPCR.

The anticholinergic compound atropine (0.02-0.04 mg/kg) can be given at initiation of CPR in cases in which the arrest rhythm is severe bradycardia, pulseless electrical activity or asystole (the majority of feline and canine CPA patients) and repeated every 3-5 minutes. Epinephrine (0.01 mg/kg) repeated every 3-5 min should be used also in case of asystole. Transthoracic defibrillation should be attempted if ventricular fibrillation/ tachycardia is the primary arrest rhythm.

Post-resuscitation care:
The last phase of CPCR consists of post-resuscitation care: protection of the heart and brain from the adverse effects of CPA, providing perfusion to vital organ systems, and addressing any underlying condition that caused CPA in the first place. Respiratory function should be supported with supplemental oxygen and many patients will need ventilator support. Crystalloid or colloid IV fluid therapy should be administered cautiously to restore and then to maintain euvolaemia. It the animal remains hypotensive despite adequate intravascular volume then vasopressors may be required. In patients with post-resuscitation myocardial dysfunction may require a positive inotrope such as dobutamine. Arrhythmias should be treated if they are compromising the patient’s haemodynamic status.

Neurologic dysfunction is common after CPCR because of cerebral oedema secondary to decreased cerebral perfusion and cerebral hypoxia. Many of these abnormalities might resolve with 1-2 days of return to spontaneous circulation. Patients should be allowed a minimum of 48 hours before any judgement on their neurological status is made. Antiepileptic treatment should be administered to any post-CPA patient with seizures and
interventions that may decrease intracranial hypertension secondary to brain oedema such as mannitol (0.5-2 gr/kg in 30-45 min) should be considered at the discretion of the veterinarian.

Cardiovascular, respiratory parameters and body temperature should be regularly evaluated. Urine output, electrolytes, blood glucose concentration, electrocardiogram, blood pressure neurologic function, and patient comfort should be monitored.

Criteria for terminating resuscitation
Various objective criteria can be used to decide the appropriate time to discontinue resuscitative efforts. Typically the duration of resuscitative efforts is most commonly used and a figure of <20 minutes is suggested.

References: