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When Things Go Wrong - Prevention and Management of Complications and Emergencies in Anaesthesia Mini Series

Session 3: Cardiopulmonary Resuscitation - What To Do When Things Go Really Wrong

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Cardiopulmonary arrest (CPA) can be defined as a sudden, cessation of functional ventilation and systemic perfusion. This will result in reduced O_2 delivery to tissues with decreased removal of CO_2. Cardiac arrest and respiratory arrest can occur at the same time but often respiratory arrest will occur first. This must be quickly treated to prevent cardiac arrest ensuing.

It is important to be able to recognise signs of impending CPA:

- Changes in the patient's respiratory rate, depth, pattern and effort
- Decreasing end tidal carbon dioxide
- Hypotension
- Cyanotic, grey or pale mucous membranes
- Capillary refill time may be prolonged
- Weak, irregular pulses, irregular heart sounds, tachycardia, ventricular premature complexes
- Bradycardia
- Sudden, unexplained increases in anaesthetic depth
- Hypothermia despite re-warming efforts

Signs of CPA

- No detectable heart sounds on auscultation
- Loss of palpable pulses and direct arterial catheter and pulse oximeter traces will be flat
- Fixed, dilated pupils (occurs within 45 seconds of arrest)
- Absence of ventilation or agonal gasping – this may be masked if the patient is being artificially ventilated
- Pale, grey or cyanotic mucous membranes. Note that mucous membranes and capillary refill time can remain normal for several minutes after arrest.
- Absence of bleeding at the surgical site, blood may appear dark if the arrest is due to hypoxaemia
- ECG tracing may be abnormal – however with pulseless electrical activity (PEA) the QRS complexes may appear normal on the ECG for several minutes after the arrest, although the myocardium will not be effectively contracting
- Collapse and loss of consciousness in the awake patient
- Loss of skeletal muscle tone and cranial nerve reflexes

Reasons for arrest

- Hypoxaemia
- Hypercarbia
- Hypotension
- Hypoglycaemia
- Hypovolaemia
- Electrolyte imbalances e.g. hyperkalaemia
- Vagal stimulation

- Overdose of anaesthetic agent
- Hypothermia
- Acid-base abnormalities
- Sensitisation of the heart to circulating catecholamines
- Severe trauma, systemic or metabolic disease
- Severe underlying cardiac or respiratory disease
- CNS e.g. herniation or traumatic brain injury
- Sepsis or Systemic inflammatory response syndrome (SIRS)

Equipment

Adequately stocked crash trolley or box comprising essential equipment for both basic and advanced life support techniques.

The box or trolley should be kept in a central, easy accessed location - regularly checked and immediately restocked after use.

The presence of checklists, algorithm charts and dosing charts have also been shown improve compliance with CPR guidelines.

The contents of the crash trolley or box will vary from practice to practice.
Typical crash trolley contents:

- Anticholinergic drugs – atropine/glycopyrolate
- Vasopressor drugs - adrenaline, noradrenaline
- Anti-arrhythmic - lidocaine
- Antagonist drugs – naloxone, flumazenil, atipamazole
- Inotropes – dopamine, dobutamine
- ET tubes, bandage to secure the tube, laryngoscope and several
- Needles and syringes
- I/V catheters, butterfly catheters
- Stylets to aid intubation
- Tracheostomy tubes
- Urinary catheters and connectors to administer oxygen or intratracheal drugs.
- Ambubag
- ECG pads
- Fluids
- Pressure bags
- Drip sets including blood filter lines and extension sets
- Surgical kit including a pre-loaded scalpel and rib retractors
- Surgical scrub solution
- Defibrillator gel

An emergency drug chart should be attached to the trolley or box giving the doses in ml for different weights of patient i.e. 2.5kg, 5kg, 10kg etc. for all drugs required in CPCR. This allows drugs to be drawn up quickly in a crash situation.

Cardio-pulmonary and cerebral resuscitation (CPCR)

Until recently there have been no specific evidence based guidelines for CPR have been available for veterinary medicine although recommendations have been published based on human clinical guidelines. The main goal of the Reassessment Campaign on Veterinary Resuscitation (RECOVER) initiative was to develop a set of clinical consensus guidelines for the practice of CPR in dogs and cats.

Our aim when performing CPCR is to achieve a return to spontaneous ventilation and circulation. We are also aiming to maximize perfusion to the heart and brain.

Speed is of the essence when faced with a patient who has suffered CPA. Survival from CPA is improved where resuscitation attempts are well organized, cohesive and led by a well-functioning knowledgeable team (Fletcher et al. 2012). It is advisable for practices to provide regular effective and standardized staff training in CPCR techniques so that vets and nurses are capable of delivering basic life support techniques. CPCR will be ineffective if performed by only one person so a well-trained team is essential.

As soon as a CPA occurs **turn off the anaesthetic** (if applicable), notify the surgeon and call for help, get someone to hit the alarm button if you have one, and begin initial basic life support procedure:

C – Circulation
A – Airway
B – Breathing

Consider peri-operative drugs that may have contributed to CPA. Reverse opioids using naloxone, benzodiazepines using flumazenil and medetomidine/ dexmedetomidine with atipamazole.

Circulation (C)

If the patient is in cardiac arrest then compressions should be started immediately and not stopped until spontaneous circulation returns or a decision is made to cease.

Closed cardiac compressions

Cardiac pump mechanism:

- Patients weighing less than 15Kg.
- Chest is compressed directly over the heart

- This action directly compresses the ventricles to create blood flow.
- Right lateral recumbency
- Heel of one or both hands compresses the chest at the fifth intercostal space directly over the heart.
- Smaller patients - use the thumb and forefingers either side of the chest.

Compress the chest by approx one third to one half and avoid excessive pressure which may lead to intrathoracic trauma.

Thoracic pump mechanism:

- Larger patients, over 15KG, or barrel chested breeds such as the bulldog.
- Lateral or dorsal recumbency.
- Compress the chest by approx one third to a half
- Compress at the widest point of the chest
- Increase in thoracic pressure causes forward blood flow into the arteries.
- Back flow is prevented by the AV valves and collapsing of the veins due to the pressure.
- The relaxation of the chest following compression and the subsequent drop in pressure facilitates venous return.

For both mechanisms the person performing compressions should be situated so that they are above the patient's chest.
Chest compressions should be performed at a rate of approx 100 -120 compressions per minute, depending on the size of the patient, with a 1:1 ratio of compression to relaxation.
The effectiveness of the chest compressions should be monitored by a person with their finger on a pulse. Effective compressions should produce a palpable pulse and pink mucous membranes.
To monitor pulmonary perfusion a capnograph should be utilized. If there is adequate delivery of blood to the lungs due to effective chest compressions then gaseous exchange will occur due to artificial ventilation resulting in increasing end tidal CO_2 levels being displayed on the capnograph. An end tidal CO_2 level above 14mmHg is a sign of good CPCR technique.

Open Chest CPCR

Indications for open chest CPCR include:

- If the chest cavity or abdominal cavity are already exposed e.g. thoracotomy or laparotomy
- Pneumothorax
- Haemothorax
- Severe chest trauma including penetrating chest wounds
- Fractured ribs
- Diaphragmatic hernia
- Flail chest

- Pericardial tamponade
- Severe hypovolaemia
- Deep chested dogs and obese patients where closed chest CCPR will not create high intrathoracic pressure
- Coagulopathies
- Septic, anaphylactic or distributive shock
- Other primary thoracic diseases e.g. neoplasia, foreign body
- If closed chest CCPR is not effective within 2-5 minutes (this last point is controversial and the time at which the chest should be entered following unsuccessful closed CPCR is the subject of much debate).

    - Mini thoracotomy is required!
    - The pericardium is torn at the apex of the heart and the heart directly compressed.
    - The compression rate remains the same as with closed compressions at approximately 100 per minute.
    - Having the chest open allows direct visualization of ventricular filling and heart rhythm
    - Open chest CPCR will require a surgical team on standby
    - Risk of sepsis, cardiac trauma, cardiac dysrhythmias and ventricular fibrillation.

Airway management (A)

Quickly establish a patent airway. The most common and effective method is endotracheal intubation. Check existing tubes to ensure they have not become dislodged or blocked. If the patient is not already intubated quickly place an endotracheal tube, secure in place and inflate the cuff if necessary. It may be necessary to suction the airway to remove any mucus, regurgitated ingesta or foreign material present.

If the patient cannot be intubated due to respiratory obstruction then oxygen may be provided in a number of ways until an emergency tracheostomy can be performed:

- An oxygen cannulae or urinary catheter may be able to pass the obstruction to allow oxygen administration
- An over the needle catheter can be placed between tracheal rings distal to the obstruction to allow oxygen to be insufflated.

Breathing (B)

Intermittent positive pressure ventilation (IPPV) should be commenced with 100% oxygen using an appropriate anaesthetic breathing system.

- Initially give the patient two breaths 1-2 seconds in duration and then assess for signs of spontaneous ventilation.
- If spontaneous ventilation does not occur then ventilations should continue at a rate of 10-12 bpm.

Cats, neonates and patients with restrictive lung disease will require smaller tidal volumes of 6-10ml/kg at higher respiratory rates 12-15bpm.

Doxapram administration is contraindicated in patients with respiratory arrest as it decreases cerebral perfusion and increases cerebral oxygen consumption and requirement (Plunkett and McMichael 2008).

Advanced life support

This includes further steps with the aim to establishing and maintaining spontaneous ventilation and circulation via the administration of drugs, appraisal of the electrocardiogram (ECG) and further interventional techniques such as defibrillation.

Drugs (D)

See table for commonly used drugs for CPCR

Atropine/ glycopyrolate:

Anticholinergics can be used to treat vagally induced bradycardias and in the treatment of ventricular asystole. Effective at reducing the effects of parasympathetic stimulation and cholinergic responses and act to increase heart rate, control hypotension and increase systemic vascular resistance. Excessive doses may produce sinus tachycardia and can predispose the myocardium to ventricular dysrhythmias.

Adrenaline (Epinephrine):

Adrenaline is a mixed adrenergic agonist and catecholamine. Drug for treatment of severe bradycardia that is unresponsive to atropine/glycopyrolate, severe hypotension and cardiac arrest. During CPCR adrenaline is administered for its α agonist effects which cause peripheral vasoconstriction thus increasing coronary and cerebral perfusion. It is believed that adrenaline is less effective in hypoxic, acidotic states. The $\beta_1$adrenergic effects of adrenaline may be harmful when treating CPA due to increased myocardial oxygen demand, exacerbating myocardial ischemia and may lead to ventricular dysrhythmias if overdosed.

Vasopressin:

Vasopressin is a nonadrenergic pressor that causes vasoconstriction. Unlike adrenaline it is not affected by acidosis and may be effective in cases where adrenaline fails. It works by stimulating V1 receptors in the smooth muscle of vessel walls and appears to cause more vasoconstriction in peripheral tissues than in the coronary and renal vasculature promoting perfusion of these areas. It is also thought to provide a dilatory effect in the vessels supplying the brain resulting in increased perfusion to this area.

Lidocaine:

A class 1b antiarrhythmic (sodium channel blocker) indicated for use in cases of atrial fibrillation, some forms of supraventricular tachycardia, ventricular tachycardia and refractory ventricular fibrillation unresponsive to CPCR techniques (inc defibrillation and pressor therapy). This drug has been largely superseded by amiodarone in human medicine and this is now the drug of choice for refractory ventricular fibrillation secondary to defibrillation.

Routes of drug administration during CPCR

The routes are listed in order of preference.

- Central venous route:
- Peripheral venous route:
- Intraosseous route:
- Intratracheal route:
- Intralingual:
- Intracardiac:

Electrocardiography (E)

It is essential to monitor the heart rhythm immediately following CPA and during CPR, this is achieved through use of an ECG. It allows monitoring of response to treatment and evaluation of the appropriate drug therapy and/or treatment to be delivered according to the rhythm. Note: a normal ECG appearance is not indicative of contractile function of the myocardium or peripheral perfusion. Spirit must NOT be used when placing an ECG on a CPA patient as defibrillation may be required.

The common arrest rhythms in veterinary patients are asystole, ventricular fibrillation and pulseless electrical activity (PEA).

Asystole:

A flat line will be seen on the ECG indicating the absence of mechanical and electrical activity.
Always check the patient's pulses and ECG connections before you assume your patient is asystolic!
Treatment requires rapid basic life support measures with minimal interruptions. No medications have been shown to be effective in the treatment of asystole (Plunkett and McMichael 2008). However atropine/glycopyrrolate and adrenaline/vasopressin remain the drug treatments of choice for this condition.

Ventricular fibrillation:

An irregular quivering of the ventricles with no effective cardiac output. The ECG will demonstrate fibrillation waves with no QRS complexes. There will be no palpable pulse. Direct current

cardioversion to defibrillate the heart is the recommended treatment. Chest compressions should be performed whilst the defibrillator is being prepared.

Defibrillation may be attempted externally with paddles which must be covered with conductive gel to prevent burning of the patient. The initial counter shock is delivered at 2-5J/Kg. Chest compressions should immediately be resumed for 2 min before reassessing the cardiac rhythm and administration of an additional shock.

Adrenaline is generally administered every 3-5 minutes if ventricular fibrillation continues after the initial countershock and subsequent 2 minutes of compressions. Lidocaine cannot convert the heart from ventricular fibrillation to normal sinus rhythm but is used to help control ventricular dysrhythmias following defibrillation.

Pulseless Electrical Activity (PEA):

Previously referred to as electrical mechanical dissociation. PEA is used to describe patients which have ECG evidence of cardiac rhythm but with an absent or very weak pulse. The waveform can range from having relatively normal QRS complexes to quite wide and bizarre complexes. Treatment involves correction of the underlying cause if possible and rapid commencement of basic life support techniques. Again no medications have been shown to be effective and the prognosis is poor for successful resuscitation (Plunkett and McMichael 2008). However adrenaline/vasopressin is still administered in PEA cases.

Fluid Therapy (F)

If CPA is due to hypovolemia then aggressive fluid therapy may be necessary. Shock rate fluids generally not administered unless the patient was hypovolemic prior to CPA. Fluid resuscitation should be approached cautiously in patients whose volume status was normal prior to arrest especially those with pre-existing lung/cardiac disease.

Prolonged life support

When spontaneous circulation and ventilation has been achieved through successful CPCR these patients require intensive treatment to provide continued support of the cardiovascular and respiratory systems. Post resuscitation patients should be carefully monitored. This will involve checking temperature, pulse rate, rhythm and character, heart rate and rhythm, resp rate, depth and effort, lung sounds, mental status and neurologic function, patient comfort, blood pressure, MM colour and CRT and urine output at regular intervals. If not already placed then arterial and jugular catheters should be placed to facilitate monitoring of central venous pressure (CVP) and direct arterial blood pressure. A continuous ECG should be connected for at least the first 24 hours post CPA. Regular blood samples should be taken to monitor packed cell volume (PCV), total solids (TS), blood glucose concentration, serum lactate concentration, electrolyte, acid-base and arterial blood gas status.

O_2 carrying capacity should be maximized by ensuring adequate volume status, blood pressure and haemoglobin concentration. Supplemental O_2 should be supplied initially at 100% immediately post CPCR and then reduced to below 60% to avoid toxicity. Continued ventilation may be required. Nutritional support should be considered and feeding tube placement or parenteral nutrition may be necessary if enteral feeding is contraindicated.

Common complications following CPA and CPCR include: cerebral oedema, hypoxia, reperfusion injury, acute renal failure, Systemic inflammatory response syndrome (SIRS), abnormal haemostatsis, multiple organs dysfunction syndrome (MODS). These patients are often at risk of further CPAs depending on the underlying cause of the initial CPA.

Note: these patients should not be allowed to become hyperthermic and that it is thought that moderate hypothermia may improve outcome.

References

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Further Reading

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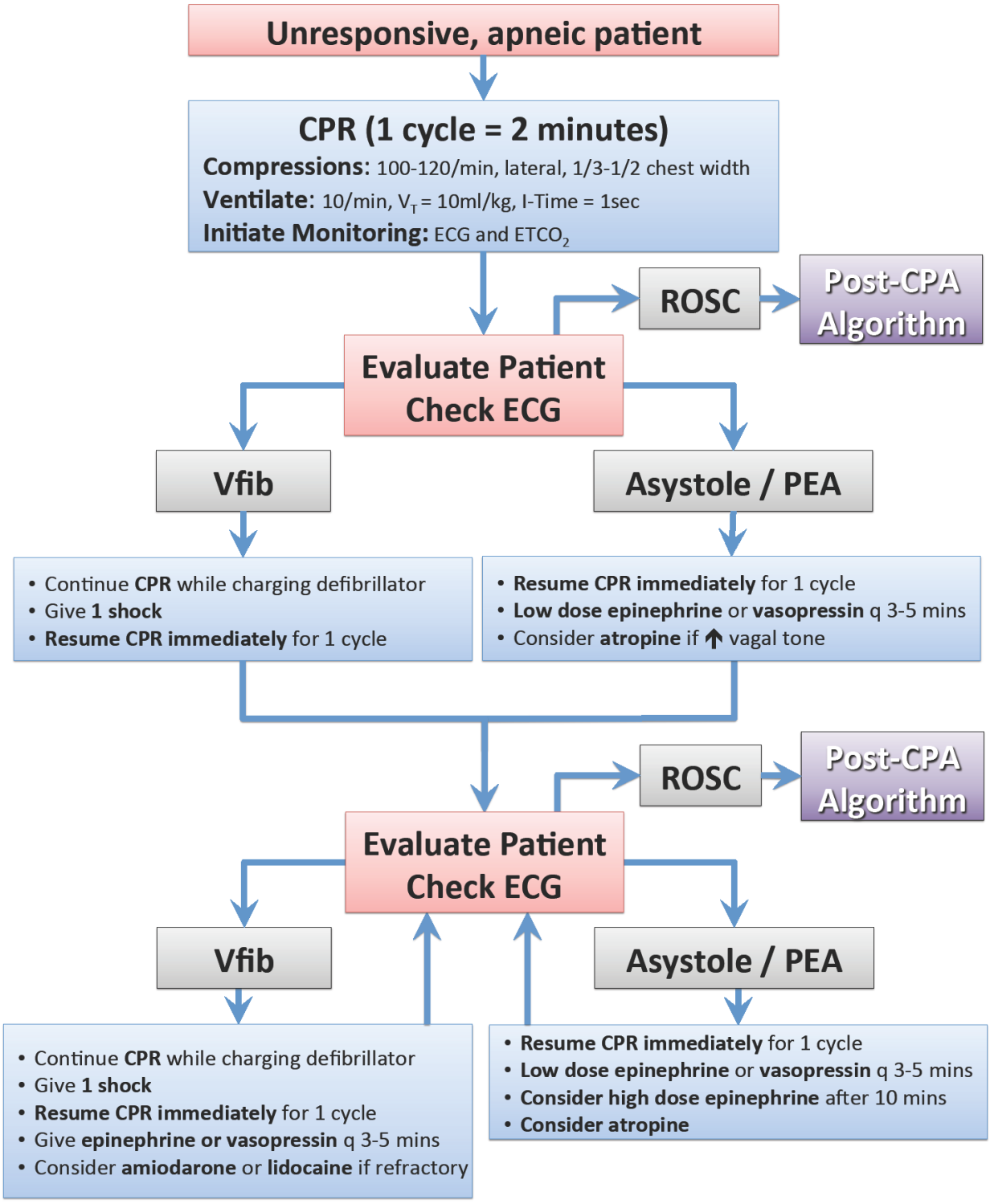
CPR Algorithm



CPR Emergency Drugs and Doses

		Weight (kg)	2.5	5	10	15	20	25	30	35	40	45	50
		Weight (lb)	5	10	20	30	40	50	60	70	80	90	100
	DRUG	DOSE	ml	ml	ml	ml	ml	ml	ml	ml	ml	ml	ml
Arrest	Epi Low (1:1000)	0.01 mg/kg	0.03	0.05	0.1	0.15	0.2	0.25	0.3	0.35	0.4	0.45	0.5
	Epi High (1:1000)	0.1 mg/kg	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
	Vasopressin (20 U/ml)	0.8 U/kg	0.1	0.2	0.4	0.6	0.8	1	1.2	1.4	1.6	1.8	2
	Atropine (0.54 mg/ml)	0.05 mg/kg	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Anti-Arrhyth	Amiodarone (50 mg/ml)	5 mg/kg	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
	Lidocaine (20 mg/ml)	2-8 mg/kg	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Reversal	Naloxone (0.4 mg/ml)	0.04 mg/kg	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
	Flumazenil (0.1 mg/ml)	0.01 mg/kg	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
	Atipamezole (5 mg/ml)	50 µg/kg	0.03	0.05	0.1	0.15	0.2	0.25	0.3	0.35	0.4	0.45	0.5
Defib Monophasic	External Defib (J)	2-10 J/kg	20	30	50	100	200	200	200	300	300	300	360
	Internal Defib (J)	0.2-1 J/kg	2	3	5	10	20	20	20	30	30	30	50