New Guidelines on Veterinary CPR

Whenever a patient goes into cardiopulmonary arrest it is a very stressful situation for the entire practice team. Stress can be minimised by ensuring all staff have a good knowledge of the CPR process and have been well prepared. Access to the correct equipment is essential and provisions should be made to ensure that this is quick to hand. Staff should be well practiced through regular mock CPR sessions, ensuring their skills are up-to-date.

Full cardiopulmonary arrest (CPA) can be defined as ‘the sudden cessation of spontaneous and effective respiration and circulation’. Cardiopulmonary cerebral resuscitation (CPCR) aims to provide circulatory and respiratory support. This should be undertaken whilst efforts are made to recommence the patient’s own circulation - the ultimate goal being the recovery of a neurologically intact animal.

Regrettably published survival rates in veterinary patients following CPCR are low (around 5–10%) and it is apparent from studies that patients fall into two categories; those that arrest due to irreversible causes (i.e. as the end stage of their disease) and those that have reversible causes such as anaesthetic overdoses or electrolyte imbalances. Careful consideration of underlying causes will determine if CPCR is actually appropriate for the individual.

<table>
<thead>
<tr>
<th>Causes of CPA</th>
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<tbody>
<tr>
<td>Cardiac disease</td>
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<tr>
<td>Pulmonary disease</td>
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<tr>
<td>Multi-system trauma</td>
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<tr>
<td>Upper airway obstruction</td>
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<tr>
<td>Traumatic brain injury</td>
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<tr>
<td>Sepsis</td>
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<tr>
<td>Coagulopathies</td>
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<tr>
<td>Severe anaemia</td>
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<tr>
<td>Hypovolaemia</td>
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<td>Hypotension</td>
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6 H’s and 6 T’s are useful abbreviations to remember when considering common reversible causes.

<table>
<thead>
<tr>
<th>H’s</th>
<th>T’s</th>
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<tbody>
<tr>
<td>Hypovolemia</td>
<td>Tablets (drug OD, accidents)</td>
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<tr>
<td>Hypoxia</td>
<td>Tamponade (cardiac)</td>
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<td>Hydrogen ion – acidosis</td>
<td>Tension pneumothorax</td>
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<tr>
<td>Hyperkalemia / Hypokalemia</td>
<td>Thrombosis, coronary (ACS)</td>
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<tr>
<td>Hypothermia</td>
<td>Thrombosis, pulmonary(embolism)</td>
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<tr>
<td>Hypoglycemia and other metabolic disorders</td>
<td>Trauma</td>
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Prompt identification and treatment of the aforementioned will result in the most favourable outcome.

**Immediate Recognition**

Anticipation and awareness of high risk or critical patients will enable identification of a pre-arrest state or an actual arrest as it occurs.
Impending CPA may include a variety of signs that differ depending on the species and the disease process they suffer from. Common signs seen in the pre-arrest setting may include altered mentation through unconscious, bradycardia or high risk tachyarrhythmias, hypotension, hypothermia, dilated to unresponsive pupils, changes in respiratory rate, depth or pattern including agonal respirations, agitation and vocalization. The veterinary nurse is vital in recognising these signs and alerting all staff. Early recognition of CPA and a rapid triage assessment will help the prognosis, facilitating early CPR and preventing onset of irreversible CPA.

**Preparation**

Successful CPCR relies on good preparation and teamwork. Nurses play a critical role in this care as they spend the majority of the time with the patients at risk, so their observations can make all the difference in vital situations. There should be access to a well-stocked crash box, containing all the likely equipment required to run a successful resuscitation attempt (see Table 1). Ideally one member of the team should have the responsibility of checking the crash trolley/box on a daily basis. Necessary resources include a central treatment room/arrest station with a hard surfaced table, stools for compressors to stand on, crash trolley with consumables and medications, oxygen, airway & ventilatory equipment, ECG with or without defibrillation, monitoring equipment, protocol flowcharts and drug dose charts. Maintain equipment and supplies with a daily check list. Team members should practice the necessary techniques on a regular basis so that CPCR can be commenced as quickly as possible. CPR ‘practice’ sessions are essential in having a well-rehearsed, coordinated team and should be carried out on a regular basis.

<table>
<thead>
<tr>
<th><strong>Crash Trolley Contents</strong></th>
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</thead>
<tbody>
<tr>
<td><strong>Endotracheal tubes (range of sizes 2.5mm-14mm)</strong></td>
</tr>
<tr>
<td><strong>Ambu bag</strong></td>
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<tr>
<td><strong>Anaesthetic breathing systems</strong></td>
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<tr>
<td><strong>Laryngoscope</strong></td>
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<tr>
<td><strong>Assorted intravenous catheters</strong></td>
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<tr>
<td><strong>Assorted hypodermic needles</strong></td>
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<tr>
<td><strong>Lactated Ringer's solution</strong></td>
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<tr>
<td><strong>Pressure bag for rapid infusion of fluids</strong></td>
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<tr>
<td><strong>50% dextrose solution</strong></td>
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</tbody>
</table>
Adhesive tape
Polyurethane urinary catheters
3-way taps
Tracheostomy tubes (range of sizes)
Tracheostomy kit
Chest drains
Thoracostomy kit
Clippers
4% chlorhexidine gluconate or 10%
povidine-iodine
70% surgical spirit
Electrocardiogram monitor, leads, clips
and conduction gel
Defibrillator
Drugs
Sterile gauze swabs
Suture material
Bandage materials

Table 1: Crash trolley contents

The resuscitation team

CPR requires a team of individuals operating efficiently and with excellent communication. A minimum of 2 people are required for effective CPCR. The ideal team consists of: (1) team leader, (2) compressor, (3) breather. If additional personnel are available, the following additional roles may be assigned: (4) drug administrator, (5) recorder. The team leader is the individual directing the CPCR process. The compressor is responsible for chest compressions and should rotate every 2 minutes with the breather to prevent tiredness and ineffective technique. The breather is responsible for ventilations and should rotate every 2 minutes with the compressor. The drug administrator draws up and administers drugs as directed by the leader, and should anticipate needs and draw up drugs that may be required in advance. The recorder maintains a record of everything that is done including timing, monitors cycles of CPR (every 2 minutes) and announces end of each cycle, and keeps track of frequency of drug administration, suggesting when administration should occur. Effective communication is essential for a successful resuscitation effort. Roles of all team members must be clearly defined. A leader must be identified immediately, and
subsequently quickly assigns roles to the other team members. Clarity of communication is another key concept. All messages should be specifically directed at an individual, and requests should be clearly and succinctly stated. Orders should not be given “to the room”, but directed at an individual, with that individual responding that he/she understands.

**Early CPR**

Resuscitation at the cellular level requires oxygen delivery to the vital organs. In recent years the sequence of Airway-Breathing-Circulation has been changed to Circulation-Airway-Breathing in all but known asphyxial causes. This means compressions are to be initiated first and foremost. Supporting arguments for this change in sequence to C-A-B state that during low blood flow states such as CPR, oxygen delivery to the heart and brain is limited by blood flow rather than by arterial oxygen content. Therefore, compressions are more important than ventilations during the first few minutes of resuscitation. Additionally, chest compressions cause air to be expelled and oxygen to be drawn in passively through the elastic recoil of the chest. This, in theory, may help maintain a higher arterial oxygen saturation until positive pressure ventilation can be initiated. Routine pulse checks are no longer advisable due to the difficulty in assessing the absence or presence of a pulse, even for experienced clinicians. It is recommended that no longer than 10 seconds should be spared between compressions. Remember that the lack of valves in the inferior vena cava allows retrograde blood flow within the venous system and therefore may produce pulsations which have no clinical relevance.

**Basic CPR procedure:**
- Push hard and fast --- depress 1/3 chest diameter, at least 100 compressions/minute
- Allow complete chest recoil
- Minimize interruptions in compressions --- not more than every 2 minutes, < 10 second duration
- Rotate compressors every 2 minutes
- 30:2 (compression to ventilation) ratio until advanced airway in place
- Avoid excessive ventilation rates --- 8-10 breaths/minute (q. 6-8 seconds)

These guidelines have been formulated to optimize blood flow with the intent of perfusing the heart and brain.
During CPR efforts, cardiac output is approximately 25-33% of normal, so optimizing your compressions is important. Compressions are performed using two different methods based on your patient size. In smaller patients (<7kg), it is feasible to directly compress the heart (cardiac pump theory) between the ribs. In larger patients, forward blood flow is related to changes in intrathoracic pressure that is transmitted to the major vessels (thoracic pump theory). Place your patient on a rigid surface, generally in lateral recumbency, and supported as needed to provide a stable compression surface. Dorsal recumbent positions may be used dependent upon a patient’s chest conformation. For small patients, place your hands directly over the heart for compressions at a rate of at least 100/minute. In larger animals, hand position is at the widest part of the thorax, lock your elbows (this is important to ensure maximal pressure is applied) and apply even pressure through the heel of your hand. Compressors will probably have to stand on a footstool in order to gain sufficient height to perform compressions effectively. “Push hard and fast” means a 1:1 ratio of compression to relaxation, allowing for complete chest recoil. Incomplete chest recoil has been associated with increased intrathoracic pressure and decreased perfusion. Compressions should be performed in two minute cycles with the break in between each cycle < 10 seconds. This break time is used for checking pulses, ECG assessment, defibrillation or performing a difficult intubation or catheter placement. After each two minute cycle, rotate compressors. Fatigue is commonly seen in compressions performed by a person for longer than a two minute cycle, which leads to decreased effectiveness.

Interposed abdominal compression is a 3-rescuer technique that includes conventional chest compressions combined with alternating abdominal compressions. The dedicated rescuer who provides manual abdominal compressions will compress the abdomen midway between the xiphoid and the umbilicus during the relaxation phase of chest compression. Hand position, depth, rhythm, and rate of abdominal compressions are similar to those for chest compressions. This technique has been shown to increase aortic diastolic pressure and venous return, resulting in improved coronary perfusion pressure. It is a viable technique to use in our veterinary patients when an extra dedicated person is available and all are well trained in performing it. This technique should not at any time impair the quality of 2-person CPR and is contraindicated with abdominal diseases, trauma or recent surgery.

In open-chest CPR, the heart is accessed through an emergency thoracotomy and compression is performed using thumb and fingers or two hands to compress, at a rate of up to 150/minute. Use of this technique generates forward blood flow and coronary perfusion pressure that typically exceeds those generated by closed chest compressions. This
technique is indicated and may be the superior technique for CPA involving pericardial tamponade, pleural space disease, penetrating chest injuries or any chest wall trauma, giant breed dogs or recent thoracic surgery. The emergency approach is at the left 6th intercostal space and using rib spreaders to maintain access. An approach through the diaphragm is available during abdominal surgery.

Excessive ventilation has been shown to have a detrimental effect, resulting in increased intrathoracic pressure and decreased perfusion. Because cardiac output is lower than normal during cardiac arrest, the need for ventilation is reduced. Until an ET tube or other advanced airway is established, ventilate at 2 breaths following 30 compressions and maintain that ratio. Deliver a tidal volume sufficient to produce a visible chest rise, each breath is given over 1 second. Once an advanced airway is in place, ventilate at a rate of 8-10 breaths per minute (every 6-8 seconds) using 100% oxygen, without a pause in compressions. Hyperventilation should be avoided as this can result in cerebral and coronary vasoconstriction and worsens ischemia. When ventilating with a pressure gauge, do not exceed 20cmH2O.

The advanced airway of choice is still an endotracheal tube; however, with the new regulations we are challenged with intubating patients in lateral recumbency during compressions. If unable to do so, delay intubation until the end of a two minute compression cycle and then intubate within the 10 second time limit. Preparation of supplies and a team approach will make this possible. Confirmation of the airway can be made by bilateral thoracic and stomach auscultation, visualization of tube in the trachea, direct palpation via the oropharynx, or capnometry. Neck palpation of “2 tubes” indicates probable oesophageal intubation, the trachea and the ET tube positioned in the oesophagus. Rescue breathing post resuscitation should be initiated at 10-12 per minute (every 5-6 seconds).

While basic CPR is the foundation of resuscitation, it is one part in the entire chain of survival. A team approach in which members have pre-planned tasks for performing additional therapies is necessary. This enables adding advanced life support measures and interventions without compromising high-quality CPR.

**Monitoring during basic life support and advanced life support**

Monitoring modalities during CPR fulfils multiple purposes:

1. Assessing the patient’s response to CPR
2. Assessing the quality of CPR delivered by the rescuer(s)
3. Triggering ALS interventions such as defibrillation
4. Identifying possible causes of CPA
5. Recognizing return of spontaneous circulation
6. Suggesting continuation vs. discontinuation of the resuscitation effort

There is unfortunately no one method of monitoring CRP which can fulfil all the above goals. The most useful equipment we have available is capnography and electrocardiogram.

If patients are intubated and being manually ventilated, capnography is a useful tool in assessing the efficacy of CPR. End-tidal CO2 (ETCO2) values provide a very close estimate of the alveolar CO2 partial pressure (VCO2), which in turn is determined by the CO2 production, pulmonary capillary blood flow (cardiac output) and CO2 elimination (alveolar ventilation). ETCO2 can be used as a measure of thoracic compressions generated pulmonary blood flow, and therefore the efficacy of CRP in generating blood flow to the brain and heart. This is not quite that straightforward as there are other variables, such as the amount of ventilation delivered. Ideally minute ventilation should be administered at a constant rate to make the best use of ETCO2 as a measure of CPR efficacy. Studies in humans suggest an ETCO2 <10mmHg is 100% predictive for non-survival. In a veterinary study by Hofmeister et al. they found only 6% of dogs with peak ETCO2 <15mmHg achieved return of spontaneous circulation (ROSC).

The following is therefore suggested as a guideline for CPR if ETCO2 is <15mmHg:

1. Push harder;
2. Push faster, with the aim of 100-120 compressions/minute;
3. Avoid ‘leaning’ on the patient, i.e. allow the chest to fully expand following each compression;
4. Avoid hyperventilation, to limit the duration of positive intrathoracic pressure;
5. Change ‘compressor’ every 2 minute to avoid fatigue related deterioration of chest compression quality;
6. Minimise interruptions in chest compressions
7. If ETCO2 is <15mmHg despite optimal CPR technique, try a different CPR method, e.g. open chest CPR.
8. If ETCO2 remains <10mmHg despite optimal CPR, discontinuation of the resuscitation attempt should be considered.

ETCO2 is a very sensitive indicator of ROSC and will almost immediately increase upon return of spontaneous circulation.
The electrocardiogram (ECG) allows the diagnosis of the arrest rhythm, the most common being asystole, pulseless electrical activity (PEA) and ventricular fibrillation (VF)/ventricular tachycardia (VT). The ECG is therefore used as a basis for decisions regarding medical management requirements and/or defibrillation (if available).

**Effective Advanced Life Support**

Advanced life support (ALS) therapies are meant to enhance Basic Life Support (BLS) efforts and improve the chance of successful resuscitation. These therapies include intravenous (IV) or intraosseous (IO) catheterization, ECG rhythm interpretations to guide drug therapy in addition to defibrillation, advanced airway management, physiologic monitoring and any other therapies tailored to a specific cause of a cardiac arrest patient. Obtaining venous access in patients in cardiopulmonary arrest can be challenging and time-consuming. Access is usually limited due to ongoing compressions and the patient’s state of vascular collapse. If IV access is not obtained quickly, IO access should be pursued. In general, this means 1-2 attempts at IV catheterization. With newer IO delivery systems on the market, IO access is quick and easy for any size patient and provides access to a non-collapsible venous plexus, enabling drug delivery similar to that achieved by peripheral venous access at comparable doses. Intratracheal is no longer recommended due to unreliable absorption, however may still be used if IV or IO access is not available. Drugs given via a peripheral IV site should be followed with a 0.9% saline flush of 2-20ml, depending on patient size, to facilitate movement into central circulation. Routine use of crystalloid fluid boluses is no longer recommended unless hypovolemia is a pre-existing condition or there is ongoing volume loss during resuscitation. Studies showed that excessive fluid resuscitation can actually decrease perfusion.

**Integrated Post-Cardiac Arrest Care**

In the post-cardiac arrest phase, the chance of re-arrest is high (68% dogs, 37% cats). Post-resuscitation requires intensive monitoring and aggressive supportive care. Maintaining adequate ventilation and oxygenation can be achieved with the use of monitoring waveform capnography, using caution not to hyperventilate and maintaining oxygen saturation >94%. Permissive hypercapnia in the post-resuscitation phase is not recommended. Hypotension is treated with IV or IO boluses of the appropriate fluid choices and vasopressors.
Therapeutic hypothermia used in the first 12-24 hours post-resuscitation (32°-34°C) may improve neurologic outcome. The efficacy of therapeutic hypothermia in improving neurological outcome following ischaemic brain injury in both humans and dogs has been proven. Active rewarming of post arrest patients should be avoided and passive rewarming carried out at a rate no faster than 0.5°C/hour. The period of hypoxia and ischaemia, no matter how short, will result in metabolic acidosis and reperfusion injury to multiple organ systems. Treatment for cerebral oedema and seizures is often necessary. Careful monitoring of CNS signs (mentation), heart rate and rhythm (ECG), peripheral pulse including arterial blood pressure measurement and palpation, packed cell volume, total protein/solids, arterial blood gas and acid-base parameters, electrolytes, and urine production are all essential. Patients should be carefully monitored for seizures and aggressively treated if they occur. Mechanical ventilation may be required for many hours post resuscitation in order to gain a successful outcome, and so the process of monitoring these patients is very demanding.

**Drugs in CPR**

In our small animal veterinary patients, the most common initial ECG rhythm is ‘pulseless electrical activity’ (PEA) formerly known as ‘electromechanical dissociation’ (EMD) – 23% of arrests. Also regularly seen are asystole (22%) and VF (19%) with sinus bradycardia in the pre-arrest environment being seen approximately 19% of the time. Atropine is an anti-cholinergic that counteracts the high vagal tone decreases in heart rate and atrioventricular nodal conduction seen in CPA. Atropine is still recommended for symptomatic sinus bradycardia. The past doses recommended for PEA/Asystole are 0.02-0.04mg/kg IV or IO q.3-5 minutes up to 3 doses. The dose for symptomatic bradycardia is usually 0.01-0.02 mg/kg IV.

Vasopressors are used adjunctively in CPR to increase aortic and diastolic pressures and therefore improve cerebral and myocardial perfusion pressures. They are indicated in PEA, asystole and refractory VF. Epinephrine is still most commonly used primarily because of its alpha-adrenergic (vasoconstrictor) properties that can increase coronary and cerebral perfusion pressures during CPR. The safety of epinephrine remains controversial because of the beta-adrenergic induced increase in myocardial oxygen demand. Based on this, dosing has changed back to using low doses at 0.01-0.02 mg/kg IV or IO. Continued use at 3-5 minute intervals is still recommended. Vasopressin is an alternative vasopressor currently recommended. It works via direct stimulation of receptors in vascular smooth muscle, has no beta-adrenergic effects and also retains effectiveness in the acidic and hypoxic environment of CPA where epinephrine will lose its vasoconstrictor effects. For these reasons AHA guidelines list vasopressin as an acceptable alternative to epinephrine. Vasopressin may be
used in place of, or alternated, with epinephrine doses. The dose is 0.2- 0.8 U/kg IV or IO q. 3-5 minutes.

Antiarrhythmics, in general, have not been shown to increase chance of survival when used for VF or pulseless VT refractory to defibrillation. Amiodarone is an antiarrhythmic that showed promise in certain clinical trials, however, is not viable in our veterinary setting. There are two different IV formulations of the drug; one has vasoactive solvents that cause hypotension and bradycardia unless delivered as a very slow infusion and the other one without these solvents is cost-prohibitive. Lidocaine is an antiarrhythmic that is widely known and used. It is thought to decrease the fibrillation threshold, however, may increase the incidence of asystole after defibrillation. It is only recommended for VF refractory to electrical defibrillation and in the absence of amiodarone. The dose is 2-4 mg/kg IV or IO (cats --- 0.2mg/kg). Magnesium sulfate is recommended only in the presence of torsades de pointes which is a polymorphic VT with a prolonged QT interval. Amiodarone and lidocaine are most useful as antiarrhythmics in the post-arrest setting. Routine use of either calcium or sodium bicarbonate is not recommended in CPA. However, in certain situations involving preexisting metabolic acidosis, hyperkalemia, or tricyclic antidepressant overdose, sodium bicarbonate can be beneficial.

References


