An Update on Veterinary CPR

Veterinary protocols for cardiopulmonary resuscitation (CPR) have been largely adapted from those promoted by the American Heart Association for people. The Reassessment Campaign on Veterinary Resuscitation (RECOVER) recently completed a systematic review of the literature relevant to veterinary CPR and developed the first evidence-based, consensus CPR guidelines for small animals (http://www.acvecc-recover.org). This web site contains thorough worksheets documenting the literature supporting the recommendations contained in this document.

BASIC LIFE SUPPORT (BLS)

Basic Life Support (BLS) should be initiated as quickly as possible following diagnosis of CPA using the Circulation, Airway, Breathing (CAB) concept. Circulation should be addressed first, as ventilation will be ineffective if there is no cardiac output, and evidence suggests that outcome worsens as delay to the initiation of chest compressions increases.

Circulation: Chest Compressions

Patients with CPA have no forward blood flow out of the heart and no delivery of oxygen to the tissues. An immediate consequence is the exhaustion of cellular energy stores, cell depolarization and thus loss of organ function. This quickly results in increasing severity of ischemic organ injury and sets the stage for escalating reperfusion injury upon reinstitution of tissue blood flow. The initial goals of chest compressions are to provide (1) pulmonary blood flow for oxygen uptake and CO2 elimination, and (2) tissue perfusion for oxygen delivery to restore cellular metabolic activity. Experimental evidence suggests that even well-executed external chest compressions produce at best 30% of normal cardiac output, making proper technique critical. Chest compressions should be started as soon as possible after diagnosis or suspicion of CPA. Delay in the start of high quality chest compressions reduces the likelihood of return of spontaneous circulation (ROSC). Chest compressions should be done with the dog or cat in lateral recumbency with a compression depth of 1/3-1/2 the width of the chest at a rate of 100-120 compressions per minute regardless of size or species. Use of aids to ensure correct compression rate, such as a metronome or a song with the correct tempo (e.g., “Staying Alive”) is recommended. Leaning on the chest between compressions must be avoided to allow full elastic recoil. Chest compressions should be delivered without interruption in cycles of 2 minutes, and a new compressor should take over after each cycle to reduce the effect of rescuer fatigue. Any interruption in compressions should be as short as possible, as it takes approximately 60 seconds of continuous chest compressions before coronary perfusion pressure (CPP) reaches its maximum. CPP in turn is a critical determinant of myocardial blood flow and the likelihood of ROSC.

The physiology of blood flow generation is fundamentally different during CPR compared to spontaneous circulation. Two distinct theories exist to explain how chest compressions lead to systemic blood flow. The cardiac pump theory is based on the concept that the left and right ventricles are directly compressed, increasing the pressure in the ventricles, opening the pulmonic and aortic valves and providing blood flow to the lungs and the tissues respectively. Recoil of the chest between compressions due to the elastic properties of the rib cage creates negative pressure within the chest, improving filling of the ventricles before the next compression. The thoracic pump theory is based on the concept that external chest compressions raise overall intrathoracic pressure, forcing blood from intrathoracic vessels into the systemic circulation, with the heart acting as a passive conduit. Given the chest wall stiffness in medium and large dogs, blood flow generated by the thoracic pump mechanism likely predominates in these patients. Therefore, it is recommended
that the chest be compressed over the highest point on the lateral thoracic wall with the patient in lateral recumbency (i.e., the widest part of the chest). In contrast, in very keel chested dogs (e.g., Doberman Pinschers, sight hounds); it is reasonable to do chest compressions directly over the heart as the cardiac pump mechanism likely predominates. In markedly barrel chested dogs (e.g., English Bulldogs), compressions over the sternum with the patient in dorsal recumbency may be more effective in eliciting the thoracic pump mechanism than lateral chest compressions. In these and other large dogs with low chest compliance, considerable compression force is necessary for CPR to be effective. The compressor should maintain locked elbows with one hand on top of the other, and the shoulders should be directly above the hands. This allows compressions to be done using the core muscles rather than the biceps and triceps, reducing fatigue and maintaining optimal compression force. If the patient is on a table and the elbows cannot be locked, a stool should be used or the patient should be placed on the floor.

Figure 1: Chest compressions in medium to large breed Dogs Left – round chested; Right – keel chested; Above – barrel chested

Most cats and small dogs tend to have higher thoracic compliance and narrower chests than larger dogs, making the cardiac pump mechanism achievable in these patients; therefore, chest compressions should be done directly over the heart. Compressions may be performed using the same two-handed technique as described above for large dogs, or may be done using a single handed technique where the compressing hand is wrapped around the sternum and compressions are achieved from both sides of the chest by squeezing. Circumferential compressions of the chest using both hands may also be considered.
Airway and Breathing: Ventilation

If an endotracheal tube and laryngoscope are available, the patient should be intubated as soon as possible. Both dogs and cats can be intubated in lateral recumbency, so chest compressions should continue during endotracheal tube placement. If an endotracheal tube is not readily available, mouth to snout ventilation will provide improved oxygenation and CO\textsubscript{2} removal. The patient’s mouth should be held closed firmly with one hand. The neck is extended to align the snout with the spine, opening the airway as completely as possible. The rescuer makes a seal over the patient’s nares with his/her mouth and blows firmly into the nares to inflate the chest. The chest should be visually inspected during the procedure and the breath continued until a normal chest excursion is accomplished. An inspiratory time of approximately 1 second should be targeted.

In non-intubated patients ventilated using the mouth to snout technique, ventilation cannot be performed simultaneously with chest compressions. Therefore, 30 chest compressions should be delivered, immediately followed by two breaths. Alternating compressions and ventilations should be continued for 2-minute cycles, and the rescuers rotated every cycle to prevent fatigue. Chest compressions and ventilations should be performed simultaneously in intubated patients because the inflated cuff of the endotracheal tube allows alveolar ventilation during a chest compression and interruptions in chest compressions are minimized. Intubated patients should be ventilated at a rate of 10 breaths per minute with an inspiratory time of approximately 1 second. If a spirometer is available, a tidal volume of approximately 10 ml/kg should be targeted. This low minute ventilation is adequate during CPR since pulmonary blood flow is reduced. Care should be taken not to hyperventilate the patient, as low arterial CO\textsubscript{2} tension leads to cerebral vasoconstriction, decreasing oxygen delivery to the brain.

ADVANCED LIFE SUPPORT (ALS)

Once BLS procedures have been implemented, the CPR team should initiate Advanced Life Support (ALS), which includes monitoring, drug therapy, and electrical defibrillation. Drug therapy is preferably administered by the intravenous or intraosseous route. Therefore, placement of a peripheral or central intravenous or intraosseous catheter is recommended, but should not interfere with continuation of BLS.
Monitoring

Many commonly employed monitoring devices are of limited use during CPR due to their susceptibility to motion artefact and the likelihood that decreased perfusion will compromise accurate readings. Low yield monitoring devices include pulse oximeter and indirect blood pressure monitors, including Doppler and oscillometric devices. The two most useful monitoring devices during CPR are the electrocardiogram (ECG) and end tidal CO2 monitor (ETCO2).

Electrocardiogram (ECG)

Although the ECG is highly susceptible to motion artefact and is of limited use during ongoing chest compressions, an accurate rhythm diagnosis is essential to guide drug and defibrillation therapy. The goal of ECG monitoring during CPR is to diagnose which of the three most common arrest rhythms are present: (1) asystole, (2) pulseless electrical activity (PEA), or (3) ventricular fibrillation (VF). The ECG should be quickly evaluated while compressors are being rotated between 2-minute cycles of CPR, the rhythm diagnosis should be called out to the group by the team leader, and differing opinions on the diagnosis should be solicited. Discussion about the rhythm diagnosis should not prevent rapid resumption of chest compressions.

End-Tidal Carbon Dioxide (ETCO2)

ETCO2 data can be used in multiple ways during CPR, and regardless of the technology used is highly resistant to motion artefact. The presence of measureable CO2 by ETCO2 monitoring is supportive of (but not definitive for) correct placement of the endotracheal (ET) tube. Because ETCO2 is proportional to pulmonary blood flow, it can also be used as a measure of chest compression efficacy under conditions of constant quality of ventilation. Upon return of spontaneous circulation (ROSC), ETCO2 dramatically increases due to the rapid increase in circulation, and therefore is a valuable early indicator of ROSC during CPR.

Drug Therapy

Depending on the arrest rhythm, the use of vasopressors, parasympatholytics, and/or anti-arrhythmics may be indicated in dogs and cats with CPA. In addition, in some cases the use of reversal agents, intravenous fluids, and alkalinizing drugs may be indicated. Strict adherence to evidence-based CPR algorithms is recommended to increase the quality of CPR, the likelihood of ROSC and the chance of survival to hospital discharge.

Vasopressors

Regardless of the arrest rhythm vasopressors are recommended to increase peripheral vasoconstriction. Because cardiac output is low even during optimal external chest compressions, shunting of blood away from the periphery and towards the core (e.g., the heart, lungs, and brain) is essential to maintain perfusion to these vital organs. Epinephrine (adrenaline) causes peripheral vasoconstriction via stimulation of α1 receptors. It is a nonspecific catecholamine that also acts on β1 and β2 receptors, but the α1 effects have been shown to be the most beneficial during CPR. Initially low doses (0.01 mg/kg IV/IO every other cycle of CPR) are recommended, but after prolonged CPR, a higher dose (0.1 mg/kg IV/IO every other cycle of CPR) may be considered. Epinephrine may also be administered via ET tube (0.02 mg/kg low dose; 0.2 mg/kg high dose) by feeding a long catheter through the ET tube and diluting the epinephrine 1:1 with isotonic saline.
Vasopressin is an alternative vasopressor that exerts its vasoconstrictive effects via activation of peripheral V1 receptors. It may be used interchangeably with epinephrine during CPR at a dose of 0.8 U/kg IV/IO every other cycle of CPR. Potential benefits of vasopressin include continued efficacy in acidic environments in which α1 receptors may become unresponsive to epinephrine and lack of β1 effects (positive inotropy and positive chronotropy), which may cause increased myocardial oxygen consumption and worsened myocardial ischemia upon ROSC. Vasopressin may be administered via ET tube using the technique described above.

Anticholinergics

Atropine is an anti-cholinergic, parasympatholytic drug that has been extensively studied in CPR. Although only a few studies have shown a beneficial effect, there is limited evidence of a detrimental effect, and atropine at a dose of 0.04 mg/kg IV/IO may be considered during CPR in dogs and cats, and is reasonable in all dogs and cats with asystole or PEA associated with increased vagal tone. Atropine may also be administered via ET tube (0.08 mg/kg).

Anti-arrhythmics

Although non-perfusing VF/ventricular tachycardia (VT) should be treated as early as possible with electrical defibrillation, patients with VF refractory to defibrillation may benefit from treatment with amiodarone at a dose of 2.5-5 mg/kg IV/IO. This drug has been associated with anaphylactic reactions and hypotension in dogs, so patients should be closely monitored for signs of peripheral vasodilation, wheals, or hives once ROSC is achieved. Treatment with diphenhydramine (2 mg/kg IM) and/or anti-inflammatory corticosteroids (0.1 mg/kg dexamethasone sodium phosphate IV) is warranted should these signs be noted. If amiodarone is not available, patients with VF refractory to electrical defibrillation may benefit from lidocaine 2 mg/kg slow IV/IO push. Although this drug has been shown to increase the defibrillation threshold in dogs in one study, benefit was evident in others.

Reversal Agents

Although specific evidence of efficacy is not available, the use of reversal agents in dogs and cats in which reversible anaesthetic/analgesic drugs were recently administered may be considered. Naloxone (0.01 mg/kg IV/IO) may be used to reverse opioids, flumazenil (0.01 mg/kg IV/IO) for benzodiazepines, and atipamezole (0.1 mg/kg IV/IO) or yohimbine (0.1 mg/kg IV/IO) for α2 agonists.

Intravenous (IV) Fluids

The routine use of IV fluids in euvolaemic or hypervolaemic patients are not recommended during CPR, but is reasonable in patients with documented or suspected hypovolemia. In euvolaemic or hypervolaemic patients, fluids administered IV serve solely to increase right atrial pressure, which results in decreased perfusion of the brain and heart and should be avoided. However, in hypovolaemic patients, IV fluids will help to restore adequate circulating volume, and will increase the efficacy of chest compressions and improve perfusion.

Corticosteroids

The routine use of high-dose corticosteroids during CPR in dogs and cats is not recommended. Although one retrospective study showed an association between administration of corticosteroids and increased rate of ROSC in dogs and cats, the type and dose of steroids administered were highly variable and the study design did not allow
determination of a cause and effect relationship (Hofmeister et al, 2009). Other studies have shown no benefit or harm from the use of steroids during CPR. Non-CPR studies have demonstrated that single high doses of corticosteroids in dogs frequently lead to gastrointestinal ulceration and bleeding, which could also cause other ill effects such as bacterial translocation. In addition, corticosteroids suppress the immune system and reduce prostaglandin production in the kidney, a primary mechanism employed by the kidney to maintain perfusion in the face of hypotension. Because the documented risks of high-dose steroids far outweigh the potential benefit shown in one study, the use of steroids is not recommended in patients with CPA.

Alkalinization Therapy

In patients with prolonged CPA of greater than 10-15 minutes, alkalinization therapy with administration of sodium bicarbonate (1 mEq/kg, once, diluted IV) may be considered. Prolonged CPA commonly leads to severe acidemia resulting from both metabolic acidosis, due to lactate and uremic acids, and venous respiratory acidosis due to inadequate peripheral perfusion and accumulation of CO2. This acidemia can cause severe vasodilation and inhibition of normal enzymatic and metabolic activity. Because these issues may be rapidly resolved once ROSC is achieved, bicarbonate therapy should be reserved for patients with prolonged CPA and with documented severe acidemia (pH < 7.0) that are not hypoventilating.

Electrical Defibrillation

Early electrical defibrillation in patients with ventricular fibrillation (VF) has been associated with increased ROSC and survival to discharge in numerous studies, and is superior to anti-arrhythmic medical therapy. The goal of defibrillation is to stop the ventricular myocardial cells from contracting by driving them all simultaneously into a refractory period, allowing the pacemakers to take over and drive coordinated contractions of the heart. If the duration of VF is known or suspected to be of duration of 4 minutes or less, chest compressions should be continued until the defibrillator is charged and the patient should then be defibrillated immediately. If the duration of VF is known or suspected to be more than 4 minutes, one full cycle of CPR should be done before defibrillating to allow the myocardial cells to generate enough energy substrate to restore a normal membrane potential, thereby increasing the likelihood of success.

Defibrillators may be either monophasic (delivering a current in one direction across the paddles) or biphasic (delivering a current in one direction, the reversing and delivering a current in the opposite direction). The use of biphasic defibrillators is recommended over monophasic defibrillators because a lower current (and hence less myocardial injury) is required to successfully defibrillate the patient. For monophasic defibrillators, an initial dose of 4-6 J/kg should be used, while biphasic defibrillation should start at 2-4 J/kg. The second dose may be increased by 50%, but subsequent doses should not be further increased.

After defibrillation, chest compressions should be resumed immediately and a full 2- minute cycle of CPR administered before reassessing the ECG and determining if the patient is still in VF and should be defibrillated again. Brief assessment of the ECG immediately after defibrillation to determine if a perfusing rhythm has resulted is reasonable, but should minimally delay resumption of chest compressions.

OPEN CHEST CPR

Direct cardiac massage during open chest CPR generates superior cardiac output compared to closed chest CPR, but is more invasive, expensive, and requires surgical closure and more intensive care if ROSC is achieved. Indications for open chest CPR include pleural
space disease, pericardial disease, flail chest or other significant thoracic wall defects. Direct cardiac massage should also be done in patients undergoing thoracic or abdominal surgery that develop CPA under anaesthesia during the surgery. In the case of abdominal surgery, an incision can be made in the diaphragm and cardiac massage performed from an abdominal approach. Finally, in very large, broad chested dogs, closed chest CPR is unlikely to be effective, and open chest CPR should be initiated as soon as possible if the owner has consented.

PREPAREDNESS

Delays in initiation of CPR for patients with cardiopulmonary arrest (CPA) consistently result in poor outcomes, making it crucial that veterinary practices are well prepared for early recognition of CPA. Studies in human medicine have shown that a combination of didactic CPR training and opportunities to practice psychomotor skills is more effective than either technique alone. Training is recommended for all veterinary personnel who may be called upon to assist in a crisis, including both technicians and veterinarians. Until structured training programs are available in veterinary medicine, practice owners must devise accessible training programs for their staff. Refresher training and drills at least every 6 months have been shown to improve performance in human medicine, and structured assessment and feedback after training maximizes effectiveness.

A fully stocked crash cart routinely audited for content should be available. Because the incidence of CPA in many practices is low, systems should be in place to ensure regular restocking of the crash cart. CPR algorithm charts and emergency drug dosing charts improve adherence to guidelines and individual performance during CPR (See examples in Figures 3 and 4). They should be available in a central location in the practice and reviewed with any staff that may be called upon to acquire drugs for Advanced Life Support efforts.

Following all CPR attempts, debriefing sessions during which team performance is discussed and critically evaluated should be held. The team leader should facilitate the discussion, but all team members should be encouraged to participate and offer their insights.
Figure 3: CPR Algorithm Chart

Figure 4: CPR Drug and Dosing Chart

DIAGNOSIS OF CARDIOPULMONARY ARREST (CPA)
CPA is an important differential diagnosis for any acutely unresponsive patient. It is a clinical diagnosis based on the presence of unconsciousness, lack of breathing and absence of a palpable pulse. Regardless of the clinician’s index of suspicion for CPA in an individual patient, a rapid assessment focused on ruling out CPA should be undertaken immediately in any unresponsive patient. A standardized approach based on evaluation of Airway, Breathing, and Circulation (ABC) will quickly identify the condition and allow immediate intervention should the diagnosis be made. Because the benefits of starting CPR immediately in a patient with CPA far outweigh the risks of performing CPR on an unresponsive patient not in CPA, the clinician should not delay starting CPR in any patient in which there is a suspicion of CPA. If CPA cannot be definitively ruled out, CPR should be initiated immediately rather than further diagnostic assessment. This is important as (1) several studies in human medicine have shown that pulse palpation is an insensitive test for diagnosis of CPA, and this may also be the case in dogs and cats, and (2) a large body of literature supports the notion that even short delays in initiating CPR in pulseless patients reduce the likelihood of successful resuscitation. Therefore, the ABC assessment should take no more than 10-15 seconds to complete.

POST-CARDIAC ARREST CARE

Patient outcome is determined by the events that led to cardiopulmonary arrest and the duration of the cardiac arrest, but also by the processes unfolding during and after reperfusion. Two-thirds of human cardiac arrest victims that achieve ROSC die during the post-resuscitation phase. In veterinary medicine, only 16% of dogs and cats initially successfully resuscitated were found to survive to hospital discharge in one study (Hofmeister et al., 2009). Abnormalities in the post-resuscitation phase result from a combination of anoxic brain injury, post-ischemic myocardial dysfunction, the systemic response to ischemia and reperfusion and the persistent precipitating pathology (e.g. underlying disease processes). Consequently, the clinical abnormalities in these post-cardiac arrest cases are highly variable and therapy should be aimed at alleviating the resulting clinical signs.

Early hemodynamic optimization, similar to algorithms described for severe sepsis and septic shock, has been shown to be effective in human CPA survivors, and should be considered in haemodynamic ally unstable small animals after cardiac arrest. Central venous oxygen saturation of at least 70% and/or normalization of lactate levels should be used as global perfusion metrics and endpoints for resuscitation. Central venous pressure monitoring is also a useful guide for fluid therapy, as left ventricular dysfunction may increase the risk for hydrostatic pulmonary oedema, while ischemia-reperfusion injury can lead to high permeability pulmonary oedema. Cerebral autoregulation of blood flow may be impaired and adequate cerebral blood flow may depend on a sufficiently high cerebral perfusion pressure, requiring a mean arterial pressure of 80 mm Hg or higher.

Mild therapeutic hypothermia (core temperature of 32-34°C [89.6o to 93.2oF]) for 12-24 hours after ROSC improves neurological outcome after CPA in humans and other species, including dogs, that remain comatose 1-2 hours after resuscitation. While therapeutic hypothermia may not be feasible in veterinary patients, rapid active rewarming post-arrest is probably detrimental, and hyperthermia is certainly harmful. It is therefore important to monitor body temperature after CPA. Active rewarming should be avoided and passive rewarming should be limited to at most 0.5°C per hour. Patients should also be carefully monitored for seizures and treated aggressively with diazepam (0.5 mg/kg IV/IO) if they occur.

Short term mechanical ventilation to assure optimal arterial oxygen tension (80 to 100 mm Hg) and CO2 levels (35-40 mm Hg) and to prevent respiratory arrest in the comatose post-cardiac arrest patient is optimal if available, but is not required for patients that are
ventilating sufficiently. In all cases, adequacy of ventilation should be monitored using end-tidal or venous/arterial blood gas analysis. Ample experimental evidence substantiates the detrimental effects of oxidative injury after ischemia-reperfusion, particularly when reperfusion occurs under hyperoxic conditions. Titration of oxygen to a normoxic level (e.g., targeting an SpO2 of 94 to 96%) is a reasonable strategy to reduce oxidative injury while maintaining adequate tissue oxygenation. Additional supportive patient care is determined by the patients' underlying disease processes and by concomitant organ dysfunctions such as ileus and renal failure.

**PROGNOSIS**

There is limited data in the veterinary literature regarding prognosis for patients receiving CPR after cardiopulmonary arrest. Although overall survival rates have been reported to be quite low, the underlying cause of the arrest may contribute significantly. Patients that experience CPA as a consequence of severe, untreatable or progressive chronic diseases are less likely to experience good outcomes. However, peri-anaesthetic CPA carries a better prognosis for survival to discharge (as high as 47% in one recent retrospective veterinary study), and aggressive, prolonged CPR attempts in these cases are reasonable. Adherence to these evidence-based CPR guidelines may help improve the likelihood of survival in these cases.