

Preparation and techniques for cardiopulmonary resuscitation

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Louise O'Dwyer MBA, BSc (Hons), VTS (ECC), DipAVN (Medical and Surgical), RVN, considers the importance of cardiopulmonary resuscitation – developed in the US more than 50 years ago.

DESPITE all the advances in technology and pharmacology, rates of success in cardiopulmonary resuscitation (CPR) have not improved dramatically over the past 50 years. The rates of success in veterinary medicine after cardiac arrest and CPR are around 13 per cent in dogs and 15.4 per cent in cats, and the rate of hospital discharge following successful CPR is less than 16 per cent, making it an unsuccessful treatment option.

In 2011, a group of dedicated specialists and professionals put together the Reassessment Campaign on Veterinary Resuscitation (RECOVER) to publish the first evidence-based consensus CPR guidelines for veterinary medicine, with these results being published in 2012.

RECOVER identified five areas to be investigated:

- preparedness/prevention;
- basic life support (BLS);
- advanced life support (ALS);
- monitoring; and

- post-arrest care.

Preparedness/prevention

RECOVER recognised training the whole veterinary team is critical for success and expecting the unexpected is part of the preparation. This training should include regular drills in the training refreshers.

CPR team

The ideal number of participants in a resuscitation attempt is three to five. In a small practice, when staffing is limited, it may be helpful to train the receptionists and kennel help to be a part of the CPR team. Each person can be taught to carry out a specific task.

In the majority of CPR situations, the team leader will be the veterinary surgeon; however, if the vet is not available, then the person with the most experience in performing CPR should lead the team. Staff will be needed to provide ventilation, chest compressions, establish intravenous (IV) lines, administer drugs, attach monitoring equipment, record the resuscitation effort and monitor the effectiveness of the team's efforts. Practice a protocol for reception, intervention and monitoring for all people in the hospital, including reception staff if necessary.

Ideally, practice CPR sessions should be held monthly. If simulation models are not available, a stuffed animal can be used as the patient during these drills. Each person should understand what his or her responsibilities would be during an arrest situation. After each practice session or true resuscitation, a self-evaluation should be performed, which should involve input from the whole team.

Resuscitation area

One of the first decisions is where will the resuscitation attempt take place. Some people prefer to perform CPR wherever the patient is kennelled and bring the resuscitation equipment and supplies to the patient. Others prefer to designate a specific area in the clinic.

When selecting an area, take into consideration the space available – will there be enough room for a CPR team (three-plus people) and equipment? An oxygen source needs to be readily available. Good lighting is a must; it facilitates endotracheal intubation and visualisation of veins, and, if open-chest massage is attempted, it will allow visualisation of internal structures.

If CPR is to be performed on a table, then the table height should be adjustable. If a table is too tall for the person performing chest compressions, it may be difficult to perform effective compressions. If the height of the table is not adjustable, then a footstool should be made available or CPR should be performed on the floor. If some form of crash cart is not used, then the drugs, ECG, and

defibrillator should be in close proximity. A shelf and a few drawers may be set aside for the emergency supplies.

Crash cart/kit

A crash cart/kit can be as simple as a plastic box or as elaborate as a mobile tool chest. Crash carts/kits help to make the resuscitation attempt more efficient by having all the supplies readily available. If a cart is used, then in addition to the endotracheal tubes, drugs, catheters, syringes and so on, other equipment may be stored on the cart, such as suction machine, ECG and defibrillator. The crash cart or kit should be checked at the beginning of each shift and restocked immediately after each use.

Recognising cardiopulmonary arrest

Existence of cardiac or cardiopulmonary arrest (CPA) must be recognised early to achieve effective, and successful, resuscitation of the patient. The “Airway, Breathing, Circulation” checks (ABCs) should be followed rapidly in the apnoeic unresponsive patient. The absence of a palpable pulse, audible heart sound, or effective ventilation (agonal breaths should not be considered effective breaths), all support the assessment of CPA.

Even under the best of circumstances it may be difficult to palpate a pulse; therefore, not much time should be spent trying to assess pulses. If there is any question CPA has taken place the patient should be treated as such until proven otherwise. If in doubt, commence CPR, do not waste time looking at the patient – checking for pulses, auscultating the thorax or looking for respirations. In patients under anaesthesia, aggressive CPR should be started as soon as possible, since we know patients under anaesthesia have an increased chance of survival over any other patient.

Basic life support

Basic life support (BLS) should be initiated as soon 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 outcome worsens as delay to the initiation of chest compressions increases. This change has come about because we know that resuscitation at the cellular level requires oxygen delivery to the vital organs. This allows for compressions to be initiated earlier on and this is vital when faced with a difficult airway or gathering supplies.

Supporting arguments for this change in sequence to CAB state that during low blood flow states such as in 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.

Circulation: chest compressions

Patients with CPA have no forward blood flow from the heart and, therefore, no delivery of oxygen to the tissues. An immediate consequence is the exhaustion of cellular energy stores, cell depolarisation and loss of organ function. This quickly results in increasing severity of ischaemic organ injury and sets the stage for escalating reperfusion injury should reinstatement of tissue blood flow be achieved.

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 per cent of normal cardiac output; this means correct, effective technique is critical.

Chest compressions should be started as soon as possible after diagnosis or suspicion of CPA. Any 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 one-third to half the width of the chest at a rate of 100 to 120 compressions per minute, regardless of size or species ([Figure 1](#)). Use of aids to ensure correct compression rate, such as a metronome or a song with the correct tempo (for example, *Staying Alive*) is recommended.

Leaning on the chest between compressions must be avoided to allow full elastic recoil; this can be achieved by ensuring the hands are lifted off the patient's chest between compressions. Chest compressions should be delivered interrupted in two-minute cycles; 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.

When comparing spontaneous circulation and the physiology of blood flow during CPR there are major differences. Two theories 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 via external force, thereby increasing pressure in the ventricles, opening the pulmonic and aortic valves and providing blood flow to the lungs and the tissues. Recoil of the chest between compressions, due to the elastic properties within the thorax, creates negative pressure in 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 probably predominates in these patients. Therefore, it is recommended the chest be compressed over the highest point on the lateral thoracic wall with the patient in lateral recumbency (the widest part of the chest).

In contrast, in very keel-chested dogs (such as sighthounds) it is reasonable to do chest compressions directly over the heart as the cardiac pump mechanism probably predominates.

In markedly barrel-chested dogs (for example, 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.

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 (circumferential compressions; [Figure 2](#)) 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

Intubation should be performed as soon as possible. Dogs and cats can be intubated in lateral recumbency, so chest compressions should continue uninterrupted during endotracheal tube placement. If an endotracheal tube is not readily available, mouth to snout ventilation will provide improved oxygenation and CO₂ 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 one 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 two-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 chest compression and interruptions in chest compressions are minimised.

Intubated patients should be ventilated at a rate of 10 breaths per minute with an inspiratory time of approximately one second. If a spirometer is available, a tidal volume of approximately 10ml/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 CO₂ tension leads to cerebral vasoconstriction, decreasing oxygen delivery to the brain.

Advanced life support

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)

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 two-minute cycles of CPR, and the team leader should be informed of the rhythm present. Any discussion

about the rhythm diagnosis should not prevent rapid resumption of chest compressions (Fletcher, 2012a).

End-tidal carbon dioxide (ETCO₂)

The use of capnography is new to CPR. Next to the ECG, ETCO₂ measurement is the only clinical monitoring modality currently recommended by the RECOVER initiative for use during CPR (Fletcher et al, 2012). Uses of ETCO₂ include early indication of cardiopulmonary arrest haemodynamic crisis, correct placement of the endotracheal (ET) tube, assessment of quality of CPR and recognition of ROSC.

Capnography and its use during CPR

Capnography allows the assessment of systemic blood flow or cardiac output. This is because ETCO₂ is proportional ultimately to pulmonary blood flow, so decreased blood flow states will be reflected in the ETCO₂ measurements. It can also be used to measure chest compression efficacy under conditions of constant quality of ventilation. Upon ROSC, ETCO₂ dramatically increases due to the rapid increase in circulation, and therefore is a valuable early detection tool for ROSC. With ROSC, increase in ETCO₂ to a normal or above normal level occurs rapidly, for example, within 30 to 60 seconds (Gudipati, 1988).

Moreover, ETCO₂ positively correlated with coronary and cerebral perfusion pressure during CPR in dogs (Sanders et al, 1985). The use of ETCO₂ measurement can also be used for prognostic information. In a human study, an ETCO₂ of less than 10mmHg after 20 minutes of CPR was shown to be 100 per cent predictive for no ROSC (Callahan et al, 1990).

In another clinical study, ETCO₂ peak values during CPR were examined. Almost none of the dogs with an ETCO₂ less than 15mmHg achieved ROSC, while five out of nine cats with an ETCO₂ of less than 20mmHg failed to be resuscitated (Hofmeister et al, 2009). In reality, this means low ETCO₂ values should initiate steps to improve the quality of CPR, such as increasing chest compression depth, compression technique, including hand position/location, avoiding leaning on the patient's chests between compressions, and compressions rate.

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Route of drug administration

The best route of drug administration in the CPR situation is via a central vein such as the jugular. Using this route, drugs are administered close to the heart. Peripheral venous drug administration tends to deliver the drug to the heart in a lower blood concentration and at a slower rate when

compared to the central venous route. Experimental studies in animals demonstrate drug delivery after peripheral injection is enhanced by following the injection with 10ml to 30ml saline flushes (depending on the size of the patient).

An alternative to IV route is intraosseous (IO). This requires placement of an intramedullary cannula inserted into the femur (through the greater trochanter), humerus (through the greater tubercle), and wing of the ilium or tibial crest. Medications and IV fluids can be injected into the medullary canal and rapid uptake is provided by the abundant endosteum-medullary blood supply.

The intratracheal route has been advocated for drug administration when venous access is not available. Some studies have indicated drug uptake from the tracheal surface during resuscitation is sporadic, undependable and delayed. Drugs that may be administered by this route include epinephrine (adrenaline), atropine and vasopressin. If this route is used, then the drug dose should be doubled and added to sterile saline or water for volume. The drugs are injected down the endotracheal tube via a long catheter that extends beyond the endotracheal tube and down to the level of the carina.

Intracardiac injections are not recommended because of the potential problems that can occur with this route, including myocardial trauma, lacerated coronary arteries, pericardial effusion, and refractory ventricular fibrillation when the heart muscle is injected with epinephrine.

Drugs

Vasopressors

Vasopressors are used adjunctively in CPR to increase aortic and diastolic pressures and, therefore, improve cerebral and myocardial perfusion pressures. They are indicated in pulseless electrical activity (PEA), asystole and refractory ventricular fibrillation (VF). Epinephrine (adrenaline) 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 (adrenaline) 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 0.01mg/ kg to 0.02mg/kg IV or IO. Continued use at three to five 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 betaadrenergic effects and also retains effectiveness in the acidic and hypoxic environment of CPA where adrenaline will lose its vasoconstrictor effects. Vasopressin may be used in place of, or alternated with, adrenaline doses. The dose is 0.2IU/kg to 0.8IU/kg IV or IO every three to five minutes (Fletcher, 2012a).

Atropine

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 and asystole are 0.02 mg/kg to 0.04mg/kg IV or IO every three to five minutes up to three doses. The dose for symptomatic bradycardia is usually 0.01mg/kg to 0.02mg/kg IV (Fletcher, 2012a).

Reversal agents: Agents such as naloxone, flumazenil and atipamezole may be considered if an opioid, benzodiazepine or alpha-2 agonist respectively has been administered recently prior to the cardiac arrest.

Crystalloid fluids: Fluids are indicated only if the patient is hypovolaemic. Lactated Ringer's or a similar solution is a reasonable choice. In one study, dextrose solutions were implicated in increased morbidity and mortality in association with cardiac arrest, therefore, dextrose should not be used. The dose of fluids in the dog is about 40ml/kg and 20ml/kg in the cat. The fluids should be given rapidly intravenously, in boluses sufficient to maintain effective circulating volume. When anaemia or hypoproteinaemia is present, whole blood or plasma or a synthetic colloid may be indicated (Fletcher, 2012a).

Sodium bicarbonate (NaHCO_3) is used to correct metabolic acidosis, which is generated by anaerobic metabolism in hypoxic tissues. NaHCO_3 may be given empirically at a dose of 1.0mEq/kg if the cardiac arrest is greater than 10 to 15 minutes. Sodium bicarbonate therapy should only be used when blood gas analysis to measure HCO_3 and base excess is available (Fletcher, 2012a).

Calcium is not recommended in the routine treatment of cardiac arrest. Calcium may be indicated when the patient is hyperkalaemic or hypocalcaemic. The dose of calcium is 0.1ml/kg of 10 per cent calcium chloride or 0.4ml/kg of 10 per cent calcium gluconate (Fletcher, 2012a).

Post-resuscitation care

In the post-cardiac arrest phase, the chance of re-arrest is high (68 per cent in dogs, 37 per cent in cats; Niles, 2011). Post-resuscitation requires intensive monitoring and aggressive supportive care ([Figure 3](#)). Maintaining adequate ventilation and oxygenation can be achieved with the use of monitoring waveform capnography, using caution not to hyperventilate and maintaining oxygen saturation more than 94 per cent. Permissive hypercapnia in the post-resuscitation phase is not recommended. Hypotension should be treated with IV or IO boluses of the appropriate fluid choices and vasopressors (Fletcher, 2012a).

Therapeutic hypothermia (32°C to 34°C) used in the first 12 to 24 hours post-resuscitation may improve neurological outcome. The efficacy of therapeutic hypothermia in improving neurological outcome following ischaemic brain injury in both humans and dogs has been proven. Active re-warming of post-arrest patients should be avoided and passive re-warming carried out at a rate no faster than $0.5^\circ\text{C}/\text{hour}$ (Smarick et al, 2012).

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 central nervous system 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 (Fletcher, 2012b). 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.

It should also be remembered CPA is commonly caused by a pre-existing disease process, such as severe sepsis, trauma, or respiratory failure; or comorbidities are present that may affect the animal's therapeutic requirements after ROSC and are considered to impact the overall quality of life. Pre-existing conditions will more often than not persist after successful initial resuscitation and will have a dual impact on the specific PCA care required and the prognostic outlook presented to the pet owner.

After the arrest, the patient should be monitored closely. Special attention should be paid to the cardiovascular, pulmonary, and central nervous system – optimising oxygenation, ventilation, circulation, metabolism and care of underlying disease processes. It's helpful to monitor as many parameters as possible for each system; this gives you a clear overview of the patient status. Post-cardiac arrest patients are one of the most challenging to deal with, but good nursing is a vital aspect in the patient's journey towards a positive outcome.

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Figure 1. Chest compressions should be done with the dog or cat in lateral recumbency with a compression depth of one-third to half the width of the chest at a rate of 100 to 120 compressions per minute regardless of size or species.



Figure 2. In cats and smaller dogs, compressions may be performed using a single-handed technique (circumferential compressions).



Figure 3. Post-resuscitation requires intensive monitoring and aggressive supportive care. Special attention should be paid to the cardiovascular, pulmonary and central nervous system – optimising oxygenation, ventilation, circulation, metabolism and care of underlying disease processes.