

**CARDIOPULMONARY CEREBRAL RESUSCITATION (CPCR)**  
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Cessation of effective circulating blood flow and ventilation constitutes cardiopulmonary arrest. Cardiopulmonary arrest is typically associated with loss of consciousness, collapse, lack of a palpable pulse, pale or cyanotic mucous membranes, lack of effective respirations, and lack of measurable blood pressure. At the time of cardiac arrest, a wide variety of cardiac dysrhythmias may be present on electrocardiogram. The dysrhythmias that occur in our canine and feline patients at the time of arrest dramatically differ from dysrhythmias that result in cardiac arrest in human. Only in rare cases can one anticipate that cardiopulmonary arrest is about to occur. Most cases that require cardiopulmonary cerebral resuscitation (CPCR) unfortunately present to you after cardiovascular collapse and pulmonary arrest have already occurred. Nonetheless, prompt recognition and rapid treatment are paramount in reestablishing both cerebral and coronary blood flow in order to have the greatest chance at a positive outcome.

In one retrospective study that investigated the outcome of cardiopulmonary arrest and CPCR in 304 dogs and 95 cats, overall outcome was less than 5% survival to discharge from hospital. Of animals successfully resuscitated, 68% of dogs and 38% of cats re-arrested within 4 hours of the initial episode. A slightly more favorable outcome may occur if cardiopulmonary arrest occurs during an anesthetic episode, most likely because the animals already have vascular access and are intubated receiving 100% inspired oxygen. Although overall outcome may not be very favorable, considerations must be made when asking clients whether they want CPCR to be performed at all in the event of an arrest. In many cases, unless the underlying cause of the arrest can successfully be treated at the time of the event (i.e. hyperkalemia secondary to urethral obstruction, tension pneumothorax secondary to trauma), the outcome is not likely to be favorable. Ethically, unless a client requests “Do Not Resuscitate” orders, attempts at resuscitation must be performed unless otherwise directed.

Cardiopulmonary-cerebral resuscitation refers to re-establishing blood flow to the cerebral and coronary systems in the event of cardiopulmonary arrest. The process by which oxygenated blood flow is re-established involves performing manual cardiac and thoracic compressions and manual ventilation until spontaneous circulation and ventilation occurs. There are typically three phases to CPCR. Phase 1 consists of Basic Life Support (BLS). Basic life

support involves manual cardiac and thoracic compression to re-establish circulation, and intubation with supplemental oxygen and artificial ventilation. Controversy exists whether to perform the “ABC’s” of CPR versus “CAB’s” in CPR. ABCs refers to “Airway”, “Breathing” and “Circulation”. Cardiac compressions begin after the patient is intubated and manual ventilation has begun. More recent evidence in a dog model of ventricular fibrillation has demonstrated that the oxygen content of blood in circulation is often adequate to start delivering oxygen to tissues and the coronary sinus if cardiac compression is started. Thus, more recent techniques implement the use of “CABs” of CPR, that is, starting manual external cardiac compressions before endotracheal intubation and ventilation. Additionally, external compression of the thoracic cage causes the animal to artificially breathe. In performing the CABs of CPR, external thoracic compression occurs usually simultaneously while a second person is intubating the patient and establishing an airway. After successful intubation and securing a patent airway, supplemental 100% oxygen is delivered, and cardiac compressions are continued. Compressions should be performed at 80 – 120 beats per minute. A simultaneous synchronized artificial breath should be performed for every chest compression. This method further increases intrathoracic pressure, generating more effective blood flow upon thoracic relaxation. Peak airway pressure should never be greater than 20 cm H<sub>2</sub>O, to prevent iatrogenic barotrauma. Effective circulation in CPR occurs by two mechanisms. The first is called the cardiac pump theory, in which direct compression of the heart from apex to base results in forward flow of blood. Unless internal cardiac massage occurs, in most cases the animal is too large for effective direct compression of the heart to occur. Even with open-chest CPR and direct cardiac massage, cardiac output achieved is usually only 50% of normal. In animals larger than 7 kg, the “thoracic pump theory” is more effective in causing forward flow of blood. External pressure on the thoracic cage creates increased intrathoracic pressure such that the change in pressure in between external compressions causes forward blood flow through passive mechanisms. Additionally, recent evidence has shown some improvement in circulation with synchronous thoracic compression with synchronous ventilation. Ventilating at the same time as external thoracic compression causes a greater change in intrathoracic pressure, and greater passive filling of great vessels upon relaxation. Generally, cardiac compressions, either direct or external, should be performed at a rate of 80 – 120 compressions per minute. The thorax should be compressed 25 – 30% of its circumference to generate the most effective change in intrathoracic pressure.

Compression and thus artificial systole should be the same length as artificial diastole or relaxation. Artificial ventilation should be synchronized at the same rate. Patients should be positioned in dorsal recumbancy if greater than 20 kg, or in lateral recumbancy if less than 20 kg. Interposed abdominal compression is also now advocated during CPR as an adjunctive therapy to increased cardiac output as well as coronary and cerebral blood flow. In this strategy, the abdomen is compressed during the period of time that the thorax is relaxed, driving forward blood flow from the abdomen toward the heart. One of the most important considerations is that if positioning and external cardiac compression is not generating a femoral pulse, the animal's position should be changed or internal cardiac massage considered. In patients with conditions that prevent a dynamic change in intrathoracic pressure such as obesity, pneumothorax, hemothorax, flail chest or rib fractures, diaphragmatic hernia, or open chest wounds, open-chest CPR should be initiated immediately, without starting closed-chest CPR at all.

Internal cardiac massage generates twice as much blood flow as external thoracic compressions. However, the overall rate of discharge from the hospital largely remains unchanged at this time in veterinary medicine. If an animal arrests under general anesthesia and is having thoracic or abdominal surgery, immediate open-chest CPR should be performed. However, if an animal presents to you after experiencing cardiopulmonary arrest, careful consideration should be weighed before opening the chest. For how long has the animal been arrested? If it has been greater than 15 – 20 minutes, the likelihood of having a successful outcome is dismal. Are you able to correct the underlying problem or problems? If not, perhaps it is not in the animal's best interest to pursue open-chest CPR. However, if the event was witness and not long ago, or if there is an underlying problem that makes closed-chest CPR ineffective, don't delay in initiating open chest CPR. Time is of the absolute essence.

To perform open chest CPR, following intubation and initiation of breathing and thoracic compressions, the patient should be placed in right lateral recumbancy and the fur over the left sixth intercostal space quickly clipped. The skin should be incised using a scalpel blade over the intercostal muscles, through the underlying fascia and fat, to the level of the intercostals muscles. A blunt stab incision should be made with a Mayo scissors into the pleural space, making sure that the assistant performing ventilation does not inflate the lungs during the stab incision to prevent iatrogenic lung injury. Once the stab incision has been made, the intercostals muscles are incised dorsally and ventrally to the level of the sternum, using care to avoid the

internal thoracic artery and the intercostals vessels located at the caudal edge of each rib. Force the ribs open and visualize the pericardial sac. Visualize the phrenic nerve and incise the pericardial sac ventral to the phrenic nerve. Exteriorize the heart from the pericardial sac and squeeze the heart from apex to base, gently avoiding placing too much tension or torque on the heart to prevent ripping the heart from the great vessels. Handling the heart during open-chest CPR allows the first-responder to directly visualize and feel the extent of cardiac filling and thus cardiac preload during resuscitation. In many cases, intravenous fluid therapy is not necessary unless hemorrhage, severe hypovolemia secondary to vomiting or diarrhea, or vasodilation secondary to anesthetic agents or sepsis have resulted in the animal's cardiopulmonary arrest. A common misconception is that all patients with cardiopulmonary arrest require large volumes of intravenous fluids. A large amount of time is usually wasted while someone attempts to secure an intravenous or intraosseous catheter. Additionally, increased diastolic filling pressures may actually decrease blood flow to the coronary sinus, thus impairing myocardial blood flow. Diastolic filling can be improved by cross-clamping the aorta during cardiopulmonary cerebral resuscitation. Once either closed-chest or open-chest CPR and basic life support consisting of airway intubation, artificial ventilation, and artificial cardiac compression (either open or closed), Phase II of CPR, or Advanced Life Support (ALS) consisting of ECG monitoring and interpretation, electrical defibrillation, and specific drug therapy should be performed. Advanced Life Support strategies can improve the chance of having a successful outcome.

Following BLS (if possible, these are performed simultaneously with a well-trained CPR team), attach an electrocardiograph monitor to the patient to determine the cardiac rhythm. Early and rapid defibrillation can be paramount to a successful outcome. Further, drugs should be administered based on a particular cardiac rhythm and timing during CPR. If a patient with a witnessed cardiopulmonary arrest is on any medication that is a potential cardiac or respiratory depressant, the offending drug must -be immediately reversed. For example, many post-operative or post-trauma patients are treated with parenteral opioid agents. Reversal with naloxone (0.02 – 0.04 mg/kg IV) should be immediately performed when initiating ALS. If the ECG rhythm indicated fine ventricular fibrillation, epinephrine (0.01 – 0.02 mg/kg IV) should be administered in an attempt to convert fine v-fib to coarse v-fib, a rhythm that may be easier to treat. Immediate electrical defibrillation (3 – 5 joules/kg externally, or 0.5 – 1.0 joule/kg

internally) should also occur with a series of three shocks occurring in rapid succession. If external or internal electric defibrillation is unsuccessful, or if an electrical defibrillator is not available, chemical defibrillators can also be used, including magnesium chloride (25 – 40 mg/kg IV), or amiodarone (5 – 10 mg/kg IV, IO) If asystole or so-called “flat-line” is diagnosed, first check the leads on the ECG. If attached to the patient properly, administer both atropine (0.04 mg/kg IV) and epinephrine (0.01 – 0.02 mg/kg IV). Electrical-mechanical dissociation (EMD), also known as Pulseless Electrical Activity (PEA) is a very difficult rhythm to treat, and has been associated with tremendously increased vagal tone. Electrical-mechanical dissociation is treated with naloxone (0.02 – 0.04 mg/kg IV) and high dose atropine (0.4 mg/kg IV). All drugs except for sodium bicarbonate that can be administered intravenously can also be administered via intratracheal route of administration, but at a higher dose. A table of drug doses and route of administration is provided for you at the end of this monograph. The use of sodium bicarbonate during CPR is very controversial, due to risk of causing hypotension, paradoxical cerebral acidosis, hyperosmolality, and hypernatremia. Sodium bicarbonate (0.5 – 1 mEq/kg IV)) should only be administered when treating severe hyperkalemia or acidosis, or when cardiac arrest and subsequent CPR attempts have been unsuccessful after 10 minutes.

Phase III of CPR consists of post-resuscitation care, including protecting the heart and brain from the adverse effects of cardiopulmonary arrest, providing perfusion to vital organ systems, and treating any underlying condition that caused cardiopulmonary arrest in the first place. This is often a very large and difficult responsibility. A spontaneous rhythm usually is generated before the patient has spontaneous respirations. Intravenous antiarrhythmic therapy in the form of lidocaine (50 – 100 mcg/kg/minute IV CRI) should be administered to prevent arrhythmias from developing. Additionally, mannitol (0.5 – 1.0 gram/kg IV over 20 minute, followed by 1 mg/kg IV furosemide 20 minutes after the mannitol) should be administered to decrease cerebral edema secondary to decreased cerebral perfusion and cerebral hypoxia. Intravenous fluids can be administered at a maintenance rate  $(30 \times \text{BW in kg}) + 70 = \text{ml/day}$ . This volume can be titrated or increased in patients with hypovolemia or vasodilation. Supplemental oxygen in the form of nasal insufflation, tracheal insufflation, or oxygen cage can be administered for supportive care. Electrocardiogram, Blood pressure, urine output should also be closely monitored, with appropriate pressor or inotropic therapy to maintain normotension and organ perfusion. Dobutamine (3 – 10 mcg/kg/min), primarily a beta-1 agonist,

can be administered as a positive inotrope to improve cardiac contractility and cardiac output without compromising organ perfusion. At the lower doses suggested, few negative side effects occur with this drug. At higher doses, tachycardia is a potential complication that should be avoided. Dopamine, with primarily dopaminergic and beta-1 effects at lower doses, can be titrated to higher doses for alpha-adrenergic pressure effects, in the event that dobutamine alone is not successful. Epinephrine, phenylephrine, ephedrine, can also be used for pressor effects.

Clearly, there is no specific way to perform successful CPR. Each case must be handled on an individual basis, taking into careful consideration patient's underlying condition and therapy, client wishes, chance for a successful outcome, and personnel available to perform CPR. Every clinic should have a designated portable crash-cart that remains fully stocked at all times. A quick reference table listing name of drug, drug dose, and dose in ml for IV and IT administration can be easily made for a wide range of body weights, then kept available near the crash cart for easy access. An emergency drug card containing the information just listed can also be made for each patient, should CPR become necessary. Team drills can be performed on cadavers or stuffed animals to help insure a practiced team approach. All of these suggestions can decrease the disorganized feeling that sometimes occurs during the chaos of an arrest! While successes are few, knowledge of what to do and practice of how to do it during an arrest can be life saving in some veterinary patients.

### **On the Horizon**

Techniques using a inspiratory impedance threshold device have been shown to improve initial outcome after CPR. An inspiratory impedance threshold device (ResQPOD® Circulatory Enhancer) causes a larger amount of negative pressure (small vacuum) to accumulate in the thorax during inspiration, and effectively pulls more blood into the heart, and this increases cardiac preload. Initial studies have been favorable in human and animals, however, the device is not routinely used in small animals at this time.

**Table of Drugs Used During and After CPR**

<b>Drug</b>	<b>Dose</b>
Epinephrine (low-dose)	0.01-0.02 mg/kg IV, IO
Epinephrine (high-dose)	0.1-0.2 mg/kg IV, IO
Atropine	0.04 mg/kg IV, IO
Calcium gluconate	50 mg/kg IV, IO
Naloxone	0.02-0.04 mg/kg IV, IO
Magnesium chloride	25 – 40 mg/kg IV
Amiodarone	5 – 10 mg/kg IV, IO
Vasopressin	0.8 ug/kg IV, IO
Sodium bicarbonate	1 mEq/kg IV, IO
Lidocaine	2 mg/kg IV, IO
Flumazenil	0.02 mg/kg IV, IO
Mannitol	0.5 – 1 g/kg IV, IO

**References available upon request.**