Managing Cardiopulmonary Arrest

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Cardiopulmonary arrest (CPA) is characterized by abrupt, complete failure of the respiratory and circulatory systems. The lack of cardiac output and oxygen delivery to tissues (DO₂) can quickly cause unconsciousness and systemic cellular death from oxygen starvation. If left untreated, cerebral hypoxia results in complete biologic brain death within 4 to 6 minutes of CPA. Therefore, prompt cardiopulmonary cerebral resuscitation (CPCR) is imperative. Veterinary technicians can play a key role in ensuring that patients receive this treatment.

Causes and Clinical Signs
In dogs and cats, common causes of CPA include anesthetic complications; vagal stimulation; hypovolemia; severe trauma, such as pneumothorax; unstable cardiac arrhythmias, such as unstable ventricular tachycardia; severe electrolyte disturbances, such as hyperkalemia; cardiorespiratory disorders, such as congestive heart failure, hypoxia, or pericardial tamponade; and debilitating or end-stage diseases, such as sepsis or cancer.

Potential signs of impending CPA include dramatic changes in breathing effort, rate, or rhythm (e.g., agonal breathing, decreased rate, sudden increased rate); the absence of a pulse; significant hypotension (i.e., systolic blood pressure <50 mm Hg [normal: >90 to 100 mm Hg]); irregular or inaudible heart sounds; changes in the heart rate or rhythm; change in mucous membrane color (e.g., white or cyanotic); fixed, dilated pupils; distressed vocalizations; and collapse.

Assessment of the patient is crucial if CPA is suspected. Before CPCR is initiated, it is essential to evaluate the patient’s responsiveness, breathing pattern, and pulse because patients in arrest are nonresponsive and apneic, with no detectable pulse.

Cardiopulmonary Cerebral Resuscitation
CPCR is initiated in three stages: basic life support (BLS), advanced life support (ALS), and postresuscitative care. Adopted from human emergency medicine, BLS involves establishing an open and clear airway, providing assisted ventilation, and performing chest compressions. These steps are often called the ABCs—airway, breathing, and circulation. ALS includes advanced care such as establishing venous access, interpretation of an electrocardiogram (ECG), drug administration, and defibrillation and is typically performed by credentialed veterinary technicians, veterinarians, or both. Postresuscitative care includes intensive monitoring as well as cardiovascular and ventilatory support.

CPCR Stage 1: Basic Life Support
Airway management involves extending the patient’s neck to straighten the airway and pulling the patient’s tongue forward. The veterinary staff should quickly examine the upper airway and initiate suctioning, if necessary. All foreign material or vomit observed in the patient’s mouth should be cleared immediately.

If the patient’s airway is fully obstructed, abdominal thrusts and finger sweeps of the pharynx can help dislodge the obstruction. An emergency tracheotomy, which is performed by a veterinarian, may be necessary if the airway obstruction is not immediately resolved. Insertion of a large-gauge needle or intravenous (IV) catheter directly into the trachea below the obstruction, along with oxygen administration, can be useful while the tracheotomy is being performed. In some cases, material fully obstructing the airway (e.g., a ball) can be manually removed with long hemostats or Doyen intestinal clamps.

After the patient’s airway has been cleared, an endotracheal tube should be placed. Tube placement should be confirmed and the tube secured and cuffed. The patient should then be ventilated with 100% oxygen. Proper ventilation is a critical component of BLS. Based on the most recent published recommendations, veterinary patients should receive 100% oxygen at a rate of 10 to 24 breaths/min. In humans, more frequent ventilation has been shown to be significantly detrimental because it can result in decreased myocardial and cerebral perfusion. Therefore, choosing a lower rate from the range of 10 to 24 breaths/min may be advised; these rates may be reexamined in future literature.

Glossary
Hemothorax—accumulation of blood in the pleural cavity
Hyperkalemia—an abnormally high concentration of potassium ions in the blood
Hypovolemia—a decreased blood volume
Hypoxia—deficiency in the amount of oxygen reaching the body tissues
Pneumothorax—accumulation of air or gas in the pleural cavity
Thoracentesis—removal of fluid or air from the chest through a needle or tube
Transcutaneous pacing (also called external pacing)—a temporary means of pacing a patient’s heart by delivering pulses of electric current through the patient’s chest, which stimulates contraction of the heart
Veterinary patients can be easily and safely ventilated with an Ambu bag (FIGURE 1) or bag-valve mask. Using an anesthesia unit can be slow and ineffective because the pop-off valve must be opened and closed repeatedly. Peak airway pressure should be <20 cm H2O. Lack of chest wall motion, poor ventilation, or absence of lung sounds should prompt an immediate search for a poorly positioned tube or a severe pleural space disorder, such as pneumothorax. In these cases, thoracentesis or thoracotomy performed by a veterinarian may be necessary.

Acupuncture has also been used to help treat CPA and respiratory depression. Needling the acupuncture point GV26 can help stimulate respiration and increase cerebral oxygen. This acupuncture point can be stimulated by inserting a regular 25-gauge needle or acupuncture needle into the nasal philtrum to a depth of approximately 10 to 20 mm and performing jabs in a hen-pecking motion while monitoring for improvement in respiration.

The goal of circulatory support during CPCR is to maximize myocardial and cerebral perfusion. Chest compressions should be performed immediately in patients without a detectable heartbeat. External cardiac massage at a rate of 80 to 120 compressions/min is recommended, but higher rates within that range seem to work better. Small patients (<15 lb [7 kg]) should receive compressions directly over one of the patient's open eyes. The presence of a "swooshing" wave sound from the Doppler unit during concurrent chest compressions can provide a crude estimate of whether forward blood flow is reaching the brain. If chest compressions are not generating adequate forward blood flow, either the patient should be repositioned and the resuscitation technique changed to increase intrathoracic pressure (BOX 1) or ICM should be considered.

CPCR Stage 2: Advanced Life Support
Establishing venous access is important but should never interfere with chest compressions or defibrillation. Venous access can be established by using methods such as intravenous catheter placement and venous cutdown, in which a small opening is created in the thoracic cavity, allowing blood to be drawn from the internal jugular vein or subclavian vein.
BOX 2. Treating Arrhythmias

Treatment of cardiopulmonary arrest depends on the type of arrhythmia, which can be effectively differentiated on an electrocardiogram (ECG) only. The two most common arrhythmias in veterinary patients are ventricular asystole and pulseless electrical activity (PEA), also known as electromechanical dissociation. Ventricular fibrillation (VF), the most common arrhythmia in humans, is not as common in cats and dogs. This may be partly due to the fact that humans commonly experience cardiac arrest for different reasons (i.e., myocardial infarction) than veterinary patients.

Ventricular Asystole
Asystole, also known as flatline, is the absence of electrical or mechanical activity from the heart. A rare P wave may be observed, but no QRS complexes are seen (FIGURE A). Transcutaneous electrical pacing (Glossary) is immediately initiated in human patients with asystole, but it has not gained widespread use in veterinary patients. Routine veterinary treatment includes IV epinephrine administered at 0.01 to 0.02 mg/kg q3–5min and IV atropine administered at 0.02 to 0.05 mg/kg q3–5min.15 One dose of IV vasopressin administered at 0.8 mg/kg is also likely to be beneficial in treating PEA.25 The use of different therapies, such as naloxone, dexamethasone, and calcium, to help treat PEA has been advocated. However, little published information supports regular use of these agents.

Ventricular Fibrillation
In patients with VF, the ECG wave form is erratic and chaotic. No clear P waves, QRS complexes, or T waves are observed (FIGURE C). Treating VF involves immediate electrical defibrillation. An initial shock of 3 to 5 J/kg is recommended; if the heart rhythm does not respond, two additional shocks, each at a 50% higher setting than the previous one, should be given in immediate succession. If, after the three progressively higher-voltage shocks, conversion to a stable rhythm is not successful, chest compressions and ventilation are continued for 2 minutes.21 Low-dose epinephrine at 0.01 mg/kg and vasopressin at 0.8 U/kg are then administered. These drugs can improve the likelihood of successful defibrillation.

Defibrillation is then resumed at 5 to 10 J/kg, followed by administration of an additional drug, such as amiodarone, lidocaine, magnesium chloride, or procainamide. After each new drug has been administered, defibrillation is again attempted at the 5- to 10-J/kg setting to restore a normal perfusing rhythm.21 The “2010 AHA Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care” recommend that health care providers continue cardiopulmonary cerebral resuscitation (CPCR) for 2 minutes after initial defibrillation before attempting defibrillation a second time or administering drug therapy in patients with VF. These recommendations are based on the understanding that biphasic defibrillators are readily used in human medicine and have a high initial defibrillation rate. Because biphasic defibrillators are not yet frequently used in veterinary medicine, human literature based on older AHA guidelines may be more applicable to treating dogs and cats. Biphasic defibrillation has been documented in small animal patients and will likely become the preferred method of small animal defibrillation in the future.26 When this more effective method of defibrillation becomes commonplace, it may make sense to continue chest compressions for a longer period.

It is difficult to successfully treat VF without a defibrillator. A sharp precordial thump administered to the chest wall may convert VF to a sinus rhythm in small patients but is unlikely to be effective in larger patients. Although pharmacologic defibrillation using agents such as potassium chloride, insulin–dextrose, and acetylcysteine has been suggested, their use is almost never effective.

Current use of defibrillators for respiratory arrest is not recommended. In human preterm infants, the drug has been shown to increase cerebral oxygen demand and, therefore, potentially worsen outcomes.27 Sodium bicarbonate should only be administered during CPR to manage severe metabolic acidosis diagnosed before the arrest, preexisting hyperkalemia, or metabolic acidosis generated by prolonged CPR efforts (>10 minutes) and anaerobic metabolism in hypoxic tissues. The routine use of calcium during CPR is no longer advised. Its use may be justified in patients with hyperkalemia or hypocalcemia or affected by a calcium channel blocker overdose. Routine use may potentiate cellular damage secondary to ischemia.28

Routine veterinary treatment includes administration of IV epinephrine at 0.01 to 0.02 mg/kg q3–5min and IV atropine at 0.04 to 0.05 mg/kg q3–5min.21 The “2010 AHA Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care” do not advocate the routine use of atropine to manage human VF because the drug has not proved to be of significant benefit for this purpose.21 One dose of IV vasopressin administered at 0.8 mg/kg is also likely to be beneficial in treating PEA.25 The use of different therapies, such as naloxone, dexamethasone, and calcium, to help treat PEA has been advocated. However, little published information supports regular use of these agents.
the skin to allow passage of a needle or cannula into a vein. The jugular vein typically yields well to catheterization during CPA and provides the shortest transit time for drugs to reach the heart. After venous access has been established, aggressive fluid administration should be considered if hypovolemia existed before the CPA or if the patient is experiencing blood loss. However, overzealous fluid administration in patients with normal body fluid volume may be detrimental. Fluid resuscitation can include administration of hypertonic saline, crystalloids, colloids, blood products, and hemoglobin-based oxygen-carrying solutions.

If an IV catheter cannot be placed initially, emergency drugs (e.g., lidocaine, epinephrine, atropine, vasopressin, naloxone) can be administered through an endotracheal tube. Typically, a long red rubber catheter is inserted down the tube, and drugs are administered through the catheter. Drug doses administered this way are normally increased two- to threefold and are followed by a small saline “chaser” to ensure successful drug passage into the lungs. For example, the dose for IV atropine is 0.02 mg/kg, but if it is administered through the endotracheal tube, the dose would be 0.04 to 0.06 mg/kg.

Intracardiac injection of medication is contraindicated, especially during closed-chest CPRC. Inaccurate injection and complications, such as vessel laceration and hemorrhage, are common. Accurate ECG interpretation, which determines the specific arrhythmia the patient is experiencing, is necessary before CPA can be treated. After the type of arrhythmia has been established, drug administration and defibrillation can be initiated. If CPA occurs as a result of anesthesia, all anesthetic agents must be immediately discontinued and their effects reversed, if possible. End-tidal carbon dioxide measurements >15 mm Hg are reportedly associated with higher survival rates. Other types of arrhythmia can usually be treated until they progress to asystole, unless the patient’s owner declines further resuscitation efforts.

**CPCR Stage 3: Postresuscitative Care**

Patients that are restored to a perfusing cardiac rhythm commonly experience rearrest—often within minutes to several hours—especially if the original cause of the CPA has not been identified. Therefore, resuscitated patients usually require substantial cardiovascular and ventilatory support during the period following CPA. Mild hypothermia after resuscitation from CPA decreases cerebral oxygen demand and has been shown to improve outcomes in dogs. Inducing mild hypothermia in patients could be considered a therapeutic option. In human patients, hypoglycemia has been shown to be associated with worse neurologic outcomes and should, therefore, be avoided after CPA.

Poor perfusion during CPA may also precipitate brain injury, disseminated intravascular coagulation, gut reperfusion syndrome, and renal failure. Therefore, intensive monitoring and aggressive supportive care are required to optimize management of blood pressure, cardiac output, oxygenation, ventilation, and vital organ perfusion.

**Conclusion**

After CPA, the success rate for recovery of veterinary patients is generally poor. A 1-week survival rate of <4% has been reported for cats and dogs that received CPRC following a rest. However, functional recovery has been reported in most animals that survive CPA.

Based on current research, resuscitation appears to be successful in patients that are treated quickly; have a reversible underlying disease process, such as anesthetic overdose, upper airway obstruction, hemorrhage, or electrolyte abnormalities; and, ideally, are not in full CPA.

**References**


1. Ventilatory rates of ________ breaths/min are currently recommended for veterinary patients in CPA.
   a. 4 to 12
   b. 10 to 24
   c. 30 to 45
   d. 60 to 82

2. Clinical signs of impending CPA include
   a. a strong pulse.
   b. audible heart sounds.
   c. fixed, dilated pupils.
   d. significant hypertension.

3. Which of the following is routinely recommended during CPCR?
   a. epinephrine
   b. calcium
   c. sodium bicarbonate
   d. doxapram

4. The ________ vein typically yields well to catheterization during CPA and provides the shortest transit time for drugs to reach the heart.
   a. jugular
   b. cephalic
   c. saphenous
   d. femoral

5. During CPCR, external cardiac massage should be performed at a rate of ________ compressions/min.
   a. 10 to 60
   b. 30 to 60
   c. 60 to 120
   d. 80 to 120

6. Which of the following statements regarding CPA is true?
   a. Published data suggest that approximately 50% of patients experiencing CPA survive for >1 week after CPCR.
   b. Functional recovery has been reported in most animals that survive CPA.
   c. Patients that experience CPA and have CPCR withheld show no increased morbidity compared with patients that receive immediate CPCR.
   d. VF is the most common arrhythmia observed in veterinary patients during CPA.

7. Mild __________ after resuscitation has been shown to improve outcomes in dogs after CPA.
   a. hypotension
   b. hypovolemia
   c. hypoxia
   d. hypothermia

8. End-tidal carbon dioxide measurements __________ have been associated with higher survival rates during CPA.
   a. <15 cm Hg
   b. >15 cm Hg
   c. <15 mm Hg
   d. >15 mm Hg

9. In patients with PEA,
   a. P waves, QRS complexes, and T waves may be observed on the ECG.
   b. no clear P waves, QRS complexes, or T waves are observed on the ECG.
   c. a rare P wave may be observed on the ECG, but no clear QRS complexes are seen.
   d. none of the above

10. Treatment of VF involves immediate
    a. insulin administration.
    b. thoracentesis.
    c. IV catheterization.
    d. electrical defibrillation.