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Cardiopulmonary Arrest & Life Support

Profile

Definition

Cardiopulmonary arrest (CPA) is defined as the abrupt cessation of cardiac pump function leading to a lack of oxygen delivery to tissues and eventually to death.

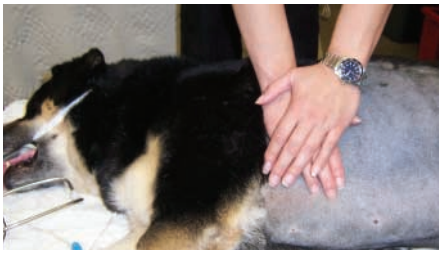
Signalment

Any incapacitated cat or dog of any age, gender, or breed is at risk for CPA that would require cardiopulmonary cerebral resuscitation (CPCR). CPCR is a technique used to provide ventilatory and cardiac support following respiratory and/or cardiac arrest, in an attempt to maintain tissue oxygenation and restore effective circulatory and neurologic function.

Causes

Causes of CPA are likely to be multifactorial, including:

- Primary cardiac or pulmonary disease
- Malignancy
- Infection
- Renal, immune-mediated, endocrine, hepatic, gastrointestinal, hematopoietic, central nervous system, or pancreatic disease
- Trauma
- Intoxication
- Complications from anesthesia/drug administration
- Upper airway obstruction
- Disseminated intravascular coagulation or cardiovascular collapse.[1,2]



Risk Factors

- Preexisting disease
- Hypoxemia, hypercarbia
- Hypotension
- Hypothermia
- Cardiac arrhythmias
- Severe anemia
- Electrolyte or acid-base abnormalities (eg, hyperkalemia, severe metabolic acidosis)
- Vagal stimulation
- Cardiac, pulmonary, or neurologic disease
- Critical illness/sepsis
- Anesthesia

Pathophysiology

- CPA results in decreased oxygen delivery and causes global ischemia.
- An intense sympathetic reflex occurs, resulting in vasoconstriction.
- The vasomotor center is stimulated but becomes inactive within 10 to 15 minutes due to lack of blood flow.[3]

Signs

- Hypotension
- Bradycardia
- Arrhythmias
- Hypothermia
- Cyanosis
- Pupillary dilation
- Changes in respiratory rate, depth, or pattern, including agonal breaths[4-6]

Diagnosis

Definitive Diagnosis

- **Cardiopulmonary arrest**: Absence of an auscultable or palpable heartbeat and lack of peripheral pulses and spontaneous respirations
- **Respiratory arrest**: Lack of spontaneous respirations with an auscultable heartbeat and presence of peripheral pulses
- **Loss of brain function**: Detected by electroencephalogram or brain stem auditory evoked potentials[7,8]

Laboratory Findings/Imaging

- Minimum database (packed cell volume, total protein, blood glucose, BUN, and/or creatinine) may show anemia or hemoconcentration, hypoproteinemia, hypoglycemia or hyperglycemia, and azotemia.
- Electrolyte evaluation (Na and K most important) may reveal hypernatremia or hyponatremia, hyperkalemia or hypokalemia, or ionized hypocalcemia.
- Arterial or venous blood gases may reveal a respiratory or metabolic acidosis.
- Serum lactate may be elevated.
- Depending on changes associated with the animal's primary disease, other tests may include complete blood count, serum chemistry, coagulation testing, blood typing, various cultures, urinalysis, radiography, and ultrasonography.

BUN = blood urea nitrogen; CPA = cardiopulmonary arrest; CPCR = cardiopulmonary cerebral resuscitation; ECG = electrocardiogram

Treatment

Inpatient or Outpatient

Patients should be treated in an intensive care unit (**Figure 1**, page 26).

Basic Life Support

- Follow the "ABCs" (airway, breathing, circulation)—assess whether the patient is breathing and if the airway is patent. If the patient is not breathing, intubate immediately using a cuffed endotracheal tube. Suctioning may be necessary.
- If intubation is not possible, perform an emergency tracheostomy using a cuffed tracheostomy tube or a modified endotracheal tube. Inflate the cuff and securely tie in place. Begin positive pressure ventilation with 100% oxygen at a rate of 10 to 24 breaths per minute (**Figure 2**).[4,9] Use higher rates in smaller animals (< 15 kg) and lower rates in larger ones (> 15 kg). Aim for normal chest wall movement and do not exceed peak airway pressures of 20 cm H_2O.[9]
- In the absence of a heartbeat and palpable peripheral pulses, cardiac compressions should be performed immediately, using either external chest compressions (closed chest CPCR) or internal cardiac massage via open chest CPCR.

Closed Chest CPCR

– Perform chest compressions at a rate of at least 100 to 120 per minute by encircling the heart in small patients (< 15 kg) and over the widest portion of the thorax in larger patients (see **Figures 3 and 4**).[6]

– Interposed abdominal compression (IAC) involves abdominal compressions between chest compressions, and has been reported to improve survival to discharge in human patients.[10]

Open Chest CPCR

– To be successful, perform open chest CPCR early. Indications for immediate internal cardiac massage include intraoperative arrest, hemoperitoneum, large dogs (> 20 kg), pericardial effusion, penetrating chest wounds or chest wall trauma (eg, rib fractures), pleural space disease (eg, diaphragmatic hernia, pleural effusion, pneumothorax), unwitnessed arrests, or if closed chest CPCR does not restore circulation within 2 to 5 minutes.[9,11,12]

– Place the animal in right lateral recumbency. Make an incision at the 6th intercostal space through the skin and muscle layers. Enter the pleural space with blunt dissection using a finger or hemostat; then use scissors to extend the incision ventrally and dorsally. Spread the ribs manually or with rib spreaders. Manually compress the heart; use of 2 hands may

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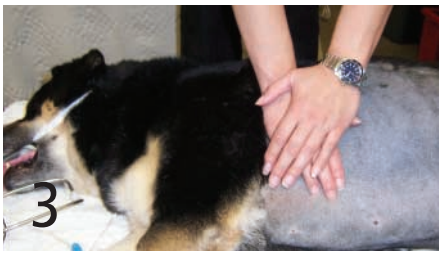
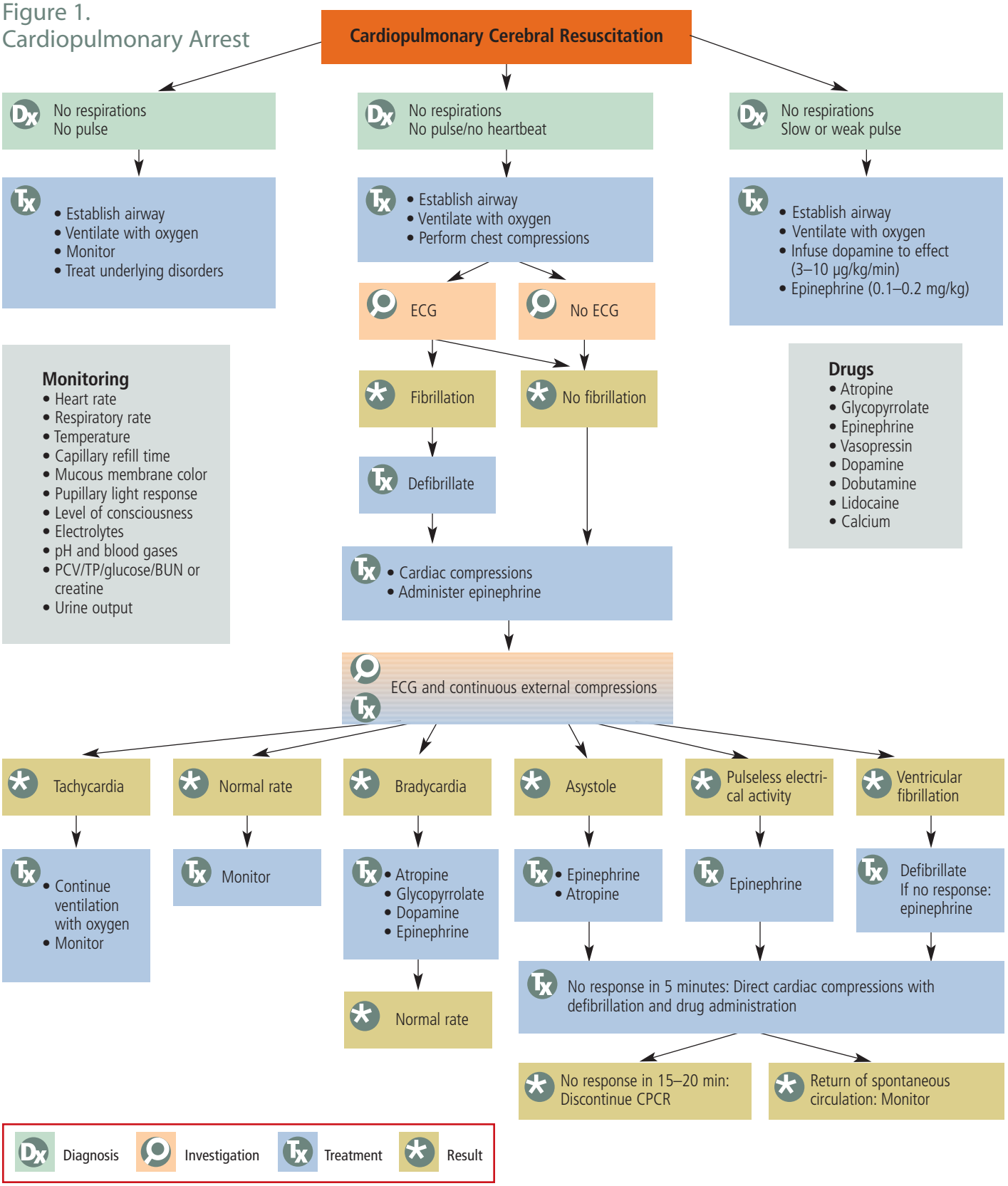


To perform external cardiac/chest compressions in a large dog, the chest wall should be depressed approximately 1 cm to 3 cm per compression, which may require a considerable amount of operator effort.

The first priority of basic life support is to ensure that the airway is patent and to initiate positive pressure respiration. This cat is being ventilated following respiratory arrest, using an Ambu bag and 100% oxygen. The cat's head is elevated to decrease intracranial pressure, vascular access has been established in the cephalic vein, a blood pressure cuff is being used on the hind leg, and ECG leads are in place for continuous cardiac monitoring.

External cardiac compressions in smaller animals such as cats or small dogs involves compressing the heart between the thumb and fingers.

Figure 1.
Cardiopulmonary Arrest



BUN = blood urea nitrogen; CPA = cardiopulmonary arrest; CPCR = cardiopulmonary cerebral resuscitation; ECG = electrocardiogram; PCV = packed cell volume; TP = total protein

Adapted from **Cardiopulmonary cerebral resuscitation.** Cooper E, Muir WW. In King LG, Boag A (eds): *Small Animal Veterinary Association Manual of Canine and Feline Emergency and Critical Care*, 2nd ed—Gloucestershire, United Kingdom: British Small Animal Veterinary Association, 2007.

be more effective than 1.[13]
  – Aortic cross-clamping may increase cerebral and myocardial blood flow. It can be performed by direct manual compression or by using a red rubber catheter, penrose drain, or Rumel tourniquet to occlude the descending thoracic aorta.[6] Gradual removal of the occlusion should occur slowly over 10 to 20 minutes once spontaneous circulation has been restored.

Advanced Life Support

Advanced life support expands on basic life support to include diagnosis of cardiac arrhythmias and administration of drug therapy, electrical defibrillation, and cardiac pacing.

- Cardiac arrhythmias
  – Place an ECG monitor on the patient as soon as possible.
  – The most common cardiac arrhythmias recognized during CPA include pulseless electrical activity asystole (PEA), ventricular fibrillation (VF), and sinus bradycardia.[14,15]
- Ventricular fibrillation
  – The only effective treatment for ventricular fibrillation is internally or externally applied electrical defibrillation. Survival is directly correlated with the time that elapses before defibrillation begins.[15]
  – For external defibrillation, the energy of the initial countershock should be 3 to 5 joules/kg. A total of 3 shocks should be given if conversion does not occur, with a 50% increase in energy for each consecutive shock. If defibrillation does not occur, CPCR should be resumed for 2 minutes,[15] followed by another series of countershocks at twice the energy level of the first set (5–10 joules/kg).
  – Internal defibrillation may be performed, using 3 countershocks of increasing energy following an initial shock of 0.5 to 1 joules/kg.

Medications

- Multiple routes are used to administer medications including intravenous (IV), intraosseous (IO), and intratracheal (IT). To optimize myocardial drug delivery, a central IV catheter is preferred; however, a peripheral catheter is adequate (preferably in a forelimb). A surgical cut-down may be necessary. IO catheters are useful in small or neonatal patients.
- When no other routes are available, IT administration may be used. Drugs that may be administered intratracheally include epinephrine, atropine, lidocaine, and vasopressin. Double the dose and dilute with sterile saline or sterile water. (Note that sodium bicarbonate should NOT be administered intratracheally due to the risk of depleting pulmonary surfactant.) Administer using a long catheter that is passed through the endotracheal tube to the carina. Following drug administration, give 2 full breaths to ensure distribution throughout the airways.

Vasopressor Therapy

- Epinephrine is indicated for the treatment of PEA, asystole, refractory VF, or pulseless ventricular tachycardia (VT). The recommended low dose of epinephrine is 0.01 to 0.02 mg/kg and the high dose is 0.1 to 0.2 mg/kg.
- It is unclear whether the use of a low or high dose has any effect on outcomes of veterinary patients. Shorter resuscitation times have been shown in some research dogs receiving higher doses of epinephrine; therefore, the high dose is currently recommended.[16]

Anticholinergic Therapy

- Atropine is indicated for animals suffering a vagally mediated arrest or for those that have developed sinus bradycardia.
- Animals with sinus bradycardia should receive low doses of atropine (0.004 mg/kg –0.01 mg/kg).[16]
- Higher doses of atropine (0.04 mg/kg) should be reserved for patients with PEA or asystole.[16]

Alternative/Additional Therapy

- Vasopressin is a potent noncatecholamine vasoconstrictor and is considered an alternative first-line vasopressor in refractory VF or pulseless VT, at a recommended 1-time dose of 0.8 unit/kg IV.[16]
- Additional drugs include calcium gluconate (for animals with documented hypocalcemia or hyperkalemia-induced cardiac arrest) or sodium bicarbonate (for animals with documented severe metabolic acidosis or following a prolonged period of resuscitation).
- Immediately following CPCR and successful restoration of circulation, measures to minimize cerebral injury may include administration of mannitol (0.5–1 g/kg IV) and elevation of the head.

Follow-up

- Appropriate management following successful CPCR is critical to survival of the patient. Monitoring should include frequent physical examinations and evaluation of blood pressure, ECG, blood gas analyses, pulse oximetry, end-tidal capnography, and urine output.
- Immediate measures should be taken to diagnose and treat the underlying cause of pulmonary arrest in order to minimize the risk of a repeat CPA.

In General

Relative Cost ($$$$$)

Depends on underlying cause of CPA and success of resuscitation. Successful resuscitation is likely to be followed by a period of intensive

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CPA = cardiopulmonary arrest; CPCR = cardiopulmonary cerebral resuscitation; ECG = electrocardiogram; PEA = pulseless electrical activity asystole; VF = ventricular fibrillation; VT = ventricular tachycardia

monitoring and treatment. In one study, the mean cost of hospitalization following CPA was $2200 (range $1300 to $3600).[1]

Cost Key

$ = < $100	$$$$ = $500-$1000
$$ = $100-$250	$$$$$ = > $1000
$$$ = $250-$500	

Client Education

Communication with owners of critically ill patients should include discussion of the possibility of CPA and the patient's prognosis. Clients should decide the level of resuscitation they desire for their animals—no resuscitation, basic life support, or advanced life support—keeping in mind that resuscitation may not be the optimal choice for patients with terminal disease.

Prognosis

Prognosis depends upon the cause of CPA, age, and condition of the animal. Patients that experience CPA due to a reversible underlying disease (eg, vagal event, anesthetic- or drug-related event, or electrolyte disturbance) have a better prognosis than those with a severe underlying disease, such as neoplasia or sepsis.[6] Mortality is significantly greater for animals that arrest as a result of anesthesia than those that arrest due to nonanesthetic causes.

Future Considerations

CPCR in veterinary medicine has not been fully evaluated. There are few retrospective studies that provide information on etiology, outcome, and prognosis. There are no prospective studies that evaluate resuscitation techniques in clinical patients. Therefore, the recommendations for resuscitative efforts in veterinary medicine have largely been adapted from current human practices. A consensus is needed on standards and protocols specific to veterinary patients such as therapies performed and drugs administered. Furthermore, further prospective studies are needed in veterinary medicine.

See Aids & Resources, back page, for references, contacts, and appendices.

Article archived on www.cliniciansbrief.com

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our authors

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