Updates in Small Animal Cardiopulmonary Resuscitation

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CPR AS A CONTINUUM OF CARE

Cardiopulmonary arrest (CPA) is a highly lethal condition in veterinary medicine with only 6% to 7% of small animals surviving to hospital discharge.¹ A comprehensive strategy is necessary to reduce mortality caused by CPA and several opportunities exist to influence outcomes positively.² Preventive measures and recognition of animals at imminent risk can lead to a reduction of the numbers that experience CPA, and preparedness of the resuscitation team and tools will optimize early and effective response to CPA. Well-executed basic and advanced life support will then increase the likelihood of a return of spontaneous circulation (ROSC). Last but not least, post-cardiac arrest care is the imperative final step to increasing rate of survival-to-hospital discharge. It addresses treatment of neurologic, myocardial, and systemic ischemia.

KEYWORDS
- Cardiopulmonary resuscitation
- Cardiopulmonary arrest
- Basic life support
- Advanced life support
- Post-cardiac arrest care

KEY POINTS
- For dogs and cats that experience cardiopulmonary arrest (CPA), rates of survival to discharge are 6% to 7%, as compared with 20% for people who experience CPA.
- To improve outcomes after CPA, a comprehensive strategy that includes preventive and preparedness measures, basic life support, advanced life support, and postcardiac arrest critical care titrated to the patient's needs is necessary.
- Optimization of each of these elements may help improve overall survival and offers an opportunity to work toward that goal.
- The Reassessment Campaign on Veterinary Resuscitation initiative recently completed an exhaustive literature review and generated a set of evidence-based, consensus cardiopulmonary resuscitation guidelines in 5 domains: preparedness and prevention, basic life support, advanced life support, monitoring, and postcardiac arrest care.

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and reperfusion injury as well as precipitating conditions. The Reassessment Campaign on Veterinary Resuscitation (RECOVER) recently completed an exhaustive literature review and generated a set of evidence-based, consensus guidelines to provide a clear basis for training and practice for the above outlined cardiopulmonary resuscitation (CPR) continuum of care.\textsuperscript{3,4} Accordingly, RECOVER provides clinical instructions in 5 domains: preparedness and prevention,\textsuperscript{5} basic life support,\textsuperscript{6} advanced life support,\textsuperscript{7} monitoring,\textsuperscript{8} and postcardiac arrest care.\textsuperscript{9} This article reviews some of the most important aspects of these new guidelines.

**PREPAREDNESS AND PREVENTION**

Early initiation of CPR for patients with CPA is of key importance. Thus veterinary practices must be well prepared for early recognition of and response to CPA. Box 1 summarizes the key recommendations for preparedness and prevention. CPR training for veterinary personnel possibly involved in resuscitation should include both a structured, didactic component and a psychomotor component with opportunities to practice technical skills.\textsuperscript{10,11} Integrated individual and team performance should be trained in mock codes with structured feedback conducted at least once every 6 months.\textsuperscript{12,13} In addition to well-trained personnel, resuscitation tools must be prepared and ready at all times, specifically a stocked crash cart and cognitive aids. A crash cart containing all necessary drugs and equipment should be maintained in the practice and should be routinely audited to ensure drugs are in date and equipment is functional. Cognitive aids, such as algorithm and dosing charts, were shown to improve adherence to guidelines and individual performance during CPR.\textsuperscript{14–16} They should be readily accessible in a standard, central location, and staff should be trained on the use of these aids regularly. After every CPR event, a debriefing session should be held, during which team performance is discussed and critically evaluated. This

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**Preparedness and prevention key guidelines**

- CPR training
  - Both didactic and hands-on training are essential
  - Refresher training should be done at least every 6 months

- Crash cart
  - Available in a central location
  - Regularly stocked and audited

- Cognitive aids
  - Algorithm, drug, and dosing charts
  - Personnel should be trained in their use

- Diagnosis of CPA
  - Standardized ABC assessment in any acutely presenting or decompensating patient
  - ABC assessment should take no longer than 15 seconds
  - If there is any doubt the patient is in CPA, CPR should be started without delay
debriefing session can improve future performance and, at the same time, serve as refresher training.17,18

Early recognition of CPA is the precondition for timely initiation of resuscitation measures and is of paramount importance. In principle, CPA should be ruled out rapidly in any acutely unresponsive patient. In nonanesthetized patients, the diagnosis can be made by physical examination alone and is defined by unconsciousness and apnea. Thus, a rapid assessment lasting no more than 10 to 15 seconds and focused on ruling out CPA should be undertaken immediately in any unresponsive patient. To that end, a standardized diagnostic approach based on evaluation of airway, breathing, and circulation (ABC) will identify CPA efficiently. If CPA cannot be definitively ruled out, CPR should be initiated immediately rather than pursuing further diagnostic assessment. Immediate CPR is important because (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; (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; and (3) the risks of performing CPR on an unresponsive patient not in CPA are small.19,20 Thus the clinician should not delay starting CPR in any patient in which there is a suspicion of CPA.

BASIC LIFE SUPPORT

Once CPA is recognized, basic life support (BLS) should be initiated as quickly as possible by following the treatment mnemonic CAB (circulation, airway, breathing). High-quality BLS is arguably the most important intervention in CPR. Box 2 summarizes the key BLS guidelines. Circulation should be addressed first by starting chest compressions immediately, because ventilation will be ineffective in the absence of blood flow, and evidence suggests that outcome worsens as delay to the initiation of chest compressions increases.19

Circulation: Chest Compressions

Patients with untreated CPA lack forward blood flow out of the heart and oxygen delivery to the tissues ceases. An immediate consequence is the exhaustion of cellular energy stores, followed by cell depolarization and compromise of organ function, which results in increasing severity of ischemic organ injury with time and sets the stage for escalating reperfusion injury on reinstatement of tissue blood flow. Thus early institution of high-quality chest compressions is the most important aspect of effective CPR. The goals of chest compressions include the following: (1) restoration of blood flow to the lungs allowing carbon dioxide (CO₂) elimination and oxygen uptake, and (2) delivery of oxygen to tissues to restore organ function and metabolism. Experimental evidence suggests that even well-executed chest compressions produce approximately 30% of normal cardiac output. Therefore, proper chest compression technique is crucial. Delay in the start of high-quality chest compressions reduces the likelihood of ROSC.

Although not well studied in dogs and cats, some experimental data and anatomic principles suggest that chest compressions should be performed with the dog or cat positioned in either left or right lateral recumbency,21 with a compression depth of 1/3 to 1/2 the width of the chest and at a rate of 100 to 120 compressions per minute, regardless of animal size or species. Use of visual or acoustic prompts to ensure correct rate of compression, such as a flashing light, a metronome, or a song with the correct tempo (eg, the BeeGee’s “Staying Alive”), is recommended. To allow full elastic recoil of the thorax, leaning on the chest between compressions must be avoided.
Excessive leaning will lead to increased intrathoracic pressure, reduced venous return to the chest and heart, and a reduction in cerebral and myocardial blood flow. Pauses in chest compressions are harmful and thus compressions should be delivered without interruption in cycles of 2 minutes. Any interruption in compressions should be as short as possible, as it takes approximately 60 seconds of continuous chest compressions before coronary perfusion pressure reaches its maximum.\textsuperscript{22} Coronary perfusion pressure in turn is a critical determinant of myocardial blood flow and increases the likelihood of ROSC. To minimize pauses, all processes requiring

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**Box 2**

**Basic life support**

*Basic life support key guidelines*

**Chest compression technique**

- Most patients in lateral recumbency
- Rate of 100–120 compressions per minute regardless of species or size
- Compress 1/3–1/2 the width of the chest
- Allow full recoil of the chest between compressions
- Minimize interruptions and delays in starting compressions
- Rotate compressor after every 2-minute cycle of CPR

**Chest compression posture**

- Lock elbows and interlock hands
- Shoulders directly above hands
- Bend at waist and use core muscles
- Avoid leaning
- If table is too tall, use a stool, climb onto table, or put patient on floor

**Chest compression hand position**

- Medium and large round-chested dogs: over highest portion of the lateral thoracic wall
- Keel-chested dogs: directly over the heart
- Small dogs and cats: directly over the heart; consider one-handed technique
- Flat-chested dogs (eg, bulldogs): dorsal recumbency; hands on sternum

**Ventilation**

- Intubated patient (preferred technique)
  - 10 breaths/minute simultaneously with compressions
  - 1 second inspiratory time
  - Approximately 10 mL/kg tidal volume
- Mouth to snout
  - Close patient’s mouth tightly
  - Make seal over both nares with mouth
  - Deliver 2 quick breaths with 1-second inspiratory time
  - 30:2 technique: 30 chest compressions, 2 quick breaths, immediately resume compressions
interruption of chest compressions, such as electrocardiogram (ECG) analysis or pulse palpation, should be executed in a coordinated fashion at the end of each 2-minute cycle. During this planned pause of a few seconds’ duration, a new compressor should take over to reduce the negative effect of rescuer fatigue on chest compression depth, rate, and leaning.

The mechanism of blood flow generation is fundamentally different during CPR compared with spontaneous circulation and 2 distinct models exist to describe how chest compressions generate systemic blood flow. According to the cardiac pump theory, 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.\textsuperscript{23} Recoil of the chest between compressions caused by the elastic properties of the rib cage creates negative intrathoracic pressure, leading to filling of the ventricles before the subsequent compression. The thoracic pump theory proposes that external chest compressions increase overall intrathoracic pressure, forcing blood from intrathoracic vessels into the systemic circulation, with the heart acting as a passive conduit.\textsuperscript{24} Recommendations for rescuer hand position during chest compressions in dogs and cats are based on these concepts and vary in accordance with the animal’s size and chest conformation.

In medium to large-breed dogs with standard round-chested conformations (eg, Labrador retrievers, Rottweilers, etc), blood flow generated by the thoracic pump mechanism likely predominates. Therefore, to maximize the intrathoracic pressure generated, it is recommended that the chest be compressed over the highest point on the lateral thoracic wall with the patient in the lateral recumbency position. To the contrary, in very keel-chested dogs (eg, sight hounds, etc), chest compressions with the hands positioned directly over the heart are reasonable, as the cardiac pump mechanism likely predominates. In markedly flat-chested dogs with dorsoventrally compressed chests like humans (eg, English bulldogs, French bulldogs, etc), compressions with the hands positioned over the sternum and the patient in the dorsal recumbency position may be considered. In these and other large dogs with low chest compliance, considerable compression force is necessary for CPR to be effective. Compression posture is essential for maximal effectiveness, and the compressor should lock the elbows with one hand on top of the other and position the shoulders directly above the hands. Using this posture engages the core muscles rather than the biceps and triceps, maintaining optimal compression force by reducing fatigue. 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 chest wall characteristics that favor the cardiac pump mechanism; therefore, chest compressions should be performed directly over the heart. Compressions may be performed using the same 2-handed technique as described above for large dogs, or alternatively by using a single-handed technique whereby 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.

\textit{Airway and Breathing: Ventilation}

Although immediate initiation of chest compressions on recognition of CPA is essential, ventilation is also important and should commence as soon as possible. If an endotracheal tube (ETT) and laryngoscope are available, the patient should be intubated. To avoid interruption in chest compressions, dogs and cats should be intubated in the lateral recumbency position. Once intubated, chest compressions and
ventilations should be performed simultaneously because the inflated cuff of the ETT allows effective alveolar ventilation during a chest compression, prevents gastric insufflation with air, and minimizes interruptions in chest compressions. Intubated patients should be ventilated at a rate of 10 breaths per minute with a short 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 because 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.

If an ETT is not readily available, mouth-to-snout ventilation will provide sufficient 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 1 second should be targeted.

In nonintubated 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 2 short breaths. Alternating compressions and ventilations at a ratio of 30:2 should be continued for 2-minute cycles, and the rescuers rotated every cycle to prevent fatigue. Because the mouth-to-snout technique requires pauses in chest compressions, it should only be used when endotracheal intubation is impossible because of a lack of equipment or trained personnel.

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. Box 3 summarizes the key ALS guidelines for veterinary CPR.

Monitoring

Many commonly used monitoring devices are of limited use during CPR because of their susceptibility to motion artifact and the likelihood that decreased perfusion and altered pulse quality will compromise accurate readings. Pulse oximeter and indirect blood pressure monitors, including Doppler and oscillometric devices, are not useful during CPR unless ROSC is restored. The 2 most useful monitoring modalities during CPR are ECG and capnography.

ECG

Although the ECG is highly susceptible to motion artifact and cannot be interpreted 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 3 most common arrest rhythms are present: (1) asystole, (2) pulseless electrical activity (PEA), or (3) ventricular fibrillation (VF).1,25,26 As interpretation of the ECG requires interruption of chest compressions, the only time the ECG should be evaluated is between 2-minute cycles of CPR while compressors are being rotated. A clear announcement of the rhythm diagnosis to the group by the team leader with an invitation to express differing opinions on the diagnosis will minimize the risk of an incorrect rhythm diagnosis. However, chest compressions should be
### Box 3

**Advanced life support**

**Advanced life support (ALS) key guidelines**

**Monitoring**

- **Electrocardiogram**
  - Apply as soon as possible during CPR
  - Determine rhythm diagnosis during intercycle pauses in compressions

- **End-tidal carbon dioxide**
  - Target minimum of 15 mm Hg as an indicator of chest compression efficacy
  - Sudden increase indicates possible ROSC

**Drug therapy**

- **Vasopressors (eg, epinephrine, vasopressin)**
  - Indicated for asystole, PEA, or refractory ventricular fibrillation
  - Divert blood from the periphery to core organs
  - Repeat every other cycle of CPR (every 4 minutes)
  - Use high-dose epinephrine only for prolonged CPR

- **Vagolytics (eg, atropine)**
  - Indicated for asystole or PEA, especially if due to high vagal tone
  - Decrease parasympathetic tone
  - Repeat every other cycle of CPR (every 4 min)

- **Reversal agents (eg, naloxone, flumazenil, atipamezole)**
  - Administer in any patients treated with reversible drugs before CPA

**Intravenous fluids**

- Use cautiously in euvolemic patients
- Administer in patients with known or suspected hypovolemia

**Corticosteroids**

- Not recommended for routine use during CPR or after ROSC
  - Consider low-dose hydrocortisone in patients after ROSC with refractory hypotension

**Defibrillation**

- **Electrical defibrillation**
  - Treatment of choice for all patients with ventricular fibrillation
  - Continue chest compressions while charging
  - Administer ONE shock
  - Immediately resume chest compressions for 2 min after defibrillation

- **Precordial thump**
  - Deliver a strong blow using the heel of the hand directly over the heart
  - Minimal efficacy—use only if electrical defibrillation is not available
resumed immediately and discussion about the rhythm diagnosis should proceed into the next cycle.

**Capnography (end-tidal CO$_2$ monitoring)**
End-tidal CO$_2$ (ETCO$_2$) can be determined noninvasively and continuously during CPR and is generally feasible to use.$^{27,28}$ The presence of measurable CO$_2$ by ETCO$_2$ monitoring is supportive of (but not definitive for) correct placement of the ETT tube.$^{29}$ Because ETCO$_2$ 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. Similarly, a very low ETCO$_2$ value during CPR (eg, <10–15 mm Hg) was found to be associated with a reduced likelihood of ROSC.$^{1,30}$ ETCO$_2$ substantially increases on ROSC, and therefore, is a valuable early indicator of ROSC during CPR.

**Drug Therapy**
Drug therapy is preferably administered by the intravenous (IV) or intraosseus (IO) route. Therefore, placement of a peripheral or central IV or IO catheter is recommended, but should not interfere with continuation of BLS. 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 (eg, naloxone, flumazenil, atipamezole), IV fluids, and alkalinizing drugs (eg, sodium bicarbonate) may be indicated. Table 1 summarizes the drugs and doses that may be of use during CPR.

**Vasopressors**
Vasopressors are recommended regardless of the arrest rhythm to increase peripheral vascular resistance, thereby increasing central arterial pressure and thus coronary and cerebral perfusion pressures. During CPR, cardiac output is low even during optimal external chest compressions. Therefore, redirecting blood flow away from the peripheral tissues and toward the core (eg, the heart, lungs, and brain) is essential to maintain perfusion to these vital organs. Epinephrine is a catecholamine that causes peripheral vasoconstriction via stimulation of $\alpha_1$-receptors, but also acts on $\beta_1$-receptors and $\beta_2$-receptors. The $\alpha_1$ effects have been shown to be the most beneficial during CPR.$^{31}$ Initially, low doses (0.01 mg/kg IV/IO every other cycle of CPR) are recommended because studies have shown that lower doses are associated with a higher survival to discharge in people.$^{32}$ However, 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 the ETT (0.02 mg/kg low dose; 0.2 mg/kg high dose) by feeding a long catheter through the ETT and diluting the epinephrine 1:1 with isotonic saline or sterile water.$^{33}$

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 acidemic environments in which $\alpha_1$-receptors may become unresponsive to epinephrine, and lack of $\beta_1$ effects that may cause increased myocardial oxygen consumption and worsen myocardial ischemia on ROSC.$^{34}$ Vasopressin may be administered via ETT using the technique described above.

**Atropine**
The use of atropine in CPR has been studied extensively.$^{35–37}$ It is an anticholinergic, parasympatholytic drug. Only a few studies have shown a beneficial effect, but there is minimal evidence of harm during CPR; atropine at a dose of 0.04 mg/kg IV/IO may be
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<th>Drug</th>
<th>Common Concentration</th>
<th>Dose/Route</th>
<th>Comments</th>
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<tr>
<td><strong>Arrest</strong></td>
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<tr>
<td>Epinephrine (low dose)</td>
<td>1 mg/mL (1:1000)</td>
<td>0.01 mg/kg IV/IO, 0.02–0.1 mg/kg IT</td>
<td>Every other BLS cycle for asystole/PEA. Increase dose ×2–10 and dilute for IT administration.</td>
</tr>
<tr>
<td>Epinephrine (high dose)</td>
<td>1 mg/mL (1:1000)</td>
<td>0.1 mg/kg IV/IO, 0.2 mg/kg IT</td>
<td>Consider for prolonged (&gt;10 min) CPR.</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>20 U/mL</td>
<td>0.8 U/kg IV/IO, 1.2 U/kg IT</td>
<td>Every other BLS cycle for IT use.</td>
</tr>
<tr>
<td>Atropine</td>
<td>0.54 mg/mL</td>
<td>0.04 mg/kg IV/IO, 0.08 mg/kg IT</td>
<td>Every other BLS cycle during CPR. Recommended for bradycardic arrests/known or suspected high vagal tone. Increase dose for IT use.</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>1 mEq/mL</td>
<td>1 mEq/kg IV/IO</td>
<td>For prolonged CPR/PCA phase when pH &lt;7.0. Do not use if hypoventilating.</td>
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<tr>
<td><strong>Antiarrhythmic</strong></td>
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<tr>
<td>Amiodarone</td>
<td>50 mg/mL</td>
<td>2.5–5 mg/kg IV/IO</td>
<td>For refractory VF/pulseless VT. Associated with anaphylaxis in dogs.</td>
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<tr>
<td>Lidocaine</td>
<td>20 mg/mL</td>
<td>2 mg/kg slow IV/IO push (1–2 min)</td>
<td>For refractory VF/pulseless VT only if amiodarone is not available.</td>
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<tr>
<td><strong>Reversal agents</strong></td>
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<tr>
<td>Naloxone</td>
<td>0.4 mg/mL</td>
<td>0.04 mg/kg IV/IO</td>
<td>To reverse opioids.</td>
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<tr>
<td>Flumazenil</td>
<td>0.1 mg/mL</td>
<td>0.01 mg/kg IV/IO</td>
<td>To reverse benzodiazepines.</td>
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<tr>
<td>Atipamezole</td>
<td>5 mg/mL</td>
<td>0.1 mg/kg IV/IO</td>
<td>To reverse α2-agonists.</td>
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<tr>
<td><strong>Defibrillation</strong></td>
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<tr>
<td>(may increase dose once by 50%–100% for refractory VF/pulseless VT)</td>
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<tr>
<td>Monophasic External</td>
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<td>4–6 J/kg</td>
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<td>Monophasic internal</td>
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<td>0.5–1 J/kg</td>
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<tr>
<td>Biphasic external</td>
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<td>2–4 J/kg</td>
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<tr>
<td>Biphasic internal</td>
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<td>0.2–0.4 J/kg</td>
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*Abbreviations:* IT, intratracheal; VT, ventricular tachycardia.
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 ETT (0.08 mg/kg). 38

**Anti-arrhythmic drugs**
The treatment of choice for nonperfusing VF/ventricular tachycardia is electrical defibrillation, which is discussed later. However, patients with VF refractory to defibrillation may benefit from treatment with amiodarone at a dose of 2.5 to 5 mg/kg IV/IO. 39 It has been reported to cause anaphylactic reactions and hypotension in some dogs, so patients should be closely monitored for signs of allergic reactions once ROSC is achieved. Treatment with diphenhydramine (2 mg/kg intramuscularly) 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, lidocaine (2 mg/kg slow IV/IO push) may be of benefit for patients with VF refractory to electrical defibrillation. Although lidocaine has been shown to increase the defibrillation threshold in dogs in one study, benefit was evident in other studies. 40,41

**Reversal agents**
Although specific evidence of efficacy is not available, the use of reversal agents in dogs and cats in which reversible anesthetic/analgesic drugs were recently administered may be considered. Naloxone (0.04 mg/kg IV/IO) can 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 fluids**
Euvolemic or hypervolemic patients are unlikely to benefit from IV fluid administration during CPR, because fluids administered IV serve solely to increase right atrial pressure, which may compromise perfusion of the brain and heart. However, in patients with documented or suspected hypovolemia, IV fluids will help to restore adequate circulating volume and may increase the efficacy of chest compressions and improve perfusion.

**Corticosteroids**
Although one prospective observational 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. 1 Other studies have shown no benefit or harm from the use of steroids during CPR. 42,43 It is well known that even a single high dose of corticosteroids can lead to gastrointestinal ulceration and bleeding in dogs, which could then lead to other ill effects, such as bacterial translocation. 44–46 Immunosuppression and reduced renal prostaglandin production, a primary mechanism used by the kidney to maintain perfusion in the face of hypotension, are also known side effects. 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.

**Sodium bicarbonate**
Administration of sodium bicarbonate (1 mEq/kg, once, diluted IV) may be considered in patients with prolonged CPA (greater than 10–15 minutes) because of the likelihood of severe acidemia resulting from metabolic acidosis (eg, lactate and uremic acids) and venous respiratory acidosis due to inadequate peripheral perfusion and accumulation of CO2. Inhibition of normal enzymatic and metabolic activity and severe
vasodilation can result from the acidemia. 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) of metabolic origin.

**Electrical Defibrillation**

Early electrical defibrillation in patients with VF has been associated with increased ROSC and survival to discharge in numerous studies. If the duration of VF is known or suspected to be 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 performed 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, then reversing and delivering a current in the opposite direction). The use of biphasic defibrillators is recommended over monophasic defibrillators because of the higher efficacy in terminating ventricular fibrillation and because a lower current (and hence less myocardial injury) is required to defibrillate the patient successfully. 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, without a pause for rhythm analysis, and a full 2-minute cycle of CPR should be administered before reassessing the ECG and determining if the patient is still in VF, requiring additional defibrillation. 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.

**POSTCARDIAC ARREST CARE**

Two-thirds of human in-hospital cardiac arrest victims that initially achieve ROSC die during the postresuscitation phase. In one veterinary study, only 16% of dogs and cats that reached ROSC after CPA were found to survive to hospital discharge. Consequently, postcardiac arrest (PCA) care plays a significant role in the management of CPA and has the potential to save many lives.

In general, patient outcome is determined by (1) patient condition, (2) events that led to CPA, (3) the ischemic injury sustained during the cardiac arrest itself, and (4) the processes unfolding during and after reperfusion. Abnormalities in the PCA phase are the consequence of the 4 following processes: (1) anoxic brain injury, (2) postischemic myocardial dysfunction, (3) the systemic response to ischemia and reperfusion, and (4) the persistent precipitating pathologic abnormality (eg, underlying disease processes). Because these elements vary between patients, the clinical phenotype of the PCA patient is highly variable and can range from virtually no detectable clinical abnormalities to devastating neurologic and multiorgan dysfunction. Consequently, a “one-size-fits-all” therapy cannot be recommended and rather should be titrated to alleviate the resulting clinical signs using critical care principles. The principles listed below have particular relevance to PCA care. 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
hemodynamically unstable small animals after cardiac arrest.\textsuperscript{52–54} Suitable resuscitation endpoints are a central venous oxygen saturation of at least 70% and/or normalization of lactate levels. Central venous pressure monitoring can be useful to limit the risk of fluid overload, given that left ventricular dysfunction predisposes the patient to hydrostatic pulmonary edema and ischemia-reperfusion injury can lead to increased endothelial permeability.\textsuperscript{55} In PCA patients, cerebral autoregulation of blood flow may be impaired after cerebral anoxia such that cerebral blood flow depends more directly on cerebral perfusion pressure, requiring a mean arterial pressure of 80 mm Hg or greater.\textsuperscript{56}

Neuroprotective measures are of particular importance after cardiac arrest, given the selective vulnerability of the brain to anoxia.\textsuperscript{57,58} Mild therapeutic hypothermia (eg, core temperature of 32–34°C [89.6–93.2°F]) for 12 to 24 hours after ROSC improves neurologic outcome after CPA in humans and other species, including dogs, that remain comatose 1 to 2 hours after resuscitation.\textsuperscript{59–62} Although therapeutic hypothermia may not be applicable broadly in veterinary patients, individual reports support that it is feasible in principle.\textsuperscript{63,64} Current experimental evidence indicates harm associated with rapid active rewarming and hyperthermia in animal models of cerebral ischemia and cardiac arrest. It is therefore important to monitor and control body temperature after CPA. Although optimal rates of rewarming are yet to be established, aggressive, active rewarming should be avoided and passive rewarming should not surpass 1.0°C (1.8°F) per hour, but rather target a slower rate of rewarming of 0.25°C to 0.5°C (0.45–0.9°F) per hour. Seizures should be treated with diazepam (0.5 mg/kg IV/IO) and/or phenobarbital (4 mg/kg IV).

Respiratory optimization is a third important element of PCA care. To assure optimal arterial oxygen tension (eg, partial pressure of arterial oxygen [PaO\textsubscript{2}] 80 to 100 mm Hg) and CO\textsubscript{2} levels (eg, partial pressure of arterial carbon dioxide [PaCO\textsubscript{2}] 35–40 mm Hg) and to prevent respiratory arrest in the comatose PCA patient, the routine use of mechanical ventilation immediately after cardiac arrest and then as needed is optimal, if available. However, this is not required for patients in whom spontaneous ventilation is adequate to maintain the above listed target blood gas values. In all cases, adequacy of ventilation should be monitored using ETCO\textsubscript{2} or arterial blood gas analysis. Experimental and human clinical evidence substantiates the harm that may be associated with hyperoxic reperfusion.\textsuperscript{65–67} As hypoxemia is also harmful, titration of supplemental oxygen to maintain normoxemia (eg, targeting an oxygen saturation [SpO\textsubscript{2}] level of 94%–96% or PaO\textsubscript{2} of 80–100 mm Hg) is therefore a reasonable strategy to reduce oxidative injury while reducing the risk of hypoxemia.

Additional supportive patient care is directed toward the patient’s precipitating disease process and concomitant organ dysfunctions (eg, ileus, acute kidney injury, etc). A summary of the key concepts for PCA care can be found in Box 4.

**PROGNOSIS**

Prognostic indicators in animals that have achieved ROSC have been poorly studied. Although low overall rates of survival have been reported, the inciting cause of cardiac arrest may be one of the most important prognostic factors. In a retrospective study examining characteristics of 15 dogs and 3 cats that survived to hospital discharge, only 3 animals had significant underlying chronic disease, whereas all other patients were considered systemically healthy before the incident.\textsuperscript{25} It is likely that patients experiencing CPA as a consequence of severe, untreatable, or progressive chronic diseases are less likely to survive to hospital discharge, even though these outcomes are confounded by euthanasia. Peri-anesthetic CPA carries a better prognosis for
survival to discharge (as high as 47% in one recent prospective observational veterinary study), and continued CPR efforts in this population are most rewarding.¹

SUMMARY

CPA is a highly lethal condition. To improve outcomes after CPA, a comprehensive strategy that includes preventive and preparedness measures, BLS, ALS, and PCA critical care titrated to the patient’s needs is necessary. Optimization of each of these elements may help improve overall survival and offers an opportunity to work toward that goal.

REFERENCES


