Cardiopulmonary Cerebral Resuscitation: Emergency Drugs and Postresuscitative Care*

Sarah Haldane, BVSc, MACVSc
Steven L. Marks, BVSc, MS, MRCVS, DACVIM
University of Illinois

ABSTRACT:
Cardiopulmonary cerebral resuscitation (CPCR) is an important component of emergency and critical care medicine. Once the basic techniques of CPCR have been instituted, advanced techniques can be used to help resuscitate patients. This article discusses emergency drugs, routes of administration, and postresuscitative care.

Once the basic techniques (i.e., “ABCs” [airway, breathing, circulation]) of cardiopulmonary cerebral resuscitation (CPCR) have been initiated, further intervention is usually indicated. This article discusses the advanced directives of CPCR, such as drug administration, fluid therapy, electrocardiography (ECG), and defibrillation. If CPCR results in successful return of spontaneous circulation (ROSC), efforts must then be directed at postresuscitative care.

ELECTROCARDIOGRAPHY
In veterinary medicine, the most commonly reported arrhythmias in cardiopulmonary arrest (CPA) are asystole, pulseless electrical activity (PEA), and ventricular fibrillation. Sinus bradycardia, sinus tachycardia, and ventricular tachycardia are frequent pre- or postarrest rhythms. Ventricular asystole is seen with end-stage cardiac, pulmonary, or multisystemic disease. Increased vagal tone may also precipitate arrest characterized by ventricular asystole. Asystole has a poor prognosis for resuscitation. When indicated, rapid and aggressive CPCR and epinephrine administration should be instituted. Vasopressin may also be indicated if patients are refractory to epinephrine. When asystole is stimulated by excessive vagal tone, atropine administration may be useful.

PEA (Figure 1), previously known as electromechanical dissociation, occurs when there is no myocardial contractility despite a normal heart rate and rhythm during ECG. PEA may be the terminal rhythm in patients with metabolic or cardiac disease,
and the prognosis for successful resuscitation is poor. PEA may also result from hypovolemia, cardiac tamponade, or pleural space disease, and the prognosis improves if the underlying cause can be rapidly corrected. Epinephrine administration is currently recommended in treating PEA, and efforts should be directed at stabilizing blood volume, oxygenation, ventilation, and circulation. Atropine, calcium chloride, calcium channel blockers, and naloxone have all been evaluated for use in PEA. Although experimental trials with atropine administration or vagotomy have been shown to improve the rate of ROSC, none of these drugs has consistently been shown to be useful in the clinical situation.

Ventricular fibrillation (Figure 2) is seen in 30% to 60% of humans and approximately 20% of animals that experience CPA. Fibrillation results in random electrical stimulation to the myocardium so that the ventricles are unable to effectively contract. Early intervention with electrical defibrillation is the treatment of choice. Electrical defibrillation involves administering a direct current, applied externally or internally. For external defibrillation, the contact areas are optimally on either side of the thoracic cavity, although in large deep-chested dogs, the paddles may be placed in dorsal and ventral positions on the same side of the chest. The contact areas should be clipped and gel applied to the defibrillator electrodes. A charge of 3 to 5 J/kg may be applied. Internal defibrillation requires an electric shock applied directly to the heart. Internal defibrillator paddles may be wrapped in saline-soaked gauze and applied on either side of the heart. A charge of 0.5 to 1 J/kg may be applied.

For both internal and external defibrillation, three shocks can be given in succession, with only a short pause to recharge the defibrillator and check the ECG tracing between each shock. The charge administered may be doubled if two shocks have not resulted in conversion to a sinus rhythm. If defibrillation is not successful, chest compressions should be resumed for 1 to 2 minutes before reapplying a charge. Care should be taken during defibrillation to ensure that personnel are not in contact with the animal or table and that there are not excessive quantities of alcohol around the external contact areas.

Fibrillation lasting longer than 5 minutes is very difficult to convert with electrical defibrillation, although success rates may be improved by providing cardiac compressions and ventilation to enhance myocardial blood flow before electrical defibrillation. Epinephrine can improve coronary perfusion pressure and may change fine fibrillation to coarse fibrillation, which is easier to convert with electrical defibrillation. However, epinephrine also increases myocardial oxygen demand and, in turn, may increase the risk of myocardial ischemia and postresuscitative arrhythmia.

In most cases, there is no indication to use antiarhythmic therapy to treat sinus tachycardia. The cause of tachycardia should be evaluated and treated when possible. Sinus tachycardia may be caused by fear, excitement, pain, fever, hypovolemia, hypoxemia, increased sympathetic stimulation, and many disease states.

Ventricular tachycardia has been defined as three or more rapid, successive, ventricular premature depolarizations or as a continuous series of ventricular beats for more than 30 seconds. It can be caused by both cardiac and noncardiac disease. Treatment should be aimed at the underlying cause. Antiarrhythmic therapy is indicated only if ventricular tachycardia is severe (i.e., a sustained heart rate >200 bpm or R on T phenomenon seen on ECG) or there is evidence of circulatory com-
promise (i.e., pale mucous membranes, weakness, syncope, peripheral pulse rate <60 bpm). Lidocaine administration may be indicated for sustained ventricular tachycardia.18

Hypothermia, increased vagal tone, anesthesia, and certain drugs may cause sinus bradycardia. If bradycardia continues despite resolution of the underlying cause, the treatment of choice is atropine. Epinephrine or dopamine may also be effective in this situation.

**DRUGS**

Many drugs have been evaluated for use in CPCR (Table 1), but very few have been proven to have efficacy during CPA. Epinephrine remains the first-line drug for treating bradycardia, asystole, ventricular fibrillation, or hypotension during CPA.3 Epinephrine is an adrenergic agonist agent with effects at both α- and β-adrenergic receptors. In CPCR, the α-adrenergic effects

administration of high-dose epinephrine, with no change or even a decrease in 24-hour survival rates.22,24

High-dose epinephrine is currently not recommended for routine use in CPCR but may be indicated if lower doses fail to achieve ROSC.3

Vasopressin is a nonadrenergic hormone that decreases renal blood flow and urine output and, in slightly higher doses, stimulates the smooth muscle of precapillary arterioles, causing vasoconstriction in the peripheral tissue beds.25 It increases myocardial and cerebral blood flow without the positive inotropic and chronotropic effects of epinephrine.25 Vasopressin has a longer onset of action than epinephrine and a long half-life (17 to 35 minutes) in circulation. Exogenous vasopressin administration has been shown to have a beneficial effect on ROSC in human and porcine experimental trials26–29 and an outcome similar to that of epinephrine in clinical trials.4,26 It has been suggested that vasopressin may be more useful than epineph-

In clinical trials, atropine has not been shown to increase survival or ROSC.

are the most useful, causing intense peripheral vasoconstriction that leads to increased mean arterial blood pressure, increased aortic diastolic pressure, and therefore increased coronary perfusion pressure. β-Adrenergic stimulation increases the heart rate and myocardial contractility and improves cerebral perfusion by vasodilating the cerebral vasculature, whereas α-agonists mediate constriction of the extracerebral carotid vessels.19–21 Other β-adrenergic effects are potentially less helpful because the positive inotropic effects may potentiate arrhythmias and increase the oxygen demand within the myocardium and cerebrum. However, studies have shown that standard-dose epinephrine has equal or increased survival-to-discharge rates compared with pure α-agonists or epinephrine combined with β-blockers.21,22

Historically, low-dose epinephrine (i.e., 0.01 to 0.02 mg/kg) has been administered to patients in which CPCR has been performed. High-dose epinephrine (i.e., 0.2 mg/kg) was shown to increase myocardial and cerebral perfusion as well as oxygen extraction and initial ROSC in human clinical trials.23 However, there has also been an increased incidence of postresuscitative complications, such as hyperglycemia, hyperkalemia, cardiac dysrhythmias, and myocardial necrosis, with epinephrine in asystolic cardiac arrest.4 However, there are concerns that its prolonged duration of action may cause post-CPA complications regarding ongoing vasoconstriction and significantly decreased renal blood flow.25

Atropine sulfate has parasympatholytic (vagolytic) effects and is recommended in treating sinus bradycardia or counteracting effects of increased vagal tone. It has also been used in combination with epinephrine to treat ventricular asystole and PEA6,7; in clinical trials, however, atropine has not been shown to increase survival or ROSC.8,30

Bretylium tosylate is a class 3 antiarrhythmic agent regarded as a chemical defibrillator; used alone, however, it is unlikely to convert ventricular fibrillation in dogs. Using bretylium may decrease the amount of electrical charge required for defibrillation.31 Bretylium decreases norepinephrine release from peripheral adrenergic nerve endings, leading to antiadrenergic and hypotensive effects. Loss of autonomic reflexes may result in impaired recovery of patients from episodes of defibrillation32; thus this drug is not widely used in veterinary medicine. Bretylium is not currently recommended for use in human CPCR because of its variable efficacy, side effects, and limited availability.1
### Table 1. Drugs Used in CPCR

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of Action</th>
<th>Doses Used in Resuscitation</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone</td>
<td>Class 3 antiarrhythmic</td>
<td>5 mg/kg IV&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Ventricular fibrillation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Refractory ventricular tachycardia</td>
</tr>
<tr>
<td>Atropine sulfate</td>
<td>Parasympatholytic</td>
<td>0.04 mg/kg IV or IO&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Sinus bradycardia</td>
</tr>
<tr>
<td></td>
<td>Decreases vagal tone to the atrioventricular node</td>
<td>0.4 mg/kg IT</td>
<td>Atroventricular block</td>
</tr>
<tr>
<td>Calcium gluconate</td>
<td>Positive inotrope</td>
<td>0.5–1.5 ml/kg IV, to effect (monitor ECG)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Hypocalcemia</td>
</tr>
<tr>
<td>(10% solution)</td>
<td></td>
<td></td>
<td>Hyperkalemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Calcium channel blocker toxicosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hypermagnesemia</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>Calcium channel blocker</td>
<td>0.25 mg/kg slow IV to cumulative dose of 0.75 mg/kg&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Supraventricular tachycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>Positive inotrope</td>
<td>5–15 µg/kg/min CRI&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Myocardial failure</td>
</tr>
<tr>
<td></td>
<td>Predominately β-agonist effects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dopamine</td>
<td>Positive inotrope with α- and β-agonist effects</td>
<td>3–10 µg/kg/min CRI&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Myocardial failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hypotension</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Positive inotrope</td>
<td>Low dose: 0.01–0.02 mg/kg IV or IO every 3–5 min until ROSC&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Asystole</td>
</tr>
<tr>
<td></td>
<td>Increases myocardial contractility</td>
<td>0.1 mg/kg IT</td>
<td>PEA</td>
</tr>
<tr>
<td></td>
<td>Peripheral vasoconstriction</td>
<td></td>
<td>Bradycardia</td>
</tr>
<tr>
<td></td>
<td>Cerebral and coronary vasodilation</td>
<td>High dose: 0.1–0.2 mg/kg IV or IO&lt;sup&gt;a&lt;/sup&gt; 1 µg/kg/min CRI</td>
<td></td>
</tr>
<tr>
<td>Furosemide</td>
<td>Loop diuretic</td>
<td>Dogs: 2–4 mg/kg IM or IV&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Cardiogenic pulmonary edema</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cats: 1–2 mg/kg IM or IV</td>
<td>Hypertension</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Class 1B antiarrhythmic</td>
<td>Dogs: 2–8 mg/kg bolus IV, IT, or IO followed by 25–100 µg/kg/min CRI&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Ventricular arrhythmia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cats: 0.25–0.5 mg/kg slow IV followed by 10–20 µg/kg/min CRI&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Note: Cats are very sensitive to the central nervous system effects of lidocaine</td>
</tr>
<tr>
<td>Magnesium sulfate</td>
<td>Antiarrhythmic: blocks calcium-dependent membrane channels</td>
<td>0.15–0.3 mEq/kg slow IV bolus (maximum 0.75 mEq/kg/day)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Refractory ventricular arrhythmia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Ventricular fibrillation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Severe hypotension</td>
</tr>
<tr>
<td>Mannitol</td>
<td>Osmotic diuretic</td>
<td>0.25–0.5 g/kg slow IV&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Cerebral edema</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Anuria</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Oliguria</td>
</tr>
<tr>
<td>Naloxone</td>
<td>Opiate antagonist</td>
<td>0.02–0.04 mg/kg IV or IO&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Reversal of opiate effects</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.3 mg/kg IT</td>
<td></td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>Alkalinizing agent</td>
<td>0.5–1 mEq/kg IV or as determined by blood gas evaluation&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Severe metabolic acidosis</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>Vasoconstriction</td>
<td>0.8 µg/kg IV or IO once&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Asystole</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Ventricular fibrillation</td>
</tr>
</tbody>
</table>

<sup>CRI</sup> = constant-rate infusion; <sup>IO</sup> = intraosseous; <sup>IT</sup> = intratracheal.


Other drugs have been advocated for managing refractory ventricular fibrillation. Amiodarone is another class 3 antiarrhythmic agent recommended for preventing fibrillation and increasing the success of electrical defibrillation. Amiodarone has been used in humans to treat ventricular fibrillation and in dogs to treat refractory ventricular tachycardia. It may prevent potassium efflux from myocytes secondary to prolonged global ischemia and therefore prevent development of terminal arrhythmia. Hypomagnesemia has been associated with myocardial Purkinje fiber excitability and consequently with generation of ventricular arrhythmia. Infusion of magnesium chloride has been used to convert refractory ventricular dysrhythmia to a normal sinus rhythm. Magnesium administration may also decrease ischemic brain injury and interfere with calcium-mediated reperfusion injury by blocking calcium-dependent membrane channels.

**Administering calcium to humans in CPA is not recommended by the American Heart Association unless there is documented hypocalcemia, hyperkalemia, or hypermagnesemia or there has been a calcium channel blocker overdose.**

Lidocaine is not indicated for ventricular fibrillation because it may increase the defibrillation threshold and make electrical defibrillation more difficult. Lidocaine may be indicated for postarrest ventricular tachycardia, although amiodarone or procainamide has recently been advocated as a first-line drug in these situations. Naloxone is an opiate antagonist. Although usually given to counteract the cardiodepressant and sedative effects of exogenously administered narcotics, naloxone can also counter the myocardial effects of endogenous opiates. Endogenous opioids are thought to play a role in depressing myocardial contractility, possibly leading to generation of PEA. Naloxone may also have mild direct vasoconstrictive, antiarrhythmic, and positive inotropic effects. In CPR, it has been administered to reverse opiate effects and make the myocardium more responsive to catecholamines; however, there is no increase in myocardial blood flow, cerebral perfusion, or survival with naloxone administration.

In hypoxic states, calcium entry into cells may increase. Increased intracellular calcium can lead to uncoupling of oxidative phosphorylation and generation of inflammatory mediators and oxygen free radicals, which are toxic to brain tissue. Increased intracellular calcium has also been implicated in myocardial stunning, in which myocardial cells surrounding a zone of ischemia become dormant, leading to an increased propensity for cardiac arrhythmia. Administering calcium to patients in CPA is not recommended by the American Heart Association unless there is documented hypocalcemia, hyperkalemia, or hypermagnesemia or there has been a calcium channel blocker overdose.

Calcium channel blockers may act on the ischemic myocardium to increase blood flow and raise the threshold of ventricular fibrillation. They may also antagonize the proarrhythmogenic effects of \( \beta \)-adrenergic stimulation following ischemic events and exogenous epinephrine administration. By blocking calcium-mediated reperfusion injury, calcium channel blockers may also improve neurologic recovery after return of spontaneous cerebral blood flow.

In low-flow circulatory states, carbon dioxide (\( \text{CO}_2 \) accumulates in peripheral vascular beds. Hypercarbia leads to decreased left ventricular performance, decreased cardiac output, and increased refractoriness of myocardial cells. In this situation, acidemia can be resolved by reinstating pulmonary blood flow, which facilitates excretion of accrued \( \text{CO}_2 \) via the respiratory system. When high levels of \( \text{CO}_2 \) are present in the blood, administering sodium bicarbonate (\( \text{NaHCO}_3 \) paradoxically worsens intracellular and cerebral acidosis by further increasing \( \text{CO}_2 \) levels via the carbonic anhydrase reaction. Adverse effects of \( \text{NaHCO}_3 \) administration include hypernatremia, hypokalemia, decreased ionized calcium concentrations, and increased incidence of cardiac arrhythmia. Overdosing \( \text{NaHCO}_3 \) leads to metabolic alkalosis, which, in turn, causes a left shift in the oxyhemoglobin dissociation curve. This means there is less offloading of oxygen from red cells in the periph-
eral tissues and, consequently, cellular hypoxia is exacerbated. However, if venous blood gas measurements reveal preexisting metabolic acidosis, NaHCO₃ administration may be indicated.

Glucose administration is not advised during CPCR because studies have shown increased morbidity and mortality associated with hyperglycemia during CPA. Glucose is a substrate for anaerobic glycolysis and lactic acid production. In brain tissue, toxic concentrations of lactic acid accumulate and cause cellular damage, leading to permanent neurologic dysfunction.

Route of Administration
The route of choice for administering drugs during CPCR is via a jugular catheter to allow simultaneous fluid and drug administration, facilitate delivery of drugs at their site of action, and decrease the possibility of extravasation of vasoactive drugs, which may cause tissue necrosis. Using a hindlimb catheter is not recommended because flow to and from the caudal half of the body is restricted during CPCR. If an intravenous catheter is not in place before CPA, intravascular access may be difficult to attain and a venous cutdown may be required. Some emergency drugs can be administered via the intratracheal route (e.g., lidocaine, epinephrine, atropine, naloxone, vasopressin) at higher doses than are usually given intravenously (Table 1). A 5- to 8-Fr urinary catheter or feeding tube may be used to deposit the drugs, diluted with sterile saline, into the small airways (Figure 3). Immediately applying positive-pressure ventilation enhances drug absorption. The efficacy of this practice was recently questioned by two human studies in which endotracheal administration of medications during CPCR was associated with a lower ROSC and survival to discharge compared with intravenous administration. These results may have been confounded by significant comorbidity in patients with difficult venous access; however, in a clinical CPCR trial in which epinephrine administration was randomly assigned to the intravenous or endotracheal route, the endotracheal route failed to significantly increase plasma epinephrine concentrations. Intraosseous access allows administration of both drugs and fluids. The marrow is close to the central circulation and does not collapse in cases of poor perfusion. The sites for intramedullary access include the femur, humerus, and tibia. Intracardiac administration of drugs is not recommended, especially in external CPCR, because of a high probability of injecting them into the myocardial muscle layer. This can result in hemorrhage, focal myocardial ischemia, and cardiac arrhythmia.

FLUID THERAPY
CPA has been shown to cause a fluid shift where the plasma volume moves from the intravascular to extravascular space. Judicious administration of an iso-

Glucose administration is not advised during CPCR because studies have shown increased morbidity and mortality associated with hyperglycemia during CPA.

Hypertonic saline has recently been reported to be more useful than normal saline or synthetic colloids in resuscitating pigs after prolonged fibrillation. Hypertonic saline improves...
intravascular volume by increasing osmotic pressure and may also prevent endothelial cell swelling, which is a common sequela to cellular hypoxia. Swollen endothelial cells cause increased resistance in the coronary and cerebral arteries, thereby decreasing blood flow and exacerbating myocardial and cerebral ischemia.\(^59,60\) Hypertonic saline should not be administered to dehydrated patients because its mechanism of action results in further intracellular fluid loss. Concurrently administering crystalloid fluids is recommended.

**POSTRESUSCITATION**

Once spontaneous circulation and heartbeat have been restored, it is important to adequately monitor and support the vital organ systems, with particular emphasis on the respiratory, cardiovascular, and neurologic systems. Almost half of postresuscitation deaths in humans occur within 24 hours of CPCR.\(^7\) Postarrest inflammatory processes, reoxygenation injury, and reperfusion failure can combine to cause metabolic derangements and cellular death, which can cause organ dysfunction up to 1 to 3 days after the initial insult.\(^3\)

Supplemental oxygen administration is recommended via oxygen cage, hood, or nasal catheter. Continued ventilatory support may be required, especially if spontaneous respiratory insufficiency is noted; however, care must be taken not to overventilate the patient because postresuscitation hypocapnia may cause cerebral vasoconstriction and worsen cerebral ischemia.\(^41\) Pulmonary edema may develop secondary to positive-pressure ventilation, thoracic compressions, and changes in pulmonary hydrostatic pressure.\(^62\)

Restoring blood volume with judicious fluid therapy is indicated to counteract fluid shifts\(^54\) and treat electrolyte imbalances that occur during CPA.\(^3\) Positive inotropic support, such as dopamine or dobutamine infusion, may be required to improve cardiac output and peripheral perfusion.\(^3\)

Neurologic dysfunction is a common but often temporary postarrest complication, and a minimum of 24 to 48 hours should be allowed to elapse before basing a prognosis on neurologic signs.\(^2\) In humans, complete recovery has occurred as much as 90 days after CPCR.\(^45,63\) Mannitol infusion may be used to counteract cerebral edema and provide some scavenging of reactive oxygen species.\(^44\) Glucocorticoids are not indicated in this situation because they do not reduce cerebral edema\(^41\) and may worsen posts ischemic neurologic injury by increasing serum glucose levels.\(^42\) Animals with mild hypothermia (\(>95^\circ F [>35^\circ C]\)) after CPA should not be aggressively rewarmed because mild hypothermia combined with vascular support to maintain blood pressure may improve cerebral resuscitation.\(^65\) There are currently no recommendations to actively cool normothermic patients.\(^3\)

**CONCLUSION**

It is important to remember that CPCR is usually not successful and good client communication is essential to prevent unrealistic expectations for patients at risk of CPA. Successful CPCR relies on patient selection, teamwork, and appropriate use of resuscitation techniques and drugs. Having a well-trained staff and adequately stocked emergency kit significantly improve the ability to provide lifesaving care to patients.

**REFERENCES**


### ARTICLE #3 CE TEST

*This article qualifies for 2 contact hours of continuing education credit from the Auburn University College of Veterinary Medicine. Subscribers who wish to apply this credit to fulfill state relicensure requirements should consult their respective state authorities regarding the applicability of this program. To participate, fill out the test form inserted at the end of this issue. To take CE tests online and get real-time scores, log on to www.VetLearn.com.*

1. Epinephrine acts as an agonist at both α- and β-adrenergic receptors. Which statement regarding CPCR is correct?
   a. The β-agonist effects of epinephrine are most useful in CPCR.
   b. α-Agonist effects cause peripheral vasodilation, leading to decreased aortic diastolic pressure and therefore increased coronary perfusion pressure.
   c. The positive inotropic effects of β-agonists increase myocardial oxygen consumption.
   d. Using pure α-agonists leads to better survival-to-discharge rates compared with using epinephrine.

2. Which drug is not indicated as an adjunct for defibrillation?
   a. amiodarone
   b. lidocaine
   c. bretylium
   d. magnesium sulfate

3. Which of the following may be used to help prevent reperfusion injury in the brain?
   a. atropine
   b. calcium
   c. glucose
   d. calcium channel blockers

4. Which drug is indicated for treating PEA?
   a. epinephrine
   b. NaHCO₃
   c. lidocaine
   d. calcium chloride

5. Which route of drug administration is not recommended during CPCR?
   a. central intravenous line
   b. intraosseous
   c. intracardiac
   d. intratracheal

6. Which of the following is not usually indicated in the postresuscitation phase?
   a. dexamethasone
   b. dobutamine
   c. oxygen
   d. mannitol

7. For which agent is transtracheal administration not recommended?
   a. epinephrine
   b. naloxone
   c. lidocaine
   d. NaHCO₃

8. Which of the following is not a common site for intramedullary catheter placement?
   a. humerus
   b. sternum
   c. femur
   d. tibia

9. Which statement best describes PEA?
   a. It is an arrhythmia that reflects uncoupling of the mechanical and electrical activity of the heart.
   b. It is an arrhythmia that can be treated effectively with lidocaine.
   c. It is often the result of electrolyte disturbances and can be easily managed.
   d. It may respond to electrical defibrillation.

10. Which statement regarding fluid therapy during resuscitation is correct?
    a. High volumes of fluid are indicated to resuscitate patients that require fluid therapy.
    b. Hypertonic saline should not be used because of the risk of increased intracranial pressure.
    c. Fluid therapy with colloids should be avoided during resuscitation.
    d. Increased right atrial pressure may lead to decreased coronary perfusion.