Misconceptions About Emergency and Critical Care: Cardiopulmonary Cerebral Resuscitation, Fluid Therapy, Shock, and Trauma*

Marie K. Holowaychuk, DVM
North Carolina State University

Linda G. Martin, DVM, MS, DACVECC
Washington State University

ABSTRACT: Diagnostic and therapeutic techniques in veterinary medicine often arise from a combination of clinical experience, expert opinion, and current scientific evidence. It is important that these techniques be continually reevaluated to avoid perpetuating outdated medical practices. As additional research is conducted, many beliefs that were once considered fact are refuted; however, reassessment of our viewpoints does not always keep pace with the rate of scientific innovation or changes in the current consensus of expert opinion. Thus common practices and procedures that were once widely accepted become obsolete. This article dispels some common misconceptions related to cardiopulmonary cerebral resuscitation, fluid therapy, shock, and trauma.

Advances in medical knowledge constantly challenge clinicians’ ability to provide the best care for their patients and the most current clinical information to their clients. Scientific research in emergency and critical care medicine has recently grown significantly in human and veterinary medicine. To provide appropriate care for veterinary patients, practitioners must have a clear and accurate understanding of the current scientific evidence in veterinary medicine.

In discussing emergency and critical care medicine with veterinary practitioners and students, we have become aware of several common misconceptions. This article is intended to clarify misunderstandings and misconceptions involving small animal emergency and critical care medicine and assist veterinarians in providing high-quality care for their patients. This article also focuses on dispelling some common misconceptions related to cardiopulmonary cerebral resuscitation (CPCR), fluid therapy, shock, and trauma.

*A companion article on misconceptions about metabolic disease and intensive care medicine starts on p. 434.
**CARDIOPULMONARY CEREBRAL RESUSCITATION**

**Misconception:** Intravenous fluid therapy should always be given during CPCR.

**Reality:** The use of intravenous fluid therapy has been advocated as standard practice in veterinary CPCR.\(^1,2\) It seems intuitive that the use of intravenous fluids during CPCR would expand blood volume and therefore increase forward blood flow to the brain and heart. Although this is true for hypovolemic patients, volume loading of euvolemic humans and animals may actually decrease myocardial and cerebral perfusion, a phenomenon that occurs despite increased forward blood flow.\(^3-6\)

The major determinant of myocardial blood flow is coronary perfusion pressure, which is defined as follows:

\[
\text{Coronary perfusion pressure} = \text{Aortic diastolic pressure} - \text{Right atrial diastolic pressure}
\]

To increase coronary perfusion pressure, the gradient between aortic and right atrial diastolic pressures must increase by increasing aortic pressure or decreasing right atrial pressure.\(^3\) Fluid therapy elevates right atrial pressure, which decreases coronary perfusion pressure by reducing the aortic to right atrial diastolic pressure gradient.\(^3-5\)

Similarly, the major determinant for cerebral blood flow is cerebral perfusion pressure, which is defined as follows:

\[
\text{Cerebral perfusion pressure} = \text{Mean arterial pressure (MAP)} - \text{Intracranial pressure (ICP)}
\]

Aggressive intravenous fluid infusion in CPCR appears to increase ICP and decrease cerebral perfusion pressure in both humans and dogs by increasing ICP more than MAP.\(^3-6\) An investigation into the potential adverse effects of volume loading during closed-chest CPCR in dogs revealed that rapid administration of 1 L of normal saline (0.9% NaCl) or 10% Dextran substantially increased total forward blood flow in euvolemic patients. However, blood flow to the cerebral hemispheres, cerebellum, brain stem, and ventricular myocardium decreased significantly.\(^6\) These deleterious changes in critical regional blood flow were accompanied by disproportionate increases in right atrial and intracranial pressures relative to the increase in aortic and mean arterial pressures, which reduced the pressure gradient across the coronary and cerebral circulations. Therefore, during CPCR, intravenous fluid therapy should be administered at high-volume rates only to animals with preexisting hypovolemia.

**Misconception:** Lidocaine is a first-line drug for the treatment of ventricular fibrillation in CPCR.

**Reality:** Lidocaine has long been a first-line drug in CPCR. However, its role as a chemical defibrillator has recently been questioned because of a lack of documented efficacy in cardiac arrest.\(^3,4\) Several canine studies\(^7-9\) have revealed that lidocaine administration increased the electrical energy required for defibrillation, often by as much as 100%; as blood concentrations of lidocaine decreased, the percentage of successful defibrillations actually increased. Lidocaine is still an excellent antiarrhythmic for the treatment of ventricular tachycardia. However, the use of lidocaine as a chemical defibrillator should be avoided because evidence indicates that lidocaine increases the defibrillation threshold and makes electrical defibrillation more difficult in dogs.\(^10-12\)

In humans, a new drug, amiodarone (a class III antiarrhythmic), is now listed ahead of lidocaine as a first-line agent in these situations. Amiodarone has been shown to increase return of spontaneous circulation and early survival compared with the use of a placebo and lidocaine for refractory ventricular fibrillation in humans.\(^13,14\) However, no veterinary studies have examined the efficacy of amiodarone in this setting.

**Misconception:** Doxapram is a first-line drug for stimulation of breathing in CPCR.

**Reality:** Doxapram is a central nervous system (CNS) stimulant that has been used in various ways in veterinary medicine.\(^15\) It is commonly used to initiate or stimulate respirations in newborn animals following dystocia or cesarean section, speed awakening from anesthesia, treat respiratory depression, and assist in the diagnosis of laryngeal paralysis in dogs.\(^15,16\) The respiratory stimulant effects of the drug led to it being stocked in many hospitals.
“crash carts” specifically for use in CPCR. Use of doxapram in this manner has fallen out of favor over the past 10 years.17–18

There are several reasons doxapram should be avoided in arrest situations. The drug’s CNS stimulatory effects include direct stimulation of the medullary respiratory centers and reflex activation of carotid and aortic chemoreceptors, causing a temporary increase in respiratory rate and tidal volume but without the expected increase in arterial oxygenation.15 The benefits due to an increased respiratory rate and volume are offset by a concurrent increase in respiratory effort leading to increased oxygen consumption and carbon dioxide (CO₂) production.15,18 This CNS stimulation generally increases the cerebral metabolic requirement for oxygen. Doxapram has also been shown to reduce cerebral blood flow as well as increase myocardial oxygen requirements in both humans and animals.19–21 When an animal experiences cardiac arrest, it is preferable to decrease cerebral and myocardial oxygen requirements because perfusion is poor and oxygen content in the blood is reduced. Increased oxygen demands can lead to cerebral and myocardial hypoxia, possibly resulting in brain injury and failure of return of spontaneous circulation. Use of doxapram also causes transient hypotension before its stimulatory effects. This can be dangerous if the drug is given to an already hypotensive animal.22

Therefore, because of the risks for cerebral and myocardial damage, use of doxapram alone or in conjunction with positive pressure ventilation is contraindicated in cases of severe respiratory depression as in cardiopulmonary or respiratory arrest. This drug should not be considered a substitute for appropriate mechanical ventilation when indicated.15 Doxapram may be a useful drug in other applications, but its use is currently not recommended for patients in cardiopulmonary arrest.

FLUID THERAPY

Misconception: Lactated Ringer’s solution is always a safe choice for fluid therapy.

Reality: Lactated Ringer’s solution is a crystalloid replacement fluid that has an osmolality similar to the extracellular fluid compartment. It is considered isotonic and has a pH of 6.5. The following electrolytes are components of the solution: 130 mEq/L of sodium, 4 mEq/L of potassium, 109 mEq/L of chloride, and 3 mEq/L of calcium.23,24 Lactated Ringer’s solution also contains 28 mEq/L of lactate, which is converted to bicarbonate by the liver. The lactate in lactated Ringer’s solution does not affect plasma lactate levels in an animal with a normally functioning liver.24

Lactated Ringer’s solution is a good replacement fluid for volume deficits. Replacement solutions contain a sodium concentration near the plasma sodium concentration and are appropriate for plasma volume expansion.24 Patients presenting in hypovolemic shock would benefit from the use of lactated Ringer’s solution for fluid resuscitation. Lactated Ringer’s solution is also appropriate in treating conditions in which added calcium and potassium are desired or a moderate sodium load is preferred over a higher sodium concentration.24 Because lactated Ringer’s solution contains the bicarbonate precursor lactate, it is considered to be an alkalinizing solution. It is indicated in patients with metabolic acidosis, which may occur in patients with diarrhea, vomiting, renal disease, and various metabolic or endocrine disorders.23

Although lactated Ringer’s solution can be used in many clinical situations, veterinarians should be aware of the contraindications for its use. A common misconception among practitioners is that lactated Ringer’s solution is a universal replacement fluid. Because lactated Ringer’s solution contains calcium, it is contraindicated for use in animals with hypercalcemia.23 Transfused blood should not be administered through an injection port of the delivery set containing lactated Ringer’s solution or any other calcium-containing solution because the calcium can activate the clotting system, causing macro- or micro-clotting in the tubing.23,25–27 Sodium bicarbonate is also incompatible with lactated Ringer’s solution, as are other specific medications that may require constant-rate infusion.23,28 Because of the inability of the liver to metabolize lactate to bicarbonate, lactated Ringer’s solution is a poor substitute.
choice for patients with hepatic disease. Therefore, fluids containing lactate are not recommended for patients with severe liver disease. A patient with metabolic alkalosis would not benefit from lactated Ringer’s solution because the summation of bicarbonate would worsen the alkaliotic state of the animal.

In addition to this, patients with neoplasia, specifically lymphoma, have been noted to be hyperlactatemic and may have a transient inability to handle increased lactate loads compared with normal dogs. Increased blood lactate values in tumor-bearing patients are believed to primarily result from increased lactate production because tumors preferentially metabolize glucose for energy using anaerobic glycolysis, thereby forming lactate as an endproduct. Lactate produced by anaerobic glycolysis results in a net gain of 2 ATP per mole of glucose substrate compared with the net gain of 38 ATP in oxidative metabolism. The patient then has to expend 12 ATP, converting the lactate back to glucose through the Cori cycle. An increase in daily energy needs of up to 20% has been theorized to occur secondary to such increased Cori cycle activity in human cancer patients, contributing to cancer cachexia. Because of these findings, the use of lactated Ringer’s solution should be avoided in lymphoma patients and possibly other cancer patients.

It is important to consider the patient as a whole before choosing a solution for fluid therapy. Knowing the components of each fluid and being aware of the patient’s needs can aid clinicians in choosing the most appropriate fluid for the patient and ultimately allow progress toward recovery.

**Misconception:** The intravenous fluid of choice for animals with severe hypernatremia is 0.45% sodium chloride (NaCl) or 5% dextrose in water. **Reality:** Treatment of an animal with hypernatremia should depend on the rapidity of the development of the hypertonic state and the severity of hypernatremia. After 24 to 48 hours of hypernatremia, brain cells produce substances called *organic osmolytes* (*idiogenic osmoles*), which increase intracellular tonicity, thereby protecting the brain from cellular dehydration. These organic osmolytes include amino acids (*e.g.*, glutamine, glutamate, taurine), methylamines (*e.g.*, phosphocreatine), and polyols (*e.g.*, myoinositol). Organic osmolytes increase cellular tonicity in the brain, thereby reducing the movement of fluid from brain cells into the extracellular space. Unfortunately, this protective mechanism is also what predisposes the brain to injury following rapid correction of hypernatremia.

If hypernatremia is corrected too rapidly with 0.45% NaCl or 5% dextrose in water, plasma tonicity is lowered abruptly. Because of organic osmolytes in brain cells, fluid is then pulled from the extracellular space into the brain, causing cerebral edema. This can be avoided by slowly reducing plasma sodium concentrations in patients with chronic hypernatremia, such as those with severe hypernatremia (>170 mEq/L) for more than 24 to 48 hours or an unknown duration. It is recommended to lower plasma sodium concentrations no more than 0.5 to 0.7 mEq/L/hr until they are within the normal range—a process that usually occurs within 48 to 72 hours. This can be more safely achieved by administering isotonic fluids with sodium contents nearer to the sodium concentration measured in serum. For example, in a dog with a sodium concentration of 180 mEq/L, 0.9% NaCl (sodium: 154 mEq/L) would be the appropriate fluid to administer because of its high sodium content. In comparison, 0.45% NaCl (sodium: 77 mEq/L) or 5% dextrose in water (sodium: 0 mEq/L) would cause serum sodium concentrations to drop very quickly. The high level of sodium contained in 0.9% NaCl would enable serum sodium concentrations to decrease more gradually, thereby reducing the risk for cerebral edema. Once the sodium concentration is reduced to near normal, the fluid may be changed to 0.45% NaCl or 5% dextrose in water.

**Misconception:** Dextrose-containing fluids provide enough energy to sustain hospitalized animals that are anorectic and need nutritional support. **Reality:** Because of the low energy content of dextrose-containing fluids and their inability to meet patient maintenance energy requirements, these fluids should not be used as the only source of nutritional support in critically ill animals.
Reality: The daily resting energy requirement (RER) or calories required for a resting animal in a thermoneutral environment can be estimated using the following formula:

\[
\text{RER (kcal/day)} = 30 \times (\text{Weight in kg}) + 70 \quad \text{for animals >}2 \text{kg [4.4 lb]}
\]

One liter of 5% dextrose in water provides approximately 170 kcal. For a 10-kg (22-lb) dog with an RER of 370 kcal/day (30 × 10 + 70), the amount of 5% dextrose required to meet these requirements would be approximately 2,180 ml (370 ÷ 170 × 1,000). Based on a daily maintenance fluid requirement of 60 ml/kg, this dog would require only 600 ml/day (60 × 10). Thus if 5% dextrose in water were used to sustain this patient’s RER, the patient’s fluid rate would need to be over three and one-half times the maintenance rate! Similarly, a cat weighing 4 kg (8.8 lb) has an RER of 190 kcal/day (30 × 4 + 70). Achieving this would require administering approximately 1,120 ml (190 ÷ 170 × 1,000) of 5% dextrose in water. The daily maintenance fluid requirement of the cat is 240 ml/day (60 × 4); therefore, this cat would receive over four and one-half times its maintenance rate!

Because of the low caloric content of 5% dextrose in water, the amount of fluid necessary to meet the RER would be incredibly high. In critically ill patients that are hypermetabolic (e.g., starvation, extensive wounds, trauma, sepsis), caloric rates one and one-half to two times higher than normal may be necessary to sustain their energy requirements. The fluid rates required to meet these needs could lead to volume overload and even death. Other potential problems with such high rates of dextrose administration include hepatic lipid accumulation and a negative nitrogen balance due to continuous stimulation of insulin secretion. Therefore, for anorectic critically ill patients, nutritional demands should be met in other ways, such as enteral nutrition via feeding tubes or parenteral nutrition. Dextrose-containing fluids are more appropriate in managing patients with hypoglycemia, such as those with insulin overdose or hepatic failure.

**SHOCK AND TRAUMA**

**Misconception:** Cats in shock are typically tachycardic.

**Reality:** Many aspects of emergency and critical care medicine are unique to cats. Their physiologic response to shock is often different than that in dogs. The daily maintenance fluid requirement of 60 ml/kg in dogs is much higher than in cats, which can be hypotensive and hypothermic. Shock in cats is typically hypodynamic and decompensatory; the hyperdynamic signs of septic shock do not commonly occur. The heart rate is often normal or slow with concurrent hypotension, instead of the typical tachycardia demonstrated by other species. A retrospective study of sepsis in 29 cats identified bradycardia as one of the most common clinical signs in this patient population. The exact mechanism for bradycardia in feline shock is unknown, but numerous factors may contribute. In cats, adrenergic receptors become refractory to catecholamines at body temperatures lower than 100°F (37.8°C), resulting in bradycardia and secondary compensatory vasoconstriction. Other mechanisms of bradycardia include cytokine-induced myocardial depression and parasympathetic stimulation. Cats with endotoxemia demonstrate decreased left ventricular contractility and increased left ventricular diastolic volume. This activates stretch receptors in the left ventricle and leads to reflex bradycardia from vagal stimulation. It has also been proposed that cats may not develop tachycardia in response to hypotension because of simultaneous baroreceptor stimulation of vagal and sympathetic fibers. To avoid misdiagnoses and inappropriate therapies, differences between cats and other species must be kept in mind when assessing patients in shock.

**Misconception:** Ventricular arrhythmias following traumatic episodes are the result of direct damage to the heart and respond to treatment only with antiarrhythmic drugs such as lidocaine or procainamide.
Reality: Although arrhythmias are common in dogs following trauma, it is important to realize that trauma to the heart itself is not a prerequisite for ventricular arrhythmia formation. Arrhythmias can occur in animals following significant traumatic episodes; however, affected animals may not have thoracic injuries. Ventricular arrhythmias, such as ventricular premature contractions and ventricular tachycardia (Figure 2), are the most common ones following trauma and often are not present until 24 to 48 hours following the traumatic episode. Although blunt trauma may lead to myocardial contusions, pericardial rents, septal perforation, and cardiac rupture, all of which may cause ventricular arrhythmias, many other disorders in dogs recently sustaining trauma may also explain the development of arrhythmias. Before instituting drug therapy to correct arrhythmias, it is essential to consider and correct concurrent abnormalities that may be contributing to arrhythmia formation.

Catecholamine release due to hypovolemia, hypotension, shock, and pain is a common cause of arrhythmia formation. Catecholamines have direct arrhythmogenic effects via enhanced influx of sodium and calcium, which increases automaticity and heart rate; increased amplitude of after potentials; and nonuniform repolarization of the myocardium, all of which may provoke arrhythmias. Hypokalemia and ischemia of the myocardium may also be induced by catecholamine release and contribute to arrhythmia formation. Reperfusion injury following recovery from hypovolemic shock may cause myocardial necrosis and arrhythmias. Similarly, acidosis alters the myocardial cell membrane and predisposes animals to arrhythmia formation. Anemia and hypoxemia, both of which are common in animals sustaining trauma, can result in myocardial tissue hypoxia as well as compensatory catecholamine release, which also can lead to the development of arrhythmias. Neurologic injury in humans with head trauma provokes activation of the sympathetic nervous system following stimulation of the hypothalamus. This results in alterations in cardiac repolarization and subsequent arrhythmia formation. It is important to remember that administration of antiarrhythmics is not a benign treatment. Most antiarrhythmics are arrhythmogenic and can worsen arrhythmias, decrease myocardial function, and result in systemic side effects. Therefore, other causes of ventricular arrhythmias in animals sustaining trauma must be carefully considered. Patients should be adequately treated with supplemental oxygen, analgesics, and intravenous fluids as well as have anemia or acid–base disturbances corrected while administration of antiarrhythmics is considered.

Misconception: Opioids are contraindicated in all patients with chest trauma because the respiratory-depressant effects of the drugs worsen the clinical signs.

**Butorphanol tartrate is one of many opioids, including morphine, hydromorphone, and fentanyl, that are safe and effective analgesics for animals that have sustained chest trauma.**
Reality: Animals that have sustained chest trauma frequently present with injuries such as rib fractures, pulmonary contusions, pneumothorax, diaphragmatic hernia, and flail chest. Rib fractures are common with any traumatic event, including motor vehicle accidents and crushing bite wounds. It is common for animals presenting with rib fractures to be hypoxemic and in shock. Hypoxemia resulting from rib fractures is due to both damage to the lung parenchyma and pain from the injuries, which causes hypoventilation. If the force sustained during a traumatic event is sufficient to fracture ribs, it inevitably ruptures alveoli and adjacent vessels and causes intraalveolar and interstitial hemorrhage, leading to formation of pulmonary contusions. The result is impaired diffusion of CO₂ and oxygen across the alveolus as well as ventilation–perfusion (V/Q) mismatch due to reduced pulmonary alveolar perfusion. In addition, pain associated with concussive injuries can restrict respiration as a result of patient reluctance to fully expand the chest during inspiration. The resultant hypoventilation exacerbates hypoxemia and further compromises the animal’s condition.

Treatment of animals sustaining chest trauma should include provision of supplemental oxygen and careful administration of analgesics to improve ventilation. Many veterinarians are hesitant to use opioids in critically ill patients that have sustained chest trauma because of concern that the respiratory-depressant effects of opioids will further decompensate the patient and worsen existing hypoxemia. However, there is a paucity of studies evaluating respiratory function in small animals during opioid administration. A study by Berg and Orton is the only one in the literature that evaluates blood gases in dogs receiving morphine and oxymorphone after intercostal thoracotomy. Hypoventilation was documented as evidenced by elevated partial pressure of CO₂; however, the degree of hypoventilation was not clinically significant and does not support withholding opioids from patients with thoracic disease. In general, most clinicians and critical care specialists believe that dogs and cats are relatively resistant to the respiratory-depressant effects of opioids and that these effects are likely only with the use of other centrally depressive drugs (e.g., anesthetic agents). Opioids can also be administered to effect, and potential reversal with naloxone is an option in case adverse effects occur. Therefore, it is safe and highly recommended to administer low doses of µ-opioids (e.g., morphine, hydromorphone, oxymorphone, fentanyl), opioid agonist–antagonists (e.g., butorphanol), or partial opioid agonists (e.g., buprenorphine). Accepted doses of µ-opioids include morphine at 0.5 to 1 mg/kg SC or IM q3–4h or fentanyl at 1 to 5 µg/kg/hr via constant-rate infusion, both of which anecdotally provide adequate analgesia without impairing ventilation. Alternatively, opioid agonist–antagonists have a ceiling respiratory-depressant effect, thereby reducing the risk for respiratory compromise. Butorphanol can be used safely at doses of 0.1 to 0.2 mg/kg IV q1h or 0.2 to 0.4 mg/kg SC or IM q1–2h.

In addition to opioid administration, in cases of rib fractures or flail chest, local analgesics can be applied at the caudal border of each rib, above and below each fracture site, and at the caudal borders of the rib cranial to and caudal to the flail segment. The duration of action of lidocaine is 1 to 2 hours, and the total dose is 1 to 2 mg/kg diluted to sufficient volume to deliver 0.5 ml at each injection site. Bupivacaine may be used at the same dose, and its effects last 4 to 6 hours. These methods enable conservative management of chest wall injuries and avoid the need for internal or external stabilization of flail segments while improving chest wall excursion and reducing hypoventilation.
CONCLUSION
A solid understanding of CPR, fluid therapy, trauma, and shock are pertinent to ensuring that emergency clinicians are able to provide effective and appropriate care for critical patients. Because these topics encompass some of the most important aspects of emergency and critical care, they are constantly being reevaluated. Reevaluation may come via new scientific evidence or modifications of expert opinion. Thus many beliefs that were previously considered correct are no longer appropriate. It is important to recognize the most up-to-date evidence and reasoning for the development of new techniques and standards to provide the best quality of care for critical patients.

REFERENCES
44. DiBartola SP: Introduction to fluid therapy, in DiBartola SP (ed): Fluid


4. Which statement regarding the use of intravenous fluid therapy in CPCR is incorrect?
   a. In euvoletic patients, an elevation in right atrial pressure secondary to fluid therapy decreases coronary perfusion pressure.
   b. Fluid therapy decreases cerebral perfusion pressure by increasing MAP more than ICP.
   c. Intravenous fluid therapy is helpful only in resuscitating hypovolemic patients.
   d. The major determinant of cerebral blood flow is cerebral perfusion pressure.

5. Which statement regarding the use of lactated Ringer's solution is incorrect?
   a. Because of the inability of the liver to metabolize lactate to bicarbonate, lactated Ringer's solution is a poor choice for patients with hepatic disease.
   b. The lactate in lactated Ringer's solution does not affect plasma lactate levels in an animal with a normally functioning liver.
   c. Lactated Ringer's solution is indicated in treating patients with metabolic alkalosis.
   d. Sodium bicarbonate is incompatible with lactated Ringer's solution.

6. Organic osmolytes
   a. are composed of fatty acids, methylamines, and polyols.
   b. decrease cellular tonicity in the brain.
   c. are produced by the brain after 24 to 48 hours of hyponatremia.
   d. reduce the movement of fluid from the brain into the extracellular space.

7. Which is not a proposed mechanism of bradycardia in feline shock?
   a. cytokine-induced myocardial depression
   b. activation of carotid and aortic chemoreceptors, leading to reflex bradycardia from vagal stimulation
   c. simultaneous baroreceptor stimulation of vagal and sympathetic fibers
   d. adrenergic receptors become refractory to catecholamines at body temperatures lower than 100°F (37.8°C)

8. Which is not a mechanism of arrhythmia formation following a traumatic event?
   a. catecholamine release due to pain
   b. reperfusion injury following recovery from hypovolemic shock
   c. alteration in the myocardial cell membrane due to acidosis
   d. activation of the parasympathetic nervous system following stimulation of the hypothalamus
9. Which statement regarding the use of opioids in patients with chest trauma is correct?
   a. Dogs and cats are relatively resistant to the respiratory-depressant effects of opioids.
   b. Use of opioids in chest trauma patients is unsafe and can lead to respiratory compromise.
   c. Buprenorphine has a ceiling respiratory-depressant effect, thereby reducing the risk for respiratory compromise.
   d. In patients with rib fractures, bupivacaine can be used as a local analgesic, and its effect can last 1 to 2 hours.

10. Which statement regarding the treatment of ventricular fibrillation in CPCR is incorrect?
   a. Lidocaine increases the defibrillation threshold and makes electrical defibrillation more difficult in dogs.
   b. Canine studies have shown that as blood concentrations of lidocaine decreased, the percentage of successful defibrillations increased.
   c. Amiodarone is a class I antiarrhythmic that has been shown to increase return of spontaneous circulation and early survival in humans.
   d. In humans, amiodarone is now listed ahead of lidocaine as a first-line drug for the treatment of ventricular fibrillation.