

Cardiopulmonary Resuscitation: Administering Fluids, Oxygen, and Drugs

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This article includes information from the June 2012 guidelines from the Reassessment Campaign on Veterinary Resuscitation (RECOVER) (BOX 1).

Cardiopulmonary arrest (CPA) is defined as cessation of spontaneous and effective ventilation as well as failure of the cardiac ventricles to contract, resulting in a lack of systemic perfusion.¹ Many diseases or injuries can cause CPA.

Having a patient with CPA is inevitable. Although cardiopulmonary resuscitation (CPR; **BOX 2**) is fairly unsuccessful in veterinary patients (survival rate: ~4% in dogs; ~9.5% in cats), initiating resuscitation as quickly as possible is imperative to improving a patient's chance of survival.¹

Because of advances in veterinary medicine, many therapies can be used during CPR. However, some drugs that were commonly used during CPR are no longer used, and, with more drugs available (**BOX 3**), selecting the right drug at the right time during CPR can be difficult.

Warning Signs of Cardiopulmonary Arrest

Recognizing the warning signs of CPA can allow early intervention or prevention of CPA. Therefore, it is important for veterinary technicians to closely monitor patients that are at risk of developing CPA. Abrupt changes in heart rate, respiratory rate, body temperature, or pulse quality may indicate that CPA is likely.² Generally, patients experience severe bradycardia or cardiac arrhythmia right before CPA. Most patients collapse, have a wide-eyed stare, and have no blink reflex, and many throw their head back and have rigid front legs right before a full CPA. Once the arrest has occurred, the legs tend to become flaccid again. Patients may experience respiratory arrest first, producing profound cyanosis. Cardiac arrest without respiratory arrest is possible.

Phases of Cardiopulmonary Resuscitation

Life support can be basic, advanced, or prolonged. Basic life support is limited to the "ABCs" (airway, breathing, and circulation), which has been proposed to be changed to "CABs" (circulation, airway, and breathing) because starting with cardiac compressions

Box 1. The Reassessment Campaign on Veterinary Resuscitation

The Reassessment Campaign on Veterinary Resuscitation (RECOVER) was formed to compile information to develop the first veterinary-specific guidelines for cardiopulmonary resuscitation (CPR). In developing the new guidelines, RECOVER's goals were:

- To facilitate an evidence-based review of the current literature on veterinary CPR
- To draft a set of clinical guidelines for veterinary CPR based on review of the literature
- To collate and incorporate feedback from the veterinary community to develop consensus CPR guidelines
- To widely disseminate the consensus veterinary CPR guidelines

In June 2012, the RECOVER guidelines ("RECOVER Evidence and Knowledge Gap Analysis on Veterinary CPR") were published in the *Journal of Veterinary Emergency and Critical Care*. [<http://onlinelibrary.wiley.com/doi/10.1111/j.1476-4431.2012.00758.x/full>] Before this, most CPR recommendations in veterinary medicine were derived from the American Heart Association standards for CPR.

For more information on RECOVER, go to <http://acvecc-recover.org>.

has been shown to improve survival.³ The goals of basic life support are to aid ventilation, oxygenation, and circulation by administering manual and external chest compressions and ventilation. In human medicine, basic life support can include defibrillation with an automated external defibrillator.⁴

Once basic life support has been initiated, whether to proceed with advanced life support should be decided quickly. Advanced life support is usually needed, and quick intervention with defibrillation and therapies (e.g., drugs, fluids, oxygen) offers the best chance of survival. Basic life support should be continued while advanced life support is prepared.

Once the heart is beating on its own, prolonged life support should be started. This may include use of a ventilator, continued

fluid therapy, and continued blood pressure maintenance. The patient must be closely monitored for changes in the cardiovascular, respiratory, or central nervous systems. Recurrence of CPA is the greatest concern after CPR.²

Routes of Administering Therapies

The intravenous route is preferred for administering drugs during CPR. Continuing chest compressions and ventilation is imperative when administering drugs. An intravenous catheter should be placed to allow multiple injections of drugs during CPR.

Ideally, a central venous catheter should be placed in the jugular vein.⁵ The disadvantages of placing a central line are that it (1) generally takes a little longer than placing a peripheral line and (2) may not be possible, depending on the size of the patient, when chest compressions are being performed; however, chest compressions should not be stopped.

Peripheral catheters are commonly placed because veterinary personnel are familiar with them and, in most cases, multiple limbs are readily available. When administered through a peripheral vein, drugs typically take 1 to 2 minutes to reach the central circulation.⁶ They take significantly less time when given through a central vein.⁶ To hasten delivery to the heart, a small bolus (10 to 20 mL; even for cats and small dogs) of isotonic crystalloids should be given after each drug is injected.⁵

If venous access cannot be obtained, drugs may be administered through the trachea by intubating the patient with an endotracheal tube, attaching a red rubber catheter to the end of the syringe, and inserting the catheter into the tube. A small amount (3 mL) of sterile water should be given afterward to flush the catheter. This ensures that the drugs reach the tracheal wall and do not sit in the endotracheal tube. Although the tracheal route is not ideal, lidocaine, epinephrine, atropine, naloxone, and vasopressin have all been shown to be absorbed from the trachea.⁴ To be effective, tracheal doses generally need to be 2 to 2.5 times higher than intravenous doses, and absorption through the trachea is slower. Therefore, attempts to gain venous access should continue.⁵

In humans, the American Heart Association (AHA) recommends using the intraosseous route if venous access cannot be obtained.⁶ Studies have shown that administration of drugs through the intraosseous route is as effective as using the central venous route.⁶ In veterinary medicine, the intraosseous route is typically reserved for use in neonates, puppies, and kittens. Intraosseous access generally takes slightly longer than accessing a vein.

The intracardiac route is never recommended because it is associated with serious complications (e.g., laceration, arrhythmias).⁵ The tracheal route is always preferred.

Intravenous Fluids

In humans, the use of intravenous fluids is recommended only when CPA is due to a hypovolemic episode, and they should be given slowly—just fast enough to maintain patency of the intravenous catheter.⁷ The fluid of choice in humans is 0.9% sodium chloride (NaCl).⁷ The restricted use of fluids is based on human studies showing that a large volume of fluid can decrease coronary perfusion

Box 2. CPR Versus CPR

The terms *cardiopulmonary cerebrovascular resuscitation* and *cardiopulmonary cerebral resuscitation* (CPCR) have been used for years in veterinary medicine. Recently, the Reassessment Campaign on Veterinary Resuscitation (RECOVER) recommended using the traditional term *cardiopulmonary resuscitation* (CPR) because it is less confusing and is used more universally.

Box 3. CPR Drug Chart

To obtain a CPR Drug Chart from the Veterinary Emergency and Critical Care Society, go to <http://www.veccs.org>, click "Shopping," and then "Posters & Charts."

pressure in patients who are not hypovolemic during CPR.^{3,8,9} A study involving dogs in the 1990s showed that high-volume boluses significantly increased total forward blood flow, decreasing blood flow to the brain and the ventricular myocardium.² These drastic changes resulted in a decrease in the average pressure gradient in the coronary and cerebral circulations.² Since 2005, the AHA has recommended against using intravenous boluses during CPR. Although they are still commonly used in veterinary medicine, in the past 2 years, there has been a trend not to use boluses unless CPA is associated with hypovolemia.

Several studies suggest that the use of hypertonic saline may improve survival compared with the use of normal saline.^{10,11} The use of hypertonic saline (also known as *low-volume resuscitation*) can rapidly increase osmotic pressure because it causes fluids to shift from the interstitial space to the intravascular space, improving venous return and cardiac output. One dose of hypertonic saline is as effective as four times the volume of isotonic saline, so a much smaller volume of the former is needed. Hypertonic crystalloids (7.5% NaCl) can be given one or two times initially at a rate of 4 mL/kg over a period of 2 minutes. If interstitial dehydration or hypernatremia secondary to a free water deficit is present, the use of hypertonic solutions is contraindicated. The effect of hypertonic saline lasts for ~20 minutes; subsequent administration of a crystalloid and/or colloid can sustain the effect. At a minimum, crystalloids must be given after hypertonic saline to replenish fluids that shifted from the interstitial space.

Solutions containing dextrose should be avoided unless there is evidence of hypoglycemia because dextrose has been associated with increased morbidity and mortality.¹

Research has shown that cooling human patients during CPR may increase the likelihood of survival.¹² In humans, ice/salt solutions have been used to decrease body temperature to 95°F. People treated with this technique had an average survival rate of 46.7%; the rate was 31% in people who were not treated with the technique.¹³ (In the same study, people who were administered CPR within 10 minutes of collapse had an average survival rate of 59.1%; the rate was 29.4% in other people.¹³) It is thought that hypothermia reduces the effects of ischemia and reperfusion injury associated with CPA. Although the use of premade ice/salt mixtures

in animals with CPA has not been studied, it has been suggested that permissive hypothermia may increase the success rate of CPR.³ Aggressive warming of patients during CPR is not recommended unless arrest occurred because of severe hypothermia.

Oxygen

Oxygen (fraction of inspired oxygen: $\geq 40\%$) should be administered quickly. Intubation and use of a bag-valve-mask resuscitator is the most effective method of delivering a high concentration of oxygen. The other delivery options include a face mask with a bag-valve-mask resuscitator or mouth-to-nose resuscitation. RECOVER recommends administration of oxygen but acknowledges that there is limited evidence to support its effectiveness over room air. For CPR to be effective, the heart and brain must be appropriately oxygenated. Myocardial oxygen delivery depends on myocardial blood flow and arterial oxygen content. Cerebral perfusion depends on cardiac output and cerebrovascular resistance. Therefore, administration of oxygen should be beneficial to CPR because the risks of hypoxemia outweigh the risks of hyperoxemia.

Epinephrine and Norepinephrine

Epinephrine (also known as *adrenaline*) is the most common drug used in CPR.⁵ Epinephrine and norepinephrine are naturally produced when the sympathetic nerves of the adrenal medullae are stimulated. In general, secretions of the adrenal medullae are 80% epinephrine and 20% norepinephrine.¹⁴ Both are considered adrenergic receptor agonists, which can be classified as α or β . α -Adrenergic receptors are responsible for vasoconstriction, while β -adrenergic receptors are responsible for vasodilation. Norepinephrine mainly excites α -adrenergic receptors and excites β -adrenergic receptors to a lesser extent.¹⁴ Epinephrine excites both receptors equally.¹⁴ When epinephrine and norepinephrine are released naturally, they tend to affect organs in the same way as the drug formulations, but the effects of the drugs last five to 10 times longer.¹⁴ Both epinephrine and norepinephrine increase heart rate, vascular contraction, and dilation of air passages.

While the drug formulations of epinephrine and norepinephrine appear to be similar, there are some notable differences. Compared with norepinephrine, epinephrine has a greater effect on β -adrenergic receptors and, therefore, greater effects on cardiac stimulation.¹⁴ However, epinephrine causes only weak constriction of blood vessels, while norepinephrine causes much stronger constriction.¹⁴ Therefore, norepinephrine increases total peripheral resistance and arterial pressure.¹⁴ Epinephrine increases arterial pressure only slightly but increases cardiac output more.¹⁴ The main effects of epinephrine (i.e., increases in coronary and cerebral perfusion pressures) are due to its effect on α -adrenergic receptors. Epinephrine is used during asystole or pulseless electrical activity. Asystole is the most common rhythm in veterinary CPA, which is why epinephrine is the most commonly used drug during CPR.¹⁵

Studies have shown that epinephrine can be absorbed intratracheally, but to have an effect, the dose needs to be five to 10 times higher than the intravenous dose.^{16,17} The AHA recommends dilution of intratracheal epinephrine in 5 to 10 mL of water or normal

saline. Studies have shown that dilution with water instead of 0.9% saline may achieve better drug absorption.⁶ Epinephrine (1:1000) can be given intratracheally at a dose of 0.03 to 0.1 mg/kg.¹⁸ While low-dose epinephrine (0.01 to 0.02 mg/kg IV) used to be considered effective, high-dose epinephrine (0.1 to 0.2 mg/kg IV) has also been discussed.^{18,19} Either dose can be given every 3 to 5 minutes intravenously. Epinephrine has been associated with increasing myocardial demand, exacerbating myocardial ischemia and ventricular arrhythmias after CPR.^{15,20} Administration of low-dose epinephrine is thought to minimize myocardial effects of the drug while increasing coronary and cerebral perfusion pressures.¹⁵ In humans, studies have proven the benefits of low-dose and high-dose epinephrine.⁴ The AHA and veterinary researchers have not taken a position on which dose is more effective. RECOVER has not taken a position either but suggests administering epinephrine during every other cycle of basic life support to avoid overdosage.²⁰

The few human and animal studies on the use of norepinephrine in CPA suggest that the drug has effects similar to those of epinephrine. The RECOVER guidelines do not include information on the use of norepinephrine.²⁰ According to the AHA, the only prospective human trial on the use of norepinephrine showed that it had no advantage over the use of epinephrine.⁶ The dose of norepinephrine is 0.1 to 0.5 g/kg/min CRI. Because of the proven history of using epinephrine during CPR, the AHA prefers epinephrine to norepinephrine.

Atropine

Atropine is a prototype antimuscarinic drug, meaning that it blocks muscarinic receptors, which are acetylcholine receptors in all effector cells of the parasympathetic nervous system (i.e., cells of muscles, glands, or organs that can respond to a nerve impulse). The heart is supplied with parasympathetic and sympathetic nerves. The parasympathetic nerves (also known as *vagi*) are mainly attached to the sinus and atrioventricular nodes of the heart.¹⁴ When stimulated, vagi release acetylcholine at their vagal ending. Acetylcholine decreases the (1) rate of the sinus node and (2) excitability of the atrioventricular junctional fibers, thus decreasing the cardiac impulse to the ventricles.¹⁴ This is also known as a *vagal response*. Vagal stimulation slows the heartbeat, and excessive stimulation can stop it entirely. Atropine inhibits acetylcholine at postganglionic parasympathetic neuroeffector sites, which helps to stop the vagal response.¹⁹

Atropine is given when vagal responses are thought to have occurred. Bradycardia usually responds to atropine. However, the AHA has stated, "No prospective controlled studies support the use of atropine in asystole or slow pulseless electrical activity arrest."⁶ Regarding asystole, the AHA has classified atropine as an indeterminate drug, meaning that it appears to have no benefit or adverse effect.⁶

The well-known adverse effect of atropine is severe sinus tachycardia.⁵ During CPA, hypoxia generally occurs. It is thought that sinus tachycardia caused by the use of atropine can increase the myocardium's demand for oxygen, predisposing the myocardium to fibrillation.⁵

Atropine can be administered in various doses intravenously or intratracheally. In patients experiencing respiratory arrest, a small dose (0.004 to 0.01 mg/kg) can be given intravenously because it has been shown to help prevent or reduce respiratory tract secretions.¹⁹ For severe bradycardia or asystole, the dose ranges from 0.02 to 0.05 mg/kg IV.¹⁹ Higher doses (>0.1 mg/kg) have been associated with worse outcomes in dogs, making it important to stay within the recommended dose ranges.²⁰

Vasopressin

Vasopressin is an effective nonadrenergic vasopressor that causes peripheral, coronary, and renal vasoconstriction.¹⁸ However, the drug also has vasodilatory effects that significantly improve cerebral and myocardial blood flow.²¹ These vasodilatory effects are due to nitric oxide—a mediator of vasodilation.²¹

Despite early promising studies, vasopressin has not been shown to be superior to epinephrine for treating CPA in humans. The AHA's position on vasopressin is, "Because vasopressin's effects have not been shown to differ from those of epinephrine in cardiac arrest, one dose of vasopressin may replace either the first or second dose of epinephrine in the treatment of pulseless arrest." While the debate continues as to whether vasopressin is superior to epinephrine in people, studies have shown that vasopressin is superior to epinephrine in animals.^{4,18} It has been shown that even in acidic and hypoxic environments, which occur during asystole, vasopressin can stimulate the V_{1A} receptor, which resides in the brain, leading to vasoconstriction.¹⁸ Epinephrine has been shown to lose some of its vasopressor effects in acidic and hypoxic environments.¹⁸

The use of vasopressin in veterinary CPR is relatively new. Vasopressin is dosed at 0.2 to 0.8 μ g/kg IV and can be used during asystole or pulseless electrical activity just like epinephrine.¹⁸ The drug has been shown to be absorbed through the trachea,⁶ and the AHA recommends using a tracheal dose that is five to 10 times higher than the intravenous dose. While epinephrine can be administered every 3 to 5 minutes during CPR, the AHA and RECOVER recommend using vasopressin instead of epinephrine for only the first or second dose, followed by administration of epinephrine thereafter.^{6,20} This recommendation is likely based on the relatively new use of vasopressin in human and veterinary medicine, whereas epinephrine has been used for over 100 years, and its efficacy is well supported by many studies.

Amiodarone

The AHA recommends administration of amiodarone for treating ventricular fibrillation or pulseless ventricular tachycardia that is unresponsive to CPR or the use of vasopressors.⁶ Intravenous amiodarone affects sodium, potassium, and calcium channels and has α - and β -adrenergic blocking properties.⁶ The AHA and RECOVER recommend the use of amiodarone before the use of lidocaine.^{6,20} A 2002 study comparing amiodarone and lidocaine found that amiodarone improved the survival rate and that lidocaine was associated with more asystole after defibrillation.⁶ The RECOVER guidelines state that of the antiarrhythmic drugs, only amiodarone "has shown consistent benefit and may be considered

in cases of ventricular fibrillation/pulseless ventricular tachycardia resistant to electrical defibrillation."²⁰ A dose of 5 mg/kg IV can be given slowly over a period of 10 minutes.²² Amiodarone can cause persistent hypotension if it is not given slowly or diluted as described in the package insert.²²

Lidocaine

The use of 2% lidocaine is strictly limited to treating arrhythmias and has no benefit during CPA.⁶ The drug is most commonly used to treat ventricular arrhythmias, including refractory ventricular fibrillation. Lidocaine works by blocking sodium from entering myocardial cells, which decreases conduction velocity and tissue excitability.⁵

Magnesium

In dogs with severe hypomagnesemia, lethal ventricular arrhythmias have been reported. Magnesium used to be given routinely to every human patient with CPA in case of a low magnesium level. However, studies have shown no improvement in CPR when magnesium is routinely given during CPR.⁶ If a low magnesium level due to disease or a metabolic disturbance is suspected, a dose of 1 to 2 g IV should be given over a period of 2 minutes.¹

Calcium

Years ago, 10% calcium was commonly used in CPR because it was thought to help improve cardiac contractility.¹ However, excessive calcium has been implicated in causing reperfusion injury and myocardial and cerebral vasoconstriction.¹ Therefore, the AHA does not recommend the use of calcium during CPR. However, much like hypomagnesemia, hypocalcemia can lead to CPA. Ideally, hypocalcemia should be confirmed through blood work before administration of calcium; at a minimum, clinical signs of hypocalcemia must be present. Administration of calcium is recommended to help protect the heart from adverse effects associated with severe hyperkalemia in veterinary patients.

Potassium

Patients with very low potassium levels (hypokalemia) can experience CPA.²⁰ Conversely, patients experiencing prolonged CPA often develop hyperkalemia.²⁰ Similarly to calcium and magnesium, potassium should be given when blood work results support the presence of hypokalemia.²⁰ It is not advised to administer potassium without documented evidence of hypokalemia.

Corticosteroids

The several studies that have used corticosteroids during CPR have had differing results.²⁰ Patients in CPA experience body-wide hypoperfusion that includes the gastrointestinal tract. Corticosteroids can have detrimental effects on the gastrointestinal tract. Currently, no evidence supports the use of corticosteroids; therefore, it is not recommended in the RECOVER guidelines.²⁰

Conclusion

It is important to fully understand which therapies are available during CPR so that a decision can be made quickly. Selecting the

right therapies for the patient and administering them quickly provide the best chance of survival.

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1. The _____ route of drug administration is preferred during CPR.
 - a. oral
 - b. intramuscular
 - c. intratracheal
 - d. intravenous
2. During CPA, the _____ vein is ideal for placing an intravenous catheter.
 - a. jugular
 - b. femoral
 - c. cephalic
 - d. saphenous
3. A large volume of intravenous fluid should be used when CPA is due to
 - a. cardiogenic shock.
 - b. hypovolemia.
 - c. obstructive shock.
 - d. any cause.
4. For low-volume resuscitation, _____ is/are used.
 - a. isotonic crystalloids
 - b. hypotonic crystalloids
 - c. colloids
 - d. hypertonic saline
5. Atropine works by
 - a. increasing the muscarinic receptors.
 - b. blocking the muscarinic receptors.
 - c. causing a vagal response.
 - d. releasing norepinephrine.
6. Atropine can be given when the patient experiences
 - a. hypertension.
 - b. tachycardia.
 - c. bradycardia.
 - d. hypotension.
7. Epinephrine should be given when the patient
 - a. experiences tachycardia.
 - b. stops breathing.
 - c. experiences asystole.
 - d. experiences hypotension.
8. Vasopressin can be used once instead of
 - a. atropine.
 - b. lactated Ringer solution.
 - c. oxygen.
 - d. epinephrine.
9. Lidocaine should be given when the patient
 - a. experiences ventricular arrhythmias.
 - b. stops breathing.
 - c. experiences asystole.
 - d. experiences bradycardia.
10. Amiodarone is used to treat
 - a. bradycardia.
 - b. hypertension.
 - c. ventricular fibrillation.
 - d. heart block.