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Special Article

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# RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 7: Clinical guidelines

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***Abstract***

**Objective** – To present a series of evidence-based, consensus guidelines for veterinary CPR in dogs and cats.
**Design** – Standardized, systematic evaluation of the literature, categorization of relevant articles according to level of evidence and quality, and development of consensus on conclusions for application of the concepts to clinical practice. Questions in five domains were examined: Preparedness and Prevention, Basic Life Support, Advanced Life Support, Monitoring, and Post-Cardiac Arrest Care. Standardized worksheet templates were used for each question, and the results reviewed by the domain members, by the RECOVER committee, and opened for comments by veterinary professionals for 4 weeks. Clinical guidelines were devised from these findings and again reviewed and commented on by the different entities within RECOVER as well as by veterinary professionals.
**Setting** – Academia, referral practice and general practice.
**Results** – A total of 74 worksheets were prepared to evaluate questions across the five domains. A series of 101 individual clinical guidelines were generated. In addition, a CPR algorithm, resuscitation drug-dosing scheme, and postcardiac arrest care algorithm were developed.
**Conclusions** – Although many knowledge gaps were identified, specific clinical guidelines for small animal veterinary CPR were generated from this evidence-based process. Future work is needed to objectively evaluate the effects of these new clinical guidelines on CPR outcome, and to address the knowledge gaps identified through this process.

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***Keywords:*** canine, cardiac arrest, defibrillation, feline

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**Abbreviations**

| | |
|---|---|
| ABC | airway, breathing, circulation |
| ALS | advanced life support |
| BLS | basic life support |
| CPA | cardiopulmonary arrest |
| CPR | cardiopulmonary resuscitation |
| $EtCO_2$ | end tidal $CO_2$ |
| ETT | endotracheal tube |
| ILCOR | International Liaison Committee on Resuscitation |
| LOE | level of evidence |
| PEA | pulseless electrical activity |
| PICO | population, intervention, control group, outcome |

| | |
|---|---|
| RECOVER | Reassessment Campaign on Veterinary Resuscitation |
| VF | ventricular fibrillation |
| VT | ventricular tachycardia |

## Introduction

The development of specific, evidence-based clinical guidelines for human cardiopulmonary resuscitation (CPR), based upon extensive surveys of the literature by the International Liaison Committee on Resuscitation (ILCOR) has allowed consistent training for human healthcare professionals and the lay public, leading directly to improved outcomes.[1–3] No comparable evidence-based guidelines have been available in veterinary medicine, although recommendations on practical execution of CPR in small animals have been published.[4–8] The absence of standardized, comprehensive training coupled with a lack of consensus on the content of the published recommendations has led to significant variability in the approach to veterinary CPR, likely to the detriment of our patients.[9]

The main goal of the Reassessment Campaign on Veterinary Resuscitation (RECOVER) initiative was to develop a set of clinical consensus guidelines for the practice of CPR in dogs and cats based upon an extensive, systematic review of the literature in the context of our target species. Although there is overlap between the literature examined by ILCOR and RECOVER, the science was interpreted based upon applicability to dogs and cats. This has led to conclusions that diverge, in some areas, from those reached by ILCOR. Based upon the results of the evidence worksheet process used in RECOVER,[10] a total of 101 clinical guidelines were developed and made available for review for a period of 4 weeks to members of the veterinary community (see Appendix I). This feedback was used to modify and refine the recommendations, yielding the final set of consensus guidelines presented in this manuscript.

In order to reflect the variability in the quality and quantity of evidence examined, each guideline developed through the RECOVER consensus process has been assigned two descriptors: (1) Class – this categorizes the risk-benefit ratio of the intervention described in the guideline, and (2) Level – this categorizes the strength of the evidence available to support the recommendation. This scheme was adapted from that used by ILCOR.[11] The individual class and level categories are detailed in Tables 1 and 2, and each guideline is labeled (Class-Level).

**Table 1:** Class descriptors for the clinical guidelines, categorizing the risk-benefit ratio associated with the intervention

| Class | Risk:benefit ratio | Clinical recommendation |
|---|---|---|
| I | Benefit >>> Risk | Should be performed |
| IIa | Benefit >> Risk | Reasonable to perform |
| IIb | Benefit ≥ Risk | May be considered |
| III | Risk > Benefit | Should not be performed |

**Table 2:** Level descriptors for the clinical guidelines, categorizing the strength of the evidence available for the recommendation

| Level | Populations studied | Criteria for recommendation |
|---|---|---|
| A | Multiple populations | Multiple high quality and/or high level of evidence studies |
| B | Limited populations | Few to no high quality and/or high level of evidence studies. |
| C | Very limited populations | Consensus opinion, expert opinion, guideline based on physiologic/ anatomic principles, standard of care |

## Small Animal Veterinary CPR Algorithm

The guidelines presented in this document cover a wide variety of CPR-related topics in 5 domains: Preparedness and Prevention, Basic Life Support (BLS), Advanced Life Support (ALS), Monitoring, and Post-Cardiac Arrest Care. The main elements of CPR and their temporal sequence have been summarized in a CPR algorithm chart (Figure 1). This algorithm was designed to deliver step-by-step prompts to the veterinary rescuer engaged in CPR and stresses the importance of early BLS interventions. The evidence reviewed strongly reinforced the importance of early delivery of high-quality chest compressions with minimal interruption. High-quality chest compressions should be delivered in uninterrupted cycles of 2 minutes with most patients in lateral recumbency, at a compression rate of 100–120/min and a compression depth of 1/3–1/2 the width of the chest while allowing for full elastic recoil of the chest between individual compressions. In addition, it is likely that early intubation and ventilation in veterinary CPR is highly valuable, with a ventilation rate of approximately 10 breaths/min, a tidal volume of 10 mL/kg, and an inspiratory time of 1 second delivered simultaneously with compressions. If intubation supplies are not available, mouth-to-snout ventilation is an acceptable alternative, and should be delivered in repeated rounds of 30 chest compressions followed by 2 rapid breaths in cycles of 2 minutes. After each 2-minute cycle of BLS, the compressor should be rotated to prevent fatigue, which may decrease the quality of chest compressions. Every effort should be made to minimize the duration of chest compression interruptions between cycles. ALS

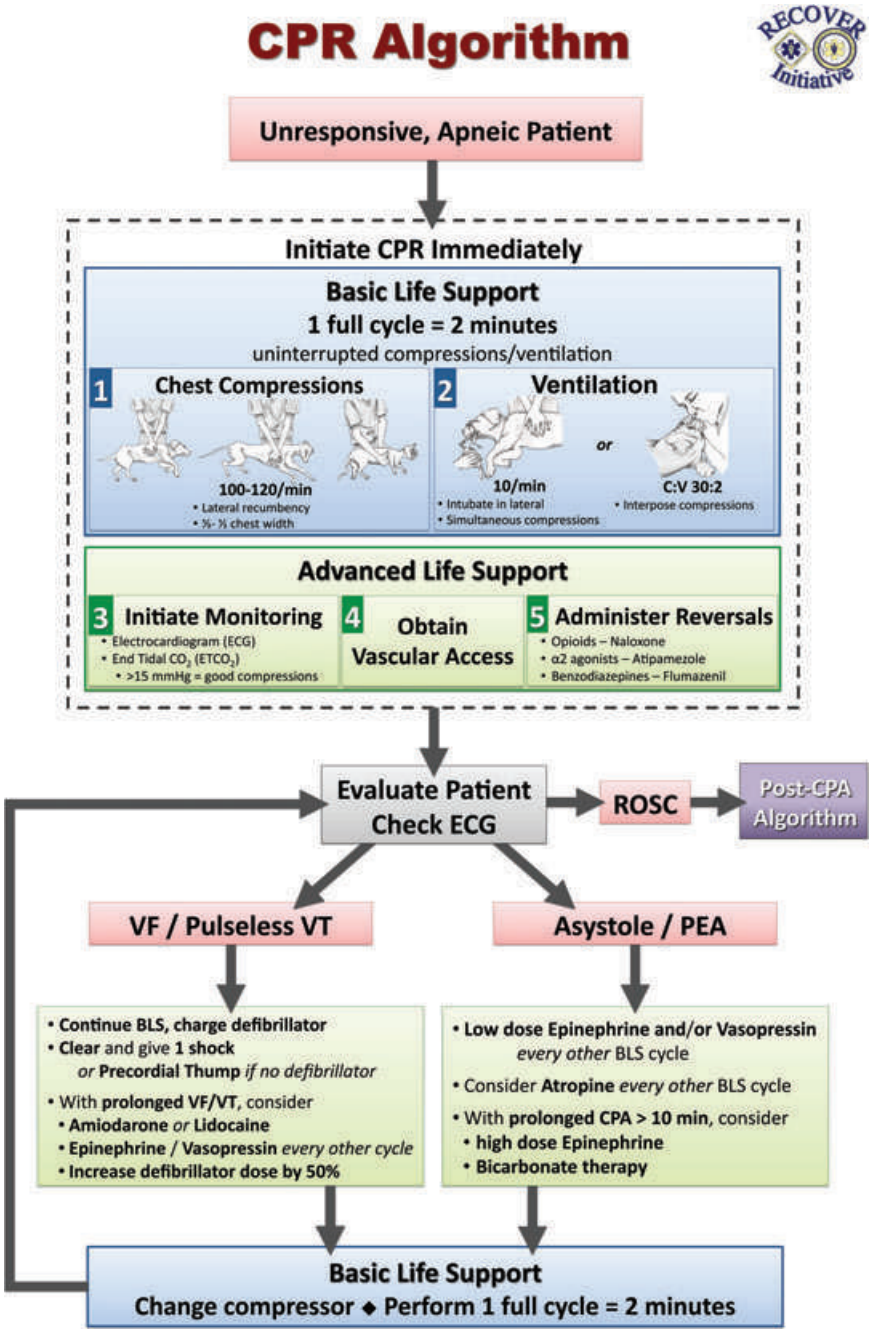


**Figure 1:** CPR algorithm chart. This chart summarizes the clinical guidelines most relevant to the patient presenting acutely in CPA. The box surrounded by the grey dashed line contains, in order, the initial BLS and ALS actions to be taken when a patient is diagnosed with CPA: (1) administration of chest compressions, (2) ventilation support, (3) initiation of ECG and $EtCO_2$ monitoring, (4) obtaining vascular access for drug administration, and (5) administration of reversal agents if any anesthetic/sedative agents have been administered. The algorithm then enters a loop of 2-minute cycles of CPR with brief pauses between to rotate compressors, to evaluate the patient for signs of ROSC, and to evaluate the ECG for a rhythm diagnosis. Patients in PEA or asystole should be treated with vasopressors and, potentially, anticholinergic drugs. These drugs should be administered no more often than every other cycle of CPR. Patients in VF or pulseless VT should be electrically defibrillated if a defibrillator is available, or mechanically defibrillated with a precordial thump if an electrical defibrillator is not available. Immediately after defibrillation, another 2-minute cycle of BLS should be started immediately. BLS, basic life support; CPA, cardiopulmonary arrest; CPR, cardiopulmomary resuscitation; C:V, compression to ventilation ratio; $EtCO_2$, end tidal $CO_2$; PEA, pulseless electrical activity; ROSC, return of spontaneous circulation; VF, ventricular fibrillation; VT, ventricular tachycardia.

interventions, including initiation of monitoring, establishment of vascular access, administration of reversal agents, vasopressor and vagolytic therapy, and defibrillation are also included in the algorithm. Recommended dosing and indications for common CPR-related drugs are included in Appendix II.

A post-cardiac arrest (PCA) algorithm chart, designed to summarize the major interventions recommended in the guidelines for patients that achieve return of spontaneous circulation (ROSC), is shown in Figure 2. The algorithm is focused on initial respiratory optimization that includes normalizing ventilation to achieve normocapnia and titration of oxygen supplementation to maintain normoxemia while avoiding both hypoxemia and hyperoxemia. Once the patient's respiratory status is assessed and a treatment plan is initiated, cardiovascular concerns are addressed. The hemodynamic optimization component is based on the concept of early goal-directed therapy, first described for patients in septic shock.[12] Arterial blood pressure is first assessed, and IV fluids, vasopressors, and positive inotropes are administered as needed to achieve normotension or mild hypertension. Severe hypertension is addressed with adjustment of vasopressors, pain management, and antihypertensives. Once arterial blood pressure targets are met, central venous oxygen saturation ($ScvO_2$) or blood lactate concentration is assessed to determine if oxygen delivery to tissues is adequate. If a deficit in oxygen delivery is noted, hemodynamic optimization is revisited and guided by oxygen delivery targets rather than arterial blood pressure targets. If oxygen delivery targets are still not met, red blood cell transfusions are administered if indicated. A PCV target of 25% is suggested, a departure from traditional early goal-directed therapy due to more recent data in humans documenting improved outcomes with more restrictive transfusion triggers.[13] Once hemodynamic optimization strategies have been initiated, neuroprotective interventions and intensive monitoring are considered based on the neurologic status of the patient. Recommended doses for common PCA-related drugs are included in Appendix II. It should be noted that this comprehensive treatment protocol is based in part on evidence specific to the PCA condition and in part on general critical care principles. Studies on the effects of these types of optimization strategies during PCA care are needed.

### *Preparedness and Prevention*

The guidelines developed through the evidence collected by this domain are based on the premise that resuscitation attempts that are organized, cohesive, and led by a well-functioning knowledgeable team adhering to evidence-based CPR guidelines should improve survival from cardiopulmonary arrest (CPA). Strengthening the links in the chain of survival, the time-sensitive, coordinated actions necessary to maximize survival from CPA, has the potential to lead to improved outcomes.[4] The guidelines derived from this domain focus on interventions involving both environmental and personnel factors that strengthen the chain of survival for dogs and cats with CPA.

#### Equipment organization and cognitive aids

An organized and efficient response to an acute medical or surgical crisis is crucial. The effects of ready access to organized and consistently audited crash carts on outcomes for patients receiving CPR have been well studied in human medicine.[14] Equipment and supply inaccessibility or failure has been implicated in delays in initiation of CPR in up to 18% of CPA cases.[15] Therefore, it is recommended that the location, storage, and content of resuscitation equipment should be standardized and regularly audited (I-A). In addition, the presence of cognitive aids such as checklists, algorithm charts, and dosing charts has been shown to improve compliance with CPR guidelines.[16] Formal training of personnel in the use of these cognitive aids is also crucial to effective utilization during a crisis.[17] Figure 1 shows an example of a CPR algorithm chart, and Figure 3 shows an example of an emergency drug and dosing chart, containing only the most commonly used drugs, separated into categories based upon indication, and provided in volume of drug to be administered by body weight to reduce dose calculation errors. Availability and clear visibility of these charts in areas in which CPA may occur, such as procedure areas, anesthesia induction rooms, and surgery suites is recommended (I-B).

#### CPR training

Adherence to CPR guidelines can only be accomplished if personnel receive effective, standardized training and regular opportunities to refresh their skills. Because high-quality CPR requires both cognitive skills to correctly perform all indicated steps in an orderly, rapid fashion as well as psychomotor skills to provide effective manual interventions such as chest compressions and ventilation, CPR training should include both didactic components targeted at cognitive performance and opportunities to practice hands-on skills with quality feedback (I-A). Effective options for psychomotor skill training include high-fidelity simulation technologies, low-fidelity task trainers, and auditory and visual feedback devices.[18–20] Regardless of the type of technology used for initial training, refresher training at least every 6 months is recommended to reduce the risk of the decay of skills (I-A). There is some evidence that the use of simulation methodologies may be most beneficial for

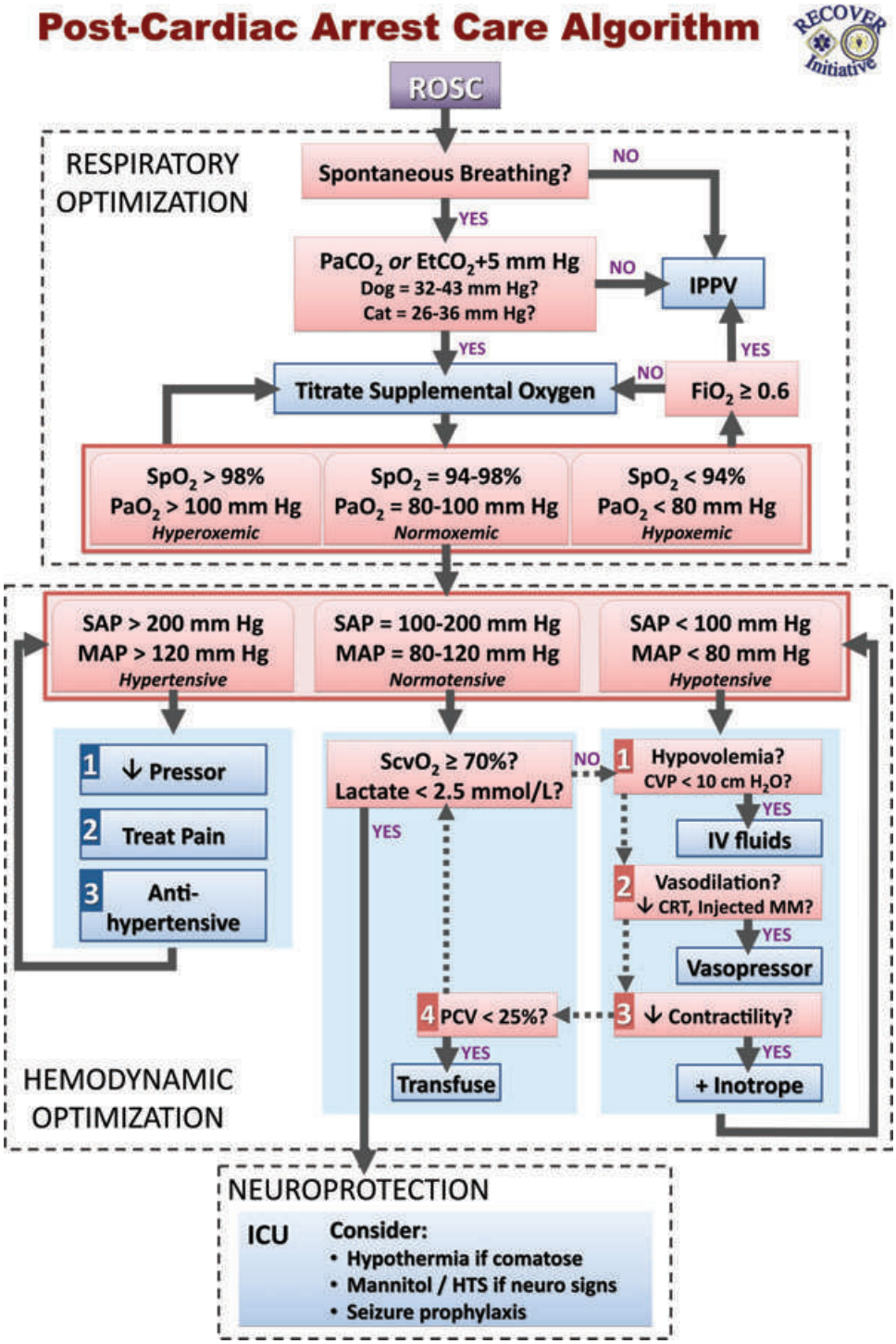


**Figure 2:** Post-cardiac arrest (PCA) care algorithm. This chart summarizes a comprehensive treatment protocol for PCA care that includes components of controlled ventilation and oxygenation, goal-directed hemodynamic optimization, and neuroprotective strategies. The sequence shown reflects the order in which each component should be assessed and treatment initiated. Assessment and initiation of treatment for the subsequent component will likely commence before the endpoints of the previous component have been completely met. Thus respiratory, hemodynamic, and neuroprotective treatment strategies will be initiated in parallel in most cases. CRT, capillary refill time; CVP, central venous pressure; $EtCO_2$, end-tidal carbon dioxide; HTS, hypertonic saline; IPPV, intermittent positive pressure ventilation; MAP, mean arterial pressure; MM, mucous membrane color; ROSC, return of spontaneous circulation; SAP, systolic arterial pressure; $ScvO_2$, central venous oxygen saturation.

# CPR Emergency Drugs and Doses

| | | Weight (kg) | 2.5 | 5 | 10 | 15 | 20 | 25 | 30 | 35 | 40 | 45 | 50 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Weight (lb) | 5 | 10 | 20 | 30 | 40 | 50 | 60 | 70 | 80 | 90 | 100 |
| | DRUG | DOSE | ml | ml | ml | ml | ml | ml | ml | ml | ml | ml | ml |
| Arrest | Epi Low (1:1000; 1mg/ml) every other BLS cycle x3 | 0.01 mg/kg | 0.03 | 0.05 | 0.1 | 0.15 | 0.2 | 0.25 | 0.3 | 0.35 | 0.4 | 0.45 | 0.5 |
| | Epi High (1:1000; 1 mg/ml) for prolonged CPR | 0.1 mg/kg | 0.25 | 0.5 | 1 | 1.5 | 2 | 2.5 | 3 | 3.5 | 4 | 4.5 | 5 |
| | Vasopressin (20 U/ml) | 0.8 U/kg | 0.1 | 0.2 | 0.4 | 0.6 | 0.8 | 1 | 1.2 | 1.4 | 1.6 | 1.8 | 2 |
| | Atropine (0.54 mg/ml) | 0.04 mg/kg | 0.2 | 0.4 | 0.8 | 1.1 | 1.5 | 1.9 | 2.2 | 2.6 | 3 | 3.3 | 3.7 |
| Anti-Arrhyth | Amiodarone (50 mg/ml) | 5 mg/kg | 0.25 | 0.5 | 1 | 1.5 | 2 | 2.5 | 3 | 3.5 | 4 | 4.5 | 5 |
| | Lidocaine (20 mg/ml) | 2 mg/kg | 0.25 | 0.5 | 1 | 1.5 | 2 | 2.5 | 3 | 3.5 | 4 | 4.5 | 5 |
| Reversal | Naloxone (0.4 mg/ml) | 0.04 mg/kg | 0.25 | 0.5 | 1 | 1.5 | 2 | 2.5 | 3 | 3.5 | 4 | 4.5 | 5 |
| | Flumazenil (0.1 mg/ml) | 0.01 mg/kg | 0.25 | 0.5 | 1 | 1.5 | 2 | 2.5 | 3 | 3.5 | 4 | 4.5 | 5 |
| | Atipamezole (5 mg/ml) | 100 µg/kg | 0.06 | 0.1 | 0.2 | 0.3 | 0.4 | 0.5 | 0.6 | 0.7 | 0.8 | 0.9 | 1 |
| Defib Monophasic | External Defib (J) | 4-6 J/kg | 10 | 20 | 40 | 60 | 80 | 100 | 120 | 140 | 160 | 180 | 200 |
| | Internal Defib (J) | 0.5-1 J/kg | 2 | 3 | 5 | 8 | 10 | 15 | 15 | 20 | 20 | 20 | 25 |

**Figure 3:** CPR drug dosing chart. Drugs are separated by indication and volumes are provided by body weight to reduce calculation errors. Defibrillator dosing is for a monphasic electrical defibrillator. Anti-arrhyth, antiarrhythmic drugs; CPR, cardiopulmonary resuscitation; Epi, epinephrine; Defib, electrical defibrillation.

this booster training.[21] Although high-fidelity simulators may carry some advantage in this type of training, simple mock codes run every 3–6 months on low-fidelity manikins are likely to improve awareness of CPR guidelines and are achievable in most small animal practice settings.

Improved learning outcomes have been documented when CPR training culminates in performance testing.[22] Therefore, regardless of the methods used for initial and refresher training, structured assessment after CPR training is recommended (I-A). In addition to assessment after didactic and psychomotor skills training, structured debriefing after a real resuscitation effort or simulated CPR, allowing participants to review and critique their performance and the performance of the team as a whole is recommended (I-A). During the debriefing, the participants should be encouraged to drive the discussion and identify for themselves the strengths and weaknesses of the team's performance. Facilitation by a team member trained in debriefing technique is useful, and care must be taken to prevent focusing on blaming individuals for poor performance. Open, honest discussion about opportunities for improvement immediately after a CPR attempt can lead to significant enhancement in CPR performance.[23–25]

**Team dynamics**

Several studies in human medicine have investigated the effect of the presence of a physician on outcomes in out-of-hospital CPA, and taken as a whole, there does not appear to be a beneficial effect on outcome of CPR from the presence of a physician acting as team leader.[26,27] Although there have been no studies investigating this question in veterinary medicine, based on the data available in human medicine, veterinarians or technicians may be considered as leaders of a CPR team (IIb-B).

Regardless of the status of the team leader, there is strong evidence in the literature that communication and team skills training can improve the effectiveness of a CPR attempt,[28] and specific leadership training is recommended for individuals who may need to lead in a CPR attempt (I-A). Crucial roles of the team leader include distributing tasks to other team members and enforcing rules and procedures. Important leadership behaviors that can improve CPR team performance include intermittently summarizing the code to ensure a shared mental model among team members, actively soliciting input from team members to encourage situation awareness and identify issues and ideas from all members of the team, and assigning individual tasks to team members rather than performing them personally to allow better attention to the global status of the code rather than a specific task. Team performance can also be enhanced by using focused, clear communication directed at individuals when tasks are assigned, and utilization of closed loop communication.[29] Closed loop communication is accomplished by a clear, directed order being given to one team member by another, after which the receiving team member repeats the order back to the requestor to verify the accuracy of the receiver's perception. This simple technique drastically reduces medical errors, especially in an emergency situation, due to misunderstanding of orders and prevents the possibility of an order not being carried out because the receiver did not hear the request.

## *BLS*

In veterinary CPR, BLS includes the recognition of CPA, administration of chest compressions, airway management, and provision of ventilation. It is imperative that BLS is provided immediately upon diagnosis or suspicion of CPA, and lay rescuers and medical professionals alike may accomplish most aspects. Numerous human and animal experimental studies have shown that the rapidity of initiation and quality of BLS performed is associated with ROSC and survival in victims of CPA.[30–32] Although BLS is considered separately from ALS and monitoring in this consensus statement, in clinical practice, the intent is that BLS will be performed simultaneously with ALS and monitoring, or that ALS and monitoring will occur as soon after initiation of BLS as possible.

**Chest compressions**

Chest compressions should be initiated as soon as possible upon recognition of CPA and if multiple rescuers are present, airway and ventilation management should not delay commencement of chest compressions.

Patient position and compressor hand placement

Due to experimental evidence suggesting higher left ventricular pressures and aortic flow in dogs in lateral recumbency compared to dorsal recumbency, and clinical data in dogs and cats showing higher rates of ROSC associated with compressions performed in lateral recumbency,[33,34] chest compressions should be done in lateral recumbency in both dogs and cats (I-B). Either left or right lateral recumbency is acceptable. However, the profound variations in chest conformation among dogs and cats suggest that a single, identical approach to chest compressions is unlikely to be optimal in all patients with CPA. There are 2 main theories describing the mechanism by which external chest compressions lead to blood flow during CPR.[35] The cardiac pump theory postulates that the cardiac ventricles are directly compressed between the sternum and the spine in patients

in dorsal recumbency or between the ribs in patients in lateral recumbency. The thoracic pump theory proposes that chest compressions increase overall intrathoracic pressure, secondarily compressing the aorta and collapsing the vena cava leading to blood flow out of the thorax. During elastic recoil of the chest, subatmospheric intrathoracic pressure provides a pressure gradient that favors the flow of blood from the periphery back into the thorax and into the lungs where oxygen and carbon dioxide exchange occurs. Although minimally studied, it is believed that the predominant mechanism in any patient will be dependent upon thoracic conformation, and it is likely that both mechanisms contribute to blood flow in most patients.

In the majority of medium, large, and giant breed dogs with rounded chests, direct compression of the heart with external chest compressions is unlikely. Therefore, the thoracic pump mechanism is likely to predominate in these patients, and chest compressions over the widest portion of the chest will allow maximal increases in intrathoracic pressure (see Figure 4a). It is therefore reasonable in most large and giant breed dogs, to deliver chest compressions with the hands placed over the widest portion of the chest (IIa-C). Conversely, in more keel-chested (narrow, deep chested) dogs such as greyhounds, the cardiac pump theory may be more easily employed with external chest compressions in lateral recumbency; therefore, in dogs with this conformation, chest compressions with the hands positioned directly over the heart is reasonable (IIa-C). (Figure 4b). In dogs with barrel-chested conformations, such as English bulldogs, sternal compressions in dorsal recumbency, directed at the cardiac pump theory, may be considered (IIb-C) (Figure 4c). Cats and small dogs tend to have higher thoracic wall compliance, and effective chest compressions using the cardiac pump mechanism can likely be achieved with a 1-hand technique with the compressor's fingers wrapped around the sternum at the level of the heart (see Figure 5a). Thus, circumferential compressions rather than lateral compressions may be considered (IIb-C). However, if the compressor becomes fatigued or an individual patient's thoracic wall compliance is lower due to age, obesity, or conformation, a 2-handed technique employing the cardiac pump mechanism can be used (Figure 5b).

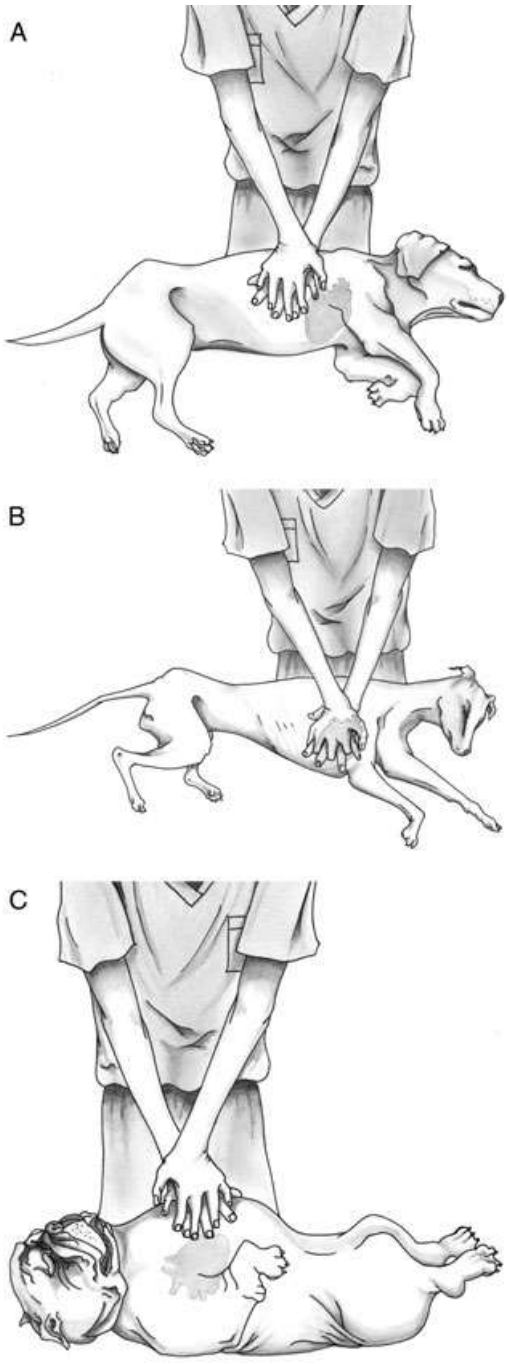


**Figure 4:** Chest compression techniques for medium, large, and giant breed dogs. (A) For most dogs, it is reasonable to do chest compressions over the widest portion of the chest to maximally employ the thoracic pump theory. Either left or right lateral recumbency are acceptable. (B) In keel-chested (ie, deep, narrow chested) dogs like greyhounds, it is reasonable to do chest compressions with the hands directly over the heart to employ the cardiac pump theory, again in either recumbency. (C) For barrel-chested dogs like English Bulldogs, sternal compressions directly over the heart with the patient in dorsal recumbency may be considered to employ the cardiac pump mechanism.

## Chest compression technique

There is strong evidence, including an experimental study in dogs documenting increased rates of ROSC and 24-hour survival, supporting a recommendation for compression rates of 100–120/min in cats and dogs (I-A).[36] However, there is also some evidence that higher compression rates of up to 150/min may be even more advantageous, and further work in this area is needed.

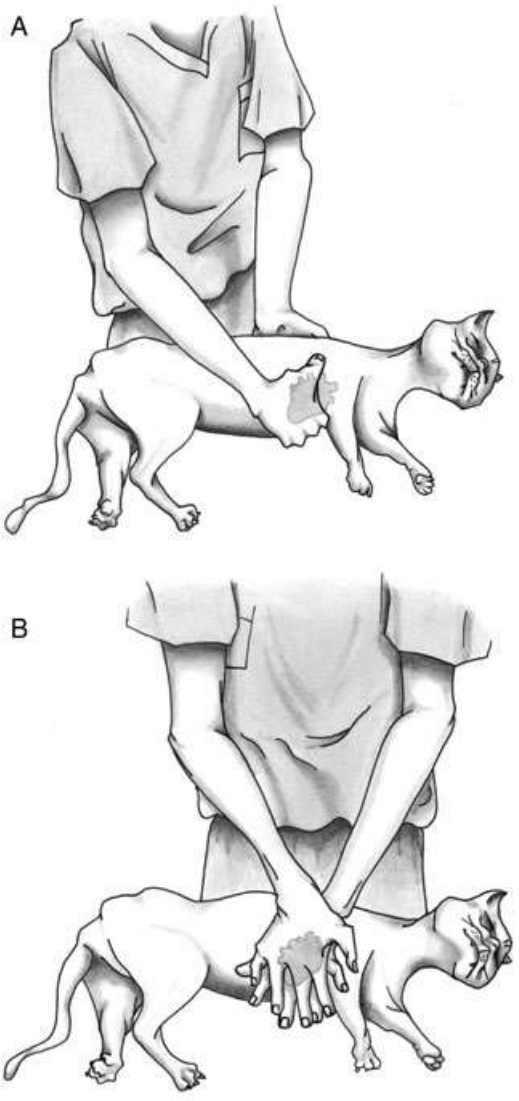


**Figure 5:** Chest compression techniques for small dogs and cats. (A) For most cats and small dogs (<10 kg) with compliant chests, the use of a 1-handed technique to accomplish circumferential chest compressions with the hand wrapped around the sternum directly over the heart may be considered. (B) An alternative chest compression method for cats and small dogs is the 2-handed technique directly over the heart to employ the cardiac pump mechanism. This method may be considered in larger cats and small dogs with lower thoracic compliance, or in situations in which the compressor is becoming fatigued while doing 1-handed compressions.

There is also good evidence to support deep chest compressions of 1/3–1/2 the width of the thorax in most patients (IIa-A), with an experimental canine study showing a linear relationship between compression depth and mean arterial pressure, and multiple human clinical trials and experimental animal studies supporting these compression depths.[37–39] Finally, experimental studies in pigs have documented reduced coronary and cerebral perfusion when full elastic recoil between chest compressions is not permitted (ie, leaning). Observational studies in people have shown a high prevalence of leaning during CPR. It is recommended that full chest wall recoil is allowed between compressions (I-A).[40,41]

**Ventilation**

Both hypoxia and hypercapnia reduce the likelihood of ROSC; therefore, securing a patent airway and providing ventilation are essential during CPR.[42,43] Although human CPR algorithms emphasize the importance of chest compressions over ventilation in BLS, there is evidence in human pediatric patients that ventilation is more important in patients with CPA not of primary cardiac origin.[44] Because the majority of canine and feline cardiac arrests are due to noncardiac root causes, early endotracheal intubation and provision of ventilation in CPR is likely to be of benefit.

Ventilation technique for intubated patients

Given the documented detrimental effects of pauses in chest compressions and the ease with which dogs and cats can be intubated, if equipment and personnel are available, rapid intubation of dogs and cats in CPA is recommended. This should be accomplished with the animal in lateral recumbency so that chest compressions may be continued during the procedure. Once the endotracheal tube (ETT) is in place, the cuff should be inflated so that ventilation and chest compressions can occur simultaneously (I-A). The ETT should be secured to the muzzle or mandible to prevent dislodgement. It may be useful for veterinarians and technicians to practice lateral endotracheal intubation in patients undergoing routine anesthetic procedures to develop and maintain these skills.

Although there are very limited data in dogs and none in cats evaluating optimal ventilation strategies for intubated patients during CPR, there are several well-controlled experimental studies in pigs as well as clinical studies in people supporting these recommendations. Higher respiratory rates, longer inspiratory times, and higher tidal volumes can lead to impaired venous return due to increased mean intrathoracic pressure as well as decreased cerebral and coronary perfusion due to vasoconstriction, and have been documented to lead to poorer outcomes in people during CPR.[45] Due to decreased pulmonary blood flow resulting from the reduced cardiac output achievable during CPR (approximately 25–30% or normal), physiologically "normal" ventilation rates are likely to lead to low arterial $CO_2$ tension. Lower respiratory rates are associated with elevated arterial $CO_2$ tension and can cause

peripheral vasodilation, worsening perfusion to the core, and cerebral vasodilation, potentially increasing intracranial pressure. Therefore, a ventilation rate of 10 breaths/min with a tidal volume of 10 mL/kg and a short inspiratory time of 1 second are recommended (I-A).

### Ventilation technique for nonintubated patients

There have been no studies examining the efficacy of mouth-to-snout ventilation in dogs and cats, although there is a case report describing successful application of this technique in a dog with traumatic cervical spinal cord injury during transport to a veterinary hospital, suggesting that it can effectively maintain oxygenation and ventilation in this species.[46] In addition, there is some evidence that effective ventilation can be accomplished in dogs using noninvasive techniques such as tight-fitting masks, but obtaining an appropriate fit and seal can be challenging.[47,48] To accomplish mouth-to-snout ventilation, the rescuer holds the patient's mouth tightly closed, places his or her mouth over the patient's nares making a seal with the snout, and blows into the nares (see Figure 6). There have been no studies investigating the optimal compression-to-ventilation (C:V) ratio during CPR in nonintubated dogs and cats and the results of studies in other species are somewhat conflicting. The preponderance of the evidence suggests C:V ratios of at least 30:2 should be maintained. Until further studies are done evaluating higher C:V ratios, a C:V ratio of 30:2 in nonintubated dogs is recommended (I-B). To accomplish this, a series of 30 chest compressions at a rate of 100–120/min is performed, followed by a brief interruption of chest compressions during which 2 breaths are delivered quickly, after which another series of 30 chest compressions are delivered.

**Figure 6:** Mouth-to-snout breathing technique. The rescuer holds the patient's mouth closed with one hand, creates a seal over the patient's nares with his or her mouth, and blows into both nares to achieve a normal chest rise.

### Cycles of CPR

Although there have been no studies in dogs and cats evaluating the optimal timing of CPR cycles, there are several high-quality prospective and retrospective studies in human medicine suggesting that uninterrupted cycles of BLS lasting 2 minutes result in better survival and neurological outcomes than shorter cycles with more frequent interruptions to chest compressions.[49,50] Therefore, chest compressions should be performed in 2-minute cycles without interruption in intubated patients when several rescuers are present, or in 2-minute cycles with brief interruptions after every 30 chest compressions to allow 2 quick breaths to be delivered using the mouth-to-snout technique if only 1 rescuer is present or the animal is not intubated (I-A). After each 2-minute cycle of compressions, the compressor should rotate to reduce lean and compromise of compression efficacy due to fatigue (I-B).

### Delay in starting CPR

Rapid diagnosis of CPA is crucial because the deleterious effects of delaying the start of BLS are significant, with reductions in survival to discharge and neurologic status reported in numerous studies.[51–53] Although not examined in veterinary medicine, several human studies have documented the poor sensitivity of pulse palpation for diagnosis of CPA.[54,55] In addition, it is common for agonal breaths to be misidentified as spontaneous breathing in people in CPA.[56] There is also strong evidence in the human literature that less than 2% of patients in CPA experience any serious harm when BLS is started, likely because patients will commonly respond to the stimulation associated with CPR.[57] Therefore, aggressive administration of CPR in patients suspected of being in CPA is recommended, as the risk of injury due to CPR in patients not in CPA is low (I-B). When assessing patients that are apneic and unresponsive, a rapid airway, breathing, circulation (ABC) assessment lasting no more than 5–10 seconds is recommended. If there is any doubt as to whether the patient has experienced CPA, CPR should be initiated immediately while further assessment to support the diagnosis of CPA is accomplished simultaneously by other personnel or after an initial cycle (2 min) of CPR.

### Interposed abdominal compressions

To facilitate venous return from the abdomen and improve cardiac output, the use of abdominal compressions

interposed with chest compressions has been extensively studied in experimental canine and porcine models as well as in human clinical trials.[58,59] There is minimal evidence of abdominal trauma due to the use of interposed abdominal compressions when rescuers are trained in the technique. Therefore, the use of interposed abdominal compressions in dogs and cats with CPA is reasonable when sufficient personnel trained in its use are available (IIa-B).

## ALS

ALS encompasses the components of veterinary CPR performed after BLS has been initiated and until ROSC is achieved. ALS includes therapy with vasopressors, positive inotropes, and anticholinergics, correction of electrolyte and acid-base disturbances and volume deficits, and prompt defibrillation. If BLS and ALS are performed promptly, initial ROSC rates may be as high as 50% in dogs and cats.[34]

### Vasopressor and vagolytic therapy

Because only 25–30% of a normal cardiac output is achieved with even high-quality external chest compressions, generation of adequate coronary and cerebral perfusion pressures during CPR requires high peripheral vascular resistance, directing more of the circulating volume to the central circulation. Vasopressors are therefore an essential component of ALS drug therapy.

#### Epinephrine

Epinephrine, a catecholamine that acts as a nonspecific adrenergic agonist, has been widely used for its vasopressor (α1) activity during CPR for decades. It also has β1 adrenergic activity, the inotropic and chronotropic effects of which are likely less crucial, and may be harmful when treating CPA due to increased myocardial oxygen demand, exacerbating myocardial ischemia, and predisposing to arrhythmias once ROSC is achieved.[60] Although higher doses (0.1 mg/kg IV) of epinephrine have been associated with increased rates of ROSC, they have not been associated with increased survival to discharge, possibly due to the exaggerated adrenergic effects.[61] Therefore, the use of low-dose (0.01 mg/kg IV) epinephrine administered every 3–5 minutes early in CPR is recommended (I-B), but high-dose (0.1 mg/kg IV) epinephrine may be considered after prolonged CPR (IIb-B). In order to minimize underdosing or overdosing during CPR, this drug should be administered during *every other* cycle of BLS.

#### Vasopressin

The vasopressor effects of vasopressin are mediated through the peripheral V1 receptor located on vascular smooth muscle. This mechanism of action is completely independent of the α1 effects of epinephrine. Unlike α1 receptors, V1 receptors remain responsive in the face of an acidic pH, and vasopressin has no inotropic or chronotropic effects that could worsen myocardial ischemia. Therefore, it has been studied as an alternative to epinephrine during CPR. Evidence of the efficacy of vasopressin compared to epinephrine in dogs and cats during CPR is limited, with 1 prospective observational study suggesting a beneficial effect of vasopressin[34] while a prospective trial in dogs found equivalent survival rates.[62] The human literature is mixed, with vasopressin potentially associated with increased survival in human patients with asystole, prolonged CPA, or hypovolemia,[63,64] but large meta-analyses have failed to show any benefit (or detriment) to the use of vasopressin over epinephrine in CPR.[65,66] Although further study is needed, the use of vasopressin (0.8 U/kg IV) as a substitute or in combination with epinephrine every 3–5 minutes may be considered (IIb-B).

#### Atropine

Atropine is a parasympatholytic agent that has been used widely in patients with CPR. Many studies have evaluated the use of atropine during CPR, and have largely shown no beneficial or detrimental effect of its use at standard dosing (0.04 mg/kg). Higher doses (0.1, 0.2, 0.4 mg/kg) have been associated with worse outcomes in an experimental study in dogs.[67] However, an experimental study showed that dogs with asphyxia-induced pulseless electrical activity (PEA) were more likely to be resuscitated when administered a combination of epinephrine and atropine than dogs administered epinephrine and 5% dextrose.[68] Although not strongly supported by the literature, atropine is most likely to be of use in dogs and cats with asystole or PEA associated with high vagal tone, and use of standard dose (0.04 mg/kg) atropine in these cases is reasonable (IIa-B). Due to the lack of any clear detrimental effect, routine use of atropine (0.04 mg/kg IV) during CPR in dogs and cats may be considered (IIb-C).

### Defibrillation

Sudden cardiac arrest due to ventricular fibrillation (VF) is common in people, and a large body of literature suggests that electrical defibrillation is the most effective therapy. Widespread implementation of by-stander-operated electrical defibrillators has been associated with marked improvement in survival in people. In a

hospital setting, current guidelines in human medicine recommend that "shockable" rhythms (VF and pulseless VT) be promptly treated with electrical defibrillation if available. Because VF and VT are the result of abnormal pacing of groups of ventricular myocardial cells by the myocardial cells themselves rather than the pacemakers, the goal of electrical defibrillation is to depolarize as many of these cells as possible, driving them into their refractory period, and stopping the random electrical and uncoordinated mechanical activity, that is, to stop the ventricles from fibrillating. If this is successful, the pacemakers may then begin driving the myocardial cells (establishing a sinus rhythm), or the patient may develop asystole. Note that either of these outcomes is considered a successful defibrillation. In the absence of an electrical defibrillator, mechanical defibrillation may be accomplished with a precordial thump, but the efficacy of this intervention is likely poor.

### Electrical defibrillation technique

Modern defibrillators use one of two main technologies: (1) monophasic, in which a unidirectional current flows from one electrode to the other, and (2) biphasic, in which current initially flows in one direction, then reverses and flows in the other direction. Biphasic defibrillators have been shown to more effectively terminate VF at lower defibrillation energy than monophasic defibrillators, in turn leading to less myocardial injury.[69] Therefore, the use of a biphasic defibrillator is recommended over a monophasic defibrillator (I-A), at a dose of 4–6 J/kg with a monophasic defibrillator or 2–4 J/kg with a biphasic defibrillator (IIa-B). If the first shock is unsuccessful, there is some evidence from experimental and clinical human studies that increasing the defibrillation energy may increase the rate of success.[70,71] Although no studies have shown a direct detrimental effect of dose escalation, there is a risk of increased myocardial damage with increasing defibrillation dose. However, in dogs and cats with VF/pulseless VT, defibrillation energy escalation (eg, 50% dose increase) is reasonable if the first countershock is unsuccessful (IIa-B).

To maximize current through the ventricles, the paddles should be placed on opposite sides of the thorax approximately over the costochondral junction directly over the heart. To facilitate this, the patient will likely have to be placed in dorsal recumbency. The use of a plastic trough may facilitate this. Defibrillator paste or gel should be liberally applied to the paddles, which must be pressed firmly against the chest to establish contact with the skin. If defibrillation patches are used, the fur must be shaved to facilitate contact, which will result in a longer pause in chest compressions. Once the defibrillator is charged, the operator must ensure that no personnel are making any contact with the patient or the table to prevent injury by announcing the intent to defibrillate with a term such as "Clear" and visually confirming that all personnel are clear before discharging the defibrillator. The person discharging the defibrillator is also at risk, and must ensure that he or she is not touching the patient or the table; the use of exam gloves can reduce the risk of contact, but he or she must ensure that no fluid, gel, or paste is bridging the cuff of the glove and allowing contact with the skin. In addition, electrical defibrillation should not be attempted if alcohol is on the fur due to the high risk of fire. The use of a posterior paddle assembly, a flat paddle replacement, can improve the efficiency and safety of defibrillation, minimizing the interruption to compressions, and eliminating the need to place the patient in dorsal recumbency. The flat paddle is coated with gel or paste and placed under the patient's thorax. Defibrillation is then accomplished using a standard hand paddle on the upward facing chest wall, and chest compressions can resume immediately while the posterior paddle is still in place (see Figure 7).

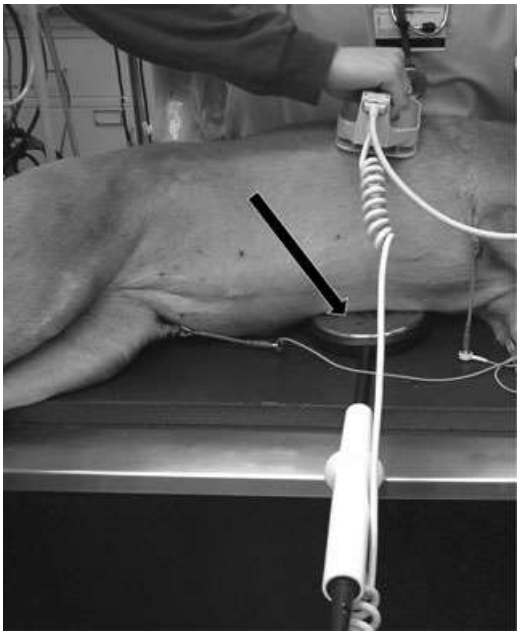

**Figure 7:** Posterior paddle assembly. The black arrow indicates the posterior paddle. The dog is laid on the posterior paddle, and when defibrillation is required, the hand paddle is placed on the opposite side of the chest directly over the heart to defibrillate. Chest compressions can then be immediately continued with the posterior paddle in place.

### Timing of electrical defibrillation

It is generally accepted that after a loss of perfusion, the ischemic heart passes through 3 phases: (1) the electrical phase during which minimal ischemic damage

occurs, lasting 4 minutes; (2) the circulatory phase during which reversible ischemic damage occurs, lasting 6 minutes; (3) the metabolic phase during which potentially irreversible ischemic damage begins to occur, and which may necessitate more advanced techniques such as therapeutic hypothermia and cardiopulmonary bypass to reverse.[72] Therefore, immediate defibrillation is recommended in cases of CPA due to VF/pulseless VT of duration of 4 minutes or less (I-B), or if VF is diagnosed during a rhythm check between cycles of CPR (IIb-B). If the patient is known or suspected to have been in VF/pulseless VT for greater than 4 minutes and is beyond the electrical phase, energy substrates are likely depleted, and the patient will most likely benefit from a 2-minute cycle of BLS before defibrillation (I-B).

Although older CPR algorithms recommended the use of 3 stacked shocks for patients with refractory VF/pulseless VT, compelling experimental data in pigs and clinical data in people showed better outcomes when a single shock was followed by a full 2-minute cycle of CPR before re-evaluating the ECG and defibrillating again.[73–75] Therefore, administration of a single shock as opposed to 3 stacked shocks is recommended, with immediate resumption of CPR in the case of nonsuccessful defibrillation (I-B).

### Precordial thump

The precordial thump was first described as a treatment option for VF in a case report in 1969 and a case series in 1970.[76,77] Briefly, this is a method of mechanical defibrillation, accomplished by striking the patient with the heel of the hand directly over the heart. Unfortunately, more recent studies have documented minimal efficacy of this technique for treatment of VF.[78–80] Although a worksheet was not completed on this topic, given that there is some limited evidence that a precordial thump may have some efficacy for the treatment of VF/pulseless VT, this intervention may be considered. However, given the overwhelming evidence of the superiority of electrical defibrillation for the treatment of VF/pulseless VT, a precordial thump should only be considered if an electrical defibrillator is not available.

## Antiarrhythmic drug therapy

The utility of antiarrhythmic agents such as amiodarone, lidocaine, and magnesium for patients with CPA due to VF/pulseless VT has been extensively studied in experimental models and clinical trials in people, and the data have been summarized in a recent meta-analysis.[81] Of the agents studied, only amiodarone has shown consistent benefit and may be considered in cases of VF/pulseless VT resistant to electrical defibrillation (IIb-B). Some studies have also shown a beneficial effect of lidocaine in patients with refractory VF/pulseless VT, although one experimental study showed an increase in the energy required to successful defibrillate dogs with induced VF.[82] However, more recent data in pigs suggested that this phenomenon occurs when using monophasic defibrillators, but not when using biphasic defibrillators.[83] Given the uniformly grave prognosis for patients in refractory VF/pulseless VT, when amiodarone is not available, lidocaine may be considered in cases of pulseless VT/VF resistant to defibrillation (IIb-B), especially when a biphasic defibrillator is used. Data on the use of magnesium are less compelling, and routine use of magnesium sulfate is not recommended for cardiac arrhythmias during CPR, although it may be considered for treatment of torsades de pointes (IIb-B). It should be recognized that the use of antiarrhythmic agents may be considered as adjunctive therapy in refractory cases, but electrical defibrillation is the recommended primary treatment for VF/pulseless VT (I-B).

## Reversal agents

Of the reversal agents available, only naloxone has been evaluated for use in patients in CPA. Although evidence of a beneficial effect is limited, in cases of opioid toxicity, naloxone should be used during CPR (I-B).[84] Even in the absence of opioid toxicity, the data available suggest that in cases of recent opioid administration, the use of naloxone during CPR may be considered (IIb-B). Although no specific studies have evaluated the use of other reversal agents, in dogs and cats that have received reversible anesthetic/sedative medication, administering reversal agents during CPR may beconsidered (IIb-C), as the potential risks associated with administration of these drugs are low. The drug and dosing chart in Appendix II contains recommended doses during CPR for naloxone (to reverse opioids), flumazenil (to reverse benzodiazepines), and atipamezole (to reverse $\alpha 2$ agonists).

## Electrolyte therapy

### Calcium

Calcium is vital for many cellular processes, including cellular communication and muscle contraction. Although hypocalcemia commonly develops in patients with prolonged CPA, the majority of studies investigating the utility of routine calcium administration during CPR demonstrated no effect on outcome or worse outcomes, suggesting that IV calcium should not be used routinely during CPR (III-B). No studies investigating the use of calcium in patients with documented hypocalcemia during CPR were identified. Given the importance of calcium for skeletal and smooth muscle contraction, intravenous calcium may be considered in dogs and

cats with documented moderate to severe hypocalcemia during CPR (IIb-C), but studies directly addressing this question are needed.

### Potassium

Hyperkalemia develops commonly in patients with prolonged CPA, and treatment of hyperkalemia during CPR using hemodialysis is associated with improved outcomes.[85] Given this evidence, documented hyperkalemia should be treated during CPR (I-B). Although hemodialysis is rarely available in veterinary clinical practice, administration of medical therapies directed at treating hyperkalemia would be reasonable.[86] Although hypokalemia has been associated with CPA in people, no studies of the efficacy of treatment of hypokalemia during CPR have been done.[87] Therefore, treatment of documented hypokalemia during CPR may be considered (IIb-C), but there is no evidence to support or refute this treatment.

## Other therapies

### Corticosteroids

Several case series and experimental studies have examined the utility of corticosteroids in CPR with mixed results, most involving multiple treatment variables other than steroids. Only one placebo-controlled randomized trial specifically investigated the efficacy of corticosteroids (dexamethasone) in people during out of hospital CPR, which showed no benefit with the use of steroids.[88] Given the lack of compelling evidence of a beneficial effect and the potential for deleterious side effects from corticosteroids,[89,90] especially in animals with poor perfusion,[91] the routine use of corticosteroids during CPR is not recommended (III-C).

### Impedance threshold device

Impedance threshold devices (ITD) have been shown to improve hemodynamics in anesthetized dogs by increasing venous return due to decreased intrathoracic pressure.[92] While some experimental studies in non-target species have demonstrated a benefit of these devices during CPR, the largest clinical trial to date failed to demonstrate any improvement in ROSC or survival to discharge in people in CPA with the use of an ITD.[93] In addition, because the device requires chest wall recoil to generate a "cracking pressure" of at least $-12$ cm $H_2O$, use is not feasible in small dogs or cats weighing less than 10 kg because they are unlikely to be capable of generating those types of pressures from elastic recoil alone. Therefore, the use of an ITD to enhance circulation is reasonable in animals > 10 kg (IIa-B), but studies to date have not demonstrated a survival advantage with their use.

### Alkalinization therapy

Severe acidemia due to metabolic acidosis is common in patients with CPA, and this acid-base disturbance can lead to detrimental metabolic dysfunction. Several experimental studies in dogs have documented improved survival with bicarbonate therapy with prolonged (>10 min) duration of CPA.[94,95] However, other experimental studies in dogs have demonstrated worse outcomes and metabolic derangements with bicarbonate therapy, especially when given early in CPR.[96] Given the evidence available, bicarbonate therapy after prolonged CPA of greater than 10–15 minutes with administration of 1 mEq/kg of sodium bicarbonate may be considered (IIb-B).

### Intratracheal drug administration

When available, intravenous or intraosseous administration of resuscitation drugs is preferred over intratracheal administration, and is associated with improved survival from CPA.[97] However, in animals in which intravenous or intraosseous access is not possible, the use of the intratracheal route for epinephrine, vasopressin, or atropine may be considered (IIb-B). The optimal location within the respiratory tract for administration of these drugs is not fully understood, nor is the optimal drug dose, or volume and type of diluent. There is some evidence that use of a long catheter advanced to or beyond the level of the carina results in higher plasma concentrations of drug than shorter catheters or direct instillation of drug into the ETT.[98] If the intratracheal route is used for drug administration during CPR, drugs should be diluted with saline or sterile water and administered via a catheter longer than the ETT (I-B). Increased doses of up to 10× standard doses (in the case of epinephrine) have been recommended, but data regarding optimal dosing are lacking.

### Supplemental oxygen administration

The use of a fraction of inspired oxygen ($FiO_2$) of 100% during CPR has been justified as a means to maximize arterial oxygen content in an effort to compensate for the decreased cardiac output (25–30% of normal) during external chest compressions. However, the presence of hyperoxia may predispose patients to increased concentrations of reactive oxygen species, worsening tissue damage during CPR. There is limited evidence in experimental animals, but the preponderance of the evidence suggests decreased neurologic injury when

oxygen supplementation is titrated to achieve normoxemia ($PaO_2$ of 80–105 mm Hg) compared to animals that are hyperoxemic.[99,100] Given this evidence, during CPR in dogs and cats, the use of an $FiO_2$ of 21% (room air) may be considered (IIb-B). However, this approach is best used in circumstances in which arterial blood gas analysis during CPR is possible so that the $FiO_2$ can be titrated to maintain normoxemia. In the absence of arterial blood gas data, the risks of hypoxemia likely outweigh the risks of hyperoxemia, and the use of an $FiO_2$ of 100% is reasonable (IIa-B).

#### IV fluid administration

A worksheet on IV fluid administration during CPR was not completed as part of the RECOVER initiative. However, the ILCOR fluid therapy worksheet (ALS-D-016A) was evaluated and guidelines extracted from evaluation of that data.[101] Multiple experimental studies in animals have shown that fluid administration during CPR in animals that are euvolemic is associated with decreased coronary perfusion pressure.[102,103] This is likely due to the fact that the administration of IV fluids predominantly increases central venous pressure, opposing blood flow to the coronary and cerebral circulation. Therefore, during CPR in euvolemic or hypervolemic dogs and cats, routine administration of intravenous fluids is not recommended (III-B). Although no specific evidence was identified, patients with preexisting hypovolemia are likely to benefit from increased circulating volume during CPR, and administration of intravenous fluids in these patients is reasonable (IIa-C).

#### Open-chest CPR

Open-chest CPR is more effective than closed-chest CPR in restoring ROSC and promoting a good outcome in canine models of VF. In practice, open-chest CPR requires significant resources, is a procedure that requires a skillful veterinary team, and demands advanced PCA supportive care. Although studies investigating the utility of open-chest CPR in veterinary medicine are lacking, in cases of significant intrathoracic disease, such as tension pneumothorax or pericardial effusion, promptly performing open-chest CPR may be considered (IIb-C).

### *Monitoring*

Two overarching clinical goals of RECOVER led to the development of a domain devoted exclusively to monitoring. First, special considerations apply to the use of familiar hemodynamic monitoring technology during CPR due to significant alterations in cardiovascular and respiratory physiology that occur under these conditions. Second, specific recommendations regarding monitoring equipment and techniques necessary for the performance of high-quality CPR are provided for practitioners aiming to update clinical CPR practices and preparedness.

Four important aspects of veterinary CPR are addressed in these monitoring guidelines. The first is focused on methods to confirm a diagnosis of CPA and endotracheal intubation. The second section, and the bulk of this domain, evaluates monitoring options during CPR, covering both commonly used monitoring protocols as well as newer options for assessing adequacy of CPR and ROSC. The third examines monitoring approaches that may be useful in patients at risk of CPA. The final section of this domain is concerned with suggested monitoring protocols for small animal patients following ROSC.

#### Diagnosing CPA

Early initiation of CPR in patients that have experienced CPA is crucial for a successful outcome; therefore, a rapid initial airway, breathing, and circulation (ABC) assessment of any unresponsive, apneic patient to rule out CPA is essential. Several monitoring techniques have been proposed to aid in this diagnostic assessment. Pulse palpation is widely employed by veterinary practitioners as part of their initial assessment of any acutely presenting patient. Although no clinical research was identified in veterinary medicine, many human studies have shown that pulse palpation is an unreliable technique to confirm CPA, and that only 2% of rescuers correctly recognize the lack of a pulse within 10 seconds.[54] The specificity of pulse palpation for diagnosis of CPA is approximately 65%, meaning that in 35% of cases, rescuers believed a pulse was present when one was not. Until studies in veterinary medicine in unresponsive, apneic dogs and cats are done, the use of pulse palpation to support a diagnosis of CPA before initiating CPR is not recommended (III-B). It may be challenging for many practitioners to begin CPR without attempting to identify a pulse. However, the data suggest that prolonged pulse palpation to refute an initial diagnosis of CPA is not beneficial, and CPR should be started immediately in any patient in which a pulse cannot be readily identified during an initial ABC assessment. Although there is evidence that Doppler blood pressure monitoring may be useful for early recognition of CPA in patients at risk of arrest, no studies investigated the ease of placement of a Doppler flow probe in patients suspected of being in CPA. Given the time associated with placing a Doppler sensor and acquiring a signal, in unresponsive, apneic dogs and cats, the use of Doppler to support a diagnosis of CPA before initiating CPR is not recommended (III-C),

unless the probe had been placed prior to CPR (eg, as part of anesthetic monitoring). Although ECG monitoring is useful during CPR to identify specific arrest rhythms that may guide ALS therapy, some rhythms (eg, PEA, pulseless VT) may appear as perfusing rhythms despite the presence of CPA, and thus have the potential to delay the start of BLS. Therefore, in unresponsive, apneic dogs and cats, the use of ECG as the sole parameter to accept or reject a diagnosis of CPA before initiating CPR is not recommended (III-B). Finally, $EtCO_2$ monitoring has been investigated as a tool for diagnosing CPA. Because of decreased pulmonary blood flow, a low $EtCO_2$ is expected in the presence of CPA. However, initial $EtCO_2$ values (ie, the first values obtained after endotracheal intubation) have been shown to be unreliable for this task in dogs, pigs, and humans. In dogs with asphyxial cardiac arrest, initial $EtCO_2$ can be higher than the prearrest mean value.[104,105] Therefore, the immediate postintubation EtCO2 value should not be used for diagnosis of CPA in dogs and cats (III-B), although subsequent values may be associated with pulmonary perfusion.

**Monitoring patients during CPA**

A large part of the monitoring domain focused on recommendations for assessments that should be performed during CPR, as well as for the appropriate application of these techniques. The following guidelines are the result of an analysis of the monitoring domain worksheets as well as worksheets from the other RECOVER domains. Of the monitoring devices evaluated, there is strong evidence to support the use of ECG and $EtCO_2$ monitoring in dogs and cats with CPA, and if they are available, these devices should be used early in any CPR attempt.

### Verification of endotracheal intubation

In contrast to the American Heart Association (AHA) guidelines for CPR in people, the RECOVER guidelines recommend early intubation and ventilation in dogs and cats in CPA because of the ease with which most dogs and cats may be intubated and the higher prevalence of asphyxial arrest in these species. Verification that the ETT is correctly placed into the trachea as opposed to the esophagus is crucial, and $EtCO_2$ monitoring has been used to assist in this verification process because $CO_2$ will not be consistently measured if the esophagus has been intubated. Based on the evidence evaluated, $EtCO_2$ monitoring is likely a valuable adjunct for verification of correct ETT placement in conjunction with direct visualization, auscultation, or observation of chest excursions in dogs and cats with CPA to verify correct ETT placement (IIa-B), but should not be used as a sole measure of correct placement (III-B). The majority of the assessed studies found that in patients with primary cardiac arrest, a low $EtCO_2$ value may be obtained despite correct ETT placement, and that more accurate evaluation of ETT placement requires other assessments as described above.[106]

### Electrocardiogram

The ECG is a valuable monitor during CPR. Although it is susceptible to artifact during chest compressions, evaluation of the ECG during intercycle pauses is recommended to obtain an accurate rhythm diagnosis and to guide ALS therapy (I-C). However, the ECG evaluation must be done rapidly, and should not significantly delay resumption of chest compressions. Chest compressions should not be stopped during a complete 2-minute cycle of CPR to allow ECG interpretation (III-B).[107] Similarly, for patients in VF, rapid assessment of the ECG to determine if VF has resolved immediately after defibrillation is reasonable, but should minimally delay resumption of chest compressions for another cycle (IIa-B). Several studies have demonstrated no harm in these short delays in chest compressions (eg,[108]), but there is also evidence that 72% of patients will develop recurrent VF within 60 seconds of defibrillation while only 20% have evidence of recurrence within 6 seconds, suggesting that an ECG rhythm diagnosis immediately after defibrillation may not be an accurate reflection of sustained defibrillation success.[109]

### End tidal $CO_2$

There is strong evidence supporting the use of $EtCO_2$ monitoring during CPR as an early indicator of ROSC (I-A) and as a measure of efficacy of CPR (IIa-B), potentially allowing rescuers to adjust their treatment to maximize perfusion during CPR. Because $EtCO_2$ is affected by both pulmonary perfusion and minute ventilation, rescuers should be cautious to maintain constant minute ventilation when using $EtCO_2$ measurement for these purposes. Multiple high-quality studies support the conclusion that sudden increases in $EtCO_2$ occur rapidly with ROSC due to an increase in pulmonary blood flow. There is limited data in dogs and cats suggesting that higher $EtCO_2$ values during CPR ($>15$ mm Hg in dogs, $> 20$ mm Hg in cats) may be associated with an increased rate of ROSC, although a statistically significant difference was only noted in dogs.[34]

### Other monitoring approaches during CPR

The evidence supporting the use of other monitoring approaches during CPR is less compelling. As described previously, although not studied in veterinary medicine, pulse palpation is not a reliable diagnostic tool for CPA

in people, and interruption of chest compressions during CPR specifically to palpate the pulse is not recommended (III-B). However, palpation of the pulse to identify ROSC during intercycle pauses in CPR is reasonable as long as it does not delay resumption of compressions (IIb-C). Pulse palpation during chest compressions is also reasonable, but should be interpreted cautiously, as retrograde flow through the venous system may be mistakenly interpreted as an arterial pulse.

Although no published data are available, the use of a Doppler flow probe during CPR has been described anecdotally as a measure of CPR quality and ROSC. Due to the lack of evidence at this time, no recommendation on the utility of this approach can be made, but Doppler signals should be interpreted with caution in patients undergoing chest compressions due to the possibility of motion artifact or detection of retrograde venous blood flow. During intercycle pauses in chest compressions, Doppler flow probe assessment may be useful, but should not delay resumption of chest compressions. Further studies are needed to assess the utility of this monitoring technology.

Audiovisual prompting and feedback devices have been shown to improve adherence to guidelines during CPR in people, but have not directly been shown to improve outcomes.[110] No studies in veterinary medicine have evaluated the use of these devices, but it is reasonable to use such devices to improve the quality of CPR (IIa-C) if they may be modified for veterinary patients and veterinary CPR goals.

Electrolyte disturbances such as hyperkalemia and hypocalcemia commonly develop with prolonged CPR, and routine monitoring of electrolytes, especially during prolonged CPR, may be considered (IIb-B).[111] In cases of CPA that are known or suspected to be due to electrolyte derangements, monitoring of electrolytes will help guide therapy and is recommended (I-C). The use of blood gases during CPR is controversial, but the available data generally support that central or mixed venous blood gases more accurately reflect ventilation and perfusion deficits than arterial blood gases. This suggests that central or mixed venous blood gas analysis to evaluate the effectiveness of CPR may be considered (IIb-B), but that arterial blood gas analysis during CPR is not recommended (III-A).[112] Quantitative VF waveform analysis using wavelet analysis has been evaluated in experimental dog and pig models, but data on its utility are limited. The major conclusions from this work are that coarse (high amplitude, low frequency) VF appears to be associated with a higher likelihood of ROSC than fine (low amplitude, high frequency) VF. This type of analysis may be considered during intercycle pauses in chest compressions (IIb-B), but more studies are needed.

**Monitoring patients at risk of CPA**

Given the grave prognosis associated with CPA in dogs and cats, early identification of at risk patients and early diagnosis of CPA are crucial for improving outcomes. Therefore, critically ill patients at risk of CPA must be vigilantly monitored. Although no specific studies investigated the effect of pre-CPA monitoring on outcome, the risk:benefit ratios of these types of monitoring approaches are highly favorable. Therefore, it is reasonable to utilize continuous ECG monitoring (IIa-C) and continuous Doppler monitoring of arterial blood flow or direct arterial pressure monitoring (IIa-C) in patients at risk of CPA. In addition, because of the close association between cardiac output and $EtCO_2$ in patients with constant minute ventilation, continuous $EtCO_2$ monitoring is recommended in intubated and ventilated patients at risk of CPA (I-A).

**Monitoring patients after ROSC**

There are limited data available to provide guidelines for monitoring of patients after ROSC; therefore, basic principles of monitoring critically ill patients should be applied. Because of the high risk of recurrence in patients with ROSC after CPA, postresuscitation monitoring should be sufficient to detect impending reoccurrence of CPA (I-C) and should be sufficient to guide therapy appropriate for the patient's condition (I-C). Based on the evidence presented above for monitoring patients at risk of CPA, minimum postresuscitation monitoring should include continuous ECG, intermittent arterial blood pressure monitoring, and assessment of oxygenation and ventilation (I-B). Other parameters that might be abnormal in patients at risk for reoccurrence of CPA include blood glucose and lactate concentrations and body temperature; PCA monitoring of these parameters may be considered depending on the patient and any underlying diseases (IIb-B). Serial body temperature measurements are also recommended in order to avoid high rewarming rates and hyperthermia.

## *PCA Care*

Many animals will ultimately die despite initial successful resuscitation, leading to the conclusion that ROSC is only an intermediate endpoint in CPR. Between 60% and 70% of human CPA victims achieving ROSC will not survive to hospital discharge,[113,114] and survival to discharge rates range from 2 to 10% for dogs and cats, despite initial ROSC in 35 to 45% of the animals.[34,115] Optimizing care after ROSC can, perhaps, positively impact outcome. Thus PCA care is an essential portion of CPA management and may be the missing link of successful CPR.[4,116] A PCA syndrome, characterized by a

combination of multiorgan failure, cardiogenic shock, anoxic brain injury, and the sequelae of preexisting diseases, is believed to be the cause of this high mortality.[117] The veterinary clinician faces the challenge of providing care to a highly heterogeneous population of patients. In addition, the goals of PCA care change over the course of the post-ROSC phase, initially focused on prevention of rearrest, and later targeting prevention of further organ injury and rehabilitation care.[117] General veterinary critical care considerations build the foundation of care for all of these patients; treatment principles specific to the PCA phase have been described. It was the focus of this RECOVER domain to examine the evidence and provide treatment guidelines for dogs and cats in the PCA phase.

The clinically relevant questions asked in the RECOVER PCA care domain centered on hemodynamic optimization strategies, control of respiratory function, mild hypothermia, and rewarming rates. Drug therapies including corticosteroids, seizure prophylaxis, hyperosmolar therapy, and metabolic protection were also examined. Finally, outcome benefits associated with combination (bundle) therapies to achieve additive or synergistic effects and referral center management of the PCA patient were investigated.

**Hemodynamic optimization strategies**

There is some limited evidence in humans that strategies targeted at ensuring adequate oxygen delivery to tissues with hemodynamic optimization algorithms may improve survival in the PCA period.[118] Use of these types of hemodynamic optimization strategies, including primary resuscitation endpoints of central venous $O_2$ saturation ($ScvO_2$) or lactate, and secondary endpoints including arterial blood pressure, central venous pressure, PCV, and arterial oxygen saturation may be considered (IIb-B) in dogs and cats in the PCA period (see Figure 2). Use of such an endpoint-driven approach to the provision of cardiovascular support allows titration of therapy to the individual needs of the patient, an important aspect given the inherent heterogeneity of PCA patients. To reach these hemodynamic goals, IV fluid therapy is often indicated. However, the routine use of large volumes of IV fluids postarrest is not recommended except in the case of strongly suspected or confirmed hypovolemia; fluid therapy should instead be adjusted according to criteria customary to veterinary small animal emergency and critical care, and should be avoided in patients with evidence of congestive heart failure (III-C). Measurement of central venous pressure in patients at increased risk of pulmonary edema may be used as an integrated part of the RECOVER PCA algorithm (Figure 2). In the PCA period, the evidence for the use of vasopressor and/or positive inotropic support to reach hemodynamic goals is generally supportive or neutral, suggesting that the use of these drugs in dogs and cats with persistent hypotension and/or cardiovascular instability is reasonable (IIa-B). In addition, there is experimental evidence in dogs that after prolonged CPA, hypertension (mean arterial pressure [MAP] > 150 mm Hg) during reperfusion and the first few hours of PCA may be associated with improved survival and neurologic outcomes.[119] Therefore, it is reasonable to assume that hypertension in the immediate PCA period in dogs and cats is beneficial (IIa-B).

**Control of respiratory function**

Ventilation impacts a number of important physiologic processes that deserve special consideration in the context of PCA care, although data describing the effect of ventilatory disturbances during the PCA period on outcome are sparse. First, ventilation is the main pathway for elimination of $CO_2$ from the body. Since the cerebrovascular reactivity to $CO_2$ is maintained after resuscitation from CPA, at least in humans, control of $CO_2$ should have an impact on cerebral blood flow.[120–122] Hypocapnia can lead to decreased cerebral blood flow potentially causing cerebral hypoxia, while hypercapnia may increase cerebral blood flow and blood volume, potentially causing increased intracranial pressure. Second, positive pressure ventilation may lead to increased intrathoracic pressure, decreasing venous return to the heart, and compromising cardiac output. The use of high tidal volumes will worsen this effect and contribute to lung injury.[123] Third, insufficient tidal volume and respiratory rate may lead to pulmonary atelectasis and hypoxemia. However, direct evidence to suggest a generally applicable ventilation strategy in PCA patients is not available. One experimental study in dogs demonstrated improved cerebral blood flow, neurologic function, and histopathologic evidence of neuronal damage in animals that were normocapnic, although multiple simultaneous interventions were employed.[124] It is reasonable to target normocapnia ($PaCO_2$ of 32–43 mm Hg in dogs and 26–36 mm Hg in cats) in the PCA period (IIa-B), and serial monitoring of $EtCO_2$ or arterial blood gases is necessary to assure adequacy of ventilation. Mechanical ventilation, ie, intermittent positive pressure ventilation (IPPV) may be necessary to achieve and maintain normocapnia and normoxia in some patients. One veterinary study found that IPPV in the PCA period is associated with poor survival.[125] This finding was likely confounded by the fact that the lung disease was severe in animals receiving IPPV, rather than documenting a detrimental effect of IPPV. Cost and case management considerations may further limit routine application of IPPV for PCA

care. It is reasonable to employ manual or mechanical ventilation in patients that are hypoventilating in the PCA period, are hypoxemic or require high inspiratory oxygen concentrations ($FiO_2 \geq 0.60$) to maintain normoxemia, or are at risk of respiratory arrest (IIa-C), but routine mechanical ventilation in all PCA patients is not recommended (III-B).

Although hypoxemia is harmful in critically ill patients, there is good evidence from multiple studies in various species, including dogs, that normoxia/normoxemia is preferable to hyperoxia/hyperoxemia in the early PCA period, probably due to the injurious effects of reactive oxygen species that are elaborated in high concentrations during reoxygenation of ischemic tissue.[126] Therefore, oxygen supplementation should be titrated to maintain normoxemia ($PaO_2 = 80$–100 mm Hg, $SpO_2 = 94$–98%) especially early after resuscitation. Both hypoxemia and hyperoxemia should be avoided (I-A).

**Hypothermia and rewarming**

The increasingly widespread use of mild therapeutic hypothermia (MTH; core body temperature of 32–34°C) in human PCA care seen today originated from two successful landmark randomized controlled trials.[127,128] The preponderance of evidence suggests that MTH has organ protective effects in PCA patients, leading to improved cardiac, and most importantly, neurologic outcomes.[129] Of key importance is that MTH is one of the few treatments that is effective when administered after reperfusion, in contrast to many other interventions that are efficacious only with pretreatment. Questions on the optimal onset and duration of hypothermia, as well as which subpopulations benefit most, still need to be addressed in human medicine. Much experimental data support the efficacy of MTH in dogs, but its benefit in clinical veterinary medicine is undocumented.[130] Safe application of MTH principles requires advanced critical care capabilities and mechanical ventilation, although it was found to be feasible in at least 1 veterinary case report.[131] Based on strong evidence from experimental studies in dogs and human clinical trials, MTH should be initiated in dogs and cats that remain comatose as soon as possible after ROSC and maintained for 24–48 hours if mechanical ventilation and advanced critical care capabilities are available (I-A). If advanced critical care capabilities including mechanical ventilation are not available, MTH should not be initiated (III-C). However, if mild accidental hypothermia is present in these cases, it is reasonable to not rapidly rewarm these patients. This is clinically relevant even without use of MTH, as many patients that experience CPA and subsequently achieve ROSC develop unintended hypothermia. Although not investigated in clinical studies targeted at rewarming in the PCA period, there are several good quality experimental studies in dogs that suggest a slow rewarming rate of 0.25–0.5°C/h is reasonable (IIa-A), and that rewarming rates of >1°C/h should be avoided (III-A).[132–136]

**Drug therapies**

The utility of other select neuroprotective and metabolic drug therapies during CPA care have been investigated. There have been no clinical studies conducted in veterinary species investigating these therapies, but some data from human clinical trials and experimental studies are available.

Corticosteroids

There is conflicting evidence in the literature regarding the utility of corticosteroids for neuroprotection in the PCA period. Although a few experimental studies have shown some benefit to their use, human clinical trials have failed to demonstrate any positive effect. Clinical trials in veterinary species have not been done. Given the limited evidence in support of a beneficial effect and the potential for severe adverse events from corticosteroids,[89,90] especially in animals with poor perfusion,[91] routine administration of corticosteroids during PCA care is not recommended (III-C). However, there is evidence that people experiencing PCA shock may have improved global hemodynamics, $ScvO_2$, and survival to discharge when treated with low-dose hydrocortisone for relative adrenal insufficiency.[137] Therefore, administration of hydrocortisone (1 mg/kg followed by either 1 mg/kg q 6 h or an infusion of 0.15 mg/kg/h and then tapered as the patient's condition allows) to cats or dogs that remain hemodynamically unstable despite administration of fluids and inotropes/pressors during PCA care may be considered (IIb-C).

Hyperosmotic therapy

Cerebral edema has been identified in people in the PCA period and is associated with poor outcome.[138] Although there is evidence of improved survival from CPA with administration of hypertonic saline or mannitol when administered during CPR, there have been no studies investigating the utility of such therapy during PCA care.[139–142] Given the documented utility of both hypertonic saline and mannitol for treating cerebral edema, the use of these drugs in dogs and cats with neurologic signs consistent with cerebral edema (eg, coma, cranial nerve deficits, decerebrate postures, abnormal mentation) may be considered (IIb-C), but the diuretic effects of

mannitol should be recognized and fluid therapy titrated to prevent development of hypovolemia.

### Seizure prophylaxis

Seizures and myoclonus occur in 5–15% of adult human patients in the PCA period and in 40% of human patients that remain comatose after ROSC.[143] Many of these seizures are nonconvulsive, and can therefore only be detected by EEG monitoring.[144] The presence of seizures is associated with poor outcomes in people, but the incidence and prognostic significance of these abnormalilties in dogs and cats in the PCA period is unknown. Prophylactic anticonvulsant therapy in the PCA period in people has been associated with reduced seizure frequency and improved outcomes in some studies, but no effect has been found in others.[117] One experimental VF study in cats showed reduced EEG evidence of seizures in the PCA period in animals treated with thiopental, but no difference in neurologic outcome.[145] Seizure prophylaxis with barbiturates (eg, phenobarbital) may be considered in dogs and cats during the PCA period (IIb-B).

### Metabolic protection

Although there is much preclinical evidence that metabolic protectants such as poly-ADP-ribose polymerase inhibitors (to prevent DNA damage), mitochondrial protectants, and antioxidants show benefit in PCA care, their clinical efficacy remains to be demonstrated.[117] The evidence, to date, can only be described as suggestive and promising, and no clinical guidelines can be developed at this time.

### Bundle therapies for PCA care

Administration of several interventions in combination has been used in treatment of complex disease states, such as cancer or sepsis, and such a bundle of therapies may also be required to address the PCA syndrome.[146–148] The concept of using a bundle of individual treatment components for PCA care has found more attention recently, as a multisystem approach is currently considered a promising strategy for PCA care in people and may include MTH, goal-directed hemodynamic optimization, controlled reoxygenation, early percutaneous coronary intervention, and glycemic control.[114,118,149–151] Although these studies demonstrated feasibility and promising trends toward benefit, no conclusive superiority of such an approach to PCA care has been demonstrated in humans compared to historic controls, and there have been no clinical studies of bundle therapies in veterinary medicine. In 1 canine cardiac arrest study, the combination of MTH (34.2°C compared to 37.6°C), hemodilution (PCV 31% versus 41%), and normocapnia (36 versus 30 mm Hg) was associated with significantly reduced neurologic deficits and histopathologic evidence of neuronal injury.[124] Applying such a bundle to veterinary PCA care is reasonable (IIa-B). In addition, controlled reoxygenation and goal-oriented hemodynamic optimization with possible inclusion of early hypertension, could be considered as additional bundle components (IIb-B). The RECOVER PCA care algorithm (Figure 2) suggests such a bundle of care, including respiratory optimization, hemodynamic optimization, and neuroprotective interventions, but its efficacy remains to be demonstrated.

### Referral center care

There is some evidence in the human literature suggesting that intensivist-led human ICUs achieve better outcomes.[152] However, there have been no clinical trials comparing outcomes during the PCA period in higher level care centers to those from centers lacking advanced care capabilities. Nevertheless, given the higher likelihood of availability of 24-hour care, intensive monitoring, and advanced therapeutics as described above in a specialty facility, referral of critically ill dogs and cats for PCA care to such facilities is reasonable (IIa-B).

## *Discussion*

This manuscript represents the culmination of the efforts of over 100 veterinary specialists tasked with developing a set of evidence-based, consensus guidelines for the clinical practice of CPR in dogs and cats. While this review resulted in the development of 101 individual clinical recommendations (see Appendix I for a complete list), careful review of the class and level descriptors for these recommendations makes it clear that the veterinary profession has much work to do. This should not be viewed as a failing of this endeavor, as identification of knowledge gaps was one of the primary goals of the RECOVER initiative, and it is our hope and expectation that the veterinary community will rise to this call to address these gaps.

The guidelines contained in this summary are the result of a consensus process. They were developed in the Fall of 2011 after completion of an evidence worksheet process, were announced and introduced at the 2011 International Veterinary Emergency and Critical Care Symposium (IVECCS) meeting, and published on the internet for public comment for a period of 4 weeks.[10] A few of the guidelines were clarified as a result of this process, but they remained largely unchanged, a testament to the thorough work and dedication of the worksheet authors. It should be noted that consensus does not imply that all evaluators fully agreed with the final guidelines, but that these guidelines and their assigned classes and levels represent compromise positions that all interested

parties could "live with." As such, we believe strongly that these guidelines represent a standard for veterinary CPR that all veterinary health care professionals should strive to master, much as the AHA Guidelines serve as the standard in human CPR. We are optimistic that, as with the development of the AHA Guidelines, providing a standard for veterinary CPR practice will lead to improved outcomes for our patients. However, the availability of guidelines is only the first step. Development of standardized training tools, evaluation of outcomes, and scientific investigation to address the many knowledge gaps identified are essential as well. As a profession, it is incumbent upon us to strive to monitor the effectiveness of this approach and to continue to refine it.

This set of guidelines should be viewed as a first step in a continuing endeavor to improve and extend our approach to CPR in veterinary medicine. It is the intention of the organizers of the RECOVER initiative that this serve as an initial foundation for veterinary CPR practice and training. A thorough review of the guidelines and the 5 domain evidence reviews in this issue of JVECC will make it clear that, like all foundations, it will require much work to maintain, strengthen, and replace the occasional misplaced or defective components. But it is our hope that it provides a solid base on which we, as a profession, can continue to build, improve, and refine our approach to CPR to better serve our patients and clients. Continued meticulous inspection of this product, vigorous debate about the conclusions drawn, and scientific investigation to identify its weaknesses and limitations are our greatest hope. We look forward to RECOVER 2017 and the opportunity to recognize the progress we are confident will be made in the next 5 years. We hope that this initial step will serve as a guide map to future research and as a means of documenting the progress to come.

### *Acknowledgments*

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### *Appendix I*

RECOVER clinical guidelines. Class and level definitions are contained in Tables 1 and 2, respectively. BLS, basic life support; CPA, cardiopulmonary arrest; IV, intravenous; IO, intraosseus; IT, intratracheal; PCA, postcardiac arrest; PEA, pulseless electrical activity; VF, ventricular fibrillation; VT, ventricular tachycardia.

| Issue | Guideline | Class-level | Worksheets |
|---|---|---|---|
| **Preparedness and prevention** | | | |
| Crash cart | Standardization and regular audit of the location, storage, and content of resuscitation equipment is recommended. | I-A | PRE01 |
| Cognitive aids | Immediate availability of cognitive aids describing the standard CPR algorithms (eg, display of algorithm and dosing charts, carrying procedural CPR checklists) is recommended. | I-B | PRE01 |
| Anesthesia-related arrests | The evidence supports that in anesthetized patients with CPA, prompt CPR should be attempted considering that these patients have a better prognosis for survival (47%) and discharge from a hospital than the overall CPR survival rate (4–9.6%). | I-B | PRE02 |
| CPR training | CPR training should include both didactic components to teach cognitive skills and high-fidelity simulation technologies that provide immediate feedback to teach psychomotor skills. | I-A | PRE03<br>PRE07 |

| Issue | Guideline | Class-level | Worksheets |
|---|---|---|---|
| | Regardless of initial training technology, refresher training every 6 months is recommended due to decay of skills. | I-A | PRE03<br>PRE07 |
| | Structured assessment after CPR training is recommended. | I-A | PRE10 |
| Leadership | Veterinarians or technicians may be considered as leaders of a CPR team. | IIb-B | PRE04<br>PRE05 |
| | Specific leadership training is recommended for individuals who may need to lead in a CPA situation. | I-A | PRE04<br>PRE05 |
| Debriefing | Debriefing after a resuscitation effort to review and critique the procedure is recommended. | I-A | PRE09 |
| **Basic life support** | | | |
| Chest compressions | In dogs and cats, chest compressions should be done in lateral recumbency. | I-B | BLS02<br>BLS06 |
| | In dogs and cats, chest compression depth of between 1/3 and 1/2 the width of the chest is reasonable. | IIa-A | BLS02 |
| | In large and giant breed dogs, chest compressions with the hands placed over the widest portion of the chest is reasonable. | IIa-C | BLS05B<br>BLS06 |
| | In keel-chested dogs, performing chest compressions with the hands directly over the heart is reasonable. | IIa-C | BLS05B<br>BLS06 |
| | In barrel-chested dogs, sternal chest compressions in dorsal recumbency may be considered. | IIb-C | BLS05B<br>BLS06 |
| | In cats and small dogs, circumferential compressions rather than lateral compressions may be considered. | IIb-C | BLS05A |
| | Chest compression rate of 100–120 compressions/min are recommended for both dogs and cats, independent of size. | I-A | BLS07 |
| | Allowing full chest wall recoil between compressions and avoiding leaning on the chest during recoil are recommended. | I-A | BLS08 |
| Ventilation | In nonintubated dogs and cats or single-rescuer CPR, a C:V ratio of 30:2 is recommended. | I-B | BLS03 |
| | In intubated, multiple-rescuer CPR, continuous chest compressions with simultaneous ventilation are recommended. | I-A | BLS03 |
| | Ventilation of dogs and cats with CPA at a rate of 10 breaths per minute with a tidal volume of 10 mL/kg and an inspiratory time of 1 sec is recommended. | I-A | BLS14<br>BLS15 |
| Cycles of CPR | Rotation of chest compressors every 2 minutes is recommended to reduce lean and compromise of compression efficacy due to fatigue. | I-B | BLS12<br>BLS18 |
| | It is recommended that CPR be performed in 2-minute cycles without interruption, and duration of pauses between cycles minimized. | I-A | BLS12 |
| Delay in starting CPR | Aggressive administration of CPR in patients suspected of being in CPA is recommended, as the risk of injury due to CPR in patients not in CPA is low. | I-B | BLS11 |
| Interposed abdominal compressions | The use of interposed abdominal compressions in dogs and cats with CPA is reasonable when sufficient personnel trained in its use are available. | IIa-B | BLS09 |
| **Advanced life support** | | | |
| Epinephrine | The use of low dose (0.01 mg/kg) epinephrine administered every 3–5 minutes early in CPR is recommended. | I-B | ALS01 |
| | The use of high dose (0.1 mg/kg) epinephrine may be considered after prolonged CPR. | IIb-B | ALS01 |
| Atropine | In dogs and cats with asystole or PEA potentially associated with increased vagal tone, use of atropine is reasonable. | IIa-B | ALS02 |
| | In dogs and cats, routine use of atropine during CPR may be considered. | IIb-C | ALS02 |
| Vasopressin | The use of vasopressin (0.8 U/kg) as a substitute or in combination with epinephrine every 3–5 minutes may be considered. | IIb-B | ALS03 |
| Defibrillation | The use of a biphasic defibrillator is recommended over a monophasic defibrillator. | I-A | ALS05 |

| Issue | Guideline | Class-level | Worksheets |
|---|---|---|---|
| | External defibrillation dosing should start at 4–6 J/kg with a monophasic defibrillator and 2–4 J/kg with a biphasic defibrillator. | IIa-B | ALS05 |
| | Administration of a single shock as opposed to 3 stacked shocks is recommended, with immediate resumption of CPR in the case of nonsuccessful defibrillation. | I-B | ALS05 |
| | Defibrillation for treatment of VF/pulseless VT is recommended over routine use of antiarrhythmic drugs. | I-B | ALS07 |
| | Immediate defibrillation is recommended in cases of CPA due to VF of duration of 4 minutes or less. | I-B | ALS08 |
| | Immediate defibrillation may be considered if VF is diagnosed during a rhythm check between cycles of CPR. | IIb-B | ALS08 |
| | A 2-minute cycle of CPR should precede defibrillation in cases of CPA due to VF of known or suspected duration of greater than 4 minutes. | I-B | ALS08 |
| | In dogs and cats with VF, defibrillation energy escalation is reasonable if the first countershock is unsuccessful. | IIa-B | ALS15 |
| Amiodarone | Amiodarone may be considered in cases of pulseless VT/VF resistant to defibrillation. | IIb-B | ALS07 |
| | When amiodarone is not available, lidocaine may be considered in cases of pulseless VT/VF resistant to defibrillation. | IIb-B | ALS07 |
| Magnesium | Routine use of $MgSO_4$ is not recommended for cardiac arrythmias, but may be considered for treatment of torsades de pointes. | IIb-B | ALS07 |
| Impedance threshold device | Use of an impedance threshold device to enhance circulation is reasonable in animals $>$10 kg. | IIa-B | ALS10 |
| Corticosteroids | The routine use of corticosteroids during CPR is not recommended. | III-C | ALS11 |
| Reversal agents | In dogs and cats that have received reversible anesthetic/sedative medication, administering reversal agents during CPR may be considered. | IIb-C | ALS13 |
| Naloxone | In cases of opioid toxicity, naloxone should be used during CPR. | I-B | ALS13 |
| | In cases of recent opioid administration, the use of naloxone during CPR may be considered. | IIb-B | ALS13 |
| Calcium | The routine use of intravenous calcium in dogs and cats during CPR is not recommended. | III-B | ALS12<br>ALS14 |
| | Intravenous calcium may be considered in dogs and cats with documented moderate hypocalcemia during CPR. | IIb-C | ALS12<br>ALS14 |
| Potassium | Documented hyperkalemia should be treated during CPR. | I-B | ALS12 |
| | Treatment of documented hypokalemia during CPR may be considered. | IIb-C | ALS12 |
| Intratracheal administration | In animals in which intravenous or intraosseous access is not possible, the use of the intratracheal route for epinephrine, vasopressin, or atropine may be considered. | IIb-B | ALS09 |
| | If the intratracheal route is used for drug administration during CPR, drugs should be diluted with saline and administered via a catheter longer than the endotracheal tube. | I-B | ALS09 |
| Supplemental oxygen administration | During CPR in dogs and cats, the use of an $FiO_2$ of 100% is reasonable. | IIa-B | ALS-CPR-A-011A (ILCOR) PA08 |
| | During CPR in dogs and cats, the use of an $FiO_2$ of 21% (room air) may be considered. | IIb-B | ALS-CPR-A-011A (ILCOR) PA08 |
| IV fluid administration | During CPR in euvolemic or hypervolemic dogs and cats, routine administration of intravenous fluids is not recommended. | III-B | ALS-D-016A (ILCOR) |
| | During CPR in dogs and cats with documented or suspected preexisting hypovolemia, administration of intravenous fluids is reasonable. | IIa-C | ALS-D-016A (ILCOR) |
| Alkalinization therapy | Alkalinization therapy after prolonged CPA of greater than 10–15 minutes with administration of 1 mEq/kg of sodium bicarbonate may be considered. | IIb-B | ALS16 |
| Open-chest CPR | In cases of significant intrathoracic disease, such as tension pneumothorax or pericardial effusion, promptly performing open-chest CPR may be considered. | IIb-C | ALS06 |

| Issue | Guideline | Class-level | Worksheets |
|---|---|---|---|
| **Monitoring** | | | |
| $EtCO_2$ | In intubated and ventilated dogs and cats, the use of $EtCO_2$ is recommended in patients at risk of CPA. | I-A | MON02 |
| | The immediate postintubation $EtCO_2$ should not be used for diagnosis of CPA in dogs and cats. | III-B | MON02 |
| | The use of $EtCO_2$ alone for verification of correct ET tube placement in dogs and cats with CPA is not recommended. | III-B | MON06 |
| | The use of $EtCO_2$ monitoring as an adjunct measure with direct visualization, auscultation, or observation of chest excursions in dogs and cats with CPA to verify correct ET tube placement is reasonable. | IIa-B | MON06 |
| | The use of $EtCO_2$ monitoring during CPR is recommended as an early indicator of ROSC. | I-A | MON10<br>MON15<br>MON22A/B |
| | The use of $EtCO_2$ monitoring as a measure of efficacy of CPR in conditions of consistent minute ventilation is reasonable. | IIa-B | MON15<br>MON23 |
| VF waveform analysis | The use of VF waveform analysis in dogs and cats with CPA may be considered, with coarse VF potentially associated with a better prognosis for ROSC than fine VF. | IIb-B | MON24 |
| Pulse palpation | In unresponsive, apneic dogs and cats, the use of pulse palpation to support a diagnosis of CPA before initiating CPR is not recommended. | III-B | MON03 |
| | Interruption of chest compressions specifically to palpate the pulse or check the ECG is not recommended. | III-B | MON11<br>MON12<br>MON14 |
| | Palpation of the pulse for detection of ROSC during intercycle pauses in CPR is reasonable, but should not delay resumption of chest compressions. | IIb-C | MON11<br>MON12<br>MON14 |
| Doppler blood pressure | In unresponsive, apneic dogs and cats, the use of Doppler to support a diagnosis of CPA before initiating CPR is not recommended. | III-C | MON04 |
| | In dogs and cats at risk of CPA, the use of continuous Doppler monitoring of peripheral arterial blood flow for early identification of CPA is reasonable. | IIa-C | MON04 |
| ECG | In unresponsive, apneic dogs and cats, the use of ECG to support a diagnosis of CPA before initiating CPR is not recommended. | III-B | MON05 |
| | In dogs and cats at risk of CPA, the use of continuous ECG monitoring for early identification of rhythm changes suggestive of CPA is reasonable. | IIa-C | MON05 |
| | Evaluation of the ECG during intercycle pauses in CPR is recommended, but should not delay resumption of chest compressions. | I-C | MON11<br>MON12<br>MON14 |
| | Rapid assessment of the ECG to determine if VF has resolved immediately after defibrillation is reasonable, but should minimally delay resumption of chest compressions. | IIa-B | MON12 |
| Feedback devices | The use of prompting or feedback devices to improve quality of CPR is reasonable. | IIa-C | MON16 |
| Blood gases | In dogs and cats with CPA, the use of central/mixed venous blood gases to evaluate effectiveness of CPR may be considered. | IIb-B | MON20 |
| | In dogs and cats with CPA, arterial blood gases are not recommended for evaluation of effectiveness of CPR. | III-A | MON20 |
| Electrolytes | Routine monitoring of electrolytes during CPR may be considered. | IIb-B | MON21 |
| | In the case of CPA known or suspected to be due to underlying electrolyte derangements, electrolytes should be monitored during CPR to inform therapeutic decisions. | I-C | MON21 |
| Postresuscitation monitoring | Postresuscitation monitoring should be sufficient to detect impending reoccurrence of CPA. | I-C | MON25 |
| | Postresuscitation monitoring should be sufficient to guide therapy appropriate for the patient's condition. | I-C | MON25 |

| Issue | Guideline | Class-level | Worksheets |
|---|---|---|---|
| | Minimum postresuscitation monitoring should include continuous ECG, intermittent arterial blood pressure monitoring, and assessment of oxygenation and ventilation. | I-B | MON25 |
| | Postresuscitation monitoring of glucose, body temperature, and blood lactate may be considered. | IIb-B | MON25 |
| **Post-cardiac arrest care** | | | |
| IV fluids | The routine use of large volumes of intraveous fluid post-arrest is not recommended except in the case of strongly suspected or confirmed hypovolemia. | III-C | PA01 |
| | IV fluids should be avoided in dogs and cats post-arrest with evidence of congestive heart failure. | III-C | PA01 |
| Goal-directed therapy | In hemodynamically unstable dogs and cats with ROSC after CPA, a hemodynamic optimization strategy that includes primary resuscitation endpoints of central venous $O_2$ saturation or lactate, and secondary endpoints including arterial blood pressure, central venous pressure, PCV, and arterial oxygen saturation may be considered. | IIb-B | PA02 |
| Vasopressors/ inotropes | The use of vasopressors and/or positive inotropes in dogs and cats with persistent hypotension/cardiovascular instability post-arrest is reasonable. | IIa-B | PA03 |
| Ventilation | In dogs and cats post-arrest, routine mechanical ventilation is not recommended. | III-B | PA06 |
| | In dogs and cats post-arrest that are hypoventilating or at risk of respiratory arrest, IPPV (manual or mechanical) is reasonable. | IIa-C | PA06 |
| | Post-arrest, a target $PaCO_2$ of 32–43 mm Hg in dogs and 26–36 mm Hg in cats is reasonable. | IIa-B | PA06 |
| Oxygenation | In dogs and cats after ROSC, inspired oxygen should be titrated to maintain normoxia ($PaO_2 = 80$–$100$ mm Hg, $SpO_2 = 94$–$98\%$); hypoxemia and hyperoxemia should be avoided. | I-A | PA08 |
| Hypothermia | In hypothermic dogs and cats post-arrest, slow rewarming at a rate of 0.25–0.5 °C/h is reasonable. | IIa-A | PA10 |
| | In hypothermic dogs and cats post-arrest, fast rewarming at a rate >1 °C/h is not recommended. | III-A | PA10 |
| | In dogs and cats that remain comatose after successful resuscitation from cardiac arrest, mild therapeutic hypothermia (32–34 °C) for 24–48 hours initiated as soon as possible after ROSC is recommended, if mechanical ventilation and advanced critical care capability is available. | I-A | PA11 |
| | In the absence facilities for mechanical ventilation and advanced critical care infrastructure, mild hypothermia should not be initiated. | III-C | PA12 |
| Corticosteroids | Routine administration of corticosteroids to cats or dogs after successful resuscitation from cardiac arrest is not recommended. | III-C | PA13 |
| | Administration of hydrocortisone (1 mg/kg followed by either 1 mg/kg q 6 h or an infusion of 0.15 mg/kg/h and then tapered a the patient's condition allows) to cats or dogs that remain hemodynamically unstable despite administration of fluids and inotropes/pressors may be considered. | IIb-C | PA13 |
| Bundle of care | Induction of mild hypothermia (34 °C) for 12 hours post-resuscitation, normocapnia (35–40 mmHg) for 24 hours, and sustained hypertension (140 mmHg, mean) for 4 hours after successful ROSC is reasonable. | IIa-B | PA19 |
| Level of care | For dogs and cats successfully resuscitated after CPA, referral to a specialty center with 24 hour care, higher healthcare provider:patient ratios, and advanced critical care capabilities is reasonable. | IIa-B | PA20 |
| Hypertension | It is reasonable to tolerate hypertension in the immediate postarrest period in dogs and cats. | IIa-B | PA04 |
| Seizure prophylaxis | Seizure prophylaxis with barbiturates may be considered in dogs and cats postcardiac arrest. | IIb-B | PA14 |
| Osmotic agents | In dogs and cats with neurologic signs consistent with cerebral edema (eg, coma, cranial nerve deficits, decerebrate postures, abnormal mentation), mannitol (0.5 g/kg) or hypertonic saline (2–4 mL/kg of the 7% solution) may be considered. | IIb-C | PA15 |

## *Appendix II*

CPR drug doses. BLS, basic life support; CPA, cardiopulmonary arrest; CRI, constant rate infusion; IV, intravenous; IO, intraosseus; IT, intratracheal; PCA, postcardiac arrest; PEA, pulseless electrical activity; VF, ventricular fibrillation; VT, ventricular tachycardia.

| | Drug | Common concentration | Dose/route | Comments |
|---|---|---|---|---|
| Arrest | Epinephrine (low dose) | 1 mg/mL (1:1000) | 0.01 mg/kg IV/IO<br>0.02–0.1 mg/kg IT | Administer every other BLS cycle for asystole/PEA.<br>Consider increasing dose 2–10× and diluting with saline or sterile water for IT administration |
| | Epinephrine (high dose) | 1 mg/mL (1:1000) | 0.1 mg/kg IV/IO/IT | Start with low dose.<br>Consider high dose for prolonged (>10 min) CPR |
| | Vasopressin | 20 U/mL | 0.8 U/kg IV/IO<br>1.2 U/kg IT | Administer every other BLS cycle.<br>Increase dose for IT use. |
| | Atropine | 0.54 mg/mL | 0.04 mg/kg IV/IO<br>0.15–0.2 mg/kg IT | May repeat every other BLS cycle during CPR.<br>Recommended in animals with bradycardic arrests and/or known or suspected high vagal tone.<br>Increase dose for IT use. |
| | Bicarbonate | 1 mEq/mL | 1 mEq/kg IV/IO | For prolonged (>10–15 min) CPR or in PCA phase to treat severe metabolic acidosis.<br>Contraindicated if patient is hypoventilating. |
| Antiarrhythmic | Amiodarone | 50 mg/mL | 5 mg/kg IV/IO | Use for refractory VF/pulseless VT.<br>Has been associated with allergic reactions/hypotension in dogs. |
| | Lidocaine | 20 mg/mL | 2 mg/kg slow IV/IO push (1–2 min) | Use for refractory VF/pulseless VT *only* if amiodarone is not available. |
| Reversals | Naloxone | 0.4 mg/mL | 0.04 mg/kg IV/IO | To reverse opioids |
| | Flumazenil | 0.1 mg/mL | 0.01 mg/kg IV/IO | To reverse benzodiazepines |
| | Atipamezole | 5 mg/mL | 100 μg/kg IV/IO | To reverse α2 agonists.<br>Note that this dose is based on a 10 μg/kg dexmedetomidine dose. If a higher dose of dexmedetomidine was administered, increase this dose accordingly. |
| Defibrillation | Monophasic external | | 4–6 J/kg | May increase dose once by 50–100% for refractory VF/pulseless VT. |
| | Monophasic internal | | 0.5–1 J/kg | May increase dose once by 50–100% for refractory VF/pulseless VT. |
| | Biphasic external | | 2–4 J/kg | May increase dose once by 50–100% for refractory VF/pulseless VT. |
| | Biphasic internal | | 0.2–0.4 J/kg | May increase dose once by 50–100% for refractory VF/pulseless VT. |
| Postarrest | Mannitol | 25% | 0.5 g/kg IV/IO over 15–20 minutes | Use in the PCA period for animals with evidence of cerebral edema (eg, abnormal mentation, cranial nerve deficits, abnormal postures). |
| | Hypertonic saline (7.2%) | 7.2% | 4 mL/kg (dog)<br>2 mL/kg (cat) IV/IO over 15–20 minutes | Use in the PCA period for animals with evidence of cerebral edema (eg, abnormal mentation, cranial nerve deficits, abnormal postures). |
| | Norepinephrine | 1 mg/mL | 0.05 – 0.1 μg/kg/min IV CRI | α1 specific adrenergic agonist.<br>Use for PCA hypotension due to vasodilation. |
| | Vasopressin | 20 U/mL | 0.5–5.0 mU/kg/min IV CRI | Nonadrenergic vasoconstrictor that acts via peripheral V1 receptors.<br>Use for PCA hypotension due to vasodilation. |
| | Dopamine | 40 mg/mL | 5–10 μg/kg/min IV CRI (β1 effects)<br>10–15 μg/kg/min IV CRI (α1 and β1 effects) | Non-specific adrenergic agonist.<br>Use for PCA hypotension due to poor cardiac contractility and/or vasodilation. |
| | Dobutamine | 12.5 mg/mL | 1–20 μg/kg/min IV CRI | β1 specific adrenergic agonist.<br>Use for PCA hypotension due to poor cardiac contractility.<br>Can cause seizures in cats. |

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