Clinical Practice Review

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Cardiopulmonary cerebral resuscitation in small animals—a clinical practice review. Part II

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Abstract

Objective: To review the principles and practice of cardiopulmonary cerebral resuscitation (CPCR) in veterinary medicine, and to incorporate recommendations from the human International Guidelines 2000 Conference on cardiopulmonary resuscitation and emergency cardiovascular care.
Data sources: Both clinical and experimental studies from the human and veterinary literature were reviewed for this manuscript.
Summary: Cardiopulmonary cerebral resuscitation consists of basic life support measures, advanced life support measures, and post-resuscitation care. Part I of this article introduced the evidence-based recommendations from human medicine and reviewed basic life support. Part II of this article reviews advanced life support, including drug therapy and electrical defibrillation based upon the electrocardiographic rhythm present at the time of cardiopulmonary arrest (CPA). Post-resuscitation care is discussed, with a particular focus on optimizing perfusion to the brain, kidneys, and gastrointestinal tract. Several currently investigational methods that may improve future patient outcomes are also addressed.
Conclusions: Advanced life support techniques provide methods to augment CPCR efforts. As with basic life support, recent recommendations to improve advanced life support in humans may be also be applied to veterinary patients. However, clinical research evaluating these interventions in veterinary CPCR is necessary. Post-resuscitation care requires vigilant monitoring and aggressive support to ensure vital organ perfusion and maximize patient outcomes.

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Introduction

Cardiopulmonary cerebral resuscitation (CPCR) is the combination of basic life support and advanced life support techniques, as well as post-resuscitation care. In human medicine, international standards for the performance of CPCR have been created, with the most recent guidelines being published in 2000.[1] The 2000 Guidelines were introduced in Part I of this article and their application to basic life support in veterinary patients was considered. The following discussion will focus on advanced life support and post-resuscitation care of patients suffering cardiopulmonary arrest (CPA). As before, potential applications of the human guidelines to veterinary CPCR will be discussed.

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Advanced life support

Advanced life support (ALS) includes determining the electrocardiographic rhythm associated with CPA and implementing specific treatment including drug therapy, electrical defibrillation, or cardiac pacing. These additional interventions augment basic life support techniques to increase the likelihood of successful resuscitation. Advanced life support requires additional

resources, and specially trained personnel. Staff must be well practiced in performing CPCR and all equipment must be readily available in one place, such as an arrest station with a crash cart. Training drills, and critically evaluating all resuscitation efforts are important to achieving successful outcomes, as CPCR techniques are learned more effectively when practiced.[1]

Determining the arrest rhythm

The number of electrocardiographic rhythms associated with CPA in small animal patients is limited. In almost all situations, the ECG will reveal sinus bradycardia, pulseless electrical activity (PEA), ventricular fibrillation (VF), or asystole. In a study of 232 cases of cardiopulmonary and respiratory arrest in small animals, electromechanical dissociation (now known as PEA) was the most common initial arrest rhythm, seen in 23.3% of cases. This was followed by asystole (22.8%), VF (19.8%) and sinus bradycardia (19.0%).[2] In comparison, VF is seen in approximately 35–60% of out of hospital CPA in human patients,[3,4] while people suffering CPA in the hospital have a much higher incidence of asystole, bradydysrhythmias, and PEA,[5] which are all rhythms that have been associated with decreased survival to discharge in these patients.[6]

Defibrillation

Rapid application of electrical defibrillation is the only effective method for converting VF to a perfusing rhythm, and the amount of time elapsed between the onset of VF and the application of countershocks is inversely correlated with survival.[1,5] A recent retrospective human study showed a 5.5% decline in survival with each minute elapsed from the time of collapse until the delivery of electrical countershocks and definitive care.[7] Because of this, in humans where VF is the initial arrest rhythm, defibrillation is recommended prior to drug therapy. Other advanced life support measures are used only after defibrillation has failed.[1,5]

As with humans, the most common way to achieve ROSC in veterinary patients is through successful defibrillation. In addition to the approximately 20% of small animal patients that have VF as the initial arrest rhythm, many animals develop VF during resuscitation (Figure 1). Accordingly, electrical countershocks should be applied without delay as soon as VF is recognized. Countershocks may be delivered externally or internally, depending upon the type of CPCR (closed *versus* open chest) being performed.

Several external defibrillation techniques can be used in small animal patients. Standard hand-held defibrillator paddles can be applied to each side of the chest with the animal in dorsal recumbency, however, this can be dangerous as the patient's legs may touch the operator during defibrillation. It is safer to place a flat paddle under the animal in lateral recumbency and a standard paddle on the upper side. Newer defibrillators have adhesive patch electrodes and remote triggering, although this requires clipping of the fur and may not be practical in emergent situations. Countershocks are applied across the chest wall, and 3 shocks are delivered in rapid succession. The energy of the initial countershock is 3–5 J/kg, and the energy of the second and third shocks are increased by approximately 50% each. Pauses should only be taken to recharge the defibrillator and reevaluate the ECG. Countershocks are discontinued if the rhythm converts from VF. If defibrillation is not successful, CPCR is resumed for 60–90 seconds,[1] and the next series of countershocks is given at twice the initial energy (5–10 J/kg). Inefficient 'arcing' of the current, usually evidenced by a burning smell, is prevented by using a copious amount of electrode gel and/or clipping hair to assure good skin contact. Excessive amounts of alcohol on the ECG leads may increase the likelihood of fire, especially in the presence of 100% oxygen.

Internal defibrillation is accomplished during open chest CPCR using special paddles. Saline-soaked sponges are placed between the paddles and the epicardium, and 3 shocks of 0.5–1 J/kg are given, using the same countershock protocol as previously described. If internal paddles are not available or if they cannot be quickly attached to the defibrillator, external defibrillation can still be successfully employed during open chest CPCR.

Hemodynamically unstable ventricular tachycardia (VT) may also be terminated by electrical countershock (cardioversion). Although VT is an extremely rare arrest rhythm in veterinary medicine, it may be seen in animals prone to malignant dysrhythmias, such as Boxer dogs with arrhythmogenic cardiomyopathy. In these situations, it is recommended that cardioversion be attempted prior to drug therapy.[1] It is also important that the delivered energy is synchronized with the QRS complex to avoid giving the countershock during the T-wave, which may result in VF.[1] Many newer defibrillators have the ability to achieve this synchronization. The energy required to convert VT is approximately 50% less than that required to convert VF.[1]

Transthoracic pacing

A feature common to many newer defibrillators is the ability to perform transthoracic (or transcutaneous) cardiac pacing, where a current is applied across the chest wall to 'capture' the electrical activity of the heart.

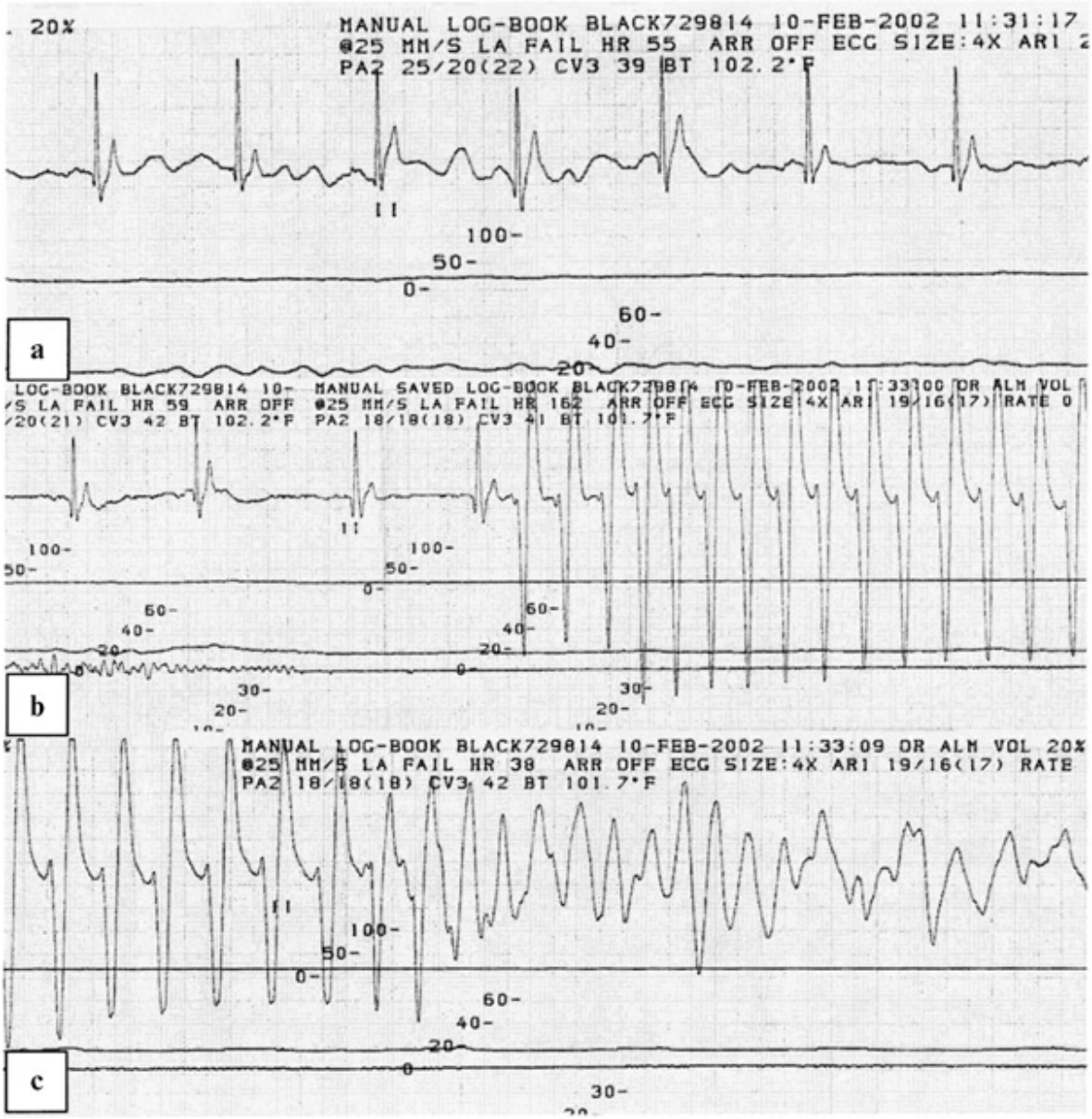

Figure 1: ECG strips from a dog suffering CPA. Systemic and pulmonary arterial tracings appear below the ECG in all panels. (a) Slow pulseless electrical activity (PEA). (b) Pulseless electrical activity progressing to ventricular tachycardia (VT). (c) Ventricular tachycardia progressing to ventricular fibrillation (VF). Note the lack of functional mechanical activity demonstrated by the absence of pressure waveforms in the systemic arterial and pulmonary arterial tracings.

Transthoracic pacing is included in the American Heart Association guidelines for the treatment of pulseless electrical activity and asystole, rhythms that are not usually responsive to electrical countershock.[1] Pacing is attempted early in the course of resuscitation, prior to drug therapy, and is used in conjunction with basic life support measures.[1] When used with appropriate sedation or anesthesia, the most common indication for transthoracic pacing is in the temporary treatment of symptomatic 3°AV block prior to surgical pacemaker implantation.[1,8]

Establishing access for fluid and drug therapy

Establishing intravenous access is vital in CPCR. A central catheter is best for drug delivery, as circulation time is decreased compared to peripheral delivery.[9] However, peripheral vascular, intraosseous, or intratracheal routes may also be used. The choice to use a particular route depends upon the individual animal and situation. Surgical cutdown to the jugular, cephalic or saphenous vein should be performed if percutaneous venous access cannot be immediately achieved. If peripheral vessels

are used, drugs should be flushed into the central circulation using volumes of 5–50 mL depending upon patient size. Intraosseous access is particularly useful in puppies, kittens and exotic species. Sites for intraosseous access include the trochanteric fossa of the femur, the proximal humerus, or the tibial crest.[10] Where other routes of parenteral access are not possible, medication may be given by intratracheal instillation. Drugs that may be delivered by this method include atropine, epinephrine, lidocaine, and vasopressin. The dosage is increased 2–2.5 times,[1] and the drug is diluted with sterile saline or sterile water (to a volume of 5–6 mL^3) in order to facilitate absorption.[11,12] It should be noted that if epinephrine is to be given intratracheally, high-dose (0.1–0.2 mg/kg) calculations should be used, as experimental evidence has shown 0.13 mg/kg to be the median effective intratracheal dose in dogs.[13] The drug is administered via a red-rubber catheter advanced through the endotracheal tube to the carina. Two full breaths are then given to distribute the medication. Finally, intracardiac injection of medication should be avoided, especially during closed-chest CPCR, where inaccurate injection is common,[14] and complications such as pneumothorax, hemopericardium, or intractable arrhythmias have been seen in human patients.[15]

Drug and fluid therapy

There has been a vast amount of research into drug and fluid therapy during CPCR. Much of this research has evaluated interventions with respect to enhancing myocardial and cerebral perfusion, and improving outcome following resuscitation. However, consistent benefits of pharmacologic therapy are sorely lacking in clinical situations.[16] Accordingly, there are no drugs that received a Class I recommendation in the 2000 Guidelines, with the exception of atropine as a Class I agent for bradycardia. The mainstays of drug treatment listed in the guidelines remain vasopressors, vagolytics, and antiarrhythmics, with recent changes only in the recommendations of specific agents and doses.[1]

Intravenous fluid therapy

The use of shock doses of intravenous fluid has been advocated as a standard practice in veterinary CPCR.[17] While this is certainly appropriate in the face of marked hypovolemia, volume loading of euvolemic patients may actually cause decreases in myocardial and cerebral perfusion, a phenomenon that occurs despite increased forward blood flow.[18] Elevation of right atrial pressure secondary to fluid therapy decreases myocardial perfusion pressure by reducing the aortic diastolic to right atrial pressure gradient. Aggressive intravenous fluid infusion may also increase intracranial pressure and decrease cerebral perfusion pressure if the rise in intracranial pressure is greater than the rise in mean arterial pressure. High-fluid rates should therefore be reserved for animals with pre-existing hypovolemia or significant ongoing losses.

Vasopressor therapy

The aim of administering adrenergic or non-adrenergic agents during CPCR is to increase aortic mean and diastolic pressure, thereby augmenting cerebral and myocardial perfusion pressure. Epinephrine has long been the vasopressor of choice in CPCR and is indicated in the management of asystole, PEA, refractory VF or pulseless VT. Although epinephrine possesses both α-adrenergic and β-adrenergic activity, it is the alpha (vasoconstrictor) effects that are of primary importance during resuscitation.[1,19,20] Potentially deleterious beta effects include peripheral vasodilation, chronotropy, and inotropy that may increase cardiac work and myocardial oxygen demand.[18,19,21–23] Both low dose (0.01–0.02 mg/kg) and high-dose (0.1–0.2 mg/kg) protocols have been suggested for use in CPCR. In human patients, high-dose epinephrine has been associated with increases in ROSC and early survival. However, no long-term benefit of high-dose epinephrine has been documented in humans, and high-dose epinephrine may be associated with myocardial dysfunction and worse neurologic outcomes.[1]

The 2000 Guidelines recommend the use of low dose epinephrine (Class Indeterminate), and the use of high-dose epinephrine is deemed acceptable in ongoing resuscitation efforts following lack of response to lower doses.[1] In all cases, doses of epinephrine are repeated at 3–5 minute intervals following an initial dose. Because there is no data concerning the impact of low *versus* high-dose epinephrine in the outcome of veterinary patients, the relevance of this recommendation to veterinary CPCR is unclear. Given experimental models that document shorter resuscitation times in dogs receiving higher doses of epinephrine,[24] initial use of high-dose epinephrine should not be discounted, although adverse effects must also be considered.

Because of concerns about potential adverse effects of adrenergic agents in CPCR, several non-adrenergic agents have been investigated. One of the most promising non-adrenergic drugs is vasopressin[a] (Pitressin, Monarch Pharmaceuticals, Bristol, TN). It is a potent vasoconstrictor when used at pharmacologic doses and achieves its effects via direct stimulation of specific vasopressin (V1) receptors in vascular smooth muscle.[1] Unlike catecholamines, this response is not blunted in the face of acidosis. Additionally, because it lacks β-adrenergic activity, vasopressin is not associated with increased myocardial oxygen demand following ROSC.[1]

In concordance with promising results in animal models, early clinical investigation in human CPA victims indicates that vasopressin is comparable, if not superior to epinephrine in achieving ROSC. These benefits may be most pronounced in prolonged arrest.[1] Vasopressin is considered an alternative first-line (Class IIb) vasopressor in shock-refractory VF or pulseless VT. Vasopressin is also regarded as a Class Indeterminate (not recommended, not forbidden) vasopressor for asystole and PEA.[1] Given once, at a dose of 0.8 u/kg IV, vasopressin may also be considered for veterinary CPCR in these same situations.

Vagolytic therapy

Because of potential parasympathetic contribution to the development of PEA or asystole, the 2000 Guidelines recommend atropine (given at dosages up to 0.04 mg/kg) in these circumstances.[1] Combined with sinus bradycardia, these rhythms are seen in nearly 70% of CPA in small animal patients.[2] Animals suffering a vagally mediated arrest often respond to atropine and oxygen therapy alone, without chest compressions. Atropine is considered a Class I agent for sinus bradycardia, however, animals with a sinus bradycardia often require significantly lower doses of atropine (0.004–0.01 mg/kg) than are considered standard for cardiac arrest. While use of low dose atropine may be associated with a transient centrally mediated exacerbation of bradycardia,[25] using lower doses in these patients may help to avoid sustained tachycardia and increased myocardial oxygen demand after administration. Thus, the full vagolytic dose of atropine (0.04 mg/kg) is reserved solely for patients with PEA or asystolic cardiac arrest.

Antiarrhythmic therapy

There is no strong evidence supporting the effectiveness of antiarrhythmic agents in long-term survival from CPCR.[1,16] In addition, many antiarrhythmic drugs increase the threshold for defibrillation or may have undesirable pro-arrhythmic effects.[1] Because of this, the 2000 Guidelines include antiarrhythmics only in protocols for VF and pulseless VT that are refractory to electrical countershock.[1]

Lidocaine has long been a first-line drug in CPCR. However, its role in resuscitation has recently been questioned due to a lack of documented efficacy in cardiac arrest.[1,16] Because of this, lidocaine is now considered a Class Indeterminate agent for shock refractory VT/VF. A new drug, amiodarone, is now listed ahead of lidocaine as a first line (Class IIb) agent in these situations. Amiodarone[b] (Cordarone, Wyeth Laboratories, Philadelphia, PA) has been shown to increase ROSC and early survival both when compared to placebo,[26] and when compared to lidocaine,[27] in out-of-hospital, shock refractory VF in human patients. Procainamide[c] (Class IIb) is also listed as an acceptable agent, however, it is not recommended due to the necessity of slow infusion rates and uncertain efficacy in cardiac arrest.[1] Magnesium sulfate is now only recommended for patients with torsades de pointes (polymorphic VT) and in known or suspected hypomagnesemia.[1] Bretylium tosylate, which has been part of previous protocols, has been removed from the guidelines completely. This stems from a lack of availability, as well as concerns over proven efficacy and a high-occurrence of side-effects.[1]

Aside from their use in VF that is refractory to electrical defibrillation, antiarrhythmic drugs are most useful in the management of post-resuscitation arrhythmias. Agents such as lidocaine or procainamide may be useful for ventricular arrhythmias, while other rapidly acting injectable drugs such as diltiazem are useful for supraventricular arrhythmias. Drugs such as esmolol[d] or amiodarone may be effective in treating both supraventricular and ventricular arrhythmias. It is important to recognize that many animals develop a stable idioventricular rhythm or a ventricular escape rhythm upon ROSC. It should be stressed that these rhythms are not indications for antiarrhythmic therapy and that suppression of these escape rhythms may prove fatal.

Buffer therapy

Sodium bicarbonate therapy during CPCR is controversial, because of potential deleterious side effects, including hyperosmolality, hypernatremia and paradoxical CNS and intracellular acidosis. Rapid administration of sodium bicarbonate has also been shown to cause transient hypotension, a consequence of the high-osmolarity of the solution.[28] In addition, sodium bicarbonate use has been associated with decreased in-hospital resuscitation rates in people.[16] However, sodium bicarbonate is recommended in cases of severe hyperkalemia, severe pre-existing metabolic acidosis, and during prolonged CPCR (greater than 10 minutes). Sodium bicarbonate should be considered only after other interventions, including intubation, ventilation, cardiac compression, defibrillation, and drug therapy have been ineffective.[1] The currently recommended dose is 1 mEq/kg, although in cases of prolonged resuscitation, additional doses of 0.5 mEq/kg may be given at 10-minute intervals.[1] Other buffers, such as THAM,[e] Carbicarb,[f] and Tribonat®[g] have been evaluated in the setting of CPCR,[29,30] however, these agents have not been included in the 2000 Guidelines.

Electrolyte therapy

Electrolyte abnormalities are common in critically ill patients and in humans having suffered CPA.[31]

Electrolyte abnormalities are also common in critically ill veterinary patients. These abnormalities, which often include derangements in potassium, calcium, and magnesium, can negatively impact cardiovascular performance. The 2000 Guidelines do not recommend the routine use of any electrolyte therapy during CPCR. However, electrolyte supplementation is recommended when a deficiency exists. Magnesium is indicated to treat ventricular arrhythmias in patients with known or suspected hypomagnesemia, as well as for patients with polymorphic VT (torsades de pointes).[1] The use of calcium during CPCR is similarly restricted, and is recommended in patients with severe hyperkalemia, hypocalcemia, and calcium channel or β-blocker overdosage. Routine use is not recommended, as calcium may theoretically potentiate cellular damage secondary to ischemia.[1,18]

Therapy for anesthetic-related arrest

Cardiopulmonary arrest associated with general anesthesia is uncommon in veterinary patients, occurring in 0.5% of dogs and 0.4% of cats in a recent one-year period at a university teaching hospital.[32] However, anesthesia-related arrests deserve special mention, because this may be one of the more treatable causes of CPA encountered by veterinarians. In many cases, animals that receive an absolute or relative overdose of an opiod, benzodiazepine, or α_2 adrenergic agonist agent as part of an anesthetic protocol may respond to prompt administration of a specific reversal agent. These reversal agents include: naloxone[h] (an opioid antagonist) at a dose of 0.02–0.04 mg/kg IV, flumazenil[i] (a benzodiazepine antagonist) at a dose of 0.02 mg/kg IV, and yohimbine[j] or atipamezole[k] (α_2 adrenergic antagonists) at doses of 0.1–0.2 mg/kg IV slowly. In some cases, especially with opiod or benzodiazepine overdoses, additional doses of reversal agents may be required due to the short half-lives of these medications.[33]

Additional considerations for peri-anesthetic arrests include discontinuing any inhalant anesthetics, as well as troubleshooting the breathing circuit to ensure adequate oxygenation and ventilation. An aggressive search for other possible underlying causes for CPA is conducted, while additional basic and advanced life support techniques are instituted. It has been noted that low doses (0.01 mg/kg) of epinephrine are effective in anesthesia-related arrests (K. Mathews, pers. comm.[l]), and this dose is recommended in the initial resuscitation of these patients.

Monitoring efficacy of resuscitation

Clinically applicable methods to monitor the efficacy of ongoing resuscitation include physical examination findings, blood gas analysis, and end-tidal capnography. Other techniques such as arterial blood pressure monitoring and determining myocardial perfusion pressure may provide additional information. However, these methods are less practical in many resuscitation situations and are usually limited to experimental models of CPA or to patients in which invasive hemodynamic monitoring is already being performed.

Femoral pulse palpation is one of the easiest and most useful measures of effective forward blood flow, however, this may be misleading. First, it has been shown that the detection of pulses can be inaccurate up to 35% of the time in human models of CPA.[34] Second, pulse pressure represents the difference between peak (systolic) pressure and baseline (diastolic) pressure. Pulse pressure does not necessarily correlate with perfusion, especially if baseline pressures are low. Also, it has been postulated that due to potential retrograde flow through the caudal vena cava, palpable femoral pulses may actually be venous, rather than arterial in origin.[1]

End-tidal capnography is a relatively simple monitoring tool if an end-tidal CO_2 monitor is available. It is useful in the setting of CPCR, because end-tidal CO_2 is linearly related to cardiac output at a constant rate of ventilation.[21] Thus, a higher end-tidal CO_2 reflects greater delivery of CO_2 (and therefore, greater blood flow) from the periphery to the pulmonary capillaries (where equilibration takes places with the alveolar gases). Experimental animal models have demonstrated a correlation between increased end-tidal CO_2 and cardiac output, coronary perfusion pressure, and successful resuscitation.[35,36] Similarly, higher end-tidal CO_2 levels have been documented in successfully resuscitated human patients.[1,37]

Relatively inexpensive blood gas analyzers are now available to veterinarians and are a commonly used monitoring tool during CPCR. However, blood gas values should be interpreted with caution, because there is a wide disparity between arterial and venous blood gases during CPCR.[38] Due to markedly decreased pulmonary blood flow, peripheral arterial blood may have normal or near-normal pO_2, pCO_2 and pH values. Conversely, because of low peripheral blood flow, the tissues experience severe hypoxia, hypercarbia and acidosis. Systemic venous and mixed venous blood reflects the consequences of this poor perfusion as significantly lower pH and significantly higher pCO_2 values.[38] Therefore, venous blood gases are a more accurate reflection of tissue acid–base status, and may potentially be used as guides for management during resuscitation.[1,21]

Myocardial perfusion pressure (aortic diastolic pressure—right atrial pressure) has been shown to correlate with successful ROSC.[1,39] Accordingly, it is probably the best means by which to monitor the effectiveness of CPCR. Unfortunately, myocardial perfusion pressure

monitoring requires central venous and arterial catheters. Thus, it is usually possible only in animals that have been previously instrumented in a critical care or research setting.

Post-resuscitation care

Once ROSC is achieved, a rapid search for underlying causes of the CPA should be performed, with specific actions taken to correct any problems. In human medicine, it is common to search for the 5 H's and 5 T's of CPA. The 5 H's consist of Hypovolemia, Hypoxia, Hydrogen (acidosis), Hyper/Hypokalemia, and Hypothermia, while the 5 T's refer to Tablets (overdose), Tamponade, Tension pneumothorax, Thrombosis of the coronary or Thrombosis of the pulmonary arteries.[1] In addition, surgery to close the thoracotomy incision should be expedited in patients receiving open-chest CPCR. Regardless of the underlying cause, most patients are vulnerable to suffering additional episodes of CPA following an initially successful resuscitation.[40] Consequently, resuscitated patients usually require significant cardiovascular and ventilatory support during the post-arrest period. The low flow state present during (or following) CPA may also precipitate renal failure, gut reperfusion syndrome ('shock gut'), disseminated intravascular coagulation, and brain injury. Because of this, intensive monitoring and aggressive supportive care is required to optimize cardiac output, blood pressure, oxygenation, ventilation, and vital organ perfusion.

Current recommendations to maximize neurologic recovery involve maintaining cerebral perfusion pressure, which is the chief determinant of cerebral blood flow following CPA.[1,17] While intracranial pressure monitoring (and determining cerebral perfusion pressure) is beyond the capability of most veterinary practices, certain steps may be taken to protect cerebral perfusion. Mean arterial pressure should be optimized with appropriate fluid therapy and vasopressor or inotropic drugs, while undue elevation of intracranial pressure may be prevented by maintaining normocapnia. Because many patients do not ventilate effectively and recurrence of respiratory arrest is common following ROSC, mechanical ventilation to prevent hypercarbia (and associated cerebral vasodilation) is often necessary. Mechanical ventilation may also be required to maintain adequate oxygenation in patients with significant pulmonary disease (either primary or secondary to resuscitation efforts). Recent evidence in humans supports the use of lung protective strategies in ventilating patients with acute lung injury (ALI) or the acute respiratory distress syndrome (ARDS),[41] disease processes that may also be present in veterinary patients suffering CPA.

Signs of elevated intracranial pressure include alterations in level of consciousness, changes in pupillary diameter and responsiveness, strabismus, and papilledema. Severely increased intracranial pressure and imminent brain herniation is often associated with bilateral mydriasis, bradycardia, hypertension, and hypoventilation. If elevated intracranial pressure is suspected, mild hyperventilation (target $PaCO_2$ of 30–35 mmHg) and mannitol administration (at doses or 0.25–1 g/kg) are indicated.[1,42] More aggressive hyperventilation (target $PaCO_2$ of 25–30 mmHg) is indicated if brain herniation appears imminent. Importantly, hyperventilation is only recommended with evidence of elevated intracranial pressure, as diminished blood flow secondary to cerebral vasoconstriction may compound ischemic damage following CPA.[1]

Hyperglycemia is associated with worse neurologic outcomes in both experimental models,[43] and human patients,[44] and should be avoided in patients following CPA. Because corticosteroids may produce hyperglycemia and lack evidence supporting their use, these drugs are not recommended in post-arrest protocols.[17,45] Other recommendations for post-resuscitation care include maintaining normal to low-normal body temperatures and specifically avoiding hyperthermia, which is associated with increased cerebral oxygen demand.[1] Recent human studies on induced hypothermia following resuscitation from cardiac arrest have demonstrated improved neurologic outcomes,[46,47] and this strategy may be incorporated into future guidelines.

Future directions

The science of resuscitation is constantly advancing, and active investigation continues in experimental models and clinical studies of CPCR. Current areas of research include improved techniques and technology for closed chest compression, as well as invasive methods to improve perfusion.[48] These artificial perfusion methods include rapid institution of cardiopulmonary bypass and extracorporeal membrane oxygenation (ECMO), as well as aortic balloon catheter perfusion techniques to selectively deliver resuscitation fluids to the cerebral and myocardial vasculature.[48] More extensive studies of amiodarone and vasopressin are underway, and novel approaches to drug therapy, including adenosine receptor antagonism with aminophylline are being researched.[48] In addition to induced hypothermia following ROSC, other modes of promoting neurologic recovery are being investigated, and these include the promotion of cerebral blood flow through moderate hypertension and hemodilution.[21]

As new information expands the horizon of resuscitation, the true measure of progress is found in the

practical application of CPCR. While the American Heart Association guidelines reflect data regarding the clinical efficacy of various human resuscitation protocols, a void exists in veterinary medicine. The reason for this is 2-fold. First, there is no consensus on standards, protocols or recommendations for performing CPCR in veterinary patients. Second, there are no prospective veterinary clinical studies focusing on resuscitation. As such, the recommendations included in this article (Table 1, 2, Figure 2) stem from current practices in both veterinary and human medicine. With the expansion of veterinary emergency and critical care medicine, there now exists the possibility of large-scale prospective clinical trials focusing on resuscitation in veterinary patients. As this vital information is obtained, future recommendations for CPCR may be more specific, and ideally, more effective for this most vulnerable population.

Summary

Advanced life support and post-resuscitation care are important adjuncts to basic life support measures and are often vital components of successful CPCR. The ability to recognize the specific rhythm associated with CPA and to institute appropriate drug therapy or electrical defibrillation requires additional equipment and training. However, this allows a veterinarian to more effectively address specific problems encountered during resuscitation. Similarly, problems anticipated during the post-resuscitation phase, which include hypotension, hypoventilation, and coagulopathy, as well as renal, gastrointestinal, and nervous system injury, may be recognized earlier with intensive monitoring and minimized with aggressive supportive care. While a wealth of clinical experience in veterinary CPCR

Table 1: Dosages of drugs commonly used in CPCR

Drug	Dose	Route	Comments
Amiodarone	5–10 mg/kg	IV	May cause hypotension
Atropine	0.04 mg/kg	IV, IT, IO	Use lower dose if perfusing rhythm
Calcium gluconate	50 mg/kg	IV, IO	Not for routine use in CPCR
Epinephrine (low dose)	0.01–0.02 mg/kg	IV, IT, IO	Repeat doses in 3–5 minutes
Epinephrine (high-dose)	0.1–0.2 mg/kg	IV, IT, IO	Repeat doses in 3–5 minutes
Flumazenil	0.02 mg/kg	IV, IT, IO	Benzodiazepine reversal agent
Lidocaine	2 mg/kg	IV, IT, IO	Increases defibrillation threshold
Magnesium sulfate	30 mg/kg	IV, IO	Only in hypomagnesemic patients
Mannitol	0.25–1 g/kg	IV, IO	For reduction of intracranial pressure
Naloxone	0.02–0.04 mg/kg	IV, IT, IO	Opiod reversal agent
Sodium bicarbonate	1 mEq/kg	IV, IO (not IT)	Not routinely recommended
Vasopressin	0.8 μ/kg	IV, IO	Longer half-life than epinephrine

IV, intravenous; IT, intratracheal; IO, intraosseus.

Table 2: Quick reference Table for veterinary CPCR

Drug (conc.)	Weight (lb) Weight (kg) Dose	5 2.5 mL	10 5	20 10	30 15	40 20	50 25	60 30	70 35	80 40	90 45	100 50
Epi low (1 : 10 000)	0.01 mg/kg	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Epi high (1 : 1000)	0.1 mg/kg	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Atropine (0.54 mg/mL)	0.05 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
Bicarb (1 mEq/mL)	1 mEq/kg	2.5	5	10	15	20	25	30	35	40	45	50
Calcium (100 mg/mL)	50 mg/kg	1	2.5	5	7.5	10	12.5	15	17.5	20	22.5	25
Magnesium (4 mEq/mL)	0.2 mEq/kg	0.1	0.25	0.5	0.75	1	1.25	1.5	1.75	2	2.25	2.5
Vasopressin (20 units/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
Amiodarone (50 mg/mL)	5 mg/kg	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Naloxone (0.4 mg/mL)	0.04 mg/mL	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Flumazenil (0.1 mg/mL)	0.02 mg/mL	0.5	1	2	3	4	5	6	7	8	9	10
External Defib	2–10 J/kg	20	30	50	100	200	200	200	300	300	300	360
Internal Defib	0.2–1 J/kg	2	3	5	10	20	20	20	30	30	30	50

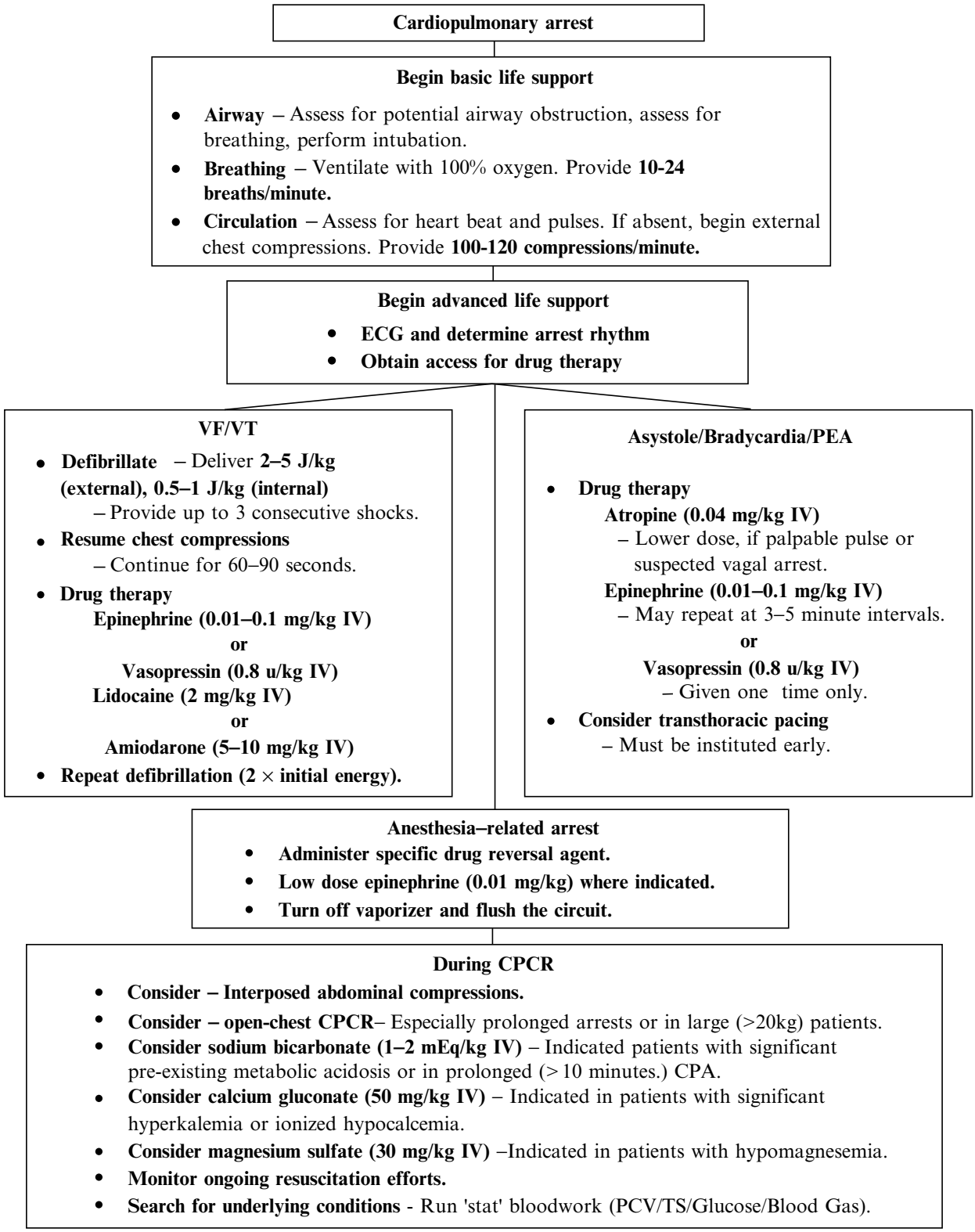

Figure 2: Algorithm for performing CPCR in veterinary patients.

exists, there is a dearth of prospective clinical research, and this may be a focus for future investigation.

Footnotes

[a] Vasopressin; Pitressin, Monarch Pharmaceuticals, Bristol, TN
[b] Amiodarone; Cordarone, Wyeth Laboratories, Philadelphia, PA
[c] Procainamide, Abbott Laboratories, North Chicago, IL
[d] Esmolol, Brevibloc, Baxter Healthcare, Westlake Village, CA
[e] THAM, Trizma base, Sigma Chemical
[f] Carbicarb
[g] Tribonat, Pharmacia and Upjohn AB, Stockholm, Sweden
[h] Naloxone; NARCAN, Endo Pharmaceuticals, Chadds Ford, PA
[i] Flumazenil; Romazicon, Roche Laboratories, Nutley, NJ
[j] Yohimbine, Lloyd Laboratories, Shenandoah, IA
[k] Atipamezole, Pfizer Animal Health, Exton, PA
[l] Dr. K. Mathews, Ontario Vet College, pers. comm.

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