Causes of cardiopulmonary arrest, resuscitation management, and functional outcome in dogs and cats surviving cardiopulmonary arrest

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Abstract
Objective: To describe the functional outcome of canine and feline survivors of cardiopulmonary arrest (CPA) and the clinical characteristics surrounding their resuscitation.
Design: Retrospective study.
Setting: Veterinary teaching hospital.
Animals: Client-owned dogs (15) and cats (3) with CPA.
Interventions: None.
Measurements and main results: Eighteen animals were identified to have survived to discharge following CPA. Cardiopulmonary arrest was associated with anesthesia with or without pre-existing disease in 10 animals, cardiovascular collapse in 5 animals, and chronic disease with an imposed stress in 3 animals. All CPAs were witnessed in the hospital. The most common initial rhythm at CPA was asystole (72%). Return of spontaneous circulation (ROSC) was achieved in less than 15 minutes from the onset of cardiopulmonary cerebral resuscitation (CPCR) in all animals. No animals had a recurrence of CPA after the initial CPA. Animals were of a wide range of ages (0.5–16 years) and breeds. Two animals were neurologically abnormal at discharge, one of which was normal at 2 months following CPA.
Conclusions: A good functional recovery after CPCR was documented in the small number of CPA survivors presented in this study. This may be due to the reversible nature of their inciting cause of CPA, early detections of CPA (‘witnessed’), and/or the animal’s underlying normal health status.

Keywords: anesthetic accident, circulation, critical care, monitoring, retrospective, survivor

Introduction
Resuscitation techniques for animals with cardiopulmonary arrest (CPA) have been well described in the veterinary literature. However, very little objective data are available surrounding the long-term treatment and quality of life of animals following resuscitation. For some small animal practitioners, the belief exists that resuscitation is futile because, although short-term success may be attainable, long-term success is unlikely. In recent years, the medical profession has expressed similar concerns due to both the escalating costs associated with the post-resuscitation patient and ethical concerns regarding quality of life post-CPA. Attention has also been focused on the likelihood of successful resuscitation of subpopulations such as the elderly, burn victims, critically ill, or in cardiopulmon-
ary cerebral resuscitation (CPCR) greater than 15 minutes. Even with successful CPCR, many human patients have severe, residual neurologic deficits due to anoxic encephalopathy or cerebral resuscitation syndrome, affecting short and long-term quality of life for the patient and their families.

Cardiopulmonary cerebral resuscitation would seem futile if survival to discharge with a good neurologic outcome were rare. In previous veterinary studies, reported survival following CPCR has been poor. Gilroy et al. reported the discharge of 4 of 18 cats (22%) following CPCR; all cats were neurologically normal. In an adjunct to their clinical study, however, the same group induced ventricular fibrillation in 22 laboratory cats and reported that 9 of the 12 successful CPCR cats suffered severe brain damage. Wingfield and Van Pelt reported that in 169 dogs with CPA, only 7 (4.1%) were discharged from the hospital. Those same authors’ results were similar for 52 cats, of which 5 (9.6%) were discharged. In that study, the neurologic status of survivors was not reported. Another author described an unpublished success rate of 25% for CPCR performed in his institution and warned of neurologic complications after CPCR, but also did not elaborate.

With such a small number of survivors to discharge described in the veterinary literature, it is hard to assess functional outcome or make assumptions regarding this population. By examining survivors of CPA, the purposes of this study were to (1) review the epidemiology of their CPA and CPCR and (2) describe their functional outcome.

**Materials and Methods**

**Animals**

Dogs and cats that experienced a documented CPA followed by successful CPCR with subsequent discharge from the hospital were eligible for study inclusion. Animals were identified by a review of case logs maintained by small animal medicine and critical care interns, residents, and faculty. Cardiopulmonary arrest was defined by the absence of both spontaneous respirations and effective circulation with arterial pulses. Animals with respiratory arrest without cardiac arrest were excluded.

**Peri-resuscitation factors**

The medical records and CPCR forms were evaluated for the following information: signalment (breed, age, gender), pre-CPA diagnosis, length of stay prior to CPA and after successful CPCR, and location of arrest within the hospital. Additionally, the inciting cause(s) of CPA, CPA rhythm diagnosis, time to return of spontaneous circulation (ROSC), any recurrent CPA events, and treatments/drugs administered during and after CPCR were identified. Return of spontaneous circulation was defined as the return of any spontaneous palpable arterial pulses. The presence or absence of a board certified criticalist (Diplomate of the American College of Veterinary Emergency and Critical Care [DAC-VECC]) or board-certified anesthesiologist (Diplomate of the American College of Veterinary Anesthesia [DACVA]) was also evaluated.

**Outcome**

The medical records were evaluated for treatments following CPCR, length of follow-up, and cost of treatment associated with that hospitalization. The presence or absence of neurological abnormalities following CPCR was also evaluated. Additionally, to assess quantitatively the functional outcome, a modified Karnovsky’s performance (MKP) criteria score was retrospectively applied to every case at the time of discharge.

**Data analysis**

The data were analyzed using commercial statistical software. The distribution of data was examined graphically. Descriptive statistics were calculated and are reported as means ± standard deviation for normally distributed data, and median and range for skewed data.

**Results**

Eighteen animals were identified with CPA and subsequent successful CPCR with survival to hospital discharge between February 1997 and January 2003. Owing to an incomplete medical records database, the...

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Table 1: Modified Karnofsky performance score criteria

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fully active, able to perform at pre-disease level.</td>
<td>0 (normal)</td>
</tr>
<tr>
<td>Activity less than pre-disease level, but able to function as an acceptable pet.</td>
<td>1 (restricted)</td>
</tr>
<tr>
<td>Severely compromised activity level; ambulatory only to the point of eating but consistently urinating and defecating in acceptable areas.</td>
<td>2 (compromised)</td>
</tr>
<tr>
<td>Completely disabled. Must be force fed. Unable to control urinations and defecations to acceptable areas.</td>
<td>3 (disabled)</td>
</tr>
<tr>
<td>Dead</td>
<td>4</td>
</tr>
</tbody>
</table>
number of animals in which CPCR was initiated without successful resuscitation during the same time period cannot be determined.

There were 15 dogs and 3 cats. Of the 15 dogs, the median age was 8 years (range 0.5–13 years). The ages of the 3 cats varied widely (1.5, 7 and 16 years of age). Gender was equally distributed with 8 females (2 intact and 6 neutered) and 10 males (4 intact and 6 neutered). Canine breeds are shown in Table 2. All cats were domestic shorthairs. The majority of animals were in the hospital for a short time (median 14 hours, range 0–8 days) prior to CPA (Table 2).

Anesthesia was considered a contributing factor for CPA in 10 of the 18 animals (55%). For 4 of the 10 animals, CPA occurred under anesthesia for elective procedures with no identifiable pre-existing diseases. In 1 otherwise healthy dog (Table 2, #1), the pop-off valve was inadvertently left closed during high-flow inhalant anesthesia. In the other 2 dogs (Table 2, #2 and #3), no identifiable cause for the arrest was determined, although in both cases, the arrest occurred within the first 10 minutes of anesthesia. In the cat (Table 2, #4), its fractious nature had resulted in the administration of a presumed relative overdose of anesthetic agents.

For the remaining 6 of the 10 animals with CPA associated with anesthesia, CPA was likely multifactorial given the presence of pre-existing conditions. Two dogs had pre-existing neurological abnormalities and arrested under general anesthesia during diagnostic imaging. One dog (Table 2, #5) arrested during myelography for cervical intervertebral disc disease. Cardiopulmonary arrest was attributed to an anaphylactic reaction to the contrast agent given since CPA occurred 1 minute after injection. A second dog (Table 2, #6), under evaluation for signs of intracranial disease, developed torsades de pointes and subsequent rapid deterioration into ventricular fibrillation during computed tomography of the brain. The intracranial disease in this dog was later attributed to milbemycin toxicity (treatment for chronic demodecosis).

Two animals arrested while undergoing anesthesia for management of pyothorax. One dog (Table 2, #7) arrested immediately following induction of anesthesia for thoracostomy tube placement. Open chest CPCR was performed with secondary lavage of the thoracic cavity and placement of thoracostomy tubes during recovery. A cat (Table 2, #8) with sepsis secondary to pyothorax and pyometra arrested during jugular

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**Table 2: Patient data**

<table>
<thead>
<tr>
<th>Breed</th>
<th>Pre-CPA length of stay</th>
<th>Location of CPA</th>
<th>CPA rhythm</th>
<th>Pre-CPA diagnosis</th>
<th>Presumed inciting event(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>#1 German shepherd</td>
<td>1.5 days</td>
<td>Anesthesia</td>
<td>Asystole</td>
<td>Cranial cruciate ligament rupture</td>
<td>Anesthesia – pop off valve closed</td>
</tr>
<tr>
<td>#2 Bichon frise</td>
<td>1 day</td>
<td>Anesthesia</td>
<td>Asystole</td>
<td>None (cryptorchid castration)</td>
<td>Anesthesia</td>
</tr>
<tr>
<td>#3 West highland</td>
<td>3 hours</td>
<td>Procedure room</td>
<td>Asystole</td>
<td>None (elect castration)</td>
<td>Anesthesia</td>
</tr>
<tr>
<td>#4 DSH</td>
<td>2 hours</td>
<td>ICU</td>
<td>Ventricular fibrillation</td>
<td>Constipation</td>
<td>Anesthesia</td>
</tr>
<tr>
<td>#5 American bulldog</td>
<td>4 hours</td>
<td>Radiology</td>
<td>Asystole</td>
<td>C5-6 intervertebral disc protrusion</td>
<td>Contrast/myelogram and anesthesia</td>
</tr>
<tr>
<td>#6 Collie</td>
<td>3 days</td>
<td>CT area</td>
<td>Ventricular fibrillation</td>
<td>Milbemycin toxicity</td>
<td>Anesthesia</td>
</tr>
<tr>
<td>#7 Golden retriever</td>
<td>1 hour</td>
<td>Procedure room</td>
<td>Asystole</td>
<td>Pyothorax</td>
<td>Anesthesia</td>
</tr>
<tr>
<td>#8 DSH</td>
<td>2 hours</td>
<td>Procedure room</td>
<td>Asystole</td>
<td>Pyothorax, pyometra, coagulopathy</td>
<td>Anesthesia recovery</td>
</tr>
<tr>
<td>#9 Pomeranian</td>
<td>14 hours</td>
<td>Procedure room</td>
<td>Asystole</td>
<td>Urethral obstruction</td>
<td>Anesthesia</td>
</tr>
<tr>
<td>#10 Sharpei</td>
<td>8 days</td>
<td>Anesthesia</td>
<td>Asystole</td>
<td>Neck bite wounds</td>
<td>Anesthesia and upper airway disease</td>
</tr>
<tr>
<td>#11 German shepherd</td>
<td>0</td>
<td>ICU</td>
<td>Asystole</td>
<td>GDV</td>
<td>GDV</td>
</tr>
<tr>
<td>#12 Rhodesian ridgeback</td>
<td>0</td>
<td>ICU</td>
<td>Asystole</td>
<td>GDV</td>
<td>GDV</td>
</tr>
<tr>
<td>#13 Border terrier</td>
<td>2 hours</td>
<td>Cardiology</td>
<td>Asystole</td>
<td>Constrictive pericardial disease with pericardial effusion</td>
<td>Pericardiocentesis</td>
</tr>
<tr>
<td>#14 Labrador cross</td>
<td>2 days</td>
<td>Ward</td>
<td>PEA – sinoatrial rhythm</td>
<td>Osteosarcoma – 1 day post amputation</td>
<td>Blood loss anemia</td>
</tr>
<tr>
<td>#15 DSH</td>
<td>6 days</td>
<td>ICU</td>
<td>Asystole</td>
<td>Dyuria, lumbosacral disease</td>
<td>Bethanecol and hypersalivating</td>
</tr>
<tr>
<td>#16 Schnauzer</td>
<td>14 hours</td>
<td>Ward</td>
<td>Ventricular fibrillation</td>
<td>Pancreatitis, chronic valvar disease</td>
<td>Restrict</td>
</tr>
<tr>
<td>#17 Poodle cross</td>
<td>8 days</td>
<td>Ward</td>
<td>PEA – idioventricular</td>
<td>Diabetic ketoacidosis</td>
<td>Restrict</td>
</tr>
<tr>
<td>#18 Maltese</td>
<td>14 hours</td>
<td>Ultrasound</td>
<td>Asystole</td>
<td>Hepatic encephalopathy – liver failure</td>
<td>Restrict</td>
</tr>
</tbody>
</table>

CPA, cardiopulmonary test; GDV, gastric dilatation and volvulus; PEA, pulseless electrical activity; DSH, domestic shorthair cat.
catheterization immediately following placement of bilateral thoracostomy tubes.

In the remaining 2 animals, CPA was attributed, in part, to anesthesia with lack of control of the airway. One dog (Table 2, #9) arrested under injectable anesthesia for relief of a urinary tract obstruction. High vagal tone secondary to urinary stimulation was also thought to play a role in this CPA. The second dog, a brachycephalic Sharpei (Table 2, #10), had suffered bite wounds to the neck necessitating daily anesthesia for wound management. The dog arrested after extubation following surgical exploration of severe bite wounds on the neck on the eighth day of hospitalization. During unsuccessful attempts to re-intubate during respiratory arrest, CPA occurred with ventricular fibrillation. The rhythm was responsive to immediate defibrillation and intubation via an emergent tracheostomy.

Five of the 18 animals (28%) arrested secondary to cardiovascular collapse. Cardiopulmonary arrest occurred on presentation for 2 dogs with gastric dilatation-volvulus (GDV) (Table 2, #11 and #12). Another dog (Table 2, #13) arrested during pericardiocentesis due to inadvertent penetration of the right atrium and subsequent pericardial tamponade. Open chest CPCR was immediately performed with subtotal pericardectomy and direct digital pressure application to the puncture in the right atrium. Blood loss into an amputation site was responsible for cardiovascular collapse in another dog (Table 2, #14). Progressive tachycardia was noted in this dog for 4 hours prior to CPA, but was inappropriately attributed to lack of sufficient analgesia rather than hypovolemia. A final cat (Table 2, #15), treated for dysuria developed CPA after treatment with bethanecol secondary to intravascular volume depletion and free water loss. Hypersalivation in the 8-hour period prior to CPA led to an increase in serum sodium to 176 mEq/L (reference range, 149–162 mEq/L), reflecting a free water deficit of approximately 80 mL/kg.

Three of the 18 animals (17%) suffered from serious chronic diseases and arrested during restraint for imaging procedures or treatments. One dog with biliary obstruction secondary to pancreatitis (Table 2, #16) developed ventricular fibrillation during restraint for a jugular catheter, and another dog with diabetic ketoacidosis (Table 2, #17) arrested during restraint for blood glucose determination. The third dog had severe liver disease (Table 2, #18) and arrested after being placed in dorsal recumbency for an abdominal ultrasound.

Documented CPA rhythms in the 18 animals included asystole in 13/18 (72%), ventricular fibrillation (VFIB) in 3/18 (17%), and pulseless electrical activity (PEA) in 2/18 (11%) (Table 2). All animals with VFIB received external defibrillation. Another dog with an initial CPA rhythm of asystole converted to ventricular fibrillation after drug therapy and then received external defibrillation. After CPA, the rhythm at ROSC was sinus for the remaining 17 animals. Return of spontaneous circulation was documented within 0–5 minutes after initiating CPCR in 13 of the 18 of animals, 5–10 minutes in 2/18, and 10–15 minutes in 3 of the 18. No animals in this study needed an active CPCR effort of greater than 15 minutes to achieve ROSC. As reported above, 2 of the 18 animals received open chest CPCR, while the remaining 16 received closed chest CPCR. All animals received positive pressure ventilation (PPV) during CPCR. None of these survivors had a recurrent CPA following the original episode.

Fluid therapy during CPCR included intravenous (IV) crystalloids (18 of the 18), synthetic colloids (2 of the 18), and packed red blood cells (1 of the 18). Epinephrine was administered to 17 of the 18 animals. One dog received epinephrine intra-tracheally at a dose of 0.08 mg/kg. In 13 animals, the IV dose of epinephrine could be accurately assessed. A wide dosage range was noted with a median dose of 0.09 mg/kg (range, 0.02–0.15 mg/kg). Of those 13 animals, 8 received 1 dose, 3 received 2 doses, 1 received 3 doses and another 4 doses of epinephrine during CPCR.

Parasympatholytics were administered IV to 16 animals including atropine (15) and glycopyrolate (1). Six animals were administered sodium bicarbonate for pre-existing metabolic acidosis or prolonged CPCR. Three of the animals with CPA attributed to anesthesia with opioids received naloxone as a reversal agent. Four dogs with ventricular tachycardia immediately following ROSC were treated with lidocaine. Only 3 of those dogs had persistent ventricular ectopy necessitating a continuous rate infusion of lidocaine, 2 of which were the dogs with GDV. Five dogs were treated with mannitol following CPCR, with 2 of these also receiving concomitant dexamethasone. Four dogs received dexamethasone alone. Other IV therapies were varied and included dextrose (n = 1), magnesium (n = 1), brytylium (n = 1), and furosemide (n = 1). After ROSC, 4 animals were maintained on vasopressors: dopamine in 2 for 2 and 35 hours, respectively, and dobutamine in 2 for 18 and 32 hours, respectively.

Cardiopulmonary cerebral resuscitation was performed under the direction of a DACVECC in 12 cases and a DACVA in 3 cases. Following ROSC, critical monitoring included the following: continuous electrocardiogram (15 of the 17), blood pressure (14 of the 17), pulse oximetry (12 of the 17), and arterial blood gases (8 of the 17).

Antibiotics were administered to 13 of the 18 animals. Eight animals were not previously on antibiotics prior...
to CPA, while 3 had a change of antibiotic after CPA. The remaining 2 animals did not have a change in antibiotic after CPA. Histamine (H2) blockers were administered in 9 animals. Seven were placed on H2 blockers after CPA; 1 animal was continued on the same regimen, while 1 was discontinued from therapy after CPA. Animals without a previous history of melena had no reported gastrointestinal hemorrhage as a consequence of CPA.

Thirteen animals (10 dogs and all 3 cats) received PPV following ROSC for 120 minutes or less (median 30 minutes, range 10–120 minutes). One dog with GDV and CPA occurring at admission was maintained on PPV for 5 hours during anesthesia for gastropexy. Four dogs were maintained on PPV following ROSC for greater than 7 hours (median 19 hours, range 7–24 hours).

Immediately following CPCR and extubation, neurologic derangements were observed in 8 animals, of which 2 were abnormal prior to CPCR. A range of deficits was observed including possible blindness, seizures, dullness, circling, and ataxia. Neurologic deficits were evident in only 2 of the 18 animals 48 hours following CPCR.

At the time of discharge, 16 of the 18 animals had MKP scores of 0. One dog with CPA during elective cruciate repair and an ROSC of 15 minutes had an MKP score of 2 at discharge. At discharge, this dog was still non-ambulatory with blindness and dementia. Four months following discharge, the dog’s MKP score had remained constant at 2 and the clients elected euthanasia due to perceived behavior problems, aggression, and circling. The second dog had an MKP score of 2 at the time of discharge, but was neurologically abnormal on admission secondary to hepatic encephalopathy. The remaining neurological deficits in this animal were decreased vision and some circling. Two months following CPA, this dog’s neurological impairments had resolved and the MKP score was 0.

Following CPA, the mean length of hospitalization was 5.1 ± 3.7 days. Long-term follow-up was unavailable for 6 animals. The remaining 12 animals had a median follow-up of 8 months (range 1–36 months). The owners of these animals reported no abnormalities in behavior at home. Only 1 dog was euthanized after discharge because of unrelated disease (metastatic pulmonary osteosarcoma). The cost of care following CPA was available for 10 animals with a median cost of $2,200 (range $1,300–3,600).

Discussion

Of the 18 CPCR survivors to hospital discharge described in this report, all but 1 had a normal neurologic outcome by the time of discharge or soon after. The 1 survivor with poor functional outcome following CPCR was subsequently euthanized. This high rate of functional outcome is similar to the success rate reported in the previous clinical veterinary study in which neurologic status was assessed. In 2 medical studies evaluating quality of life after hospital discharge in adults, CPA survivors exhibited a similar health-related quality of life (physical, psychological, and social function) as compared with other ICU survivors. In a study of pediatric burn patients surviving after CPA, only 1 of 17 long-term survivors had persistent neurologic abnormalities. Other medical studies have reported similarly low rates of neurologic abnormalities in survivors to discharge. Whether a good quality of life following CPCR is related to a particular subpopulation or disease in veterinary medicine remains to be seen.

The use of scoring systems to examine quality of life is controversial and focuses mainly on subjective criteria. Since many of these concepts apply only to humans, formulating an animal score is difficult. The use of the MKP score by veterinary oncologists has been described as a prognostic indicator and focuses on an animal’s ability to function (i.e., alertness, appetite, activity, and elimination behaviors). Given the serious neurological impairments that can occur secondary to CPA, a qualitative assessment of functional status following CPCR may hold prognostic significance. A prospective application of the MKP score in CPA survivors would be necessary to evaluate its usefulness.

In previous veterinary reports, anesthesia and drug reactions were suspected as the inciting cause of CPA in the overwhelming majority of CPA survivors. Kass and Haskins reported that dogs and cats with CPA secondary to a non-drug or anesthetic reaction were on average 11 times more likely to die in the first 3 days post-CPCR than animals that arrested from drug or anesthetic reactions. Owing to the lack of a control population in this study, a similar comparison is not possible, but a reaction to contrast material injected during a myelogram was documented in one dog.

Anesthesia was a contributing factor to CPA in over half of the animals in this study, but almost one-third suffered solely from cardiovascular collapse. The increased frequency of CPA secondary only to cardiovascular collapse in this study may reflect the larger number of survivors reported here or the actual causes of hypovolemia in this population. In animals with cardiovascular collapse in this study, hypovolemia occurred relatively acutely, was witnessed in the hospital, and addressed quickly and definitively. From the previous studies and the data in this study, it
would seem that animals without serious underlying disease prior to CPA and/or reversible causes of CPA are more likely to have a successful outcome following CPCR.

Three animals suffered from chronic disease, but arrested secondary to an imposed stress. The impact of restraint and applied stress to an already debilitated animal can exacerbate underlying cardiovascular and metabolic derangements. Interestingly, all 3 animals were dogs suffering from metabolic diseases.

The majority of the survivors in this study had CPCR performed under the supervision of a trained criticalist or anesthesiologist. Previous reviews of CPCR from veterinary teaching institutions have reported variable levels of training in the personnel performing CPCR.8–10 The level of personnel training may impact both the success of CPCR, and post-resuscitation monitoring and support. Medical studies have documented an improved outcome if physicians and nurses trained in advanced life support deliver CPCR.20,21

The age of the animal did not seem to play a role in this population. Eleven of these animals were between 7 and 16 years of age. There has been controversy in the human literature regarding the impact of age on survival after CPA,5,22 but this may not be as important in dogs and cats as long as the cause of CPA is reversible.

Asystole was the most common CPA rhythm diagnosis in this study. In a previous study, PEA (also known as electromechanical dissociation), asystole and VFIB were relatively equal in distribution for animals with documented CPA,11 but the authors did not include information on survival. In humans, the rhythm diagnosis at CPA is significantly correlated with successful CPCR due to the high rate of VFIB, and the success of early detection and defibrillation.5,17 The predominance of asystole as the CPA rhythm diagnosis may reflect the lack of similar risk factors for VFIB in the veterinary population or an inability to convert VFIB (i.e., non-survivors.) Given the lack of a comparison group, strong assumptions cannot be made.

The time of ROSC for all animals reported was below 15 minutes. Kass and Haskins10 reported an average length of CPCR of 17 minutes in dogs and cats with no difference between survivors and non-survivors. In their clinical study, Gilroy et al. found that no cat was discharged with duration of CPA greater than 15 minutes.8 The neurologic impact of prolonged ROSC has been documented experimentally. Severe neurologic deficits were seen in dogs with arrests lasting longer than 12 minutes.23 In medical studies, there is some variability regarding the impact of ROSC time on survival reflecting the differences in study populations that include treatable VFIB.5,14 Of the 3 animals in this study with an ROSC greater than 10 minutes, 1 was neurologically impaired at discharge and subsequently euthanized. Shorter times for ROSC may reflect a more reversible cause of CPA and an increased likelihood of survival.

No survivor in this study suffered any further CPA events or arrested outside of the hospital, 2 factors that have a high impact on survival in the medical literature. Wingfield and Van Pelt9 reported 2 dogs that were discharged with more than 1 CPA. Kass and Haskins10 describe 1 dog with cardiac disease that re-arrested and survived to discharge. Re-arrest may reflect the severity of underlying disease and/or inadequate correction of the inciting cause of CPA.

The lack of out-of-hospital survivors in the veterinary literature and in this study likely reflects the actual causes of CPA and the inherent lack of field CPCR in veterinary medicine. In the medical literature, the success rates for CPCR performed outside the hospital are lower than for patients in the hospital.18 With the advent of portable defibrillators, higher survival rates are now being seen with VFIB out-of-hospital, but this success still relies on prompt (<8 minutes) defibrillation and delivery to the hospital17 It is unlikely that an animal presented to a veterinary hospital with CPA will survive unless the duration of CPA has been short (<8 minutes).

Regarding arrest management, 2 survivors received open chest CPCR while the remainder were resuscitated with external compressions alone. Those 2 dogs had clear indications to pursue emergent thoracotomy (pyothorax and pericardial tamponade) and did not receive closed chest CPCR. No survivors in this study had open chest CPCR performed late in CPCR after initial attempts with external compressions had failed. The veterinary guidelines for the application of open chest CPCR are varied,1–4 but some authors have argued that open chest CPCR should be performed within 2 minutes of CPA, given its proven ability to increase cardiac output above that of external compressions.1,4 This may be especially true in large breed dogs or animals with contraindications to external compression (e.g., rib fracture, post-operative thoracic surgery).

The majority of the animals in this study received epinephrine and an anticholinergic during CPCR. The dose of epinephrine varied widely. Recent clinical evidence shows that high-dose epinephrine (0.2 mg/kg) may not improve survival over the standard dose (0.02 mg/kg) used in humans.24,25 The recommendation to use the standard dose is also supported by experimental evidence documenting post-resuscitative tachycardia, systemic hypertension, myocardial necrosis, and greater early mortality after administration of
high dose epinephrine. Although epinephrine remains classified as an ‘Indeterminate/Experimental’ intervention in CPR according to the American Heart Association, atropine has been proven to decrease cholinergic-mediated hypotension, bradycardia, and vasodilation. A large majority of the survivors in this study received atropine for asystole or bradycardia.

Limitations of this study include those inherent in a retrospective study, including availability and completeness of medical record information. Data could not be standardized for each animal. A controlled prospective study would be required to compare survivors and non-survivors in order to assess accurately such factors as time of ROSC, age, and therapies administered. The use of CPR protocols and standardized doses should also be encouraged to allow comparisons of treatment modalities.

In conclusion, a good functional recovery following CPR was documented in the majority of animals in this study. It may be presumed that the risk of severe anoxic encephalopathy should not deter clinicians from performing CPR in animals with witnessed CPA secondary to a potentially reversible cause. These circumstances include anesthesia in both healthy and diseased animals, correctable cardiovascular collapse, and imposed stressors in a metabolically compromised animal. Prompt reversal of the inciting cause in these animals may lead to long-term survival and good functional outcome. Further research into the survival following CPA in these subpopulations is warranted, as it may be higher than previously reported.

Footnotes

a SPSS for Windows 10.1.0, Chicago, IL.
b Omnipaque (iohexol) injection, Amersham Health, Cork, Ireland.
c Propoflo, Abbott Laboratories, N. Chicago, IL.
d Urecholine, Sidmark Laboratories Inc., East Hanover, NJ.
e Hetastarch, Abbott Laboratories, N. Chicago, IL.
f Robinul, A.H. Robbins, Richmond, VA.

References


