Prognostic indicators for dogs and cats with cardiopulmonary arrest treated by cardiopulmonary cerebral resuscitation at a university teaching hospital

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Objective—To determine the association among signalment, health status, other clinical variables, and treatments and events during cardiopulmonary cerebral resuscitation (CPCR) with the return of spontaneous circulation (ROSC) for animals with cardiopulmonary arrest (CPA) in a veterinary teaching hospital.

Design—Cross-sectional study.

Animals—161 dogs and 43 cats with CPA.

Procedures—Data were gathered during a 60-month period on animals that had CPA and underwent CPCR. Logistic regression was used to evaluate effects of multiple predictors for ROSC.

Results—56 (35%) dogs and 19 (44%) cats had successful CPCR. Twelve (6%) animals (9 dogs and 3 cats) were discharged from the hospital. Successfully resuscitated dogs were significantly more likely to have been treated with mannitol, lidocaine, fluids, dopamine, corticosteroids, or vasopressin; had CPA while anesthetized; received chest compressions while positioned in lateral recumbency; and had a suspected cause of CPA other than hemorrhage or anemia, shock, hypoxemia, multiple organ dysfunction syndrome, cerebral trauma, malignant arrhythmia, or an anaphylactoid reaction and were less likely to have been treated with multiple doses of epinephrine, had a longer duration of CPA, or had multiple disease conditions, compared with findings in dogs that were not successfully resuscitated. Successfully resuscitated cats were significantly more likely to have had more people participate in CPCR and less likely to have had shock as the suspected cause of CPA, compared with findings in cats that were not successfully resuscitated.

Conclusions and Clinical Relevance—The prognosis was grave for animals with CPA, except for those that had CPA while anesthetized. (J Am Vet Med Assoc 2009;235:50–57)

Cardiopulmonary cerebral resuscitation is the attempt to restore spontaneous circulation in a patient with CPA. Cardiopulmonary cerebral resuscitation encompasses a variety of treatments and typically includes establishment of an airway, provision of intermittent positive-pressure ventilation, and provision of circulatory support by mechanical (chest or direct cardiac compressions) and pharmacologic means. The goal is ROSC, return of spontaneous respiration, and prevention of permanent dysfunction of the CNS.

The incidence and prevalence of CPA in hospitalized and outpatient dogs and cats are unknown. The incidences of death during anesthesia in dogs and cats have been recently reported as 0.17% and 0.24%, respectively. This represents 17 and 24 deaths/10,000 anesthetized patients, respectively, and contrasts with a rate of 2 to 5 deaths/10,000 anesthetized patients in humans. To our knowledge, the incidence of CPA in dogs and cats hospitalized or evaluated because of health emergencies has not been reported. However, the authors’ clinical impression is that CPA happens more commonly in dogs and cats treated in intensive care units or emergency departments than in those that are anesthetized.

Few reports have been published on CPCR in veterinary patients. Four of these are retrospective case series. Three of those were published in 1992, and in the other report, discharge from a hospital was the only outcome addressed for dogs and cats that survived CPA. In none of these studies were statistical
models applied to determine variables that may be used to predict success or failure. Also, the clinical practice of CPR may have advanced and changed during the past 15 years. The purpose of the study reported here was to collect information about dogs and cats that had CPA in a veterinary teaching hospital and to determine the association among signalment, health status, other clinical variables, and treatments and events during CPR and ROSC.

Materials and Methods

Animals—Every dog and cat that had CPA and underwent CPR during a 60-month period (February 2003 through February 2008) in the Veterinary Teaching Hospital of the University of Georgia was eligible for inclusion in the study. This included dogs and cats hospitalized in the intensive care unit, managed by the anesthesiology section, evaluated by the emergency service, and hospitalized or managed by any other clinical service. Dogs and cats that had respiratory arrest without cardiac arrest and those that had CPA without an attempt at CPR were not included.

Cardiopulmonary arrest was diagnosed by the supervising clinician and was characterized by loss of spontaneous circulation (ie, undetectable heart sounds as evaluated via direct auscultation and loss of palpable pulses) and respiratory arrest. Return of spontaneous circulation was defined as audible heart sounds, electrical activity (evaluated by use of ECG) coincident with heart sounds, palpable pulses, and spontaneous respiration.

Data collection—A standardized form was used for gathering data about eligible dogs and cats. Technicians in the intensive care unit and interns, residents, and faculty of the small animal hospital were interviewed regularly to ensure that a form was completed for all dogs and cats that had CPA. Each form was completed by the clinician supervising the CPR effort or by the technician directly working with the supervising clinician. After each CPR effort, one of the authors interviewed the clinician who supervised the CPR effort to clarify details and obtain an accurate description of events. Appropriate changes were then made to the data for each patient on the basis of the interview. The total number of patients managed by the anesthesia service during the same 60-month period was calculated by reviewing daily records maintained by the service.

Monitoring capabilities for PETCO₂ were added in the intensive care unit 6 months after the start of the study. A critical care service staffed by American College of Veterinary Emergency and Critical Care board-certified veterinarians during the day was established and certified veterinarians during the day was established and monitored 52 months after the study began (ie, 8 months before study termination).

A BC5 was assigned on the basis of a 9-point scale. An ASA status (I = healthy patient, II = mild systemic disease, III = moderate-severe systemic disease, IV = disease that is a constant threat to life, and V = patient not expected to survive 24 hours) was assigned on the basis of each patient's underlying disease. Medical problems were subdivided into the following categories: cardiovascular, neurologic, pulmonary, hemolyphatic, gastrointestinal, MODS, other, and unknown. The cardiovascular designation was assigned to patients with cardiac disease (eg, congestive heart failure). The neurologic designation was assigned to patients with any central or peripheral neuropathy. The pulmonary designation was assigned to patients with dyspnea, abnormal findings on thoracic radiographs, abnormal results of arterial blood gas analysis, or abnormal values for pulse oximetry. The hemolymphatic designation was assigned to patients with coagulopathies (including disseminated intravascular coagulation), anemia, or hemolymphatic neoplasia (eg, leukemia or splenic neplasia). The gastrointestinal designation was assigned to patients with hepatic or gastrointestinal tract disease (eg, ileus, vomiting, or intestinal resection). The MODS designation was assigned to patients with physiologic derangements of the endothelial, cardiopulmonary, renal, nervous, endocrine, and gastrointestinal systems associated with the progression of uncontrolled systemic inflammation and disseminated intravascular coagulation. Published guidelines for the diagnosis of this syndrome were used to retrospectively assign these patients. The other designation was assigned to patients with a disease or condition (eg, bite wounds) that could not be classified into one of the other categories. The unknown designation was assigned when the primary clinician managing the patient did not have a tentative diagnosis. When a patient was classified into > 1 category, it was given an additional designation of multiple, which indicated multiple problems that did not meet the criteria for a designation of MODS.

The suspected cause of CPA, means of identifying CPA, and initial arrhythmia were provided by the clinician supervising the CPR effort and were based on the clinician's perception at the time of the CPR effort and in retrospect during the interview after the CPR effort. The numerous categoric variables for suspected cause and initial signs of CPA were coded as detected or not detected, and those for treatments given during the CPR effort were coded as administered or not administered. Patient position was coded as laterally recumbent or not laterally recumbent. The initial arrhythmia and route of drug administration were characterized as categoric variables. Patients were considered anesthetized when CPA happened during the time they were receiving inhalant or injectable anesthetics for the purpose of providing anesthesia and analgesia, muscle relaxation, and narcosis. Patients recovering from anesthesia (ie, after extubation) or that were only heavily sedated were not considered anesthetized.

Statistical analysis—Normality of continuous data was assessed by use of the Kolmogorov-Smirnov test. Variables were compared between animals of each species in which CPR was successful (ie, there was ROSC) and those in which CPR was not successful (ie, lack of ROSC). Continuous data that were normally distributed were compared via t tests and included variables for age, body weight, BS5, number of people who participated in the CPR effort, duration of the CPR effort, and PETCO₂ values. Data that were nonparametric or discontinuous (ie, all other quantitative variables) were analyzed by use of the Mann-Whitney U test. Categoric data were analyzed via the χ² test. Significance for reporting these values independently was set at P < 0.01.

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Separate logistic regression models were constructed for each species, with ROSC as the dependent variable. To construct each model, variables from the aforementioned univariate analyses with values of $P \leq 0.1$ were included as independent variables. Models were designed by use of stepwise selection with the likelihood ratio method, with cutoffs of $P \leq 0.05$ for entry and $P \geq 0.10$ for removal of independent variables. Interaction terms were created by one of the authors (EHH) on the basis of physiologic rationale.

**Results**

During the 60-month duration of the study, 204 animals (161 dogs and 43 cats) had CPA (Table 1). Cardiopulmonary cerebral resuscitation was successful in 56 (33%) dogs and 19 (44%) cats. Twelve (6%) animals (9 dogs and 3 cats) survived and were discharged from the hospital, which represented 6% (9/161 dogs) and 7% (3/43 cats), respectively, of all animals with CPAs and 16% (9/56 dogs) and 16% (3/19 cats), respectively, of all animals successfully resuscitated. For dogs that were successfully resuscitated, those that were not discharged from the hospital included 30 (54%) that had another CPA and did not recover and 17 (30%) that were euthanatized. For cats that were successfully resuscitated, those that were not discharged from the hospital included 7 (37%) that had another CPA and did not recover and 9 (47%) that were euthanatized. For animals that had another CPA, the second CPA was between 1 minute and 5 days later (median, 15 minutes).

Nineteen animals (12 dogs and 7 cats) had CPA while anesthetized; these animals had ASA status that ranged from 1 in some animals to IV in other animals. Of those 19 anesthetized animals, 10 (6 dogs and 4 cats) did not survive to be discharged from the hospital, including 4 (2 dogs and 2 cats) in which ROSC was not achieved. During the same period, 11,379 animals were managed by the anesthesia service. This represented a CPA rate of 16.7/10,000 anesthetized animals and a fatality rate of 8.8/10,000 anesthetized animals (Table 1). Dogs that were anesthetized at the time of CPA had significantly more fatality rates than were cats that were not anesthetized at the time of CPA. Cats that were anesthetized at the time of CPA tended to have lower fatality rates than were dogs that were not anesthetized at the time of CPA. Of all animals discharged after successful resuscitation, 9 of 12 were anesthetized at the time of CPA. This included 6 of 9 dogs and all 3 cats discharged from the hospital.

Eighteen animals (14 dogs and 4 cats) were considered dead at the time of arrival at the emergency service. Of those, 2 (1 dog and 1 cat) had successful ROSC, although neither survived to be discharged from the hospital. Median estimated interval between CPA and arrival at the emergency service was 5 minutes (range, 1 to 60 minutes). For each of the 2 animals that were successfully resuscitated, estimated interval from CPA to arrival at the emergency service was 1 minute.

The suspected cause of CPA in the 204 animals was hypoxemia (n = 74 [36%]), other (43 [21%]), shock (37 [18%]), anemia (26 [13%]), malignant arrhythmia (16 [8%]), MODS (12 [6%]), cerebral trauma (11 [5%]), or anaphylactoid reaction (2 [1%]), with some animals having > 1 suspected cause. Cardiopulmonary arrest was identified by the detection of agonal gasps (n = 77 [38%]), apnea (66 [32%]), collapse (43 [21%]), a fixed gaze (29 [14%]), lack of a pulse (evaluated via digital palpation or by use of a probe for Doppler ultrasonography) or a rapid decrease in PetCO$_2$ values (26 [13%]), or an arrhythmia (evaluated via continuous ECG monitoring; 23 [11%]) or by other methods (ie, detection of vomiting followed by collapse, hypoxentilation, or seizures; 13 [6%]). Some animals had CPA identified by > 1 sign. More than half (15/26 [58%]) of the animals with CPA identified by detection of lack of a pulse or an abrupt decrease in PetCO$_2$ values were anesthetized at the time of CPA.

For the 161 dogs, 80 (50%) had asystole, 37 (23%) had bradycardia (sinus or other causes), 17 (11%) had EMD, 12 (7%) had other arrhythmias, 11 (7%) had ventricular fibrillation, 2 (1%) had ventricular tachycardia, and 2 (1%) had atrial tachycardia as the initial arrhythmia. The 43 cats, 17 (40%) had asystole, 9 (21%) had bradycardia, 8 (19%) had EMD, 5 (12%) had other arrhythmias, 2 (5%) had ventricular tachycardia, 1 (2%) had ventricular fibrillation, and 1 (2%) had atrial tachycardia as the initial arrhythmia. The proportions of arrhythmia types were not significantly different between dogs and cats.

The supervisor of the CPCR effort was an intern for 90 of 204 (44%) patients, a resident for 63 (31%) patients, or a technician for 50 (24%) patients. Of the 204 patients, 106 (52%) had CPA between 5 PM and 8 AM (ie, outside of regular business hours).

Treatments given to the 204 animals included external chest compressions (n = 193 [95%]), atropine (0.05 mg/kg [0.023 mg/lb]; 189 [91%]), low-dose epinephrine (0.01 mg/kg [0.0045 mg/lb]; 144 [71%]), crystalloid fluids (121 [59%]), high-dose epinephrine (0.1 mg/kg [0.045 mg/lb]; 62 [30%]), abdominal compressions (41 [20%]), sodium bicarbonate (31 [15%]),

<table>
<thead>
<tr>
<th>Variable</th>
<th>Dogs</th>
<th>Cats</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ROSC achieved (n = 56)</td>
<td>ROSC not achieved (n = 105)</td>
</tr>
<tr>
<td>Age (y)</td>
<td>7.1 ± 4.3</td>
<td>8.8 ± 3.7</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>16.4 ± 15.0</td>
<td>21.3 ± 17.1</td>
</tr>
<tr>
<td>BCS</td>
<td>5.0 ± 1.6</td>
<td>5.4 ± 1.4</td>
</tr>
<tr>
<td>ASA status</td>
<td>3.6 ± 0.9</td>
<td>3.6 ± 1.0</td>
</tr>
</tbody>
</table>

The BCS was scored by use of a 9-point scale, and the ASA status was scored by use of a 5-point scale.

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Table 1—Mean ± SD values for signalment variables of dogs and cats with CPA who underwent CPCR during a 60-month period.
electrical defibrillation (31 [13%]), dobutamine administered via continuous rate infusion (25 [12%]), dopamine administered via continuous rate infusion (24 [12%]), vasopressin (0.4 U/kg [0.18 U/lb]; 15 [7%]), mannitol (13 [6%]), hydroxyethyl starch (12 [6%]), naloxone (13 [6%]), lidocaine (7 [3%]), internal cardiac compressions (7 [3%]), and corticosteroids (7 [3%]). All animals received ≥ 2 treatments.

During CPCR, drugs were administered IV via a peripheral vein catheter in 134 of 204 (66%) animals, a central venous catheter in 37 (18%) animals, or intratracheally in 24 (12%) animals, with some animals receiving drugs by > 1 route. Chest compressions were performed while an animal was in lateral recumbency for 169 of 193 (88%) animals or while in other positions for 24 (12%) animals.

Monitoring of PETCO2 was instituted during CPCR efforts for 47 of 161 (29%) dogs and 19 of 43 (44%) cats. The mean highest recorded PETCO2 was significantly (P < 0.001) higher in successfully resuscitated dogs (36.6 mm Hg) than in dogs that did not achieve ROSC (10.3 mm Hg), but was not significantly (P = 0.08) different between successfully resuscitated cats and cats that did not achieve ROSC (23.5 vs 14.3 mm Hg; Table 2). For dogs, 17 of 18 (94%) with a PETCO2 < 15 mm Hg were not successfully resuscitated, whereas 25 of 29 (86%) with a PETCO2 ≥ 15 mm Hg were successfully resuscitated. For cats, 5 of 9 with a PETCO2 < 20 mm Hg were not successfully resuscitated, whereas 9 of 10 with a PETCO2 ≥ 20 mm Hg were successfully resuscitated.

The mean duration of CPCR was significantly (P = 0.002) shorter in dogs that were successfully resuscitated (12 minutes) than in dogs that were not successfully resuscitated (15 minutes; Table 2). Dogs that achieved ROSC received significantly (P = 0.001) fewer doses of epinephrine (1.8), compared with the number of doses (2.4) administered to dogs that were not successfully resuscitated. There was no significant difference between dogs and cats with regard to the proportion of animals that had ROSC or survived until discharge from the hospital. For 1 dog discharged from the hospital, CPCR duration was 20 minutes; CPCR duration was < 5 minutes for the other 8 dogs discharged from the hospital. For the 3 cats discharged from the hospital. For the 3 cats discharged from the hospital.

Table 2—Mean ± SD values for continuous variables in dogs and cats with CPA that underwent CPCR during a 60-month period.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Dogs</th>
<th>Cats</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ROSC achieved (n = 56)</td>
<td>ROSC not achieved (n = 108)</td>
</tr>
<tr>
<td>No. of people who participated in the CPCR effort</td>
<td>6.3 ± 3.1</td>
<td>6.1 ± 2.5</td>
</tr>
<tr>
<td>Duration of CPCR effort (min)</td>
<td>11.5 ± 9.7*</td>
<td>14.9 ± 7.9</td>
</tr>
<tr>
<td>No. of doses of atropine</td>
<td>1.8 ± 1.2*</td>
<td>2.4 ± 1.2</td>
</tr>
<tr>
<td>Peak PETCO2†</td>
<td>36.6 ± 19.7*</td>
<td>103 ± 10.2</td>
</tr>
</tbody>
</table>

*Within a variable for a species, the value for animals in which ROSC was achieved differed significantly (P < 0.002; univariate analysis) from the value for animals in which ROSC was not achieved. †Peak PETCO2 values were obtained in 26 dogs and 13 cats that achieved ROSC and 21 dogs and 6 cats that did not achieve ROSC.

Table 3—Results of multivariate logistic regression analysis for variables associated with the likelihood of ROSC in 161 dogs with CPA that underwent CPCR.

<table>
<thead>
<tr>
<th>Variable</th>
<th>OR</th>
<th>95% CI</th>
<th>P value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment with mannitol</td>
<td>18.4</td>
<td>1.2–290.4</td>
<td>0.038</td>
</tr>
<tr>
<td>Treatment with lidocaine</td>
<td>57.6</td>
<td>4.9–70.6</td>
<td>0.001</td>
</tr>
<tr>
<td>Treatment with fluids</td>
<td>4.6</td>
<td>1.5–14.4</td>
<td>0.008</td>
</tr>
<tr>
<td>Treatment with dopamine</td>
<td>19.3</td>
<td>3.7–101.1</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Treatment with corticosteroids</td>
<td>272.6</td>
<td>2.5–1,000.0</td>
<td>0.020</td>
</tr>
<tr>
<td>Treatment with vasopressin</td>
<td>13.1</td>
<td>1.7–96.5</td>
<td>0.013</td>
</tr>
<tr>
<td>Duration of CPCR effort†</td>
<td>0.912</td>
<td>0.857–0.971</td>
<td>0.004</td>
</tr>
<tr>
<td>Anesthetized at the time of CPA</td>
<td>8.2</td>
<td>1.1–61.6</td>
<td>0.041</td>
</tr>
<tr>
<td>No. of doses of epinephrine†</td>
<td>0.49</td>
<td>0.30–0.80</td>
<td>0.004</td>
</tr>
<tr>
<td>Patient had multiple disease conditions</td>
<td>0.192</td>
<td>0.054–0.695</td>
<td>0.011</td>
</tr>
<tr>
<td>Received chest compressions when positioned in lateral recumbency</td>
<td>46.6</td>
<td>4.1–535.6</td>
<td>0.002</td>
</tr>
<tr>
<td>Suspected cause of CPA is other than hemorrhage or anemia, shock, hypoxemia, MODS, cerebral trauma, malignant arrhythmia, or an anaphylactoid reaction</td>
<td>3.9</td>
<td>1.2–12.5</td>
<td>0.022</td>
</tr>
</tbody>
</table>

*Values were considered significant at P < 0.05. For continuous variables, the odds ratio (OR) represents the factor by which the odds of achieving ROSC are increased (for values > 1) or decreased (for values < 1) for each 1-unit increase in change in the variable. The unit for duration of CPCR effort is minutes.

CI = Confidence interval.
CPR duration was 1 minute, 5 minutes, and 15 minutes, respectively.

Dogs that were successfully resuscitated were significantly more likely to have been treated by administration of mannitol, lidocaine, fluids, dopamine, corticosteroids, or vasopressin; been anesthetized at the time of CPA; received chest compressions while positioned in lateral recumbency; had other suspected causes of CPA (ie, did not have hemorrhage or anemia, shock, hypoxemia, MODS, cerebral trauma, a malignant arrhythmia, or an anaphylactoid reaction as the suspected cause) and were significantly less likely to have been treated by administration of multiple doses of epinephrine, had a longer duration of CPA, or had multiple disease conditions, compared with findings in dogs that were not successfully resuscitated (Table 3). Return of spontaneous circulation was achieved for 5 of 6 dogs that received corticosteroids, whereas ROSC was achieved for 51 of 155 dogs that did not receive corticosteroids. For 8 dogs that received vasopressin, 5 had ROSC, compared with 51 of 153 dogs that did not receive vasopressin.

Cats that were successfully resuscitated were significantly more likely to have had more people who participated in the resuscitation effort and were significantly less likely to have had shock as the suspected cause of CPA, compared with findings in cats that were not successfully resuscitated (Table 4). Variables that did not affect ROSC in either species included body weight, BCS, age, ASA status, method of identification of CPA, initial arrhythmia, experience level of the person who supervised the CPR effort, time that CPA occurred, or route of drug administration.

**Discussion**

Return of spontaneous circulation was achieved in 56 of 161 (35%) dogs and 19 of 43 (44%) cats in the study reported here. Resuscitation rates of 13% and 28% for dogs and 15%, 42%, and 61% for cats have been reported. For humans who had CPA during hospitalization, resuscitation rates of 46% to 61% have been reported. For dogs, the resuscitation rate in the study reported here was higher than the rates reported in other studies, which possibly reflects an improvement in the quality of care for CPA since 1992. However, the resuscitation rate for cats in the present study was not higher than the rates reported in other studies and the resuscitation rates for both dogs and cats were far lower than those reported for humans, implying that management of dogs and cats with CPA can be improved.

Survival until discharge from the hospital was accomplished in 9 dogs and 3 cats, of which 6 dogs and all 3 cats were anesthetized at the time of CPA. At our hospital, vascular access is established in anesthetized animals and they are closely monitored, at a minimum, by use of Doppler ultrasonography. Monitoring via Doppler ultrasonography enables audible detection of a cessation of blood flow or slowing of the pulse rate, which allows for rapid intervention. Furthermore, advanced monitoring techniques, such as evaluation of P_{ETCO₂}, heart rhythm via ECG, and arterial blood pressure via direct or indirect methods, are used in many anesthetized animals and also enable rapid intervention for those with CPA.

In the study reported here, 12 of 204 (6%) patients with CPA survived and were discharged from the hospital, compared with rates reported in the veterinary literature of 5%, 3%, and 22% for patients with CPA. Compared with reported rates of 15% to 32% for humans who had CPA during hospitalization and were discharged from the hospital with intact neurologic function, the rate in the study reported here for dogs and cats with CPA that survived to be discharged from the hospital was dramatically lower. In contrast to humans, who frequently have CPA secondary to ventricular fibrillation attributable to ischemic cardiac disease, dogs and cats frequently have CPA because of diseases involving multiple organ systems; therefore, their health may be more compromised at the time of arrest. Regardless, a rate of 6% for survival to be discharged from a hospital for all dogs and cats with CPA, with a rate of 1% for dogs and cats in which CPA was not related to anesthesia, represents a grave prognosis.

For patients that were anesthetized at the time of CPA, the rate of survival until discharge from the hospital (9/19 [47%]) was dramatically and significantly higher than the rate (3/185 [2%]) for patients not anesthetized at the time of CPA. For the 12 patients that survived and were discharged from the hospital, 9 had CPA while anesthetized; this proportion is higher than the proportion (18 patients survived and were discharged, of which 10 had CPA while anesthetized) in another report. This suggests that in anesthetized patients with CPA, CPR should be attempted because these patients have a fair prognosis for survival and discharge from a hospital.

A fatality rate of 8.8/10,000 anesthetized patients in the study reported here is lower than the estimated rate of 25/10,000 anesthetized patients in another report. This difference may be because anesthesia-related death has been defined as an event that happens within 48 hours after the episode of anesthesia, whereas in the study reported here, only anesthetized patients within the veterinary teaching hospital (regardless of whether they were managed by the anesthesia service) were included. It is also possible that there are differences in the clinical practice of anaesthesiology at our institution, compared with the practice at other institutions. At our teaching hospital, every anesthetized patient managed by the anesthesia service undergoing surgery is monitored by use of Doppler ultrasonography for detection
of pulses, evaluation of PETCO₂, and measurement of end-tidal concentrations of halogenated inhalant anesthetics. In addition, direct monitoring of arterial blood pressure is performed for many patients, including some of those with an ASA status of I or II. These measures may improve safety, compared with that provided by use of monitoring protocols that only include indirect monitoring of arterial blood pressure via an automated blood-pressure cuff. Monitoring of end-tidal concentrations of halogenated inhalant anesthetics appears to have improved the quality and safety of anesthesia in horses at our institution.16

None of the animals that had CPA outside of the hospital survived until discharge. This is in contrast to the discharge rate of 8.1% for humans who have CPA outside of a hospital.17 This difference may represent a more rapid response time for humans, in which the arrival of an ambulance and emergency medical technicians can enable prompt provision of emergency medical services, compared with the response time for animals. Cardiopulmonary cerebral resuscitation for humans may be initiated in an ambulance during transport, whereas CPCR is not often provided for animals during transport. Bystander-initiated CPCR is also an important component of the success rate for humans who have CPA outside of a hospital, although the success rate is significantly less than the success rate for humans who have CPA while in a hospital.18 The wide availability of automated external defibrillation machines in public places has also significantly improved ROSC for humans with CPA in certain circumstances.19 Animal CPCR courses are sporadically available, and an expansion of this education to more pet owners may result in more attempts to use CPCR in dogs and cats with CPA outside of a hospital. Currently, however, it is not recommended that CPCR be performed on animals that have CPA outside of a hospital.

For dogs, the proportion of arrhythmias in the study reported here is different from the proportion reported in another study.3 In that study,2 23% of dogs had EMD, 23% had asystole, and 20% had ventricular fibrillation, compared with 11%, 50%, and 7% of dogs, respectively, that had those arrhythmias in the study reported here. It is possible that dogs in the present study had a longer interval between the onset of CPA and the initiation of ECG recordings, which may explain the lower proportions of ventricular fibrillation and EMD and the higher proportion of asystole, compared with the proportions in the other study;3 ventricular fibrillation and EMD, when untreated, typically progress to asystole.20 Also, diagnosing EMD requires an accurate trace from an ECG, and when ECG leads are not properly attached to a dog with EMD, it may produce a trace more consistent with a diagnosis of asystole. Alternatively, the difference in proportions may represent a shift in disease states of patients that were hospitalized in the present study, compared with the disease states of patients in the report2 from 1992.

Peak PETCO₂ is an accurate predictor of death in humans with CPA,21 and this finding was true for dogs in this study. One cat that died had a peak PETCO₂ of 33 mm Hg, and this value qualified as an outlier. When the value for that cat was removed, peak PETCO₂ differed significantly (P = 0.02) between cats with ROSC and cats that were not successfully resuscitated. Monitoring of PETCO₂ is a noninvasive way of estimating blood flow through the lungs during CPA. It is easy to implement and, on the basis of the data reported here, provides objective values that a clinician can use to estimate the likelihood of ROSC. Dogs with a peak PETCO₂ < 15 mm Hg and cats with a peak PETCO₂ < 20 mm Hg are not likely to be successfully resuscitated. Treatment efforts in those patients should be changed (ie, converted from external chest compressions to internal cardiac compressions) or ceased.

Cats had higher PETCO₂ during resuscitation efforts than those values in dogs. External chest compressions in cats and other animals with body weights < 10 kg frequently produce a cardiac pump mechanism in which the strength of the compressions is transmitted directly to the heart.3 In larger animals, external chest compressions cause forward flow via a thoracic pump mechanism in which forward cardiac flow is accomplished by changes in intrathoracic pressure.2 The thoracic pump mechanism appears to be less effective at generating a high PETCO₂ during CPR, and this may reflect a less effective generation of cardiac output by use of this technique.

Several variables were significant in dogs for predicting the likelihood of ROSC. As expected, increasing the duration of CPR efforts had an inverse relationship with outcome, such that longer resuscitation efforts were less likely to result in a positive outcome.22 When time passes without effective blood flow, myocardial ischemia develops, which makes it progressively less likely to achieve successful resuscitation. Surprisingly, the number of doses of epinephrine administered also decreased the likelihood of ROSC, independent of duration of CPR efforts. This may be attributable to increased myocardial oxygen demand, which is an effect of epinephrine, coincident with decreased delivery of oxygen, thus exacerbating hypoxia and ischemia.23

Dogs with multiple disease conditions were approximately 20% as likely to be successfully resuscitated as dogs without multiple disease conditions, which is similar to findings in humans.24 This suggests that aggressive CPR efforts should not be initiated in dogs with CPA attributable to multiple disease conditions.

After controlling for other factors, dogs that received chest compressions when positioned in lateral recumbency had greater odds of achieving ROSC than dogs that received chest compressions when positioned in dorsal recumbency. This is contrary to some recommendations;4 however, the findings in the study reported here were significant. For this reason, it is recommended that CPR be performed with the animal positioned in lateral recumbency. Dogs that had a cause of arrest other than those traditionally associated with CPA were more likely to be successfully resuscitated. This may have reflected the influence of an anesthesiarelated cause in addition to vagally mediated CPA, neither of which was included as a proximate cause of CPA on the standardized form.

Administration of mannitol improved outcomes in dogs. It is recommended that mannitol be administered after successful CPR to minimize the development of cerebral edema that results from the cerebral ischemia.
associated with the CPA. It is possible that mannitol administered during CPCR efforts has some cerebral protective effects, thus improving neurologic outcome. Administration of lidocaine resulted in an improved outcome in dogs. Use of lidocaine for the treatment of ventricular tachyarrhythmias during CPA is considered an alternative treatment in humans, but it does not dramatically improve outcome. It is possible that lidocaine is not as beneficial as amiodarone, with which it has often been compared in human studies, but lidocaine may have some beneficial effects. Lidocaine is a rapid-acting sodium channel blocker that may protect against ischemia but that also may decrease the chance of successful defibrillation.

Administration of isotonic fluids improved outcomes in dogs of the study reported here. In the authors’ clinical experience, dogs in which CPA is secondary to active hemorrhage are more likely to be successfully resuscitated; however, the study reported here did not control for this disease group. Therefore, it is possible that in the present study, the effect of administration of fluids reflected a correction of a disease condition that causes hypovolemia, rather than reflecting other effects associated with fluid treatment. However, it is also possible that administration of fluids improved outcome by improving venous return and thus cardiac output. Administration of fluids via the same IV catheter into which drugs are administered also has the effect of ensuring that the drugs are adequately flushed from the catheter into a patient’s bloodstream.

Dopamine administered via continuous rate infusion improved the likelihood of ROSC in dogs in the study reported here. Dopamine is a direct-acting inotrope and also indirectly increases norepinephrine concentrations. Administration of dopamine is advocated for hypotension during the period after ROSC is achieved, and the positive inotropic effects of dopamine may have had a role in ROSC. The inotropic effects of dopamine may have been additive with the inotropic effects of epinephrine because epinephrine was commonly administered to patients that received dopamine. Pigs with CPA that receive dopamine have superior mesenteric blood flow; this may imply that dopamine infusion improves organ perfusion during CPA, which would improve prognosis.

A small number of dogs received corticosteroids. Thus, the larger proportion of dogs that had ROSC and received corticosteroids, compared with the proportion of dogs that had ROSC and did not receive corticosteroids, may be a spurious finding or may be attributable to the timing of corticosteroid administration. In the study reported here, we did not specifically assess the time during the CPCR effort when drug treatments were administered. Although every effort was made to ensure that the drug treatments recorded on the form were administered before ROSC, it is possible that some drug treatments were administered after ROSC was achieved. However, the magnitude of the effect of administration of corticosteroids during CPA suggests that this treatment deserves consideration for potential beneficial effects. It is possible that corticosteroids were administered to those patients with diseases (eg, anaphylactoid reaction or asthma) in which it was believed this treatment would be beneficial, which would explain the beneficial effect. Hydrocortisone given at the time of arrival in the emergency department significantly improves ROSC in humans who have CPA outside of a hospital. Indiscriminate use of corticosteroids may not yield the same results as those in the study reported here. However, given the relatively low cost of corticosteroids and low morbidity associated with administration of a single dose of short-acting corticosteroids during CPCR, this treatment in selected patients with CPA appears warranted.

Treatment with vasopressin improved the likelihood of ROSC in dogs. Although vasopressin has provided promising results in animals used to evaluate CPCR, in large clinical trials of humans who had CPA outside of a hospital, a benefit of administering vasopressin rather than epinephrine was not detected. However, vasopressin is superior to epinephrine for the treatment of humans with CPA who have an initial arrhythmia of asystole. Because most animals in the study reported here and in other studies have an initial arrhythmia of asystole or EMD, the beneficial effects of vasopressin are in agreement with findings in humans and in other animals used for evaluation of CPCR. Vasopressin was only available at our hospital during the final 12 months of this study, so it is possible that the incidence of ROSC may have increased if more patients had received vasopressin during the study period. Given these findings, vasopressin may be beneficial when used in addition to epinephrine during CPCR in dogs.

In contrast to the results for dogs, relatively few independent variables predicted the likelihood of ROSC in cats. The number of cats in the present study was small, considering the number of covariates and compared with those for the dogs. This small number decreased the power of the statistical model for cats and could possibly have been a reason there were relatively few independent variables in the model. Cats with CPA attributable to shock had poorer odds for ROSC, compared with the odds for ROSC in cats with CPA attributable to other reasons. This implies that cats with shock have a grave prognosis and should be treated aggressively to prevent progression to CPA.

Increasing the number of people who participated in a CPCR effort improved the likelihood for ROSC in cats, but not in dogs. This finding appears contradictory because in the authors’ clinical experience, CPCR efforts in dogs require more people to allow exchange of individuals to enable effective performance of chest compressions. This finding was also independent of time of day and experience level of the person who supervised the CPCR effort; these variables would be expected to impact outcome and be associated with the number of people who participated in a CPCR effort. It is possible that a larger resuscitation team reflected more individuals that were present when a cat initially had CPA, thus decreasing the interval from onset of CPA to initiation of CPCR efforts.

No significant effect was detected for the experience level of the veterinarian supervising the CPCR effort (ie, intern, resident, or faculty member). This is inconsistent with evidence from human medicine, in which more experienced resuscitation teams have im-
proved outcomes, compared with outcomes for less experienced teams. It may be that in veterinary medicine, the treatments are sufficiently straightforward such that there is not a difference in outcome based on experience level. It may also reflect the severity of disease in patients with CPA: no matter the experience level of the supervisor, the patient will die.

In the study reported here, we identified several clinical variables and treatments significantly associated with the likelihood of ROSC in dogs and cats with CPA. However, the prognosis for survival and discharge from the hospital was grave for all patients, except for those that had CPA while anesthetized. Clients should be made aware of the grave prognosis associated with CPA. The decision to perform CPRC should be determined by the supervising clinician, who should discuss the prognosis with owners and avoid futile CPRC efforts in extremely ill patients.

a. Copies available from author on request

References