Indications for and outcome of positive-pressure ventilation in cats: 53 cases (1993–2002)

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Objective—To determine indications for and outcomes of positive-pressure ventilation (PPV) in cats, document ventilator management, and identify factors associated with outcome.

Design—Retrospective study.

Animals—53 cats that underwent PPV.

Procedure—Information on signalment, history, concurrent diseases, clinical findings, results of venous blood gas analyses and clinicopathologic testing, treatment, ventilator settings, and outcome was retrieved from the medical records. Data for cats that survived were compared with data for cats that died or were euthanatized while undergoing PPV.

Results—PPV was initiated for management of respiratory failure (36 cats [68%]), cardiac arrest (9 [17%]), neurologic impairment (6 [11%]), and nonresponsive hypotension (2 [4%]). Eight cats (15%) survived, 19 (36%) died, and 26 (49%) were euthanatized while undergoing PPV. Cats that survived had a longer duration of ventilation than did those that died or were euthanatized and had a significantly higher incidence of ventilator-associated pneumonia. Signalment and ventilator settings were not associated with outcome. Cats that had no clinical evidence of pulmonary disease but required PPV because of primary neurologic disease had a higher survival rate (2/6) than did cats that required PPV because of respiratory failure (5/36), cardiac arrest (1/9), or nonresponsive hypotension (0/2).

Conclusions and Clinical Relevance—Results suggest that the survival rate for cats requiring PPV may be lower than reported survival rates for dogs. Death was attributable to progressive respiratory failure, nonresponsive hypotension, kidney failure, or neurologic impairment. (*J Am Vet Med Assoc* 2005;226:924–931)

Positive-pressure ventilation (PPV) is an invasive method of respiratory support that requires intensive monitoring and sophisticated instrumentation.[1] It provides support for 2 aspects of pulmonary function: elimination of carbon dioxide and oxygenation of arterial blood. The clinical decision to use PPV is always made on the basis of the condition of each individual patient, but guidelines suggest that PPV should be ini-

tiated if the **arterial partial pressure of carbon dioxide ($PaCO_2$)** is > 50 mm Hg or the **arterial partial pressure of oxygen (PaO_2)** is < 50 mm Hg while the patient is receiving oxygen supplementation.[2] Because some ventilator modes assume most or all of the **work of breathing (WOB)**, PPV may also be used in patients that are developing signs of respiratory muscle fatigue, even if arterial blood gas values have not yet deteriorated. Positive-pressure ventilation improves pulmonary function by increasing the **tidal volume (V_T)** and recruiting atelectatic alveoli, particularly if **positive end-expiratory pressure (PEEP)** is maintained.[3]

Even if they initially have normal lungs, spontaneously breathing animals with severe hypotension, cardiovascular collapse, or neurologic disease may not ventilate or oxygenate optimally and may expend considerable amounts of energy on respiratory muscle function. In such animals, implementation of PPV under heavy sedation allows more intensive continuous monitoring; eliminates the risk of respiratory arrest; ensures that PaO_2 and $PaCO_2$ are maintained within acceptable limits; and, by assuming all of the WOB, prevents the expenditure of valuable energy on respiration. Therefore, even though it does not improve hemodynamic function, PPV can be used to minimize the risk of cardiac arrest in animals with cardiovascular collapse and in animals that have been resuscitated following respiratory arrest or previous cardiac arrest. Finally, because hyperventilation can cause cerebral vasoconstriction, PPV can also be used to treat high intracranial pressure.

Although there are many experimental studies of PPV in dogs and cats, little information is available about ventilator management and outcomes in companion animals with naturally occurring disease. A few case series and individual case reports[1,3-14] describing dogs and cats that underwent PPV have been published, but little specific information about PPV in cats is available. For instance, a previous study[1] described 34 dogs and 7 cats that underwent PPV but did not distinguish between the 2 species with regard to management, complications, and outcome. The overall survival rate in that study was 39%, with higher mortality rates in animals < 1 year or > 11 years old, animals that weighed < 10 kg (22 lb), animals that developed PPV-related complications, and animals that required PPV because of severe lung disease. A more recent study[5] documented a 50% survival rate in 40 dogs and cats that required PPV for > 24 hours because of ventilatory failure but an 11% survival rate in 45 dogs and cats that required PPV for > 24 hours because of hypoxemic respiratory failure. Other studies have documented a 30% survival rate in dogs with severe pulmonary

contusions resulting from trauma[3] and a 71% survival rate in dogs with cervical spinal cord injury.[4]

The purpose of the study reported here was to determine indications for and outcomes of PPV in cats, document ventilator management, and identify factors associated with outcome.

Criteria for Selection of Cases

The computer database of the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania was searched to identify cats examined between 1993 and 2002 that underwent PPV. Cats were eligible for inclusion in the study if PPV had been used for respiratory support in the emergency room or intensive care unit and a complete medical record was available for analysis. Cats were excluded if PPV had been used during anesthesia for a surgical or diagnostic procedure or if the medical record was incomplete.

Procedures

The medical records were reviewed, and information on signalment, history, clinical findings, results of clinicopathologic testing and diagnostic imaging, management, and outcome was recorded. If available, results of venous blood gas analyses performed within 4 hours prior to the institution of PPV were recorded to evaluate pulmonary function while the cat was breathing spontaneously.

Cats were assigned to 1 of 4 categories (ie, respiratory failure, cardiac arrest, neurologic impairment, and nonresponsive hypotension) on the basis of the underlying reason why PPV was initiated. If > 1 reason was identified for initiating PPV, the cat was assigned to a category on the basis of the primary underlying condition. Cats were considered to have required PPV because of respiratory failure on the basis of compatible clinical signs (ie, dyspnea, tachypnea, and abnormal auscultation findings), results of pulse oximetry, **venous partial pressure of carbon dioxide ($PvCO_2$)**, and results of thoracic radiography or if they had experienced agonal breathing or respiratory arrest. Cats were considered to have required PPV as a result of cardiac arrest if they had undergone cardiopulmonary resuscitation following cardiac arrest, defined as the absence of auscultable heartbeats. Cats were considered to have required PPV because of neurologic impairment if ventilation was inadequate secondary to primary CNS dysfunction. Cats were considered to have required PPV because of nonresponsive hypotension if after surgery, blood pressure, measured by means of Doppler sphygmomanometry, remained < 80 mm Hg despite treatment with catecholamines and fluids.

Serial measurements of rectal temperature, heart rate, blood pressure measured by means of Doppler sphygmomanometry, urine output, and **oxygen saturation determined by means of pulse oximetry (SaO_2)** obtained after initiation of PPV were recorded, along with **fractional inspired oxygen concentration (FIO_2)**, **peak inspiratory pressure (PIP)**, mean airway pressure, PEEP, respiratory rate, inspiratory time, inspiratory flow rate, and V_T. To facilitate manipulation of the data, mean or median values were calculated for each 6-hour period of PPV for up to 72 hours for each of these parameters.

Complications associated with PPV, such as pneumothorax and ventilator-associated pneumonia, were also recorded. A diagnosis of pneumothorax was made on the basis of radiographic findings or if air was obtained by means of thoracocentesis after initiation of PPV. Ventilator-associated pneumonia was diagnosed if bacterial culture of an endotracheal lavage sample taken after initiation of PPV yielded bacterial growth or if there was radiographic evidence of worsening alveolar pulmonary disease after initiation of PPV. Cats for which results of bacterial culture of an endotracheal lavage sample were positive prior to initiation of PPV or with primary lung disease prior to initiation of PPV were not included in this category unless a change in bacterial flora was documented.

The medical record of each cat was analyzed to identify organ failure that developed after the initiation of PPV and was, therefore, thought to contribute to the death of the cat. Organ failure was defined as cardiac arrest, nonresponsive hypotension (ie, blood pressure < 80 mm Hg despite treatment with catecholamines and fluids), respiratory failure (ie, progressive respiratory dysfunction as evidenced by decreased SaO_2; increased $PvCO_2$, PEEP, FIO_2, respiratory rate, or PIP; or a change in the ratio of inspiratory to expiratory time), neurologic dysfunction (ie, decreased neurologic function as evidenced by negative results for a brainstem auditory evoked response test; fixed, dilated, nonresponsive pupils; or a lack of need for anesthesia to maintain airway intubation), and renal dysfunction (ie, development of oliguria, defined as urine output ≤ 0.5 mL/kg/h [0.23 mL/lb/h], or anuria despite treatment to maintain hydration and arterial blood pressure and administration of diuretics). Cats that developed anuria secondary to hypotension were considered to have both renal dysfunction and nonresponsive hypotension.

Cats were considered to have survived if they were weaned off the ventilator. Cats were considered to have not survived if they died or were euthanatized while still receiving PPV. With the exception of blood glucose concentration, there were no significant differences between cats that died and cats that were euthanatized. Therefore, these cats were grouped as nonsurvivors for subsequent analyses.

Statistical analyses—Normally distributed data were represented as mean ± SD, and values for cats that survived were compared with values for cats that did not survive by use of t tests and 1-way ANOVA. Nonparametric data were represented as median and range, and values for cats that survived were compared with values for cats that did not survive by use of Wilcoxon rank sum or Kruskal-Wallis tests. All analyses were performed with standard software[a]; values of $P < 0.05$ were considered significant.

Results

Fifty-three cats met the criteria for inclusion in the study. Of these, 8 (15%) survived and 45 (85%) died or were euthanatized while undergoing PPV.

Cats ranged from 0.2 to 16.7 years old (median, 8.9 years). There was no significant difference in age

between cats that survived (median, 5.45 years; range, 0.55 to 15 years) and cats that died or were euthanatized (median, 8.9 years; range, 0.2 to 16.7 years). Twenty-nine (55%) of the cats were castrated males, 20 (38%) were spayed females, 2 (4%) were sexually intact males, and 2 (4%) were sexually intact females. Mean ± SD body weight was 4.9 ± 1.7 kg (10.8 ± 3.7 lb; range, 2.5 to 10 kg [5.5 to 22 lb]). Forty-one cats (77%) were domestic shorthairs, and 5 (9%) were Persians. The remaining 7 cats included a Himalayan, Serval, Maine Coon, Lynx point, Angora, Siamese, and domestic longhair. There were no significant differences between survivors and nonsurvivors with regard to sex distribution, body weight, or breed distribution.

Indications for PPV—The underlying reason for initiation of PPV was identified as respiratory failure in 36 (68%) cats, cardiac arrest in 9 (17%), neurologic impairment in 6 (11%), and nonresponsive hypotension in 2 (4%).

An attempt was made to categorize cats that required PPV because of respiratory failure according to the primary underlying lesion. Ten of the 36 (28%) cats had parenchymal lung disease (toxoplasmosis pneumonia [n = 3], bacterial pneumonia [2], acute respiratory distress syndrome with diabetic ketoacidosis [2], severe anaphylactic reaction to a vaccine [1], pulmonary thromboembolism [1], and lung disease of unknown cause [1]), 8 (22%) had pulmonary edema secondary to cardiac disease (endomyocarditis [7] and congestive heart failure [1]), and 4 (11%) had respiratory failure secondary to trauma (vehicular trauma [3] and high-rise fall [1]). Seven (19%) cats with respiratory failure had pleural space disease (mediastinal lymphosarcoma [n = 2], chylothorax [2], pyothorax [1], pleural effusion of unknown cause [1], and congestive heart failure [1]); 4 (11%) had upper airway obstruction (tracheal neoplasia [2], necroulcerative tracheitis [1], and bronchial mass [1]); 1 (3%) had hypoventilation associated with hypokalemia attributed to underlying renal disease; 1 (3%) had respiratory arrest associated with hyponatremia and seizures following caesarean section; and 1 (3%) had combined respiratory and cardiac arrest secondary to renal failure, hyperthyroidism, and pemphigus foliaceus.

Five (14%) of the 36 cats that required PPV because of respiratory failure survived. Cats that survived had pulmonary contusions secondary to vehicular trauma, pneumonia, tracheal neoplasia (squamous cell carcinoma), endomyocarditis, and hypokalemia-induced hypoventilation. Four cats were discharged from the hospital; the cat with hypokalemia-induced hypoventilation was weaned off the ventilator but was euthanatized 2 days later at the owner's request.

Six of the 9 cats that required PPV because of cardiac arrest had undergone surgery (balloon dilation of an esophageal stricture, endoscopic cystotomy, thoracotomy for tracheal mass removal, ventral bulla osteotomy, lung lobectomy for pulmonary adenocarcinoma, and cholecystojejunostomy) and had cardiac arrest in the postoperative period. The remaining 3 had not undergone surgery, and cardiac arrest occurred during venipuncture, secondary to pyrethrin toxicosis, and during catheter placement.

Only 1 of the 9 cats that underwent PPV because of cardiac arrest survived. This cat received PPV for 18 hours after cardiac arrest secondary to pyrethrin toxicosis.

Underlying abnormalities in the 6 cats that required PPV because of neurologic impairment included brainstem tumor removal, laryngeal paralysis, and aspiration pneumonia (n = 1); cerebellar herniation following tetralogy of Fallot surgery (1); postoperative craniotomy (2); *Cryptococcus neoformans* infection (1); and meningioma and lymphosarcoma (1). Two of these 6 cats survived. One of these was the cat that required PPV following brainstem tumor removal, laryngeal paralysis, and aspiration pneumonia; the other was a cat that underwent bulla osteotomy and craniotomy because of meningioma and lymphosarcoma.

One of the 2 cats that required PPV because of nonresponsive hypotension had undergone splenectomy; the other had undergone exploratory surgery because of cholangitis. Neither cat survived.

Clinicopathologic testing prior to PPV—Results of venous blood gas analyses performed within 4 hours prior to initiation of PPV were available for 21 cats. Mean ± SD venous pH was 7.23 ± 0.14 (range, 7.02 to 7.53; reference range, 7.30 to 7.40), mean Pv_{CO_2} was 56.2 ± 22 mm Hg (range, 23.6 to 110.5 mm Hg; reference range, 33 to 43 mm Hg), and mean base excess was –4.4 ± 7.8 mmol/L (range, –25 to 7.5 mmol/L; reference range, –1 to –7 mmol/L). Median venous lactate concentration was 2.0 mmol/L (n = 11; range, 0.7 to 16.4 mmol/L; reference range, 1 to 2 mmol/L).

Median PCV was 31% (n = 50; range, 13% to 65%; reference range, 32% to 48%), and median total solids concentration was 6.1 g/dL (n = 50; range, 3.8 to 9.3 g/dL; reference range, 6 to 8.6 g/dL). Mean ± SD plasma sodium concentration was 148.5 ± 7.3 mmol/L (n = 20; range, 137 to 167.5 mmol/L; reference range, 148 to 157 mmol/L), median plasma potassium concentration was 3.63 mmol/L (n = 20; range, 2.4 to 7.6 mmol/L; reference range, 3.6 to 4.6 mmol/L), and mean plasma ionized calcium concentration was 1.06 ± 0.18 mmol/L (n = 20; range, 0.7 to 1.34 mmol/L; reference range, 1.1 to 1.22 mmol/L). Mean plasma glucose concentration was 214 ± 113 mg/dL (n = 20; range, 25 to 468 mg/dL; reference range, 67 to 168 mg/dL).

Results of clinicopathologic testing prior to initiation of PPV were not significantly different between cats that survived, cats that died, and cats that were euthanatized, except that plasma glucose concentration for cats that survived (n = 4; median, 173 mg/dL; range, 107 to 197 mg/dL) was significantly lower than the concentration for cats that were euthanatized (9; median, 272 mg/dL; range, 190 to 423 mg/dL). Cats that died had a median serum glucose concentration of 116 mg/dL (n = 7; range, 25 to 468 mg/dL).

Ventilator variables—Duration of PPV ranged from 0.75 to 176 hours (median, 11 hours). Median duration of ventilation was significantly (P = 0.002) longer for survivors (n = 8; median, 32 hours; range, 12 to 176 hours) than for nonsurvivors (45; median, 7.5 hours; range, 0.75 to 65.5 hours).

During the first 6 hours of PPV, mean ± SD F_{IO_2} was 0.77 ± 0.22 (n = 45; range, 0.4 to 1.0); for hours 6

through 12, mean F_{IO_2} was 0.59 ± 0.24 (n = 24; range, 0.24 to 1.0); for hours 12 through 18, mean F_{IO_2} was 0.52 ± 0.18 (n = 18; range, 0.26 to 0.9); for hours 18 though 24, median F_{IO_2} was 0.45 (n = 13; range, 0.25 to 0.9); for hours 24 through 48, mean F_{IO_2} was 0.51 ± 0.19 (n = 9; range, 0.34 to 0.9), and for hours 48 through 72, median F_{IO_2} was 0.46 (n = 5; range, 0.4 to 0.76). Mean F_{IO_2} was not significantly different between survivors and nonsurvivors during the 72 hours of ventilation.

During the first 6 hours of PPV, mean $\pm$ SD PIP was 22.5 ± 8.4 cm H_2O (n = 37; range, 8.5 to 41 cm H_2O), mean airway pressure was 8.8 ± 4.4 cm H_2O (n = 36; range, 1 to 19 cm H_2O), and median PEEP was 3.15 cm H_2O (n = 38; range, 0 to 11 cm H_2O). Mean inspiratory flow rate was 8.6 ± 2.5 L/min (n = 38; range, 4 to 13 L/min), median inspiratory time was 1 second (n = 36; range, 0.5 to 2 seconds), and mean set respiratory rate was 24 ± 7 breaths/min (n = 45; range, 13 to 43 breaths/min). Mean V_T recorded on the ventilator during the first 6 hours of PPV was 116 ± 42 mL (n = 29; range, 29 to 187 mL), which was equivalent to 23.7 ± 8.6 mL/kg (10.8 ± 3.9 mL/lb). None of these values changed significantly during the 72 hours of ventilation, and none differed significantly between survivors and nonsurvivors.

The mode of ventilation was recorded in 18 cats. Volume-limited ventilation was used in 15 cats, and pressure-limited ventilation was used in 3. Cats were ventilated in assist-control or synchronized intermittent mandatory ventilation modes.

Airway management and sedation—In all cats, endotracheal intubation was performed to allow PPV. In 1 cat, a tracheostomy tube was also used because of severe maxillary and mandibular trauma. Forty-seven cats were anesthetized during PPV. Anesthetic agents that were used included fentanyl (n = 28), diazepam (19), hydromorphone or oxymorphone (12), pentobarbital (11), ketamine (6), and propofol (5). In 31 cats, > 1 drug was used for sedation. In addition to sedation, 13 cats required neuromuscular blockade; agents used included cisatracurium, atracurium, pancuronium, and vecuronium. Only 1 of the cats that required neuromuscular blockade (cisatracurium) survived; this cat had a tracheal tear after being hit by a car. Six cats did not require any sedation or neuromuscular paralysis to tolerate endotracheal intubation. Four of these cats had been resuscitated following cardiac arrest, and 2 had undergone craniotomy. One of the 2 cats that had undergone craniotomy survived; the remaining 5 cats died or were euthanatized.

Monitoring—Mean $\pm$ SD rectal temperature during the first 6 hours of PPV was $36.4 \pm 1.6°C$ ($97.6 \pm 2.8°F$; n = 47; range, $32.8°$ to $38.9°C$ [$91.3°$ to $102.6°F$]), and mean heart rate was 174 ± 36 beats/min (n = 50; range, 93 to 250 beats/min).

During PPV, attempts were made to maintain blood pressure, measured by means of Doppler sphygmomanometry, > 90 mm Hg through administration of catecholamines and fluids as necessary. Thirty-one of the 53 cats (58%) required administration of catecholamines, including 1 or more of the following drugs:

epinephrine (n = 19), dopamine (18), phenylephrine (3), and dobutamine (1). Nine of the 31 cats required administration of > 1 catecholamine. During the first 6 hours of PPV, mean $\pm$ SD blood pressure was 95 ± 24 mm Hg (n = 46; range, 49 to 149 mm Hg); for hours 6 through 12, mean blood pressure was 101 ± 28 mm Hg (n = 28; range, 45 to 154 mm Hg); for hours 12 though 18, mean blood pressure was 101 ± 24 mm Hg (n = 24; range, 30 to 152 mm Hg); for hours 18 through 24, mean blood pressure was 100 ± 24 mm Hg (n = 15; range, 69 to 160 mm Hg); for hours 24 through 36, mean blood pressure was 98 ± 19 mm Hg (n = 9; range, 65 to 133 mm Hg), and for hours 36 through 48, median blood pressure was 96.4 mm Hg (n = 6; range, 77 to 150 mm Hg). There were no significant differences between survivors and nonsurvivors in regard to rectal temperature, heart rate, or blood pressure at any time period.

Pulse oximetry was performed continuously during PPV, and ventilator settings and F_{IO_2} were adjusted to maintain SaO_2 > 93%. In 6 cats, SaO_2 could not be measured during the initial 6 hours of PPV because the signal was inadequate. For the remaining cats, median SaO_2 during this time was 93% (n = 47, range, 73% to 99%). Median SaO_2 during this period for survivors (n = 6; 94.8%; range, 90% to 99%) was not significantly different from median value for nonsurvivors (41; 92%; range, 73% to 99%).

Complications of PPV—Fifteen of the 53 cats (28%) developed pneumothorax during PPV, of which 3 survived. Seven of 16 cats with PIP > 25 cm H_2O developed pneumothorax, as did 8 of 20 cats with PIP < 25 cm H_2O. Cats with PIP > 25 cm H_2O included 9 with pulmonary parenchymal disease, 4 with pleural disease, 1 with upper airway obstruction, 1 with cardiac arrest, and 1 with nonresponsive hypotension. Pneumothorax occurred in 12 of 36 cats that underwent PPV because of respiratory failure, 1 of 9 cats that underwent PPV because of cardiac arrest, 1 of 6 cats that underwent PPV because of neurologic impairment, and 1 of 2 cats that underwent PPV because of nonresponsive hypotension. There was no significant association between PIP > 25 cm H_2O and development of pneumothorax or between presence of pulmonary parenchymal disease and development of pneumothorax.

Results of bacterial culture of endotracheal lavage fluid samples were available for 15 cats. A single organism was isolated from 7 cats, and multiple organisms were isolated from 7 cats. In the remaining cat, bacterial culture did not yield any growth. Organisms identified included *Escherichia coli* (n = 10), *Acinetobacter* spp (6), *Enterobacter cloacae* (2), α-hemolytic *Streptococcus* spp (1), *Klebsiella* spp (1), *Bordetella bronchiseptica* (1), *Staphylococcus* spp (1), *Enterococcus* spp (1), and *Pseudomonas* spp (1). Eight of the 53 cats (15%) fulfilled the strict criteria for diagnosis of ventilator-associated pneumonia. Ventilator-associated pneumonia was identified in 4 of the 8 cats that survived and 4 of the 45 cats that did not survive. Incidence of ventilator-associated pneumonia was significantly ($P = 0.013$) higher in survivors than in nonsurvivors.

Outcome—Eight (15%) cats survived, 19 (36%) died, and 26 (49%) were euthanatized while undergoing PPV. Of the 19 cats that died of cardiac arrest while being ventilated, 10 had nonresponsive hypotension, 8 had progressive respiratory dysfunction, 3 had neurologic dysfunction, and 3 had renal dysfunction. Of the 26 cats that were euthanatized, 15 had progressive respiratory dysfunction, 7 had nonresponsive hypotension, 7 had neurologic dysfunction, 3 had infectious diseases, and 2 had renal dysfunction. Three of the nonsurvivors did not have any evidence of primary organ failure and were euthanatized at the owner's request. Of the 8 survivors, 7 had no evidence of organ failure while undergoing PPV. One cat had hypotension and neurologic deterioration but improved with supportive care.

Discussion

In the present study, we identified 53 cats that underwent PPV during a 9-year period, of which 8 (15%) recovered and were weaned from the ventilator. In contrast, 3 previous studies[1,3,4] from our institution that primarily included dogs documented survival rates of 39%, 30%, and 70%. Thus, our results suggest that cats requiring PPV may have a lower survival rate than previously reported for dogs. In previous studies[1,3] involving dogs, the mortality rate was higher among smaller animals. Because of their small size, cats may be technically and mechanically more difficult to ventilate and monitor, which may help to explain the high mortality rate found in the present study. In a previous study[1] involving dogs and cats, very young and older animals had higher mortality rates, whereas in the present study, we did not identify any association between age and outcome.

The decision to initiate PPV in the cats in the present study was made on the basis of clinical evidence of pulmonary or cardiovascular system failure, with PPV initiated because of respiratory failure in 36 (68%) cats, cardiac arrest in 9 (17%), hypoventilation associated with CNS impairment in 6 (11%), and nonresponsive hypotension in 2 (4%). Similarly, indications for PPV in 7 cats described in a previous study[1] included cardiac arrest (n = 3), acute upper airway obstruction following mandibular surgery and bulla osteotomy (2), hypoventilation secondary to hyperosmolar diabetic coma (1), and primary lung parenchymal disease (1). The relatively larger number of cats in the present study that underwent PPV because of primary lung disease may represent a change in attitude toward cats, a better understanding of feline physiology, or an increase in proficiency in PPV in the intervening years between studies. Despite changes in attitudes and expertise, the decision to initiate PPV in the present study appeared to have been made when cats were in the late stages of disease, with PPV initiated in many cats after cardiac arrest, and this likely contributed to the poor outcome.

In awake animals with respiratory distress, especially cats with dyspnea, there are few safe and practical ways to assess pulmonary and cardiovascular function. Diagnostic testing and even physical examination may be limited because such patients often do not tolerate handling and manipulation. The clinical diagnosis of respiratory failure is typically quite obvious, and the decision to initiate PPV is logical if the animal is experiencing severe dyspnea. In critically ill humans, the metabolic cost of breathing can be as high as 25% to 50% of total oxygen consumption,[15] compared with 2% of total oxygen consumption in healthy humans. Therefore, in addition to improving pulmonary gas exchange, by assuming most or all of the WOB, PPV might allow energy to be redirected towards other vital body functions. Little information was available about the cardiovascular status of many of the cats included in this study until after they had been anesthetized and intubated for PPV.

Collection of arterial blood samples is difficult in cats and associated with unacceptable risks in cats with dyspnea that are not anesthetized. Thus, results of analyses of venous blood gas samples collected prior to initiation of PPV were used to assess ventilation. Although $PvCO_2$ is always higher than $PaCO_2$, the difference is usually only 4 to 6 mm Hg.[16] However, high $PvCO_2$ may also result from hypoperfusion and is not specific for hypoventilation. Moderate hypoventilation may have been present in most cats in the present study prior to initiation of PPV, as evidenced by high $PvCO_2$ and acidemia, but values did not differ significantly between survivors and nonsurvivors. Results of clinicopathologic testing prior to initiation of PPV were not significantly different between cats that survived, cats that died, and cats that were euthanatized, except that plasma glucose concentration for cats that survived was significantly lower than the concentration for cats that were euthanatized, possibly representing more severe stress hyperglycemia in cats that were euthanatized.

Even in anesthetized cats, arterial catheters are technically difficult to place and maintain. Thus, in most cats in the present study, decisions about ventilator settings were made on the basis of results of venous blood gas analyses, pulse oximetry, and end-tidal capnography. Ventilator settings were chosen to maintain SaO_2 values > 93%. During the first 6 hours of PPV, mean FiO_2 was 0.77, but this was decreased to 0.59 during the next 6 hours. In cats that survived to the second and third days of PPV, the FiO_2 was further decreased as attempts were made to wean cats off the ventilator. There were no significant differences in SaO_2 or FiO_2 between survivors and nonsurvivors, but this finding is difficult to interpret in light of the small number of animals. Ventilator settings for airway pressures, PEEP, and V_T were adjusted to maintain an optimal SaO_2 while using the lowest possible FiO_2, which may also have masked differences between groups.

Respiratory rates during PPV for cats in the present study were within the physiologic range. However, mean V_T (23.7 mL/kg) was higher than values traditionally (10 to 15 mL/kg [4.5 to 6.8 mL/lb]) or currently (6 to 10 mL/kg [2.7 to 4.5 mL/lb]) recommended.[17] However, reported V_T values may have been higher than the actual volumes delivered as these values were obtained from the digital readout of the ventilator, rather than by spirometry. Compliance of the ventilator tubing likely resulted in overestimation of the volume actually delivered to the patient.

During PPV, airway pressures are typically monitored continuously to provide an estimate of pulmonary mechanics and prevent barotrauma. Assuming that V_T is not excessive, higher airway pressures reflect poor lung compliance or increased airway resistance secondary to lung disease.[1] It is recommended that PIP be maintained < 25 cm H_2O to minimize the risk of barotrauma and volutrauma.[17-19] For cats in the present study, mean ± SD PIP was 22.5 ± 8.4 cm H_2O, indicating that some cats had a higher PIP than recommended. These cats likely had decreased lung compliance and more severe pulmonary abnormalities, although the high V_T may also have contributed to the high PIPs.

A previous study[3] in dogs that underwent PPV found that PIP increased gradually in nonsurvivors as a result of worsening lung mechanics, whereas in survivors, PIP tended to remain stable or decrease. In the cats in the present study, however, PIP and V_T were not significantly different between survivors and nonsurvivors. Barotrauma and volutrauma associated with excessively high PIPs (> 40 cm H_2O) is one of the primary adverse effects of PPV. Alveolar overdistension can result in increased microvascular permeability and an inflammatory response, along with pulmonary interstitial emphysema, pneumomediastinum, pneumoretroperitoneum, or pneumothorax.[14,20-22] In the present study, pneumothorax developed in 15 (28%) cats. However, development of pneumothorax was not significantly associated with outcome, PIP, or detection of pulmonary parenchymal disease. This contrasts with results of a previous study,[1] which found that development of pneumothorax was associated with a lower survival rate. The low number of cats in the present study may have masked a true significant difference.

Another potential adverse effect of PPV is oxygen toxicosis, which can occur if patients are maintained on 100% oxygen for > 18 to 24 hours.[23-25] Oxygen toxicosis results from free radical formation, which causes alveolar inflammation and leakage of proteinaceous fluid, and atelectasis associated with alveolar nitrogen washout.[26-28] Although F_{IO_2} was high at the beginning of PPV, in most cats the F_{IO_2} was decreased to safer values (ie, < 0.60) within 12 hours, thereby minimizing the risk of oxygen toxicosis. If hypoxemia still exists despite high F_{IO_2}, PEEP can be used to increase functional residual capacity and recruit alveoli. Whereas use of PEEP may result in higher PIPs, preventing the cycle of repeated alveolar recruitment and collapse reduces ventilator-induced lung injury.[17] In the present study, high PEEPs (> 10 cm H_2O) were not used in most cats and only 10 cats were ventilated with PEEP between 8 and 9 cm H_2O.

Progressive worsening of respiratory tract disease was a primary contributor to outcome in 23 of the 45 (51%) cats that did not survive in the present study. Other causes of death included cardiac arrest (19/45 [42%]), nonresponsive hypotension (17/45 [38%]), neurologic dysfunction (10/45 [22%]), and renal dysfunction (5/45 [11%]). In most instances, it was impossible to determine whether nonresponsive hypotension was a result of the underlying disease or the PPV in these cats. Most often, blood pressure could not be measured prior to induction of PPV but preexisting hypotension was likely. On the other hand, animals with previously adequate cardiovascular function may have decreases in blood pressure after initiation of PPV. Positive-pressure ventilation decreases venous return to the heart as a result of increased intrathoracic pressure, right ventricular dysfunction, altered left ventricular distensibility, and low cardiac output.[29] These effects are exaggerated when PEEP, which acts to increase intrathoracic pressure, is used.[30-33]

Cats that required PPV following cardiac arrest or to assist in management of nonresponsive hypotension generally had poor outcomes. Although the Doppler blood pressure measurements that we reported here during PPV did not appear to be low (< 90 mm Hg), 31 (58%) cats required administration of catecholamines to maintain adequate blood pressures. Furthermore, cats in which blood pressure was so low that the Doppler device could not detect blood flow were not included in the calculation of mean blood pressure. As a result, the reported mean value was an overestimation of the actual mean value for all of the cats. Results of a previous study[1] confirm that cardiovascular collapse is a poor prognostic indicator in animals undergoing PPV. In that study, only 2 of 21 animals with hypotension during PPV survived, despite fluid therapy and administration of vasopressors. In the present study, all but 3 of the 31 cats that received catecholamines died or were euthanatized.

In the present study, median duration of ventilation was significantly longer for cats that survived than for cats that died or were euthanatized. Subjectively, cats that maintained adequate cardiovascular stability appeared to survive longer and, therefore, have a greater chance of recovering pulmonary or neurologic function and being weaned off the ventilator. Death secondary to cardiovascular collapse, if it occurred, often happened relatively soon after the initiation of PPV (< 48 hours).

Failure of other organs often follows cardiovascular collapse and hypotension, and occurred frequently in nonsurviving cats in the present study. Ten of the 45 (22%) cats that did not survive had progressive neurologic deterioration as evidenced by a lack of brainstem auditory evoked responses and the presence of fixed, dilated, nonresponsive pupils or the lack of a need for anesthesia to maintain airway intubation. Cerebral hypoxia resulting from the underlying disease, rather than PPV itself, appeared to be responsible for this progression, although we cannot rule out the possibility that PPV contributed to the neurologic deterioration.

Decreased urine output was documented in 5 of the 45 (11%) cats that did not survive in the present study, even though attempts were made to optimize hydration and blood pressure in all cats through administration of crystalloid and colloid fluids and vasopressors. Diuresis with furosemide or mannitol was attempted when a cat was determined to have oliguria or anuria, and cats were only included in this category if they failed to respond to diuresis. Clearly, oliguria can be a physiologically appropriate response, but if severe hypotension is ongoing and accompanied by PPV, intrinsic renal failure secondary to decreased renal

perfusion and acute tubular necrosis is a predictable consequence.[34,35] In these patients, it is difficult to distinguish appropriate oliguria from oliguria secondary to intrinsic renal failure if the hypotension is not responsive to treatment.

Sepsis and septic shock are common complications in critically ill patients that require PPV. Such patients are often profoundly debilitated and immunosuppressed prior to initiation of PPV, and placement of an endotracheal tube bypasses the normal airway defense mechanisms, predisposing patients to development of ventilator-associated pneumonia. Ischemia of the gastrointestinal tract mucosa secondary to high gastric venous pressure can result in bacterial translocation, and sepsis may develop as a sequela of pneumonia or bacterial translocation.

In the present study, bacterial growth was obtained from endotracheal lavage fluid samples from 14 cats but only 8 cats fulfilled the strict criteria for a diagnosis of ventilator-associated pneumonia. The incidence of ventilator-associated pneumonia was probably underestimated because endotracheal lavage was performed in few cats and it was often difficult to distinguish radiographic changes of pneumonia from those associated with an underlying primary pulmonary condition. Ventilator-associated pneumonia occurred in a significantly higher percentage of survivors (4/8 [50%]) than nonsurvivors (4/45 [9%]), which likely reflected the longer duration of PPV in cats that survived.

General recommendations to minimize the risk of ventilator-associated pneumonia include maintenance of aseptic technique during all interventions, including changing the endotracheal tube and suctioning the airway.[36] In addition, use of suction of the pharynx and around the top of the inflated cuff of the endotracheal tube before cuff deflation may help prevent ventilator-associated pneumonia. Because the endotracheal tube impairs the function of the mucociliary escalator and creates airway inflammation, large amounts of tenacious mucus can accumulate and occlude the tube and provide an excellent medium for bacterial growth.[29] Inspired gases should be humidified to minimize airway injury and moisten the mucus, allowing it to be aspirated more easily during suctioning.[37,38] Finally, atelectasis supports bacterial growth and, therefore, should be minimized by frequently turning the patient from one side to the other. Nutritional support should be prioritized as a negative energy balance can result in impaired cell-mediated and humoral immunity, delayed wound healing, and increased susceptibility to infection and shock.[39,40]

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a. Intercooled Stata for Windows 7.0, StataCorp, College Station, Tex.

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Selected abstract for *JAVMA* readers from the *American Journal of Veterinary Research*

Assessment of the reliability of plasma electrophoresis in birds
### Karen L. Rosenthal et al

AJVR

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Objective—To determine the reliability of plasma electrophoresis (EPH) in psittacine birds.

Animals—93 psittacine birds.

Procedure—Jugular venipuncture was performed on 93 awake psittacine birds. The plasma was centrifuged, separated, aliquoted into duplicate samples, frozen, and sent to 2 commercial laboratories that routinely perform avian EPH. Samples from 51 birds were sent to laboratory A, and samples from 42 birds were sent to laboratory B. The reliability of EPH results within each laboratory was assessed, but not between laboratories. To determine the reliability (agreement between duplicate samples) of total protein; albumin; prealbumin; and α_1-, α_2-, β-, and γ-globulin concentrations, the intraclass correlation coefficient (r_i) was calculated.

Results—Both laboratories had excellent agreement between samples for measurement of total protein concentration and only good agreement for albumin concentration. Except for the prealbumin concentration measured at laboratory B, both laboratories had poor agreement for all other values of the EPH.

Conclusions and Clinical Relevance—These data indicate that plasma EPH for measuring prealbumin and α_1-, α_2-, β-, and γ-globulin concentrations may not be a reliable tool for assessing avian health. Small amounts of these proteins in birds plus human variation in reading the EPH curves may lead to variable results. Avian veterinarians should cautiously interpret results from plasma EPH assays for these protein fractions. (*Am J Vet Res* 2005;66:375–378)
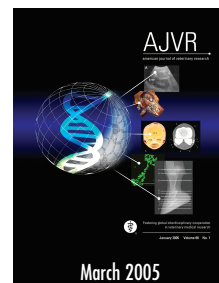
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