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Retrospective Study

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Outcome of positive-pressure ventilation in dogs and cats with congestive heart failure: 16 cases (1992–2012)

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Abstract

Objective – To describe the indications, duration of ventilation, underlying cardiac diseases, and outcome of dogs and cats undergoing positive-pressure ventilation (PPV) for treatment of congestive heart failure (CHF).
Design – Two-site retrospective study (1992–2012).
Setting – Two university small animal teaching hospitals.
Animals – Six cats and 10 dogs undergoing PPV for CHF.
Interventions – None.
Measurements and Main Results – Medical records were searched to identify patients requiring PPV for treatment of pulmonary edema secondary to CHF. Sixteen animals fulfilled these criteria. Patient signalment, duration of PPV, underlying cardiac disease, arterial or venous blood gas values, pharmacologic therapy before, during, and after PPV, anesthetic drugs, complications, and outcome were recorded. Overall survival to discharge was 62.5% (10/16). Mean (±SD) duration of PPV was 30.8 ± 21.3 hours and average time from presentation for CHF to initiation of PPV was 5.9 ± 6.4 hours. Azotemia at the time of initiation of ventilation, development of anuria or oliguria, and use of pentobarbital for anesthesia were negatively associated with survival ($P = 0.011$, $P = 0.036$, and $P = 0.036$, respectively). Survival-to-discharge rate was 77% (10/13) for patients treated after 2005 and those not receiving pentobarbital. There was no significant effect attributed to age, sex, weight, species, nature of heart disease, furosemide dose, length of ventilation, use of vasopressors, first-time CHF events, or plasma lactate concentration on survival to discharge.
Conclusions – Dogs and cats requiring PPV for CHF have a good overall prognosis for hospital discharge and require PPV for a relatively short duration. Azotemia, oliguria or anuria, and the use of pentobarbital are negatively associated with outcome.

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Keywords: cardiogenic, cardiovascular, heart failure, parenchymal disease, pulmonary edema, respiratory tract, small animal

Introduction

Congestive heart failure (CHF) is a clinical syndrome in which systolic or diastolic cardiac dysfunction

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Abbreviations

CHF	congestive heart failure
CVD	chronic myxomatous valvular disease
ISACHC	International Small Animal Cardiac Health Council
NCSU	North Carolina State University
PPV	positive-pressure ventilation
PEEP	positive end-expiratory pressure
VAP	ventilator-associated pneumonia
UGA	University of Georgia

leads to activation of the renin-angiotensin-aldosterone and sympathetic nervous systems, producing hypervolemia and vasoconstriction, followed by vascular congestion and edema.[1] The most life-threatening consequence of this fluid accumulation occurs in the setting of left-sided CHF, in which increased cardiac filling pressures lead to increased pulmonary capillary hydrostatic pressure and fluid extravasation into the pulmonary parenchyma, producing pulmonary edema.[2] Pulmonary edema can lead to hypoxemia and necessitates timely medical intervention.[3]

The prognosis and treatment of animals with CHF varies with severity of disease. Reported survival-to-discharge rates for dogs and cats hospitalized for the treatment of CHF of various etiologies are 56–80%.[4,5] While symptoms of CHF can often be successfully managed with medical therapy consisting primarily of diuretics, positive inotropic agents, vasodilators, and oxygen support, those with severe pulmonary edema may require positive-pressure ventilation (PPV) to support ventilation and to maintain blood oxygen tension in the acute setting.[6,7] PPV in patients with CHF helps to recruit alveoli, improve pulmonary gas exchange, reduce the work of breathing, and decrease cardiac afterload.[8] In human patients with moderate-to-severe CHF who are able to ventilate without assistance, noninvasive positive airway pressure provided through continuous positive airway pressure or bilevel positive airway pressure ventilation may help to alleviate signs and improve oxygenation.[9] In others, endotracheal intubation for mechanical ventilation is required.[10] Continuous positive airway pressure improves oxygenation in sedated, healthy dogs and cats; however, its feasibility has not been examined in clinical patients.[11,12] Therefore, endotracheal intubation with PPV is currently the most practical modality available to aid veterinary patients requiring extended ventilatory support.

There are no studies in the veterinary literature that specifically examine patients with CHF that require ventilatory support. The cases reported comprise small subgroups within larger studies examining CHF or mechanical ventilation in dogs and cats.[4,13–16] In many of these reports, no information regarding the clinical course of these patients is available to guide prognostication or clinical decision making. The objective of the present study was to examine the indications for ventilation, duration of ventilation, underlying cardiac disease, and outcome in dogs and cats undergoing PPV for treatment of CHF. We hypothesized that dogs and cats requiring PPV for severe CHF would be ventilated for relatively short durations of time and would have a good prognosis for discharge from hospital.

Materials and Methods

The computerized medical records of the North Carolina State University (NCSU) Veterinary Teaching Hospital and the University of Georgia (UGA) Veterinary Teaching Hospital were searched for all patients undergoing PPV between January 1, 1992 and December 31, 2012. Each medical record was reviewed to identify cats and dogs undergoing PPV for treatment of isolated left-sided CHF. The nature, severity, and extent of heart disease were documented in all cases by either echocardiography or necropsy. Inclusion criteria included the presence of cardiac pathology believed to be severe enough to cause left-sided heart failure and radiographic or histopathologic evidence of cardiogenic pulmonary edema. All diagnostic investigations were performed by board-certified specialists (veterinary cardiology, radiology, and pathology). Use of these inclusion criteria resulted in all cases included in the study being classified as International Small Animal Cardiac Health Council (ISACHC) stage IIIb (advanced heart failure with hospitalization recommended).[22]

Cases were excluded if a noncardiac indication for PPV was cited, if radiographic findings consistent with cardiogenic pulmonary edema were not reported, if echocardiography or necropsy was not performed to confirm the presence of heart disease severe enough to cause cardiogenic pulmonary edema, or if medical records were incomplete. Cases were also excluded if complicating noncardiogenic pulmonary disease (eg, neoplasia, pneumonia, noncardiogenic pulmonary edema) was present radiographically prior to initiation of ventilation. Cases were also excluded if there was other pathology that could have contributed to impaired ventilation (eg, cervical myelopathy). Finally, cases were excluded if there was no intent to treat (eg, if the purpose for ventilation was to obtain diagnostics prior to a decision to euthanize). Animals were not excluded if mild, historic lower or upper airway disease was present and this disease was believed to be well controlled at the time of ventilation.

The following information was recorded for each case fulfilling inclusion criteria: patient signalment and body weight; duration of PPV; type of cardiac disease diagnosed; time from initial clinical diagnosis of cardiac disease (if known) to CHF; duration of hospitalization prior to PPV; clinical signs at presentation; mechanical ventilator settings; complete blood count and serum biochemical values; arterial or venous blood gas values; pharmacologic therapy before, during and after PPV; drugs used to induce and maintain anesthesia during PPV; complications during PPV and outcome. Outcome was classified as death during PPV, survival to extubation but death prior to discharge, and survival to discharge. Data

were also collected on patients that survived past discharge until the completion of the manuscript (February 2013).

For the purposes of this study, azotemia was defined as a serum creatinine ≥123.8 μmol/L (1.4 mg/dL). Ventilator-associated pneumonia (VAP) was defined as radiographic or cytologic evidence of pneumonia (ie, suppurative inflammation with infectious organisms identified by tracheal wash or bronchoalveolar lavage) that developed after a patient had been on PPV for more than 24 hours in a patient with no prior radiographic or cytologic evidence of pneumonia. Oliguria was defined as urine production less than 0.5 mL/kg/h and anuria was defined as no discernable urine production.

Statistical Methods

Statistical analyses were performed using a commercially available software package.[a] When comparing types of heart disease, groups were classified as chronic myxomatous valvular heart disease (CVD), cardiomyopathy, or other (endocarditis, CVD with hypertensive cardiomyopathy or 3rd degree AV block). Continuous data are represented as mean ± SD. Survival times after discharge are presented as median (range). One-way ANOVA was used to compare quantitative variables to survival to discharge. Fisher's exact tests or chi-square analyses were used to examine categorical variables (eg, canine/feline, recent/past ventilation, presence of azotemia, first CHF episode, use of vasopressors, category of heart disease) with survival to discharge. P values < 0.05 were considered significant and corrections were not made for multiple comparisons.

Results

A total of 216 patients underwent PPV during the study period (35 at UGA, 181 at NCSU). From these cases, 16 patients fulfilled the criteria for inclusion in the study. Of the 16 patients included, 6 were cats, and 10 were dogs. Seven were spayed females, 8 were neutered males, and 1 was an intact male. Represented canine breeds included Chihuahua (2), Dachshund (2), Pomeranian (2), Coton de Tulear (1), Shetland Sheepdog (1), Basset Hound (1), and mixed breed (1). Represented feline breeds included domestic short hair (5) and domestic long hair (1). Mean age at time of PPV for all patients was 9.2 ± 3.3 years. The mean age for cats at the time of PPV was 7.4 ± 3.7 years and the mean age for dogs at the time of PPV was 10.3 ± 2.6 years. Underlying cardiac diseases identified in the canine patients of this study included CVD (4), CVD with suspected acute chordae tendineae rupture (3), CVD with 3rd degree atrioventricular block (1), CVD with hypertensive cardiomyopathy (1), and aortic valve endocarditis (1). In cats, underlying cardiac disease included hypertrophic cardiomyopathy (5) and dilated cardiomyopathy (1). For the purposes of analysis, there were thus 7 patients with "uncomplicated" CVD, 6 patients with cardiomyopathy (primary or secondary to thyrotoxicosis), and 3 patients classified as "other" (endocarditis [1], CVD with 3rd degree AV block [1], and CVD with hypertensive cardiomyopathy [1]).

Patients included in the study had various comorbidities including controlled idiopathic epilepsy (3), asymptomatic tracheal collapse (2), suspected lower airway disease (2), untreated hyperthyroidism (1), uncontrolled hypertension (1), lymphosarcoma (1), controlled hypothyroidism (1), and controlled immune-mediated polyarthritis (1). Eight of 16 patients were being treated for cardiac disease at the time of CHF development and hospitalization for PPV. All 8 of these patients were receiving an oral angiotensin converting enzyme inhibitor, 5 were receiving furosemide, and 2 were receiving pimobendan. Other cardiac medications with which patients in this study were being treated at the time of admission included nitroglycerin, digoxin, atenolol, and amlodipine.

The decision to initiate PPV was made based on the subjective perception of respiratory distress in combination with the presence of blood-gas-derived parameters indicative of impaired oxygenation ($PaO_2 < 60$ mm Hg with oxygen supplementation or the need for > 60% oxygen supplementation to maintain a $PaO_2 >$ 60 mm Hg) or ventilation ($PaCO_2$ or $PvCO_2 > 60$ mm Hg), when available. The perception of respiratory distress was based on observations of clinical signs including lateral recumbency, irregular respiratory pattern, mydriasis, short periods of apnea, or red-tinged fluid from the mouth or nares. Various combinations of injectable anesthetic drugs were used to both induce and maintain patients on PPV; selection of induction and maintenance drugs was based upon the attending clinician's discretion. The use of each drug individually and in combination with other drugs was compared with survival to discharge. All patients in this study received benzodiazapines during PPV. There was no significant relationship found between the use of ketamine ($P = 0.12$), etomidate ($P = 1.0$), propofol ($P = 0.12$), neuromuscular blocking agents ($P = 0.50$), opioids ($P = 0.38$), or a combination of opioid/benzodiazepine/propofol ($P = 0.12$) and survival to discharge. Only the use of barbiturates (as the sole maintenance agent in 1 case and as part of a multidrug anesthesia regimen in 2 cases) was significantly negatively correlated with survival to discharge ($P = 0.036$).

One of 3 types of critical care ventilators was used to ventilate each patient in the study.[b,c,d] Initial ventilator settings were established at the discretion of the clinician

overseeing the case. A combination of clinical preference, arterial or venous blood gas analysis, and response to therapy was used to determine initial ventilator settings. Synchronized intermittent mandatory ventilation was the mode of ventilation used for all patients. Specific ventilator settings, including the degree of positive end-expiratory pressure (PEEP), were only available for 5 of the 16 patients in the study. Initial PEEP for these 5 patients ranged from 6 to 12 cm H_2O and PEEP was gradually titrated to 2–3 cm H_2O prior to extubation. All patients were continuously monitored with a temperature probe, electrocardiogram, and pulse oximeter. Patients were started on PPV with an initial FiO_2 of 1.0, and subsequent arterial or venous blood gases were used to guide therapy at the attending clinician's discretion. Arterial blood pressure was monitored utilizing either direct or indirect methods. Careful nursing care was practiced in all patients to minimize ventilator-induced complications, as previously described.[17] Withdrawal of ventilator support occurred at the discretion of the attending clinician but in general did not occur before a patient was reliably generating spontaneous breaths and maintaining arterial oxygenation (SpO_2 of >95% or $PaO_2 > 80$ mm Hg) while breathing an FiO_2 between 0.21 and 0.40, with minimal PEEP (less than 3 cm H_2O) and pressure support by the ventilator. Spontaneous breathing trials toward weaning of ventilator support were performed on each patient under the direction of the attending clinician.

The duration of hospitalization prior to initiation of PPV was 5.9 ± 6.4 hours. Duration of PPV was 30.8 ± 21.3 hours. Of the 16 patients undergoing PPV, 11 (68.8%) survived to extubation and 10 (62.5%) survived to discharge. Six of the 10 dogs (60%) in the study survived to discharge while 4 of the 6 cats (66%) survived to discharge.

When grouped for statistical evaluation, 4 of the 6 patients with primary cardiomyopathy survived and 5 of the 7 patients with CVD survived to discharge ($P = 1.0$). Euthanasia prior to extubation was performed in 4 patients following the development of oliguria or anuria (3), or the loss of corneal reflex with fixed and dilated pupils (1). One patient died during mechanical ventilation due to cardiac arrest secondary to refractory ventricular tachyarrythmias. One dog was successfully extubated, but collapsed and died while defecating approximately 34 hours postextubation and prior to hospital discharge. In the 10 patients surviving to discharge, hospitalization time after extubation was 63.0 ± 57.6 hours.

Surviving patients were managed following discharge at the discretion of the attending clinician. In the 6 that died during the follow-up period, median survival time following discharge was 60 days (range, 45–1,151 d). Three patients died due to recurrent heart failure, 2 were euthanized due to renal failure, and 1 died of unknown causes. Four of the 10 patients were still alive at the time of manuscript submission, thus far surviving 106, 145, 240, and 440 days following discharge.

Complications encountered during PPV included VAP (4), oliguria/anuria (3) fever/pyrexia (2), superficial corneal ulceration (2), mild pneumothorax (believed to be associated with thoracocentesis) (1), ulcerative tracheitis (discovered on necropsy) (1), anisocoria and nystagmus (both of which resolved after naloxone administration) (1), development of atrial fibrillation (1), and ventricular ectopy (1).

A significant effect of gender ($P = 0.30$), age ($P = 0.83$), or species ($P = 0.79$) on survival to discharge was not identified. The institution at which the patients were treated was also not associated with survival ($P = 0.33$). The average duration of PPV, time from presentation to initiation of PPV, total furosemide dose (the sum of furosemide that was administered in the hospital prior to PPV plus that administered while on PPV; milligrams per kilogram), furosemide dose administered while receiving PPV (milligrams per kilogram), and furosemide dose normalized for duration of PPV (milligrams per kilogram per hour while receiving PPV) were not associated with survival to discharge (Table 1). Comparison of survival-to-discharge rates in patients undergoing PPV for a first-time CHF event (11/16 cases; survival rate 63.6%) to those of patients with a history of having experienced a prior episode of CHF (5/16 cases; survival rate 60.0%) revealed no significant difference ($P = 0.89$).

Plasma lactate values, obtained either before or immediately after initiation of PPV, were available for 12/16 patients in this study; mean lactate was 0.32 ± 0.33 mmol/L (2.85 ± 2.97 mg/dL; range 0.056–1.19 mmol/L [0.5–10.7 mg/dL]). There was no relationship between initial plasma lactate and survival to discharge ($P = 0.069$). Serum creatinine concentrations were available in 15/16 animals in the present study; the mean concentration was 114.9 ± 78.7 μmol/L (1.3 ± 0.89 mg/dL). Serum creatinine >123.8 μmol/L (1.4 mg/dL) prior to or within the first hours of initiation of PPV was inversely correlated with survival; none of the 4 animals classified as azotemic by this criterion survived to discharge ($P = 0.011$). There was no significant relationship between chronic furosemide administration and azotemia at time of presentation ($P = 0.68$), nor was there a correlation between type of heart disease and the presence of azotemia at presentation ($P = 0.42$).

The year during which patients underwent PPV was significantly correlated to survival to discharge. Patients undergoing PPV for CHF prior to 2005 were less likely to survive to discharge when compared to those ventilated after 2005 ($P = 0.036$). The combined

Table 1: Characteristics survivors to discharge and nonsurvivors to discharge who underwent positive-pressure ventilation for congestive heart failure

Variable:	Survivors – mean ± SD	Nonsurvivors – mean ± SD	*P*-value
Age at time of PPV (y)	9.1 ± 3.1	9.2 ± 3.8	0.86
Body weight (kg)	7.25 ± 4.69	10.93 ± 8.16	0.26
Time from presentation to PPV (min)	327 ± 310	376 ± 441	0.50
Pre-PPV serum lactate (mg/dL)	2.2 ± 1.8	6.3 ± 6.2	0.069
Pre-PPV serum creatinine (mg/dL)	0.86 ± 0.27	1.97 ± 1.1	0.011
Total (before + during PPV) furosemide dose (mg/kg)	13.5 ± 8.3	15.1 ± 7.3	0.70
Furosemide dose during PPV (mg/kg)	9.3 ± 6.7	10.1 ± 4.8	0.82
Duration of PPV (h)	28.7 ± 18.3	34.3 ± 27.2	0.62
Furosemide dose during PPV (mg/kg/h)	0.56 ± 0.26	1.1 ± 1.5	0.29

PPV, positive-pressure ventilation.

survival-to-discharge rate for all patients placed on PPV after 2005 was 77%. This included 6 out of 9 dogs (66%) and 4 out of 4 (100%) cats. Three patients were ventilated prior to 2005 (1 in 1992, 1 in 1995, and 1 in 1996), all of which were anesthetized with pentobarbital (either as part of a multidrug regimen or as a single maintenance agent) and none of which survived to discharge.

Discussion

The overall survival-to-discharge rate for patients undergoing PPV for treatment of CHF in the present study was 10/16 (62.5%). This rate exceeds that found in most other studies examining PPV in dogs and cats. Three large retrospective veterinary studies have described outcomes of PPV in dogs and cats ventilated for various reasons; in these, overall survival rates range from 15% to 39%.[13–15] Survival rates were lower for patients with pulmonary parenchymal disease than for patients with extra-pulmonary disease. Focused reports examining outcomes in dogs undergoing PPV for treatment of lower motor neuron disease, brachycephalic syndrome, and cervical spinal disorders have documented survival rates of 21%, 27%, and 71%, respectively.[16,18,19] The higher rate of survival found in the present study, which is comparable to that reported for patients with cervical spinal disorders undergoing PPV, is likely a reflection of the fact that most patients comprising these 2 groups have readily treatable conditions. It is of particular interest that the survival-to-discharge rate for cats in the present study (66%) was considerably higher than those reported previously for cats undergoing PPV for various reasons (15–42%).[13–15] In a study by Lee and colleagues of PPV in cats, only 1 of 53 cats was ventilated for CHF and the clinical course of this cat was not specified.[15]

In patients with cardiogenic pulmonary edema, PPV with varying degrees of PEEP supports oxygenation, helps to recruit alveoli, improves lung compliance, and decreases the work of breathing in patients until pharmacologic therapies can resolve the edema.[20] In addition, as confirmed by this study, patients with CHF require PPV for relatively short periods of time, decreasing the risk of life-threatening complications such as VAP, which typically occurs 48–72 hours after the onset of invasive ventilation in human patients.[21] Shorter durations of ventilation may also help to prevent financial limitations from influencing treatment decisions.

The survival rate for patients in the present study was slightly lower than that reported in the largest veterinary study of patients with acute CHF (80%).[4] However, comparison between the 2 studies is difficult. All cases in the current study would be classified as ISACHC stage IIIb.[22] Of the 145 cases reported in the study by Goutal and colleagues, 61 dogs and 45 cats were classified as stage IIIb; however, details regarding the severity of clinical signs and degree of pulmonary edema were not reported. Although 4/145 cases in that report underwent PPV, those patients' specific clinical courses were not described, nor were the criteria for initiating PPV. The decision to initiate PPV at both NCSU and UGA is based upon the severity of clinical signs in combination with blood-gas-derived indicators of declining pulmonary function when available. The only other veterinary study describing outcomes in dogs and cats undergoing PPV for pulmonary edema reported a 23% (3/13) survival-to-discharge rate.[13] However, no differentiation between cardiogenic and noncardiogenic pulmonary edema was made, making it difficult to draw conclusions between outcomes in the 2 studies.

Because only 6 patients died during our follow-up period, comparison of survival times following discharge of the patients in the present study to those reported for patients experiencing CHF of various etiologies is difficult. In dogs with ISACHC stage III CHF due to CVD, median survival time is approximately 9 months[23] and cats with CHF due to hypertrophic cardiomyopathy, survival times range from 3 to 18 months.[24–26] When survival times to date of the 4 living patients are

included in analysis, median survival time increases to 126 days; this value would be expected to increase further as the follow-up period continues. Based on this information, it is not clear whether animals with advanced CHF requiring PPV support have a long-term prognosis that is different from those in which more conservative management is sufficient.

We found no significant relationship between species, gender, age at time of ventilation, total furosemide dose, preventilation lactate, duration of ventilation, duration of hospitalization prior to PPV, institution where the patient was treated, use of vasopressors during PPV, or first-time failure events and survival to discharge. Other studies have found significantly lower survival-to-discharge rates in ventilated patients associated with increasing age and shorter durations of ventilation.[13,15]

Patients in the current study had various comorbidities at the time of presentation. CHF was likely directly associated with hyperthyroidism in 1 patient, and systemic hypertension in another. While potentially contributory, the significance of other comorbidities (such as the presence of lymphosarcoma in patient 1 and airway disease in patients 2 and 11) with respect to clinical course is unclear. The remainder of the concurrent diseases found in these animals is not thought to have contributed significantly to their morbidity and mortality.

Based on our findings, patients that were azotemic prior to initiation of PPV had a significantly worse prognosis than those that were not azotemic. This is in contrast to the findings of Goutal and others, who reported no correlation between azotemia and survival.[4] Because most of the animals in the present study were treated with furosemide prior to admission, the etiology of the azotemia is unclear and may have been multifactorial. However, chronic administration of furosemide prior to admission for CHF was not significantly related to azotemia at the time of presentation. In people treated for acute decompensated CHF, an increased serum blood urea nitrogen (BUN) or creatinine at the time of hospital admission is associated with a significantly higher mortality rate.[27,28] Human patients with CHF and renal insufficiency do not respond to diuretics as efficiently as those with normal renal function.[6] Lack of significant response to diuretics (as demonstrated by the need for increased doses, or for other interventions such as hemodialysis with ultrafiltration) can prolong the duration of CHF, increase the cost of treatment, necessitate other diuretic therapies, and result in a worse prognosis for the patient.[29,30]

In the present study, animals that were treated with PPV for CHF prior to 2005 were significantly less likely to survive to discharge than were those treated after 2005. When the 3 cases undergoing PPV prior to 2005 were excluded from analysis, survival-to-discharge rate for patients of this study increased to 77%. One possible explanation for the increased survival after 2005 may be the differences in sedation protocols between these groups. In the 3 cases ventilated prior to 2005, pentobarbital was used as part of a balanced anesthetic technique in 2 cases and as the sole maintenance agent in 1 case to maintain anesthesia whereas in those ventilated after 2005, patients received combinations other drugs for anesthetic maintenance (propofol, fentanyl, midazolam, ketamine, and morphine). Pentobarbital can increase afterload, transiently increase heart rate, generate potent negative inotropic effects, and has an overall myocardial depressive effect when administered to dogs.[31] In patients with severe cardiac disease, the cardiovascular effects of pentobarbital may have contributed to increased mortality. Improved survival rate after 2005 may also reflect the timing of the development of a fully staffed emergency and critical care program at both institutions. NCSU had a mature emergency and critical care program by 2005 and UGA's program was fully in place by 2007. Critically ill humans are more likely to survive to discharge when attended to by more experienced and trained staff.[32,33] Finally, due to the low numbers of patients, a type 1 error is possible and significance may have been found when none truly existed.

Complications encountered in this study were attributable to the presence of underlying heart disease, therapy utilized during PPV, or the altered physiology of mechanical ventilation. Cardiac arrhythmias are commonly associated with severe cardiac disease and were not unexpected in these animals.[34] VAP was present or suspected in 25% of the patients (4/16). Two of these patients were diagnosed via trans-tracheal washes and signs consistent with pneumonia, 1 was diagnosed based on radiographs and 1 was diagnosed at necropsy. While this rate is slightly higher than those found in other veterinary studies,[13–15] it is consistent with the 15–30% reported in human medicine.[35,36] Because patients were excluded from the study if radiographic evidence of pneumonia was found before or immediately after initiation of PPV, radiographic patterns or cytology obtained from lung washes that was consistent with pneumonia were presumed to be associated with PPV. All animals diagnosed with pneumonia were ventilated for greater than 24 hours and 3 of the 4 were mechanically ventilated for greater than 48 hours. In 1 patient, evidence of mild bronchopneumonia was found incidentally during necropsy. The development of pneumonia was not significantly associated with survival to discharge ($P = 0.12$).

Three patients developed oliguria or anuria while receiving PPV and none of these patients survived to discharge ($P = 0.036$). All of these patients were azotemic at the time of presentation. Decreased urine production

has been associated with an increased mortality rate in other studies of acute renal failure in dogs and cats.[37,38] All 3 patients were treated with furosemide. Mannitol and dopamine (2 cases) and diltiazem (1 case) were also utilized to encourage urine production.

Other complications encountered during PPV included 2 cases of corneal ulceration, consistent with the rates found in other studies.[13] There was a mild increase in rectal temperature in 2 patients. In 1 of these patients, pneumonia was believed to be the source of the fever. In the other patient, the cause of the elevated temperature was never discovered but resolved after the administration of antibiotics. Pneumothorax was found in 1 patient in this study, which was believed to have occurred secondary to thoracocentesis for treatment of pleural effusion. This rate of pneumothorax is lower than the rate reported in 1 study[14] and consistent with the rate reported in another.[13] Barotrauma-induced pneumothorax was not believed to be present in any patients in this study.

There are several limitations to the present study. First, the retrospective nature of the study limited the data that could be examined. Data were not available on ventilator settings in 11 of the 16 animals in the study, precluding statistical examination of ventilator settings on outcome. Additionally, controlling for variables such as the duration of hospitalization prior to PPV, drugs used for sedation, dose of furosemide, and nature of heart failure was not possible in this study. Second, the small number of patients increases the potential for both type 1 and type 2 errors for all measured variables. The degree to which concurrent systemic and respiratory diseases impacted overall pulmonary function and therefore the outcome of mechanical ventilation is not clear, but these factors may have influenced the results reported here. Finally, there was no standardization of ventilation protocols between institutions. Clinicians at both institutions treated each case individually based on their clinical judgment, which contributed to variability in case management.

This study suggests that there is a relatively good overall prognosis for discharge from the hospital in dogs and cats with CHF that require mechanical ventilation. The presence of azotemia prior to PPV may confer a worse prognosis. Larger studies are needed to prospectively evaluate animals with severe CHF that are managed with PPV versus those that are not, specifically focused on ideal ventilator settings and anesthetic protocols.

Footnotes

[a] Minitab 16—Minitab Inc, State College, PA.
[b] Respironics Esprit Ventilator—Philips Healthcare, Andover, MA.
[c] Maquet Servo I and Seimens 300A Servo—Maquet Getinge Group, Wayne, NJ.
[d] Bear 3 Ventilator—Bear Medical Systems, Inc, Riverside, CA.

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