Indications, management, and outcome of long-term positive-pressure ventilation in dogs and cats: 148 cases (1990–2001)

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Objective—To determine outcome of positive-pressure ventilation (PPV) for 24 hours or longer and identify factors associated with successful weaning from PPV and survival to hospital discharge in dogs and cats.

Design—Retrospective case series.

Animals—124 dogs and 24 cats that received PPV for 24 hours or longer.

Procedures—Medical records were reviewed for signalment, primary diagnosis, reason for initiating PPV, measures of oxygenation and ventilation before and during PPV, ventilator settings, complications, duration of PPV, and outcome. Animals were categorized into 1 of 3 groups on the basis of the reason for PPV.

Results—Group 1 patients received PPV for inadequate oxygenation (67 dogs and 6 cats), group 2 for inadequate ventilation (46 dogs and 16 cats), and group 3 for inadequate oxygenation and ventilation (11 dogs and 2 cats). Of the group 1 animals, 36% (26/73) were weaned from PPV and 22% (16/73) survived to hospital discharge. In group 2, 50% (31/62) were weaned from PPV and 39% (24/62) survived to hospital discharge. In group 3, 3 of 13 were weaned from PPV and 1 of 13 survived to hospital discharge. Likelihood of successful weaning and survival to hospital discharge were significantly higher for group 2 animals, and cats had a significantly lower likelihood of successful weaning from PPV, compared with dogs. Median duration of PPV was 48 hours (range, 24 to 356 hours) and was not associated with outcome.

Conclusions and Clinical Relevance—Results suggested that long-term PPV is practical and successful in dogs and cats. (*J Am Vet Med Assoc* 2007;230:64–75)

Positive-pressure ventilation provides artificial support of lung function and is becoming a standard of care in many veterinary intensive care units. In addition to understanding the principles of mechanical ventilation, provision of PPV requires continuous monitoring and intensive nursing care. Although it has been well established that animals can be maintained on PPV for short periods, either during a surgical procedure or in the critical-care setting, there is limited information available regarding support of patients for longer periods. Long-term PPV requires appropriate facilities and equipment, a team of trained caregivers, and a substantial emotional and financial commitment from owners. Information about indications, complications, and outcome of these patients may improve patient care and allow better counseling of owners when the initial decision is made to ventilate.

The indications for PPV can be broadly categorized into 2 groups: inadequate blood oxygenation because of severe diffuse parenchymal pulmonary disease and inadequate elimination of carbon dioxide because of neurologic or muscle disease. The success of ventilator support varies with the underlying problem. Survival rates have been reported to range from 20% to 33% for veterinary patients with parenchymal pulmonary disease and 57% to 71% in patients with neuromuscular disease.[1-5] These studies included animals ventilated for various periods (from as little as 45 minutes to as long as 312 hours) and were based on small numbers of patients (from 3 to 53). Specific weaning and survival rates for animals receiving long-term PPV have not been reported.

Long-term PPV may be associated with more complex patient management issues and higher ventilator-associated

Abbreviations

PPV	Positive-pressure ventilation
F_{IO_2}	Fraction of inspired oxygen
Ppeak	Peak airway pressure
PEEP	Positive end-expiratory pressure
P:F	Pa_{O_2}:F_{IO_2} ratio
LMN	Lower motor neuron
ARDS	Acute respiratory distress syndrome
A-C	Assist-control
SIMV	Synchronized intermittent mandatory ventilation
bpm	Breaths per minute
CPAP	Continuous positive airway pressure
CRI	Constant rate infusion
VAP	Ventilator-associated pneumonia
CPR	Cardiopulmonary resuscitation

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complication rates, compared with short-term PPV. The feasibility and effectiveness of this level of patient support have not been described for veterinary patients.

The purpose of the study reported here was to determine the indications for and management and outcome of long-term PPV and identify factors associated with the duration of PPV and successful weaning and survival to hospital discharge in dogs and cats. We hypothesized that the severity of the initial disease process and the magnitude of required ventilator settings could be used to predict the likelihood of successful weaning from PPV and survival to hospital discharge in patients receiving PPV for 24 hours or longer.

Criteria for Selection of Cases

Medical records from the small animal intensive care unit of the Veterinary Medical Teaching Hospital, University of California, Davis, Calif, were searched to identify cases of dogs and cats that received long-term PPV from January 1990 to December 2001. Records were included if animals had ≥ 24 hours of continuous PPV. For animals that had more than 1 long-term ventilator episode, only the first ventilator episode was included in the study.

Procedures

Information obtained from the medical records included signalment; body weight; clinical signs; initial arterial or venous blood gas results; pulse oximetry readings; thoracic radiographic findings, when available; underlying disease diagnosis; initial ventilator settings; duration of ventilation; clinical course during PPV; final ventilator settings; and for those patients successfully weaned off the ventilator, clinical outcome subsequent to PPV. The following measurements were recorded: F_IO_2, minute ventilation, tidal volume, respiratory rate, Ppeak, and PEEP. In addition to the underlying disease diagnosis, the nature of any pulmonary disease was also categorized. This diagnosis was based on the history; underlying disease process; radiographic appearance; laboratory data; and, when available, postmortem findings.

Animals were categorized according to the reason they needed PPV into 1 of the 3 following groups: inadequate oxygenation caused by parenchymal pulmonary disease that was not responsive to oxygen therapy (group 1); inadequate ventilation caused by neurologic, muscle, or large airway disease (group 2); or both inadequate oxygenation and ventilation (group 3). Categorization of animals was based on physical examination findings, thoracic radiography results, and results of arterial blood gas analyses.

Animals were included in group 1 if there was evidence of severe hypoxemia that was unresponsive to oxygen therapy, calculated inadequate oxygenation, or radiographic evidence of pulmonary infiltrates and no evidence of inadequate ventilation. Severe hypoxemia was defined as the physical examination finding of cyanosis, a pulse oximetry measurement of < 90%, or Pa_{O_2} < 60 mm Hg. In general, a patient was considered unresponsive to oxygen therapy when severe hypoxemia could not be resolved with an inspired oxygen concentration of 60%.

Inadequate oxygenation was defined as an alveolar-arterial P_{O_2} gradient > 25 mm Hg when breathing room air or a P:F < 300 on any F_IO_2. The alveolar-arterial gradient ($P_{AO_2} - Pa_{O_2}$) was calculated by use of the following formula:

$$(150 - Pa_{CO_2}/0.9) - Pa_{O_2}$$

where 150 is the partial pressure of inspired oxygen at sea level when breathing room air (21% oxygen).

The P:F was calculated by use of the following formula:

$$\text{Measured } Pa_{O_2} \text{ (mm Hg)}/F_IO_2 \text{ (0.21 – 1.0)}$$

Radiographic evidence of pulmonary infiltrates was considered representative of the disease process at the commencement of PPV if such evidence was obtained within the 24 hours preceding or 12 hours after initiation of PPV. The severity of pulmonary infiltrates was categorized as mild, moderate, or severe on the basis of the radiologist's report. A small number of animals had inadequate oxygenation with only mild pulmonary infiltrates on thoracic radiographs. If arterial blood gas measurement revealed persistent inadequate oxygenation after initial stabilization on PPV, these patients were assigned to group 1.

Animals were included in group 2 if they had inadequate ventilation in the absence of parenchymal pulmonary disease. Causes of inadequate ventilation were neurologic diseases localized to the brain or cervical spinal cord, diffuse LMN disorders or generalized muscle diseases (the term neuromuscular will be used to describe all these disease processes in this report), or collapse of large intrathoracic airways that did not respond to medical treatment. Inadequate ventilation was defined as physical examination findings of apnea or weak ventilatory efforts associated with minimal air movement, Pa_{CO_2} > 55 mm Hg, or venous blood Pv_{CO_2} > 60 mm Hg; Pv_{CO_2} was only used when Pa_{CO_2} was not available. Animals were also categorized to group 2 if PPV was implemented to manage intracranial pressure. A small number of patients had inadequate ventilation and coexisting pulmonary infiltrates on preventilation radiographs. If arterial blood gas analysis after initial stabilization on PPV revealed normal oxygenating ability, these patients were assigned to group 2. Those with persistent abnormalities in oxygenation were assigned to group 3. Animals were included in group 3 if they fulfilled the inclusion criteria for groups 1 and 2.

Clinical outcome was either death during PPV, successful weaning from PPV but death prior to hospital discharge, or survival to hospital discharge. Spontaneous death was not differentiated from euthanasia. Successful weaning was defined as a patient being able to maintain adequate oxygenation and ventilation (Pa_{O_2} > 60 mm Hg and Pa_{CO_2} < 60 mm Hg) with or without oxygen supplementation and without excessive respiratory effort as determined via clinical evaluation.

One ventilator model[a] was used during most of the study; a second ventilator[b] was also used in the last

year of the study. All animals were initially stabilized on PPV with an F_{IO_2} of 1.0, and a process of trial and error was used to determine the initial ventilator settings. This process was most commonly guided by arterial blood gas measurement; in the absence of arterial blood gas measurements, venous blood gases and pulse oximetry were used. After initial stabilization, arterial blood gases were generally monitored every 4 hours; measurements were made more frequently if there were concerns regarding patient stability, and in some stable, long-term patients, arterial blood gases were measured less frequently. All patients were continuously monitored throughout the ventilation period with an ECG, temperature probe, and pulse oximeter. Continuous direct arterial blood pressure was monitored in those patients with an indwelling arterial catheter. All other patients received frequent arterial blood pressure monitoring performed by use of indirect methods. Patient care was performed as described.[6]

Statistical analysis—Normally distributed data are reported as mean ± SD values, and nonparametric data are reported as medians and ranges. Unconditional logistic regression was used to measure the magnitude of association between potential determinants of outcome (species, sex, age, weight, reason for mechanical ventilation, duration of PPV, P:F before PPV, Ppeak and PEEP at initial stabilization, P:F after initial stabilization on the ventilator, Ppeak and PEEP at the time of weaning, and P:F at weaning) and the following 3 outcomes: weaning, discharged from the hospital, and development of pneumothorax. The assumption of linearity was verified for duration, P:F before PPV and at weaning, and Ppeak and PEEP at initial stabilization and at weaning. Results are presented as odds ratios and 95% confidence intervals.

Cox proportional hazards regression analysis was used to measure the magnitude of association between potential factors (species, sex, age, weight, reason for PPV, occurrence of pneumothorax, P:F before PPV, Ppeak and PEEP at initial stabilization, and P:F after initial stabilization on the ventilator) and the duration of PPV. Continuous variables were checked for linearity, and when necessary, polynomial terms were added to the model to improve fit. Results are presented as hazard rate ratios and 95% confidence intervals.

Likelihood ratio tests were used to evaluate improvement in model fit. For all comparisons, $P \leq 0.05$ was considered significant.

Results

From January 1, 1990, to December 31, 2001, 540 animals were ventilated; 148 animals (124 dogs and 24 cats) were identified that were ventilated for 24 hours or longer. Of the 124 dogs, 55% were male and 45% were female; 58% of cats were male and 42% were female. Ages ranged from 10 weeks to 16 years, with a median age of 8 years. Mean body weight of the dogs was 29.4 kg (64.7 lb), median weight was 28.5 kg (62.7 lb), and range was 0.84 to 62 kg (1.8 to 136.4 lb). Group 1 contained 67 dogs and 6 cats, group 2 contained 46 dogs and 16 cats, and group 3 contained 11 dogs and 2 cats.

Underlying disease—The underlying diseases in group 1 included renal failure (10/73 [14%]), neoplasia (9/73 [12%]), cardiac disease (7/73 [10%]), recovery after craniotomy (7/73 [10%]), sepsis (5/73 [7%]), immune-mediated disease (5/73 [7%]), and trauma (5/73 [7%]). Three (4%) animals each had toxicosis, intracranial disease, recovery after laparotomy, cervical spinal cord disease, pneumonia, or airway collapse. Diagnoses in the remaining 7 cases included diffuse LMN disease (n = 2), gastrointestinal disease (2), tetanus (1), recovery after thoracotomy (1), and no diagnosis (1). The major pulmonary disease processes in these patients were aspiration pneumonia (20/73 [27%]), pulmonary edema (13/73 [18%]), ARDS (12/73 [16%]), pneumonia (10/73 [14%]), pulmonary contusions (5/73 [7%]), no diagnosis (3/73 [4%]), pulmonary hemorrhage (2/73 [3%]), atelectasis (2/73 [3%]), recovery after CPR (2/73 [3%]), pulmonary thromboembolism (2/73 [3%]), small airway disease (1/73 [1%]), and pyothorax (1/73 [1%]).

Underlying disease processes in group 2 were intracranial disease (15/62 [24%]), recovery after CPR (9/62 [15%]), cervical spinal cord disease (8/62 [13%]), toxicosis (7/62 [11%]), diffuse LMN disease (7/62 [11%]), and recovery after craniotomy (7/62 [11%]). The remaining diagnoses included intrathoracic airway collapse (4/62 [7%]), sepsis (2/62 [3%]), tetanus (1/62 [2%]), recovery after laparotomy (1/62 [2%]), and myopathy (1/62 [2%]). Forty-two percent (26/62) of these animals had high P_{CO_2} before PPV, and 20% (12/62) were ventilated as a preventive measure after CPR or for management of intrathoracic airway collapse. Sixteen percent (10/62) of patients were apneic, 11% (7/62) were ventilated to manage intracranial pressure, and 11% (7/62) were considered clinically at risk of hypoventilation but did not have substantially high P_{CO_2} at the time PPV was started. In group 3, the underlying disease processes were diffuse LMN disease (3/13 [23%]), intracranial disease (2/13 [15%]), and recovery after CPR (2/13 [15%]). The diagnoses in the remaining patients included 1 case each of the following: trauma, toxicosis, head trauma, cervical spinal disease, recovery after thoracotomy surgery, and sepsis. The concurrent pulmonary diseases in these animals were aspiration pneumonia (4/13), pulmonary contusions or hemorrhage (3/13), no diagnosis (2/13), ARDS (2/13), pulmonary edema (1/13), and pneumonia (1/13).

Outcome—Overall, 41% (60/148) of all animals were successfully weaned from PPV and 28% (41/148) survived to discharge. In group 1, 36% (26/73) of animals were weaned from PPV (24 dogs and 2 cats). Sixteen of 73 (22%) survived to discharge (15 dogs and 1 cat). In group 2, 50% (31/62) of animals were weaned from PPV (27 dogs and 4 cats). Twenty-four of 62 (39%) animals survived to discharge (20 dogs and 4 cats). In group 3, 3 of 13 dogs were weaned from PPV. One of 13 survived to hospital discharge. This gave an overall successful weaning rate in dogs of 44% and survival to hospital discharge rate of 29%. The successful weaning rate for cats was 25%, and the survival to hospital discharge rate was 21%. In group 1, 10% (7/73) of animals (all dogs) required a second episode of PPV during the same hospitalization period. Of these, 2 dogs were

eventually successfully weaned from PPV. The weaning and survival to hospital discharge rates according to the primary pulmonary disease process in group 1 were aspiration pneumonia, 50% (10/20) weaned and 30% (6/20) survived to hospital discharge; pulmonary edema, 5 of 13 weaned and 3 of 13 survived to hospital discharge; ARDS, 1 of 12 weaned and survived to hospital discharge; pneumonia, 4 of 10 weaned and 1 of 10 survived to hospital discharge; and pulmonary contusions, 3 of 5 weaned and 2 of 5 survived to hospital discharge.

The weaning and hospital discharge rates according to the primary neuromuscular disease process in group 2 were intracranial disease, 4 of 15 weaned and 2 of 15 survived to hospital discharge; recovery after CPR, 1 of 9 weaned and survived to hospital discharge; cervical spinal cord disease, 5 of 8 weaned and 4 of 8 survived to hospital discharge; toxicosis, 7 of 7 weaned and 6 of 7 survived to hospital discharge; diffuse LMN disease, 6 of 7 weaned and 4 of 7 survived to hospital discharge; and recovery after craniotomy, 5 of 7 weaned and survived to hospital discharge.

Increased age (P = 0.014), decreased P:F prior to starting PPV (P = 0.011), and a higher PEEP setting when initially stabilized on the ventilator (P = 0.037) were associated with a lower likelihood of successful weaning. When adjusted for the reason for PPV, cats had significantly (P = 0.035) lower likelihood of successful weaning, compared with dogs. When adjusted for species, group 2 animals had a significantly (P = 0.032) higher chance of successful weaning **(Table 1)**.

Age was significantly inversely associated with survival to discharge from the hospital; for every increase in age of 1 year, the log odds of survival was reduced by 10% (P = 0.027). Animals in group 2 had a significantly (P = 0.035) increased chance of successful discharge from the hospital in comparison to group 1 animals. Other variables significantly associated with survival to discharge from the hospital were increased P:F before starting PPV (P = 0.013), lower PEEP when initially stabilized on the ventilator (P = 0.007), increased P:F at weaning ($P < 0.001$), and lower PEEP at weaning ($P < 0.001$; **Table 2**).

Preventilation assessment—Thoracic radiographs were obtained before PPV in 82% (60/73) of the patients in group 1, all of which had abnormalities. Sixty-

Table 1—Results of logistic regression analyses of factors potentially associated with successful weaning in 148 episodes of long-term PPV in dogs and cats.

Variable	Odds ratio*	95% Confidence interval	*P* value
Species†			
Dog	1.00	—	—
Cat	0.33	0.12–0.93	0.035
Sex			
Male (sexually intact)	1.00	—	—
Male (castrated)	1.17	0.46–2.96	0.74
Female (sexually intact)	1.10	0.33–3.66	0.88
Female (spayed)	1.34	0.53–3.43	0.54
Age			
Linear (y)	0.87	0.78–0.97	0.014
Inverse (y)	0.47	0.21–1.02	0.056
Weight (kg)			
Dog	1.01	0.99–1.03	0.34
Cat	1.15	0.61–2.18	0.67
Reason for PPV‡			
Inadequate oxygenation (Group 1)	1.00	—	—
Inadequate ventilation (Group 2)	2.22	1.07–4.59	0.032
Inadequate oxygenation and ventilation (Group 3)	0.57	0.14–2.30	0.43
Duration of PPV (per 6-h increment)	1.02	0.99–1.05	0.30
Pneumothorax			
Absent	1.00	—	—
Present	0.15	0.02–1.21	0.074
P:F (50-unit change in ratio)			
Before PPV	1.21	1.04–1.40	0.011
At initial stabilization	1.08	0.96–1.21	0.19
Ppeak at initial stabilization (cm H_2O)	0.97	0.92–1.02	0.22
PEEP at initial stabilization (cm H_2O)	0.90	0.82–0.99	0.037

*Odds ratios for continuous variables refer to a 1-unit change unless otherwise specified. †Adjusted for the reason for PPV. ‡Adjusted for species.
— = Referent category. To convert kilograms to pounds, multiply by 2.2.

Table 2—Results of logistic regression analyses of factors potentially associated with discharge from the hospital in 148 episodes of long-term PPV in dogs and cats.

Variable	Odds ratio*	95% Confidence interval	*P* value
Species†			
Dog	1.00	—	—
Cat	0.64	0.22–1.85	0.41
Sex			
Male (sexually intact)	1.00	—	—
Male (castrated)	1.12	0.41–3.05	0.83
Female (sexually intact)	1.06	0.29–3.92	0.93
Female (spayed)	1.02	0.36–2.86	0.97
Age (y)	0.90	0.82–0.99	0.027
Weight (kg)			
Dog	1.00	0.97–1.02	0.88
Cat	1.00	0.98–1.03	0.81
Reason for PPV‡			
Inadequate oxygenation (Group 1)	1.00	—	—
Inadequate ventilation (Group 2)	2.25	1.06–4.78	0.035
Inadequate oxygenation and ventilation (Group 3)	0.30	0.04–2.46	0.26
Duration of PPV (per 6-h increment)	1.01	0.98–1.04	0.69
Pneumothorax			
Absent	1.00	—	—
Present	0.27	0.03–2.22	0.22
P:F (50-unit change in ratio)			
Before PPV	1.20	1.04–1.39	0.013
At initial stabilization	1.11	0.98–1.25	0.095
At weaning	1.58	1.30–1.91	< 0.001
Ppeak (cm H_2O)			
At initial stabilization	0.94	0.89–1.00	0.047
At weaning	0.94	0.91–0.98	0.006
PEEP (cm H_2O)			
At initial stabilization	0.84	0.75–0.96	0.007
At weaning	0.65	0.54–0.79	< 0.001

See Table 1 for key.

two percent (37/60) had moderate pulmonary abnormalities, and 25% (15/60) had severe pulmonary abnormalities. In group 2, 48% (30/62) of animals had thoracic radiography before PPV; of these, 57% (17/30) had no abnormalities, 30% (9/30) had mild pulmonary changes, and 2 patients had radiographic evidence of atelectasis. Of the group 3 patients, 9 of 13 had thoracic radiography before PPV; these revealed mild (n = 2), moderate (5), or severe (2) pulmonary abnormalities.

The diagnostic information assessed before starting PPV included arterial blood gases in 62% (92/148) of all animals, venous blood gases in 11% (16/148), and pulse oximetry in 3% (4/148). Cyanosis was detected in 3% (4/148) of animals, apnea in 11% (16/148), and excessive respiratory effort in 31% (46/148; some patients had more than 1 diagnostic test). In 22% of dogs and 50% of cats, no blood gas or pulse oximetry measurements were performed before starting PPV. In groups 1, 2, and 3, mean ± SD Pa_{O_2} was 80 ± 39 mm Hg, 189 ± 120 mm Hg, and 84 ± 22 mm Hg, respectively. Mean ± SD Pa_{CO_2} before PPV was 43 ± 11 mm Hg, 73 ± 26 mm Hg, and 69 ± 18 mm Hg, respectively, and P:F was 110 ± 68 mm Hg, 266 ± 216 mm Hg, and 116 ± 76 mm Hg, respectively.

Initiation of PPV—Pentobarbital, with or without diazepam, was the most common induction (29%) and maintenance (64%) agent. Nineteen percent (28/148) of animals did not need anesthetic induction for intubation; of these, 68% (19/28) had been intubated during a resuscitation procedure. Overall, 18% (27/148) of patients did not need maintenance anesthesia. Twenty-three of these animals were in group 2. Endotracheal tube intubation was used for PPV in 92% (67/73) of animals in group 1, 73% (45/62) of animals in group 2, and 10 of 13 animals in group 3. A temporary tracheostomy tube was placed in the remaining animals.

The first mode of ventilation in most (90%) animals was continuous mandatory ventilation; volume A-C ventilation was used in 57%, and pressure A-C ventilation was used in 33%. Of the group 1 animals, 51% were initially stabilized with volume A-C and 47% with pressure A-C ventilation. In group 2, 68% were initially stabilized with volume A-C, 21% with pressure A-C, and the remainder with SIMV. In group 3, 6 of 13 animals were initially stabilized with volume A-C, 4 were stabilized with pressure A-C, and 3 were stabilized with SIMV. The ventilator settings at which animals were considered initially stabilized were minute ventilation

of 365 ± 181 mL/kg (166 ± 82 mL/lb) in group 1, 367 ± 327 mL/kg (167 ± 149 mL/lb) in group 2, and 337 ± 111 mL/kg (153 ± 50 mL/lb) in group 3. Tidal volume was 18 ± 13 mL/kg (8 ± 6 mL/lb) in group 1, 17 ± 7 mL/kg (8 ± 3 mL/lb) in group 2, and 14 ± 2 mL/kg (6 ± 1 mL/lb) in group 3. Mandatory respiratory rate was 21 ± 9 bpm in group 1, 18 ± 7 bpm in group 2, and 24 ± 9 bpm in group 3. Peak airway pressure was 25 ± 7 cm H_2O in group 1, 18 ± 7 cm H_2O in group 2, and 22 ± 8 cm H_2O in group 3. Positive end-expiratory pressure was 6 ± 3.5 cm H_2O in group 1, 2 ± 2.4 cm H_2O in group 2, and 6 ± 3.0 cm H_2O in group 3.

After initial stabilization on the ventilator, arterial blood gas measurements were performed in 100% of animals in group 1, 79% of animals in group 2, and 13 of 13 animals in group 3. Those animals in group 2 that did not receive arterial blood gas measurements were monitored via venous blood gas measurements and pulse oximetry. Mean ± SD Pa_{O_2} was 139 ± 60 mm Hg in group 1, 204 ± 108 mm Hg in group 2, and 96 ± 9 mm Hg in group 3. The P:F was 226 ± 113 in group 1, 353 ± 213 in group 2, and 237 ± 106 in group 3. Mean Pa_{CO_2} was 38 ± 7 mm Hg across all groups.

Nutrition—Nutritional support was given in 38% (56/148) of all animals at a mean ± SD of 29 ± 26 hours after beginning PPV. Four animals were already receiving nutrition at the time PPV was initiated. In 30% (22/73) of animals in group 1, nutritional support was started during PPV, compared with 45% (28/62) in group 2 and 6 of 13 in group 3. Nutrition was provided by IV (38% [21/56]), nasogastric (27% [15/56]), or gastrotomy (21% [12/56]) tube. Also, in group 2, 8 animals ate voluntarily. Of the animals in which enteral nutrition was started, it was stopped in 59% (16/27) of animals because of complications of regurgitation in 8 animals, high gastric residual volumes in 7 animals, and aspiration of gastric contents in 1 animal. Nutritional support was continued throughout the PPV period in the remaining animals.

Duration—Median duration for all 148 ventilator episodes was 48 hours (range, 24 to 356 hours) and was similar among group 1 (47 hours; range, 24 to 218 hours), group 2 (48 hours; range, 24 to 356 hours), and group 3 (52 hours; range, 24 to 355 hours). There were no significant differences in duration among the groups, and none of the variables tested was significantly correlated with duration of PPV (**Table 3**).

Weaning from PPV—Synchronous intermittent mandatory ventilation was the most commonly used ventilator mode for weaning (47% [29/60]), and CPAP was used as a weaning mode in 23% (14/60) of animals. In those animals successfully weaned from the ventilator, the final ventilator settings prior to disconnection were determined. Mean ± SD minute ventilation was 436 ± 221 mL/kg (198 ± 100 mL/lb) in group 1, 304 ±

Table 3—Results of Cox proportional hazards analysis of factors potentially associated with duration of ventilation in 148 episodes of long-term PPV in dogs and cats.

Variable	Odds ratio*	95% Confidence interval	*P* value
Species†			
Dog	1.00	—	—
Cat	1.00	0.43–2.36	0.99
Sex			
Male (sexually intact)	1.00	—	—
Male (castrated)	1.00	0.48–2.09	0.94
Female (sexually intact)	0.96	0.37–2.49	0.94
Female (spayed)	1.09	0.52–2.27	0.83
Age (y)	0.99	0.93–1.05	0.64
Weight (kg)			
Dog	1.00	0.98–1.02	0.94
Cat	0.99	0.60–1.61	0.96
Reason for PPV‡			
Inadequate oxygenation (Group 1)	1.00	—	—
Inadequate ventilation (Group 2)	0.88	0.51–1.52	0.64
Inadequate oxygenation and ventilation (Group 3)	0.33	0.10–1.11	0.073
Pneumothorax			
Absent	1.00	—	—
Present	0.27	0.04–1.92	0.19
P:F (50-unit change in ratio)			
Before PPV	1.00	0.91–1.10	0.98
Initial stabilization	1.02	0.78–1.33	0.69
Ppeak at initial stabilization (cm H_2O)	0.97	0.93–1.01	0.15
PEEP at initial stabilization (cm H_2O)	0.95	0.88–1.03	0.26

See Table 1 for key.

108 mL/kg (138 ± 49 mL/lb) in group 2, and 295 ± 141 mL/kg (134 ± 64 mL/lb) in group 3. Mean peak airway pressure was 17 ± 7 cm H_2O in group 1, with a mean PEEP of 2.5 ± 1.7 cm H_2O. In group 2, the Ppeak was 15 ± 7 cm H_2O with a mean PEEP of 1.1 ± 1.7 cm H_2O, and in group 3, the Ppeak was 21 ± 0.7 cm H_2O with a mean PEEP of 4.3 ± 2.5 cm H_2O. Animals successfully weaned from PPV had a P:F prior to disconnection from the ventilator of 334 ± 109 in group 1, 390 ± 189 in group 2, and 369 ± 133 in group 3. Mean $F_{I}O_2$ setting at the time of weaning was 0.65 ± 0.50 in group 1, 0.27 ± 0.10 in group 2, and 0.33 ± 0.15 in group 3. Mean Pa_{CO_2} at weaning was 37 ± 7 mm Hg, 36 ± 8 mm Hg, and 40 ± 10 mm Hg in groups 1, 2, and 3, respectively.

Of the 59% of animals that were not weaned from the ventilator, minute ventilation at the time of death or euthanasia was 665 ± 668 mL/kg (302 ± 304 mL/lb) in group 1, 478 ± 563 mL/kg (217 ± 256 mL/lb) in group 2, and 389 ± 236 mL/kg (177 ± 107 mL/lb) in group 3. Peak airway pressure was 22.9 ± 16.6 cm H_2O with a PEEP of 8.9 ± 4.7 cm H_2O in group 1, 23.5 ± 7.4 cm H_2O with a PEEP of 3.3 ± 3.9 cm H_2O in group 2, and 28.8 ± 16.9 cm H_2O with a PEEP of 5.7 ± 3.2 cm H_2O in group 3. The P:F of these animals just prior to death or euthanasia was 200 ± 134, 288 ± 171, and 156 ± 131 in groups 1, 2, and 3, respectively. Mean F_{IO_2} setting at this time was 0.63 ± 0.26 in group 1, 0.40 ± 0.18 in group 2, and 0.66 ± 0.24 in group 3, and the mean Pa_{CO_2} was 51 ± 32 mm Hg in group 1, 38 ± 10 mm Hg in group 2, and 38 ± 16 mm Hg in group 3.

Complications—Pneumothorax occurred in 10 of 148 (7%) animals (6 dogs and 1 cat in group 1 and 3 dogs in group 2). Median time at which pneumothorax occurred was 13.5 hours (range, 2 to 78 hours) after initiating PPV. Prevalence of pneumothorax was not significantly different between groups 1 and 2 ($P = 0.30$). None of the variables tested were significantly associated with development of pneumothorax (**Table 4**).

Of the 148 ventilator episodes, 37 medical records contained insufficient detail to accurately determine the prevalence of complications. Of the remaining 111 episodes, oral ulceration was reported in 8% (9/111) of animals and corneal ulceration in 5% (6/111) of animals. Tracheal tube occlusion occurred more than once in 14% (15/111) of animals, and tracheal tube disconnection or dislodgement was reported in 14% (15/111) of animals. Overall, excessive airway fluid was noted in 24% of animals, and two thirds of these animals were in group 1. Gastric distension requiring decompression was reported to occur more than once in 7 group 2 animals, and 4 animals in group 2 chewed through the ventilator circuit more than once; all of these were awake animals managed with a temporary tracheostomy tube.

Postmortem examination was performed in 47% (41/88) of the animals that died or were euthanatized during PPV. Of the necropsies performed, 83% (34/41) of animals had pulmonary lesions and 17% (7/41) had none. Of the patients in group 1 that had necropsies, 96% (24/25) had pulmonary lesions; in group 2, 7 of 13 necropsies revealed pulmonary lesions, and in group 3, 3 of 3 necropsies revealed pulmonary lesions. Twenty-three percent (14/62) of the group 2 animals acquired parenchymal pulmonary disease during the ventilation period, as determined by use of bacteriologic culture of specimens from the airway or radiographic, postmortem, or clinical evaluation. The nature of the acquired pulmonary disease was considered to be pneumonia in 9 animals; 2 dogs had ARDS at postmortem, 2 had diffuse lung injury after CPR, and 1 dog had diffuse pulmonary congestion of unknown etiology at postmortem. The 9 animals with pneumonia included 1 dog with cervical spinal cord disease and a

Table 4—Results of logistic regression analyses of factors potentially associated with the occurrence of pneumothorax in 148 episodes of long-term PPV in dogs and cats.

Variable	Odds ratio*	95% Confidence interval	*P* value
Species†			
Dog	1.00	—	—
Cat	0.56	0.07–4.60	0.59
Sex			
Male (sexually intact)	1.00	—	—
Male (castrated)	1.78	0.18–17.88	0.63
Female (sexually intact)	8.29	0.85–81.19	0.069
Female (spayed)	1.26	0.11–14.54	0.85
Duration of PPV (per 6-h increment)	0.98	0.92–1.05	0.62
Reason for PPV‡			
Inadequate oxygenation (Group 1)	1.00	—	—
Inadequate ventilation (Group 2)	0.48	0.12–1.94	0.30
P:F (50-unit change in ratio)			
Before PPV	0.97	0.75–1.26	0.83
At initial stabilization	0.79	0.62–1.03	0.077
Ppeak at initial stabilization (cm H_2O)	1.07	0.99–1.17	0.090
PEEP at initial stabilization (cm H_2O)	1.10	0.94–1.28	0.26

See Table 1 for key.

collapsing trachea in which a tracheal stent was placed during the ventilation period and 1 dog with organophosphate toxicosis that had aspiration pneumonia. In 3 dogs suspected of developing pneumonia, endotracheal lavage samples were obtained for bacteriologic culture; a single organism was cultured in each case (*Klebsiella* spp [n = 2] and *Escherichia coli* [1]).

Discussion

This study found that the survival rate from long-term PPV correlated with the underlying disease process. Group 2 animals had a significantly higher likelihood of survival to hospital discharge, compared with groups 1 and 3. Spontaneous death was not differentiated from euthanasia in this study because, in most instances, euthanasia was performed because of a combination of poor prognosis and financial considerations. The outcome statistics in this study may, therefore, be an underestimation of the true weaning and survival rates that could be achieved in the absence of euthanasia. Meaningful comparison of survival rates between this study and previous veterinary studies was difficult because of the various study designs. All previous studies include short-term PPV episodes, some studies report weaning rates, and others report only hospital discharge rates.

Outcome statistics are consistently worse for animals with parenchymal pulmonary disease, compared with neuromuscular disease. In a previous study[1] of 34 dogs and 7 cats with heterogeneous lung disease, an overall survival to hospital discharge rate of 20% was reported. In another study,[3] 3 of 10 dogs requiring PPV for pulmonary contusions survived to hospital discharge. In the present study, 22% of animals with parenchymal pulmonary disease survived to hospital discharge. A retrospective study[7] of human intensive care patients treated with > 24 hours of PPV revealed a survival to discharge (from the intensive care unit) rate of 29.3% in the group with oxygenation impairment and 57.8% in the group with ventilatory insufficiency. This implies that diseases causing neuromuscular dysfunction are more amenable to treatment than those causing pulmonary abnormalities. In addition, pulmonary disease typically necessitates more aggressive ventilator settings. This increases the risk of ventilator-induced lung injury and further deterioration in pulmonary function and may propagate a systemic inflammatory response.[8,9]

In the present study, cats had a significantly lower chance of successful weaning from PPV, compared with dogs. A recent retrospective study[5] also revealed a low weaning rate for cats; the overall weaning rate was 15% (8/53) for cats requiring PPV for any reason, but the survival to hospital discharge rate was not reported. It is not clear why cats requiring PPV do not do as well as dogs. Although 2 previous studies[1,3] revealed lower survival rates in dogs of smaller body weights, weight was not significantly associated with successful weaning or survival to hospital discharge in the present study. Monitoring oxygenation is certainly more challenging in cats; regular arterial blood sampling is often not feasible, and pulse oximeters may not consistently provide measurements. This may contribute to less than ideal ventilator management and subsequent poorer outcomes. Because PPV weaning and survival rates vary with underlying disease processes, it is also possible that cats often require PPV for more severe disease processes than dogs.

The survival rate in group 1 varied according to the primary pulmonary disease process; for example, patients with pulmonary contusions and aspiration pneumonia had better weaning (57% and 60%, respectively) and survival to hospital discharge rates (40% and 30%, respectively) than patients with ARDS, which had the lowest weaning and survival to hospital discharge rate (1/12 for both). The outcome of patients with pulmonary contusions in this study was similar to that reported previously.[3] A study[3] of 10 dogs requiring PPV for thoracic trauma and pulmonary contusions reported a 4/10 weaning rate and 3/10 hospital discharge rate. In a retrospective study[2] of dogs with pulmonary contusions, 1 of 3 dogs requiring PPV survived to hospital discharge.

Acute respiratory distress syndrome was associated with the lowest survival rate of all pulmonary disease processes in the present study, and this survival rate (1/12) was low, compared with human patients in which survival rates have been reported to range from 42% to 75%.[8,10-13] In addition, patients with ARDS had the lowest survival rate (48%) of all disease processes in a prospective, multicenter study[14] of adult human PPV patients. Mortality rates for ARDS have not been previously reported in veterinary patients. The low survival rate in the present study likely reflected the disease severity; the inexperience in management of this disease; and, possibly, the diagnostic criteria used for diagnosing ARDS. Diagnosis of ARDS in this study was based on postmortem findings in 8 patients and the development of acute severe hypoxemia and moderate-to-severe diffuse pulmonary infiltrates in the absence of evidence of fluid overload or cardiac failure in the remaining 4 patients. All 4 animals had an underlying primary disease process known to cause ARDS. These may be insensitive diagnostic criteria and possibly only identified the most severely affected patients. Further investigation of veterinary ARDS patients is required before accurate prevalence and mortality rates can be determined.

The survival to hospital discharge rate of animals with neuromuscular disease processes in the present study (39%) was lower than previously reported.[1] A 57% hospital discharge rate was reported for dogs and cats requiring PPV for ventilation failure.[1] This difference was attributed to a difference in the disease processes in the study group.

The survival to hospital discharge rates for group 2 animals in this study varied with the underlying disease process. For example, survival to hospital discharge rate for animals with toxicoses was 86%; for diffuse LMN disease was 57%; for recovery after craniotomy was 71%; and for cervical spinal cord disease was 50%, compared with a poorer survival rate for animals with intracranial disease of 13%, and rate of recovery after CPR of 11%. Of the dogs with cervical spinal cord disease that required PPV in this study, 5 of 8 were weaned and 4 of 8 survived to hospital discharge. This was lower than the survival rate of 10 of 14 reported in a pre-

vious study[4] of dogs with cervical spinal cord disease requiring PPV. The previous study[4] included short-term PPV episodes and may not be directly comparable. Feline patients in group 2 had a worse outcome than the group as a whole; 4 of 16 cats were successfully weaned from PPV, and all 4 survived to hospital discharge. Ventilatory failure in cats may be associated with diseases with a worse prognosis than in dogs. Toxicoses, diffuse LMN disease, and cervical spinal cord disease were uncommon diseases in cats, whereas ventilatory failure caused by intracranial disease was overrepresented.

Animals with both inadequate oxygenation and inadequate ventilation at the time of initiation of PPV had the lowest weaning and survival rates in this study. These patients had both an important neuromuscular disease process and an important parenchymal pulmonary disease, or they had primary pulmonary disease which had led to respiratory muscle fatigue. This group contained animals with greater severity of illness, and the lower weaning and survival rates were not surprising.

Positive end-expiratory pressure improves pulmonary mechanics and gas exchange by improving pulmonary compliance, recruiting collapsed alveoli, and preventing cyclic alveolar collapse.[13,15] Higher PEEP values were used in group 1 and 3 animals when they were initially stabilized on the ventilator than in group 2 patients in this study. Requirement for increased PEEP when animals were initially stabilized on the ventilator was associated with a reduced likelihood of weaning and survival to hospital discharge, and reduced PEEP at weaning was associated with an increased likelihood of survival to discharge. These results may have reflected the use of higher PEEP settings in patients with more severe disease processes. Furthermore, because PEEP can lead to increased mean airway pressure and lung volumes, patients on higher PEEP settings may be at greater risk of ventilator-associated lung injury.[13]

Age was associated with outcome of PPV patients in a previous veterinary study[1] and several human studies.[13,16,17] King and Hendricks[1] reported that animals < 1 year old or > 11 years old had a higher mortality rate. In the present study, every increase in age of 1 year reduced the odds of survival to hospital discharge by 10%. Increased age is well correlated with negative outcome and is incorporated into the Acute Physiology and Chronic Health Evaluation II scoring system that is widely accepted in the human literature as a general measure of disease severity.[18]

The P:F is indirectly proportional to the severity of lung injury.[19,20] In the present study, a higher P:F prior to PPV was significantly associated with successful weaning from PPV and survival to hospital discharge and a higher P:F at weaning was significantly associated with survival to hospital discharge. The P:F is a significant predictor of death in human ARDS patients.[12,19] Because the P:F is a reflection of the severity of lung injury, its relationship to outcome is understandable. The P:F provides a simple measure of pulmonary oxygenating ability that is comparable across varying F_{IO_2} concentrations and is useful for evaluation of PPV patients.

Although arterial blood gas measurement is a valuable diagnostic tool, arterial blood sample collection can be challenging in small patients and the restraint required for the technique may be detrimental to animals with substantial respiratory distress. Pulse oximetry is often used as a surrogate measure of Pa_{O_2} in such patients. However, in 22% of dogs in the present study and 50% of cats, no blood gas or pulse oximetry measurements were performed before PPV was begun. Ventilation was initiated in these animals in response to clinical findings such as excessive respiratory effort; apnea; weak, ineffective respiratory efforts; and cyanosis. Severe respiratory distress is a life-threatening condition, and in many cases, PPV can be a life-saving intervention. Delaying PPV to perform diagnostic tests can be dangerous in this patient population; the decision to begin PPV may need to be made on the basis of clinical signs alone.

Patients with normal neurologic function require anesthesia to allow intubation and mechanical ventilation. In this study, pentobarbital, with or without diazepam, was most often used for anesthetic induction and maintenance. Although pentobarbital is the authors' anesthetic of choice for long-term mechanical ventilation, it does have adverse effects. Short-term (1 to 2 days) pentobarbital CRI is associated with prolonged hyperreflexic recoveries; this problem can be minimized by a benzodiazepine CRI during the recovery period. After 3 to 5 days of a pentobarbital CRI, dogs appear to have extreme dysphoria for 12 to 24 hours after recovering consciousness. This can be minimized by maintaining anesthesia with a propofol CRI for 24 hours beyond termination of the pentobarbital CRI so that the animal recovers from propofol rather than from pentobarbital. After approximately 1 week of a pentobarbital CRI, dogs may have seizures upon recovery. This can be minimized by administration of phenobarbital (approx 4 mg/kg [1.8 mg/lb], q 12 h, started approx 3 days prior to terminating administration of pentobarbital) in addition to a benzodiazepine CRI.

Most (92%) animals in group 1 were managed with an endotracheal tube, whereas 27% of group 2 animals were ventilated via a temporary tracheostomy tube. In animals with neurologic abnormalities and that cannot resist PPV, a tracheostomy tube may allow the patient to be conscious. This improves neurologic monitoring; in addition, many animals will eat and drink normally and are able to interact with their owners. In contrast, animals with normal neurologic function require general anesthesia to tolerate PPV, and consequently, tracheostomy tubes offer little advantage.

The goal of PPV is to maintain adequate oxygenation and ventilation with the least aggressive ventilator settings possible. The blood gas goals were a Pa_{O_2} of 80 to 120 mm Hg, with a minimum of 60 mm Hg, and a Pa_{CO_2} of 35 to 45 mm Hg, with a maximum of 60 mm Hg. When ventilating animals with intracranial disease, the Pa_{CO_2} is more strictly controlled between 35 and 45 mm Hg. When initially stabilized on the ventilator, the animals in groups 1 and 2 of this study had Pa_{O_2} measurements of 139 ± 60 mm Hg and 204 ± 108 mm Hg, respectively. The P:F at this time was 226 ± 113 in group 1, 353 ± 213 in group 2, and 237 ± 106 in group 3. Patients are initially stabilized on PPV with an F_{IO_2} of 1.0, and the F_{IO_2} is thereafter reduced to achieve a desired Pa_{O_2}. The ultimate aim is to decrease the F_{IO_2} to

< 0.6 to minimize the possibility of oxygen toxicosis.[21] In animals that have severe abnormalities in oxygenation such that the F_{IO_2} cannot be adequately reduced, increases in PEEP and Ppeak are made in an effort to increase the oxygenating efficiency of the lungs. The minute ventilation is manipulated to obtain a desired P_{aCO_2}.

Controlled mandatory ventilation was the mode used for initial stabilization in 90% of patients in this study because it transfers most, if not all, of the work of breathing to the ventilator. Volume and pressure A-C ventilation were used with similar frequency in all 3 groups. Modes of ventilation that rely on intermittent or continuous spontaneous breathing such as SIMV and CPAP were rarely used for initial stabilization.

Minute ventilation is the product of respiratory rate and tidal volume. Typical values in healthy dogs are from 150 to 250 mL/kg (68 to 114 mL/lb).[22,23] Total minute ventilation includes the dead space volume and overrepresents alveolar minute ventilation at higher respiratory rates with reduced lung function.[24] The minute ventilation delivered to patients during initial stabilization in the present study exceeded 300 mL/kg (136 mL/lb) in all groups; however, the mean P_{aCO_2} (a reflection of alveolar minute ventilation) for all groups on initial stabilization was 38 mm Hg. The ventilator used most commonly in the present study did not compensate for the volume of breathing circuit expansion and gas compression during the inspiratory phase. Recorded tidal and minute volumes, therefore, overrepresented the tidal and minute volumes actually delivered to the patient. Newer ventilators compensate for breathing circuit expansion and gas compression and provide more accurate measures of delivered volumes. The minute ventilation was similar in all 3 groups. This was most likely a result of the ventilation strategy, which included titration of minute ventilation to achieve a desired P_{aCO_2} of 35 to 45 mm Hg. The similarity between the total measured minute ventilation and the mean P_{aCO_2} of each group was a reflection of the lungs' ability to adequately maintain normal P_{aCO_2} concentrations provided there was adequate alveolar minute ventilation despite the presence of parenchymal pulmonary disease and inadequate oxygenating ability.

The general recommendation for PPV of healthy lungs is a Ppeak of 10 to 20 cm H_2O.[6] Lung disease decreases pulmonary compliance, and such patients often require higher airway pressures for effective ventilation. This was reflected by the Ppeak in the groups 1 and 3 animals in the present study, 25 ± 6.7 cm H_2O and 22 ± 7.8 cm H_2O, respectively, compared with 18 ± 7.0 cm H_2O for the group 2 animals, which had more normal lung function.

In the present study, nutritional support was administered to 38% (56/148) of the animals and was started at 29 ± 26 hours after initiation of PPV. Median duration of ventilation was 48 hours, and in many animals, it was deemed acceptable to wait until after the animals were weaned off the ventilator to start nutritional support. Complications with enteral nutrition were regurgitation and high gastric residual volumes. Feeding was stopped in these animals because of concerns of increased risk of aspiration. Aspiration of gastric contents is a cause of VAP, and this complication is associated with increased morbidity and mortality rates.[25] Eight of the animals in group 2 that were managed awake by use of a temporary tracheostomy tube were able to eat and drink normally without complication.

A study[26] of 5,915 human patients revealed that the primary diagnosis was the most important determinant of PPV duration. Parenchymal pulmonary diseases such as ARDS and pneumonia as well as neuromuscular diseases were associated with increased length of ventilation. A previous veterinary study[1] revealed that the mean duration of PPV was significantly longer in patients with inadequate ventilation, compared with patients with inadequate oxygenation. The duration of ventilation of patients in the present study was not significantly different among groups and could not be correlated with any of the variables tested. From the present study, it is apparent that animals can be successfully supported on PPV for at least 14 days. Since the time of this study, a dog with cervical spinal cord disease was successfully weaned after 30 days of ventilation. In addition, the duration of PPV was not a negative predictor of successful weaning or survival to hospital discharge. King and Hendricks[1] also found no significant difference between the duration of PPV in survivors and nonsurvivors, although there was a higher mortality rate in the initial 24 hours of ventilation, compared with the mortality rate of animals ventilated longer than 24 hours. In contrast, a retrospective study[5] of feline PPV patients revealed that the median duration of ventilation was significantly longer in survivors, compared with nonsurvivors. Because that study[5] included short-term PPV episodes, perhaps this finding also reflects a higher mortality rate early in the ventilation period.

Weaning from PPV requires the patient to perform a gradually increasing proportion of the work of breathing. The level of ventilatory support is slowly reduced to reestablish adequate respiratory muscle function and to ensure that the animal can maintain oxygenation and ventilation requirements. In the present study, the 2 most common weaning modes were SIMV (29/60) and CPAP (14/60); SIMV is a combination of mandatory and spontaneous breaths. The weaning process involves a gradual reduction in the number of mandatory breaths. In comparison, CPAP is a completely spontaneous ventilator mode and weaning can be achieved by a gradual reduction in the positive airway pressure. When most of the work of breathing is being performed by the patient and respiratory function remains adequate, discontinuation of ventilator support can be considered. The mean F_{IO_2} at the time of weaning in group 1 was 0.65 ± 0.5. Many patients required ongoing oxygen supplementation after weaning. Young age, high P:F before starting PPV, and low PEEP on initial stabilization on PPV were all associated with successful weaning. Because these findings indicate less disease severity, it is not surprising that such patients had a better chance for successful weaning from PPV.

The prevalence of pneumothorax in human PPV patients ranges from 2.9% to 42%.[8,13,14,27,28] The role of high inspiratory pressures and volumes in the development of pneumothorax is controversial. In several recent human studies,[8,13,28,29] the magnitude of ventila-

tor settings could not be correlated with the prevalence of pneumothorax. In the present study, pneumothorax occurred in 7% (10/148) of animals and was not significantly associated with outcome. In 2 previous veterinary studies,[1,5] pneumothorax occurred in 28% and 29% of animals.[1,5] In human medicine, the prevalence of pneumothorax varies with the primary disease process. In the present study, there was no significant difference in the frequency of pneumothorax between the groups 1 and 2 animals. In the 2 previous veterinary studies,[1,5] pneumothorax occurred more frequently in animals with parenchymal pulmonary disease, although these findings were not tested statistically. The overall low rate of occurrence of pneumothorax in the present study may have made it difficult to detect a difference in frequency between the group 1 and 2 animals.

Ventilator-associated pneumonia is defined as the development of nosocomial pneumonia after 48 hours or longer of PPV in patients that had no previous evidence of pneumonia.[25] In patients with preexisting pulmonary disease, it would be impossible to differentiate VAP from deterioration of the primary disease process in the absence of routine bacteriologic culture of specimens obtained from the airways. Group 2 animals in the present study started PPV with no evidence of parenchymal pulmonary disease. Of these patients, 23% (14/62) had acquired pulmonary disease during the ventilation period. Nine had pneumonia on the basis of radiographic evidence, bacteriologic culture, and necropsy findings; one of these animals had a witnessed aspiration event, and another developed pneumonia subsequent to placement of a tracheal stent for tracheal collapse. The most likely cause of pneumonia in the remaining 7 animals was VAP (prevalence, 7/62). This was likely to be an underestimation of the prevalence of VAP because routine thoracic radiography and bacteriologic cultures were not performed. Ventilator-associated pneumonia was reported in 15% (6/41) of dogs and cats in 1 study[1] and 15% (8/53) of cats in another study.[5]

Oral ulceration occurs in patients during PPV as a result of the loss of normal oral hygiene mechanisms and mechanical injury from teeth and equipment such as orotracheal tubes.[30] Oral ulceration was reported in 8% (9/111) of patients in the present study. A previous study[30] in our intensive care unit revealed a 90% prevalence of oral lesions during mechanical ventilation. The prevalence has been reduced by the introduction of an oral hygiene protocol. Prevalence in the present study may have been underrepresented because many of the staff considered oral lesions a normal consequence of PPV and therefore did not record it as a problem. Oral ulceration is a primary source of bacterial proliferation; bacteria can then either colonize the airways, leading to VAP, or gain direct access to the systemic circulation, leading to sepsis.[31-33] Diligent attention to oral hygiene and prevention of pressure between the endotracheal tube or teeth and the mucous membranes of the tongue and lips is an essential aspect of the management of ventilated patients.

Corneal ulceration occurred in 5% (6/111) of animals in this study. To our knowledge, this has not been previously reported in the veterinary literature. Perhaps the longer duration of PPV in the present study amplified the problem. In 1 patient that developed severe bilateral corneal ulceration, it was considered to be a contributing factor to the owner's decision for euthanasia. Corneal ulceration was believed to be caused by corneal dehydration or trauma resulting from the loss of normal eyelid closure and tear production in the anesthetized patient. Ocular care is important in the care of comatose or anesthetized patients, and lateral temporary tarsorrhaphy may be indicated in some animals.

Breathing circuit issues included tracheal tube occlusion, disconnection of the breathing circuit, accumulation of airway secretions, and patients chewing through the breathing circuit. These types of complications occur frequently and are often viewed as routine by the technical staff. As a result, these issues are likely to be underreported. Tracheal tube occlusion occurs with endotracheal and temporary tracheostomy tubes and can cause acute life-threatening airway obstruction. One dog in this study died as a result of difficulties in the timely replacement of a dislodged temporary tracheostomy tube. Tube occlusion can be caused by accumulation of airway sections or tube kinking. Regular tube suctioning and changing of tracheal tubes are recommended to minimize occlusions. Tracheal tube patency should be assessed on an ongoing basis. Tracheal tube disconnections can also be immediately life threatening, especially in patients that do not have adequate ventilatory capability. Most ventilators sound an alarm with the loss of airway pressure, and capnometers will also display appropriate changes. The patients in group 2 that were ventilated without sedation with a temporary tracheostomy tube had some unique complications. In addition to frequent circuit disconnections related to patient movement, there were several incidents of dogs chewing through the ventilator circuit. Gastric distension from aerophagia was also a problem for some of these animals and generally appeared to occur in association with increased breathing effort. These issues highlight some of the management challenges associated with conscious animals that are still dependent on PPV.

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b. Esprit Ventilator, Respironics Inc, Murrysville, Pa.

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