

Pulmonary function, ventilator management, and outcome of dogs with thoracic trauma and pulmonary contusions: 10 cases (1994–1998)

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Objective—To document pulmonary function, ventilator management, and outcome of dogs with thoracic trauma that required mechanical ventilation because of severe pulmonary contusions.

Design—Retrospective study.

Animals—10 dogs that required mechanical ventilation because of severe pulmonary contusions caused by blunt thoracic trauma.

Procedure—Signalment, historical data, arterial blood gas values, oxygen tension-based indices, ventilator settings, peak inspiratory pressure, positive end-expiratory pressure, tidal volume, and minute ventilation values were retrieved from medical records.

Results—All 10 dogs required positive-pressure ventilation because of dyspnea following trauma and had severely abnormal pulmonary function. Survival rate to discharge was 30%. Dogs were categorized into 2 groups; group A included 5 dogs in which pulmonary function improved during ventilation, whereas group B included 5 dogs that were euthanized because of progressive lung dysfunction ($n = 4$) or cardiac arrest (1). Mean \pm SD body weight of group-A dogs (30.9 ± 15.9 kg [68 ± 35 lb]) was significantly greater than that of group-B dogs (7.6 ± 1.8 kg [16.7 ± 4 lb]). Dogs with improved lung function had peak inspiratory pressure that decreased progressively, whereas lung compliance deteriorated in dogs in group B.

Conclusions and Clinical Relevance—Dyspneic dogs with severe pulmonary contusions may require and benefit from positive-pressure ventilation. Prognosis is better for dogs that weigh > 25 kg (55 lb). (*J Am Vet Med Assoc* 2000;217:1505–1509)

Pulmonary contusions and respiratory distress are often sequelae to blunt thoracic trauma. During the early phase of injury after blunt trauma, several mechanisms lead to the formation of pulmonary contusions and progressive lung dysfunction.¹ Initially, transmission of the mechanical force to the thoracic wall causes an increase in tissue pressure, followed by a sudden decrease in pressure, which results in tearing, edema, and hemorrhage of the lung parenchyma. There is a proportional relationship between the compressive force and the inward displacement of the thoracic wall, which also leads to crushing injury of the lung parenchyma. In addition, the damaged lung

parenchyma has impaired clearance of fluid and cells. Lastly, the lung parenchyma has the potential to become lacerated during the injury, and blood from the injured segments of the lung may spread to uninjured segments, which further impairs lung function.^{1,3} It has been suggested that the progressive nature of pulmonary contusions commonly seen in clinical medicine is attributable to multiple injuries and administration of fluids.⁴

Most animals with pulmonary contusions respond to supportive care, rest, and oxygen supplementation.⁵ In some animals, pulmonary function declines so severely that respiratory arrest is imminent if intervention with mechanical ventilatory support is not pursued. Little information is available regarding the pulmonary function, management, and outcome of dogs with thoracic trauma that have been mechanically ventilated.

The purpose of the study reported here was to retrospectively evaluate the medical records of dogs that needed ventilatory support because of severe pulmonary contusions associated with blunt thoracic trauma. We wished to determine clinical course and outcome, characterize pulmonary function, and identify prognostic indicators that may allow us to improve management in the future.

Criteria for Selection of Cases

We reviewed medical records from 1994 to 1998 to identify dogs with radiographic evidence of pulmonary contusions that were mechanically ventilated because of severe respiratory distress secondary to blunt thoracic trauma. Patients with blunt thoracic trauma commonly have multiple cardiopulmonary injuries that, in addition to pulmonary contusions, may contribute to progressive cardiopulmonary dysfunction, tissue hypoxia, and mortality. These injuries include pneumothorax, hemothorax, myocardial-pericardial contusions, pericardial effusion, arrhythmias, rib fractures, or flail chest.^{6–8} Although our main objective was to characterize the clinical findings in dogs with pulmonary contusions, we realistically could not exclude dogs that had multiple thoracic injuries, considering the nature of our study group. Dogs that obviously were ventilated for reasons not related to their pulmonary contusions were excluded.

Procedures

Data regarding medical procedures and pulmonary function before, during, and after positive-pressure ventilation (PPV) were compiled. History, signalment, clinical signs at referral, body weight, initial problem list, thoracic radiographic findings, fluid therapy, ven-

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tilator^a management, arterial blood gas (ABG)^{b,c} measurements, complications, and outcome were recorded.

The ABG results and ventilator settings were analyzed during 3 selected time periods. When available, ABG measurements taken before ventilation were recorded. When dogs were on the ventilator and settings appeared stable, a representative ABG measurement and the ventilator settings were recorded. Representative samples of ABG measurements and ventilator settings were taken if there was a substantial change in ventilator settings and a correlating ABG measurement was available after the ventilator setting change. Typically, these samples were obtained every 12 hours until weaning from the ventilator or death. If a major clinical change developed within each 12-hour period, the ventilator settings and ABG measurements were recorded.

The ratio between the PaO₂ and the corresponding fraction of inspired oxygen (FIO₂) was calculated to standardize and compare oxygenation among dogs. Peak inspiratory airway pressure (PIP), positive end-expiratory pressure (PEEP), respiratory rate, PaCO₂, and duration of ventilation were recorded. Minute ventilation per kilogram of body weight and tidal volume (TV) per kilogram of body weight were also calculated.

The dogs were categorized into 2 groups. Group A included dogs in which pulmonary function improved during ventilation, whereas group B included dogs that had progressive lung dysfunction.

Ventilator settings and other variables were compared among dogs and between groups. Data were analyzed to determine whether certain variables may help to predict outcome. Variables that had only a single value for each patient were analyzed by use of the Student *t*-test to compare groups A and B. Variables that had multiple values in individual dogs during the course of treatment were analyzed by use of regression analysis, and slopes and intercepts were calculated for each variable in each patient. Mean slopes and intercepts of the groups were compared by use of the Student *t*-test. Values were calculated as mean \pm SD unless otherwise indicated, and significance was set at $P \leq 0.05$.

Results

Ten dogs were eligible for inclusion in the study. Eight dogs were female (4 spayed), and 2 dogs were sexually intact males. There were 4 mixed-breed dogs and 1 each of the following breeds: Golden Retriever, Great Dane, Jack Russell Terrier, Shih Tzu, Yorkshire Terrier, and Cocker Spaniel. Mean and median ages were 4 ± 4.2 years and 2 years, respectively, with a range of 5.5 months to 12.5 years. Mean body weight of the dogs was 19.2 ± 16.3 kg (42.3 ± 35.8 lb) with a median body weight of 10.5 kg (23.1 lb) and a range of 5 to 50 kg (11 to 110 lb).

Mean elapsed time from trauma to evaluation at the emergency room was 2 ± 1.57 hours, excluding 1 dog that arrived 18 hours after trauma occurred. Most dogs ($n = 8$) were hit by a car, 1 was dragged by a car, and 1 fell from a 12-foot deck.

At referral, mean heart rate was 152 ± 32 beats/min, mean respiratory rate was 47 ± 24 breaths/min, and 6 of

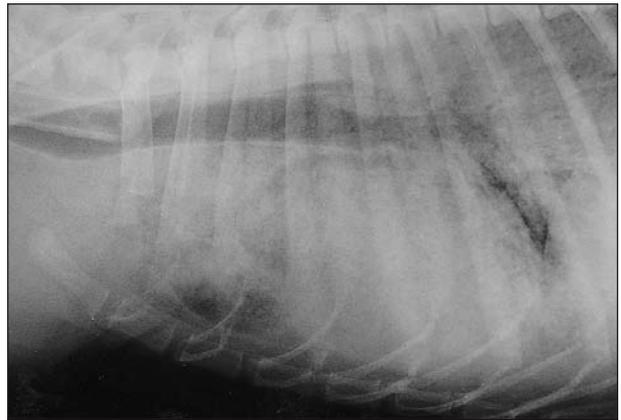


Figure 1—Lateral thoracic radiographic view of a dog with severe lung contusions.

10 dogs had initial rectal temperature < 36.6 C (98 F). Eight of the dogs were originally assessed as being hemodynamically unstable. All of the dogs had radiographic evidence of pulmonary contusions (Fig 1). In addition, concurrent traumatic injuries were severe. Two dogs had rib fractures, 7 had pneumothorax, 4 had hemothorax, and 3 had hemoperitoneum. All dogs with pneumothorax were treated by placement of thoracostomy tubes, and continuous negative-pressure thoracic drainage was used. Only 1 dog had concurrent hemoperitoneum and hemothorax. Four dogs had ventricular arrhythmias, and of these 4 dogs, 1 had concurrent hemoperitoneum, 2 had hemothorax, and 1 had pneumothorax. Three of 4 dogs with ventricular arrhythmias weighed > 25 kg (55 lb), and all 4 dogs with ventricular arrhythmias were in group A. Only 2 dogs had concurrent long-bone fractures, and both of these dogs weighed > 25 kg.

For all dogs, routine supportive care, including oxygen supplementation, was provided. The clinical decision to initiate PPV was based on the presence of severe dyspnea or tachypnea ($n = 10$), verified hypoxemia (7), hypercarbia (2), signs of pain or flail chest (1), or respiratory arrest (1), despite aggressive supportive care. Mean time from arrival to intubation and ventilation was 16.5 ± 17.2 hours (range, 0.25 to 53 hours), and mean duration of treatment with a ventilator was 32 ± 18.2 hours (range, 8 to 77 hours). After induction of anesthesia and intubation, all dogs were immediately placed on assist-control ventilation, except for 1 dog that was spontaneously breathing while intubated.

Various anesthetic protocols were used throughout the ventilatory period. All 10 dogs received diazepam and an opioid anesthetic such as oxymorphone, hydromorphone, or fentanyl, 8 dogs received a barbiturate (either pentobarbital or thiopental), 7 dogs received a nondepolarizing neuromuscular blocker (either atracurium or cis-atracurium), 1 dog received etomidate, and 1 dog received propofol.

Various drugs were administered during hospitalization. Seven dogs received corticosteroids either prior to or at initial evaluation. All 10 dogs received furosemide during the ventilatory period. Three of the 4 dogs that had ventricular arrhythmias were treated with continuous infusions of lidocaine. Nine of the 10

Table 1—Arterial blood gas measurements (mean \pm SD) obtained before initiation of positive-pressure ventilation (PPV; $n = 7$) and during PPV at initial ventilator settings (10) in dogs with pulmonary contusions caused by thoracic trauma

Blood gas measurement	Variable					
	PaO ₂ (mm Hg) ^a	Paco ₂ (mm Hg) ^b	pH ^c	Base excess (mmol/L) ^d	FiO ₂ ^e	PaO ₂ :FiO ₂ ^f
Before PPV	55.84 \pm 17.45	37.79 \pm 9.76	7.35 \pm 0.15	-3.6 \pm 7.37	0.76 \pm 0.23	77.49 \pm 24.8
Initial PPV	147 \pm 78.8	52.57 \pm 23.58	7.29 \pm 0.17	-3.34 \pm 5.21	0.79 \pm 0.27	201 \pm 92.5

^aPaO₂ reference range, 91 to 118 mm Hg (FiO₂ = 0.21). ^bPaco₂ reference range, 26 to 41 mm Hg. ^cpH reference range, 7.36 to 7.47. ^dBase excess reference range, -4 to 4 mmol/L. ^eFiO₂ reference range, 0.21 to 1. ^fPaO₂:FiO₂ reference range, 350 to 500.

dogs were treated with various antimicrobials (alone or in combination) during ventilation, including ticarcillin-clavulanate ($n = 5$), ampicillin (4), enrofloxacin (3), metronidazole (2), cefazolin (1), and gentamicin (1). Endotracheal washes were performed within the first 24 hours of starting PPV in 7 dogs and submitted for bacteriologic culture and susceptibility testing. Bacteria were cultured from specimens from 3 dogs, including *Acinetobacter* spp ($n = 2$), *Staphylococcus* sp (1), *Escherichia coli* (1), *Enterococcus* sp (1), and *Clostridium* sp (1).

All 10 dogs received IV administration of fluids prior to or upon initial evaluation as well as combinations of isotonic crystalloid solutions, hypertonic saline (7.5% NaCl) solution, hetastarch, dextran 70, plasma, or packed RBC. During PPV, all 10 dogs received crystalloid solutions at mean fluid administration rate of 3.8 ± 1.7 ml/kg (1.7 ± 0.8 ml/lb) of body weight/h. Six dogs received hetastarch during ventilation at mean administration rate of 0.8 ± 1.6 ml/kg (0.4 ± 0.7 ml/lb)/h.

Complications during or following ventilation were as follows: desaturation ($n = 3$), pneumonia (3), tachycardia (3), iatrogenic pneumothorax (2), hypotension (2), pancreatitis (2), accidental extubation (1), pitting edema (1), decreased urine output (1), corneal ulcer (1), urinary tract infection (1), acute respiratory distress syndrome (1), and anemia (1).

Arterial blood gas measurements obtained before ventilation were available in 7 of 10 dogs (Table 1). Mean PaO₂ before ventilation was 55.84 ± 17.45 mm Hg, despite mean FiO₂ of 0.76 and mean PaO₂:FiO₂ of 77.49 ± 24.8 . After intubation and stabilization at the initial ventilator settings, mean PaO₂ increased to 147 ± 78.8 mm Hg ($n = 10$), with mean FiO₂ of 0.79 and mean PaO₂:FiO₂ of 201 ± 92.5 . The PaO₂ and PaO₂:FiO₂ values were significantly different ($P = 0.03$ and 0.01 , respectively) immediately after initiation of mechanical ventilation, compared with those values obtained prior to ventilation.

After the dogs were stabilized on the ventilator, the initial ventilator settings were recorded in 9 of the 10 dogs. One dog was intubated but breathing spontaneously; therefore, ventilator settings were not applicable. Mean PIP was 29.2 ± 6 mm Hg, with mean PEEP of 4.9 ± 4.2 mm Hg. Mean minute ventilation was 0.74 ± 0.96 L/kg (0.34 ± 0.44 L/lb)/min, and mean tidal volume was 27 ± 19.9 ml/kg (12.3 ± 9.0 ml/lb). Mean respiratory rate was 23 ± 9.1 ($n = 10$).

Regarding outcome, 3 dogs survived to discharge. Additionally, 2 other dogs had improved lung function during ventilation, as judged by evaluation of blood gas values and ventilator settings. One of these 2 dogs was in a condition appropriate for removal from the ventilator, but the owner decided to euthanize the dog because she did not want to continue care. The other dog was weaned from the ventilator because of improvement in pulmonary function, judged on the basis of results of blood gas analysis. However, this dog's condition eventually declined because of traumatic pancreatitis, and the dog died of acute cardiopulmonary arrest 72 hours after removal from the ventilator. The 5 dogs that had improved pulmonary function while being ventilated were placed in group A.

The other 5 dogs, which had a decline in pulmonary function during mechanical ventilation, were placed in group B. Four of these dogs were euthanized because of progressive lung dysfunction, and 1 dog had cardiac arrest and died while being ventilated (the same dog had respiratory arrest prior to ventilation and had the lowest PaO₂ of all 10 dogs).

A significant ($P = 0.03$) difference was detected between body weights of dogs in group A (30.9 ± 15.9 kg [68 ± 35 lb]) and body weights of dogs in group B (7.6 ± 1.8 kg [16.7 ± 4 lb]). A significant ($P < 0.001$) difference was also detected between mean slope of the PIP values in group A (-0.01 ± 0.13) and mean slope of the PIP values in group B (0.52 ± 0.16). Mean initial PIP and PEEP values for group A were 30 ± 9.42 and 5.75 ± 3.69 mm Hg, respectively, and for group B were 28.6 ± 3.5 and 4.2 ± 4.9 mm Hg, respectively. Significant differences between groups were not detected for initial PEEP values ($P = 0.76$) or PIP values ($P = 0.69$). Mean preweaning or preeuthanasia PIP and PEEP values for group A were 29.5 ± 13.77 and 1 ± 0.82 mm Hg, respectively, and for group B were 42.8 ± 7.98 and 5.2 ± 1.6 mm Hg, respectively.

A significant difference between the 2 groups was not detected in the slope of PaO₂:FiO₂, minute ventilation per kilogram, Paco₂, TV per kilogram, respiratory rate, or duration of ventilation.

Discussion

Clinical evidence of pulmonary contusion has been reported in as many as 50% of animals with thoracic trauma.⁶⁻⁸ The management of pulmonary contusion is controversial, with ongoing debate over use of fluid administration, antimicrobials, corticosteroids,

and diuretics.^{1,3,4,7,9} Most animals with pulmonary contusions have mild to moderate lung injury, however, and usually respond well to supportive care with rapid resolution of hypoxemia.⁵ Some animals such as those of the study reported here have such severe dyspnea and hypoxemia that invasive support of respiratory function is required.

The clinical decision to initiate PPV in the dogs reported here was made on the basis of the severity of respiratory distress, hypoxemia, and hypercarbia. All of the dogs were judged clinically to be extremely dyspneic. Thoracic radiographic findings, characterized by an alveolar pattern, confirmed the presence of pulmonary contusions and revealed concurrent injuries including pneumothorax, hemothorax, and fractured ribs. Although radiographic findings are helpful in establishing a diagnosis of pulmonary injury,⁵ severity of radiographic changes cannot be used to directly quantify the severity of pulmonary dysfunction. This is especially true in the first 24 to 36 hours after injury, because pulmonary contusions often worsen during this initial period.^{3,7} In addition, although fluid administration during ventilation seemed appropriate for the dogs reported here, many of the dogs had been treated by administration of fluids prior to referral. Therefore, the progression of lung dysfunction in these dogs may have been caused by a combination of pulmonary contusions and fluid accumulation in the alveoli.

Lung function may be evaluated by measurement of gas exchange and estimates of airway and lung mechanics. Information about lung mechanics can be derived indirectly from the ventilator settings.¹⁰ The efficacy of gas exchange in the dogs of the study reported here was assessed by use of ABG analysis and calculation of the PaO₂:FIO₂. The PaO₂:FIO₂ was chosen as the optimal oxygen tension-based measurement, because the dogs were receiving variable FIO₂. Although calculation of the PaO₂:FIO₂ is not identical to calculating the respiratory index, it is probably one of the most accurate and clinically applicable means of comparing intrapulmonary shunt fractions when variable FIO₂ are being used.¹¹⁻¹³

Arterial blood gas values were determined in 7 dogs prior to ventilation while they were receiving oxygen supplementation. Despite an estimated FIO₂ of 0.5 to 1, mean PaO₂ was only 55 mm Hg, with a range of 43 to 94 mm Hg. Mean PaO₂:FIO₂ prior to PPV was 77.5, which confirmed a profound abnormality of gas exchange (reference range, 350 to 500¹³). Most of the dogs were normocarbic or hypocarbic at referral, with mean PaCO₂ of 37.8 ± 9.8 mm Hg. Evidence of hypocarbia attributable to pulmonary contusion has been reported.⁷ As hypoxemia becomes severe enough to trigger respiratory effort, it overrides the control exerted by CO₂, resulting in hyperventilation and hypocarbia.^{7,14} Although hyperventilation is occurring, PPV is the only means to improve oxygenation if simple oxygen supplementation is inadequate. The PaCO₂ was > 50 mm Hg in 2 dogs prior to ventilation, which indicated hypoventilation or hypercarbia secondary to increased physiologic dead space; PPV may be indicated in management of such dogs.

After initiation of PPV, the PaO₂:FIO₂ was greatly

improved in most dogs in the study reported here and increased to a mean value of 201. Positive-pressure ventilation improves oxygenation by decreasing the work of breathing, increasing TV, and recruiting atelectatic alveoli, especially if PEEP is used.^{10,15,16} Although there was an overall improvement in oxygenation, the wide range of values indicated that response to PPV was quite variable in individual patients. For example, the lowest PaO₂ value of 43.9 mm Hg was obtained in a dog that was ventilated because of respiratory arrest that occurred 15 minutes after referral; despite PPV, this dog's PaO₂ was never > 58 mm Hg. Similarly, values obtained in a Yorkshire Terrier were also extreme; PaCO₂ was 105.6 mm Hg, which was probably partially attributable to incorrect initial ventilator settings as well as severe lung disease (PaO₂:FIO₂, 159.5). In this dog, the PaCO₂ eventually decreased to 45 mm Hg with PaO₂:FIO₂ of 61.9 but increased to 80 mm Hg prior to euthanasia (PaO₂:FIO₂, 122.50).

The initial ventilator settings indicated that the mean PIP of 29.2 mm Hg was slightly high. Optimal values for PIP are < 25 mm Hg, and higher pressures reflect the severity of the lung disease, which is attributable to poor lung compliance and increased airway resistance.¹⁰ High peak airway pressures are more likely to result in barotrauma and lead to iatrogenic pneumothorax. Unfortunately, plateau pressures were not available in the study reported here; these would help determine whether increases in PIP were truly caused by poor lung compliance or increased airway resistance attributable to mucus plugs. Peak inspiratory pressures are also increased by the addition of PEEP. Most of the dogs in our study were initially treated with PEEP at approximately 5 cm H₂O in an attempt to increase functional residual capacity and recruit atelectatic alveoli.

Mean minute ventilation for the dogs was high at 0.74 L/kg/min (reference range, 0.21 to 0.26 L/kg [0.1 to 0.12 L/lb)/min¹⁷), and mean recorded TV was increased at 27 ml/kg (reference limit, 16.6 ml/kg [7.5 ml/lb)¹⁷). High minute ventilation reflects the requirements of a hypoxemic or hypercarbic ventilator patient with severe lung disease. However, our values for TV were an overestimation of the actual volumes administered, because they are reported on the basis of the digital ventilator readings rather than from independent spirometer measurements. In our experience, the digital ventilator readout often overestimates the volume actually delivered to the patient, because some of the gas is redistributed throughout the compliant tubing instead of into the lungs. This effect is most important in small dogs and causes erroneously high readings.

Although 4 dogs were successfully weaned from the ventilator, the overall survival rate to discharge was 30%. The most common causes of progressive lung dysfunction in this type of patient include development of secondary pneumonia or progression of injury to acute respiratory distress syndrome.^{18,19} It must be remembered that pulmonary contusions were not the only causes of morbidity or mortality in the dogs reported here. Pneumothorax developed in 9 of the 10 dogs, including 2 dogs with rib fractures and 2 dogs that developed pneumothorax iatrogenically because of PPV.

In the other 5 dogs, pneumothorax was most likely caused by alveolar disruption secondary to sudden lung compression when the glottis was closed.¹ In all dogs, pneumothorax was managed by placement of thoracostomy tubes and a continuous negative-pressure thoracic drainage system. Multiple injuries and complications, including flail chest, pneumonia, anesthesia, drug administration, fluid therapy, hemothorax, hemoperitoneum, myocardial contusion, and ventricular arrhythmia may have been additional contributing factors.

The goal of our statistical analyses was to determine differences between dogs in which lung function improved and those in which lung function deteriorated. Results of these analyses must be interpreted cautiously for several reasons. The sample size was small and may not accurately represent this patient population. Dogs were allocated into 2 groups on the basis of their clinical course and pulmonary function, and the subsequent comparison of these groups introduced some bias in the data analysis. Additionally, we must acknowledge that 4 of the dogs were euthanized because of profound clinical deterioration, and it was impossible to determine whether their condition would have eventually improved. Conversely, 1 owner chose euthanasia despite substantial improvement in the dog's pulmonary function, and it is impossible to predict whether that dog would have been successfully weaned off the ventilator.

It is interesting that dogs in the more successfully treated group were significantly larger than those with deteriorating lung function. Larger dogs may have sustained less injury than small dogs because of their size and may have larger lung surface area and, therefore, more reserve lung function. However, these explanations are inconsistent with the fact that the initial PaO₂:FIO₂ were not significantly different between the 2 groups either prior to ventilation or at initial ventilator settings. It is also possible that the degree of myocardial injury may be higher in small dogs, compared with large dogs, if they sustained similar thoracic impact, but ventricular arrhythmias were only detected in large dogs. The fact that ventilation treatment is more successful in large dogs has been reported¹⁰ and may reflect the fact that it is technically easier to mechanically ventilate a large patient than a small one.

The PIP generally remained stable or decreased in dogs in group A, whereas PIP typically increased gradually in dogs in group B. A significant difference between mean slopes for this variable was detected between the 2 groups. This finding was somewhat intuitive, because progressive lung dysfunction is expected to lead to worsening lung compliance and airway resistance, which result in increasing airway pressures. In addition, dogs with poor oxygenation while being treated with ventilation are more likely to receive PEEP, which also contributes to an overall increase in PIP.

Significant differences between groups were not detected for PEEP or PIP at initial ventilator settings. Despite an increase in PIP over time in group B, the PEEP did not change, which implies that the dogs with decreased lung function had an increase in PIP attributable to worsening lung compliance or increases in airway resistance.

It may be expected that improvement in gas exchange would also develop in dogs in group A, whereas deterioration may be expected in dogs in group B, but differences in PaO₂:FIO₂ were not detected between groups. We surmise that significant differences may have been detected if our study population had been larger.

^aNewport Breeze pediatric ventilator, Newport Medical Instruments Inc, Newport Beach, Calif.

^bStat portable clinical analyzer, Heska Corp, Fort Collins, Colo.

^cStat Profile M, Nova Biomedical Corp, Waltham, Mass.

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