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Case Report

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Management of acute respiratory distress syndrome in a French Bulldog using airway pressure release ventilation

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Abstract

Objective – To describe the successful clinical management of a dog with acute respiratory distress syndrome (ARDS) using airway pressure release ventilation (APRV).
Case Summary – An 18-month-old female French Bulldog was presented for routine ovariohysterectomy and correction of brachycephalic airway obstruction syndrome. Following the surgical procedures, the dog developed aspiration pneumonia and ARDS. Her clinical condition failed to improve with conventional pressure-support mechanical ventilation and she was subsequently managed with APRV. She recovered fully and exhibited no clinical or radiographic abnormalities during follow-up examinations.
New or Unique Information Provided – This is the first reported use of APRV to manage refractory hypoxemia associated with ARDS in a dog. This alternative mode of mechanical ventilation can be considered a feasible alternative in canine patients with ARDS.

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Keywords: APRV, ARDS, brachycephalic, dogs, mechanical ventilation

Introduction

Patients that develop significant respiratory compromise, such as acute lung injury (ALI) or acute respiratory distress syndrome (ARDS) often require mechanical ventilation. Mortality rates in human patients with ARDS remain high and survival to discharge is less than 10% in veterinary medicine.[1,2] In human medicine, research has led to the development of a number of strategies to improve the management and outcome of patients with ARDS. These strategies include the use of low tidal-volume ventilation,[3] restrictive fluid strategies,[4] prone positioning,[5] as well as alternative therapies, such as extracorporeal ventilation and the use of "open lung" modes of ventilation. The open lung approach minimizes atelectasis by using a higher than conventional positive end expiratory pressure (PEEP) to optimize gas exchange. Modes of ventilation that employ the open lung approach include airway pressure release ventilation (APRV) and high-frequency oscillatory ventilation.

APRV uses the concept of open lung ventilation combined with spontaneous ventilation in order to improve arterial oxygenation and minimize ventilator-induced lung injury. APRV varies markedly from conventional mechanical ventilation. During APRV, a high continuous positive airway pressure (CPAP) is maintained and the patient is allowed to breathe spontaneously. Each

Abbreviations

ALI	acute lung injury
ARDS	acute respiratory distress syndrome
APRV	airway pressure release ventilation
BPAP	bilevel positive airway pressure
CPAP	continuous positive airway pressure
FIO_2	fraction of inspired oxygen
I:E	inspiratory:expiratory ratio
MAP	mean arterial pressure
PF	PaO_2/FIO_2 ratio
PEEP	positive end expiratory pressure
SpO_2	oxygen saturation

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period of high airway pressure is interrupted by a very short period of low airway pressure. During this low-pressure phase that facilitates expiration, PEEP is maintained to conserve alveolar recruitment. It is the ratio of time spent at the high airway pressure to time spent at the low airway pressure that determines the rate of mandatory mechanical ventilation, with further spontaneous breaths allowed throughout the ventilator cycle to enhance physiologic recruitment of alveoli. APRV improves many physiologic endpoints of mechanical ventilation such as gas exchange, cardiac output, arterial oxygenation, and systemic blood flow in canine experimental models, as well as human clinical and experimental studies.[a,b,6–8] In this report, we describe the use of APRV to successfully manage ARDS in an 18-month-old French Bulldog with aspiration pneumonia.

Case Summary

An 18-month-old female French Bulldog presented to the surgical service for an ovariohysterectomy and upper airway examination. She had no prior medical concerns and abnormalities on physical examination at the time of presentation were limited to stertorous breathing and stenotic nares. Thoracic radiographs were performed prior to anesthesia and did not reveal any pulmonary abnormalities.

The following day, an upper airway exam was performed under general anesthesia and confirmed the presence of an elongated soft palate, everted laryngeal saccules, stenotic nares, and normal laryngeal function. A routine palatoplasty, ventriculectomy, and wedge rhinoplasty were performed, followed by an ovariohysterectomy. The anesthetic protocol consisted of hydromorphone[c] premedication (0.05 mg/kg IV), followed by propofol[d] (10 mg/kg IV total, titrated to effect) for the upper airway examination and ventriculectomy, and inhalant anesthesia with isoflurane[e] inhalant (1.5–2.0%/v) for the palatoplasty and ovariohysterectomy. She received an additional dose of hydromorphone (0.025 mg/kg IV) for analgesia upon recovery from anesthesia.

The dog initially recovered well following the procedures. She recovered in an oxygen cage with an inspired oxygen concentration of 40% and had an oxygen saturation (SpO_2) of 98% with minimal evidence of upper airway obstruction. Ten hours postoperatively, the dog developed progressively increased respiratory effort and hypoxemia (SpO_2 = 88%) while receiving oxygen supplementation via the oxygen cage and flow-by oxygen provided at 6 L/min. She was sedated with diazepam[f] (0.3 mg/kg IV) and ketamine[g] (5.5 mg/kg IV), endotracheally intubated, and administered flow-by oxygen supplementation, which resulted in improvement of her SpO_2 to 96%. Sedation was administered approximately every 60 minutes, as intermittent boluses of diazepam and ketamine as described above, to effect. Empiric antimicrobial therapy with ampicillin[h] (22 mg/kg IV q 6 h) and enrofloxacin[i] (10 mg/kg IV q 24 h) was initiated. Thoracic radiographs were repeated and showed evidence of a cranioventral pulmonary alveolar pattern consistent with aspiration pneumonia.

The dog's condition deteriorated 2 hours after intubation when she developed a progressive decrease in her SpO_2 (83%) despite continued oxygen supplementation (flow-by oxygen, 6 L/min). Mechanical ventilation was initiated on day 3 (Table 1) using a volume-controlled anesthesia ventilator[j] and inhalant anesthetic machine[k] with a heat-moisture exchange device[l] because the critical care ventilators were in use on other patients. Oxygen was delivered using a flow meter (1.5 L/min) and medical air was initially delivered at 300 mL/min and increased to 1.5 L/min 6 hours later. Because the fraction of inspired oxygen (FIO_2) was unknown, the P/F ratio could not be calculated. Sedation was maintained with fentanyl[m] (6 μg/kg/h) and ketamine[g] (0.5–1 mg/kg/h). Further sedation was achieved with dexmedetomidine[n] (0.5–1 μg/kg/h) and midazolam[o] (0.5–1 mg/kg/h). A nasoesophageal tube, urinary catheter, and dorsal pedal arterial catheter were placed. The dog was ventilated for 12 hours, until one of the critical care ventilators[p] became available. Initial ventilator settings are available in Table 1. Six hours following initiation of mechanical ventilation on day 3, the dog developed persistent hypotension (mean arterial pressure (MAP) of 45–68 mm Hg) and tachycardia (175–220/min) that was unresponsive to crystalloid[q] (55 mL/kg IV) and colloid[r] (20 mL/kg IV) therapy and she ultimately required treatment with dobutamine[s] (3–5 μg/kg/min) and norepinephrine[t] (0.2–0.8 μg/kg/min). Administration of norepinephrine improved MAP to approximately 70 mmHg and the addition of dobutamine further improved MAP to approximately 80 mmHg. Septic shock was suspected with pneumonia as the primary site of infection. Cytologic examination of airway secretions obtained by endotracheal lavage revealed suppurative inflammation and the presence of intracellular coccoid bacteria; therefore, clindamycin[u] (10 mg/kg IV q 12 h) was started. A sample submitted for aerobic culture subsequently grew *Escherichia coli* susceptible to ampicillin that the dog was already receiving.

After 12 hours of mechanical ventilation (day 4), the dog was transferred to a critical care ventilator and placed on pressure control ventilation plus assist (PCV+A). A complete blood count and serum biochemical profile were performed and revealed a normal

Table 1: Daily ventilation parameters during mechanical ventilation

	Day 3*	Day 4	Day 5	Day 6	Day 7	Day 8§	Day 9	Day 10
Ventilator mode	Volume controlled	PCV+A	PCV+A	PCV+A	PCV+A	APRV	APRV	APRV
I:E ratio	1:2	1:1.8	1:1.9	1:2–1:2.9	1:3, 1:1.4, 1.3:1, 2.3:1	4.6:1	5.5:1	5.5:1
Peak pressure/*P* High	22–28	26	25–27	23–26	20–21	24–27	23–25	24
P Mean	N/A	13–14	14–15	12–14	14–16	20–22	20	20
PEEP/*P* low	5	8–10	9–10	10	9–10	1–2	1	1

	Day 11	Day 12	Day 13‖
Ventilator mode	APRV	APRV	APRV
I:E ratio	8:1	8:1	8:1
Peak *P*/*P* High	24	21–22	20–24
P Mean	20	17–18	15–21
PEEP/*P* low	1	1	1

Note: Where two numbers are listed, they represent the range of values obtained during the day.
PCV+A, pressure control ventilation + assist; APRV, airway pressure release ventilation; Peak *P*, peak airway pressure during PCV + A ventilation; *P* High, the pressure setting during continuous positive airway pressure phase of APRV ventilation.
*Initiation of mechanical ventilation—ventilation was performed using a volume-controlled anesthesia ventilator for 24 hours prior to transitioning to critical care ventilator.
§Ventilation changed from PCV+A to APRV.
‖Day of weaning from continuous mechanical ventilation.

Table 2: Daily oxygenation parameters during mechanical ventilation

	Day 3*	Day 4	Day 5	Day 6	Day 7	Day 8§	Day 9	Day 10
Ventilator mode	Volume controlled	PCV+A	PCV+A	PCV+A	PCV+A	APRV	APRV	APRV
FiO_2	N/A	0.50–0.60	0.40–0.50	0.45–0.50	0.45–0.55	0.45–0.55	0.45–0.60	0.35–0.45
PaO_2 (mm Hg)	84.4–117	79.7–100	73.1–85.1	70.6–86.3	69.4–111	85.4–155	74.5–140	104–371
$PaCO_2$ (mmHg)	34.1–48.9	20.2–21.6	31.0–39.4	45.2–48.4	47.9–51.2	31.4–52.8	45.0–65.1	35.4–40.9

	Day 11	Day 12	Day 13‖
Ventilator mode	APRV	APRV	APRV
FiO_2	0.35–0.40	0.35	0.35
PaO_2 (mm Hg)	110–183	120–144	140–528
$PaCO_2$ (mmHg)	37.9–49.8	50.4–56.8	41.4–57.7

Note: Where two numbers are listed, they represent the range of values obtained during the day.
PCV+A, pressure control ventilation + assist; APRV, airway pressure release ventilation.
*Initiation of mechanical ventilation—ventilation was performed using a volume-controlled anesthesia ventilator for 24 hours prior to transitioning to critical care ventilator.
§Ventilation changed from PCV+A to APRV.
‖Day of weaning from continuous mechanical ventilation.

neutrophil count with a mild left shift (band neutrophil count = 0.34×10^9/L [0.34×10^3 cells/μL]; reference interval: 0.0–0.3×10^9/L [0.0–0.3×10^3 cells/μL]) and an increased creatinine kinase (11,384 U/L; reference interval: 40–255 U/L) concentration. Fresh frozen plasma transfusions (30 mL/kg total volume) were administered because of prolongation of the activated clotting time (220 s; reference interval: 90–120 s) and hypoalbuminemia (14 g/L [1.4 mg/dL]; reference interval 29–43 g/L [2.9–4.3 mg/dL]). Due to repeated occlusion of the endotracheal tube with airway secretions, the endotracheal tube was replaced four times during the first 36 hours of intubation. Therefore, a revision of the palatoplasty was also performed due to loosening of the sutures, likely secondary to repeated endotracheal intubations.

The dog's vasopressor dependence, ventilatory status, and PaO_2/FIO_2 (PF) ratio remained unchanged during days 4 and 5 (Table 2, Figure 1). A CBC and biochemical profile performed on day 6 revealed a mature neutrophilia (neutrophil count = 12.63×10^9/L [12.63×10^3 cells/μL]; reference interval: 2.9–10.6^9/L [2.9×10^3–10.6^3 cells/μL]) with a left shift (band neutrophil count = 0.31×10^9/L [0.31×10^3 cells/μL]; reference interval: 0.0–0.3×10^9/L [0.0–0.3×10^3 cells/μL]) and evidence of toxic change, as well as mild hyperbilirubinemia (19 μmol/L [1.1 mg/dL];

reference interval: 0–4 μmol/L [0–0.23 mg/dL]) and elevated alkaline phosphatase (537 U/L; reference interval: 22–143 U/L). The dog was successfully weaned from vasopressor support on day 6.

Thoracic radiographs on day 7 showed a bilateral pulmonary interstitial and alveolar pattern. Although her septic shock resolved and she was no longer vasopressor dependent, there was no improvement in her ventilatory parameters and her gas exchange remained compromised as indicated by a persistently decreased PF ratio (Figure 1). Therefore, transition to ventilation with APRV was initiated.

Transition to APRV was accomplished by gradually increasing the inspiratory time and decreasing the expiratory time to increase the inspiratory:expiratory (I:E) ratio over 24 hours. During pressure control ventilation, the initial I:E ratio was 1:3 and was gradually increased every 2–4 hours (1:1.4, 1.3:1, 2.3:1) to create an inverse I:E ratio. An inverse I:E ratio of 2.3:1 was well tolerated by the dog and maintained for 12 hours. On the morning of day 8, the dog was switched to APRV (initial I:E ratio 5:1, increased to 8:1 after 72 hours). Twelve hours after the onset of APRV, the dog's PaO_2 increased to 155 mmHg with a PF ratio of 344 and a $PaCO_2$ of 31.4 mmHg. Thereafter, the dog exhibited mild daily improvements in her oxygenation (Table 2, Figure 1).

On day 13, the dog was extubated and maintained on flow-by oxygen supplementation. One hour later, she demonstrated signs of a marked upper airway obstruction, was reintubated, and a temporary tracheostomy was performed. The dog was mechanically ventilated with APRV for an additional 48 hours, during which time her sedative medications were gradually weaned and mechanical ventilation was intermittently discontinued for 1–4 hours at a time and was re-initiated once the dog began to show evidence of respiratory fatigue. In this case, respiratory fatigue was diagnosed based on clinical visualization of her thoracic wall movements. This patient's respiratory effort became increasingly shallow and her respiratory rate markedly decreased when she became fatigued. When this patient became fatigued, mechanical ventilation was re-instituted using APRV because the patient would not breathe spontaneously when mechanical ventilation was re-initiated. When the patient resumed taking spontaneous breaths, mechanical ventilation was discontinued for another period of time. On day 15, mechanical ventilation was discontinued for 4–12 hours at a time, until the patient showed evidence of respiratory fatigue based on clinical visualization and ventilation with CPAP was implemented during periods of rest.

Samples from the airway were obtained by endotracheal lavage and swabs of the tracheostomy site were submitted for aerobic culture on day 16 and ultimately revealed growth of a resistant *Pseudomonas aeruginosa*. Antimicrobial therapy was subsequently switched to ceftazidime[v] (30 mg/kg IV q 8 h). Repeat thoracic radiographs were performed on day 17 and showed a mildly improved bilateral interstitial and alveolar pattern, consistent with improvement of her pneumonia and ARDS. The dog was discontinued from mechanical ventilation and her tracheostomy tube was removed. She remained in the oxygen cage ($FIO_2 = 35\%$) for the following 36 hours after which oxygen supplementation was discontinued.

She was discharged on day 22, following normal lab work and thoracic radiographs that showed a primarily right sided, diffuse interstitial pattern, consistent with mild improvement in her pneumonia. She was given ceftazidime (25 mg/kg SQ q 8 h) to continue until follow-up. The dog returned for reevaluation 4 and 12 days following discharge. There was continued radiographic improvement of her pneumonia and she was reportedly normal at home. Her antimicrobial therapy was discontinued at the time of her final recheck examination.

Discussion

Mechanical ventilation is indicated in cases of severe hypoxemia, hypercapnea, and respiratory fatigue or failure. Survival rates in people ventilated for the development of ARDS are highly variable, but have increased over the last decade, with an overall survival rate of approximately 57%.[9] The mortality rate for veterinary patients undergoing mechanical ventilation varies quite significantly depending on the underlying disease. Overall, survival rates are much lower than in human medicine and range from 28 to 39%.[1,2,10] Specifically, cases ventilated for hypoxemia have a reported survival to discharge of 11–22% compared to those ventilated for hypercapnea whose survival is reported as 39–50%.[1,2] Though there is limited information in veterinary medicine regarding the specific prognosis for patients with ARDS, reports are infrequent[11] and available information suggests a survival rate of approximately 5%.[2] A recent study of mechanical ventilation of brachycephalic dogs revealed that aspiration pneumonia was the most common underlying disease and that survival to discharge was 27%. In that study, none of the dogs ventilated for hypoxemia survived to discharge.[12]

The dog in the present report was diagnosed with ARDS based on her acute onset of tachypnea and hypoxemia, history of recent surgery and suspicion for aspiration pneumonia, bilateral radiographic pulmonary infiltrates, PF ratio of <200, and a neutrophilic fluid retrieved by endotracheal lavage. These findings are consistent with the recently published veterinary

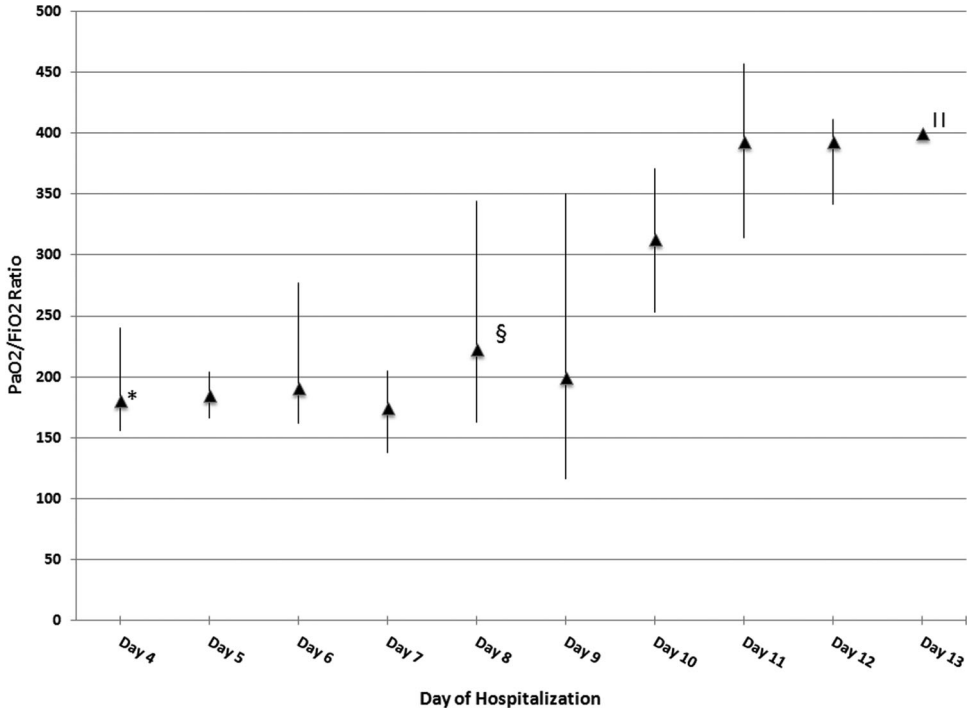

Figure 1: Daily trends in PaO_2/FiO_2 ratio during mechanical ventilation. The vertical bars are representative of the range of values obtained in the 24-hour period. The gray triangles represent the median daily value. PCV+A, pressure control ventilation + assist; APRV, airway pressure release ventilation.

*Initiation of mechanical ventilation with PCV + A.

§Ventilation changed from PCV+A to APRV.

‖Day of weaning from mechanical ventilation.

consensus definitions for the diagnosis of ALI or ARDS.[13] A limitation in this case was that an echocardiogram was not performed to definitively rule out cardiogenic pulmonary edema as a cause for her hypoxemia. In this case, a presumptive diagnosis of noncardiogenic edema was made based on the radiographic pattern of distribution (bilaterally cranioventral with the right middle lung lobe being most severely affected), and based on the lack of clinical and radiographic evidence of cardiac disease (no auscultable cardiac arrhythmias or murmurs, no evidence of jugular venous distension, normal size of the cardiac silhouette on radiographs with normal-sized pulmonary vasculature).

The decision to switch from conventional pressure-control ventilation to APRV was made because of the dog's refractory hypoxemia (persistently low PF ratio) on day 7 of hospitalization. APRV, also known as biphasic ventilation or bi-level ventilation, is not a new mode of ventilation and was first described in 1987.[14] Although APRV is the most common name for this mode of ventilation, manufacturers have developed a number of trademarked, brand names for this type of ventilation including BiVent, BiLevel, and DuoPAP. It is important to note that bilevel positive airway pressure (BPAP) is a noninvasive type of CPAP and that BPAP is not an appropriate synonym for APRV. Additionally, BiPAP is a registered trademark, referring specifically to the use of BPAP in a specific mechanical ventilator and these are not synonyms for APRV.[w]

The premise of APRV, a form of open-lung ventilation, is to keep the alveoli open at the end of expiration in order to reduce shearing injury to the alveoli during prolonged mechanical ventilation. Computerized tomography scans in people during APRV ventilation demonstrate significantly decreased proportions of atelectatic lung and increased proportions of normally aerated lung in comparison to patients receiving pressure support ventilation.[15] APRV differs dramatically from conventional pressure-support modes of ventilation (Figure 2). During APRV, there is a continuous level of high positive airway pressure (CPAP phase). The maintenance of continuous airway pressure enables increased alveolar recruitment. The duration of this CPAP phase (inspiratory time) is termed T high and the airway pressure (CPAP) during this phase is termed P high. With heterogenous lung disease, alveoli may have variable time constants and individual alveoli may vary greatly in the pressure and time required for opening of

the alveoli and exhalation of inspired air. By increasing the time for alveolar opening, alveoli that are slower to open can be recruited in a low-pressure environment. Likewise, the peak increase in arterial oxygen concentration is seen 8–16 hours after the induction of APRV, indicating that the rerecruitment of alveoli is likely slow and steady, rather than instantaneous.[16]

It is the termination of the CPAP phase that allows for clearance of carbon dioxide during a period of elastic recoil. The period of low pressure (*P* low) is called the release phase. The *P* low phase is the period of PEEP and is also the period of expiration. The *P* low phase is set for a very short period of time (*T* low) in order to prevent alveolar de-recruitment. During *P* low, the alveoli remain open due to the presence of a constant level of intrinsic positive end expiratory pressure, either set by the ventilator, or intrinsic to the patient. APRV is a is a time-cycled, pressure-limited time-triggered form of ventilation that is cycled based on the length of time set for *T* high and *T* low. During APRV, the time of *P* high is much greater than that of *P* low. This leads to a pronounced inverse I:E ratio. Conventional pressure-controlled ventilation modes typically aim for an I:E ratio of 1:2 to 1:3, whereas, the I:E ratio during APRV is typically 8:1 to 10:1. Therefore, in order to transition a patient from conventional modes of ventilation to APRV, a period of adjustment involving a gradual transition to an inverse I:E ratio is required in order to maximize patient tolerance to APRV. In this case, a period of 12 hours was chosen in order to slowly transition the dog to an inverse ratio. She was then ventilated for 12 hours at this inverse ratio before she was switched into APRV.

To transition from conventional mechanical ventilation modes to APRV, initial ventilator settings for APRV are adapted from the patient's current conventional settings.[17] The patient's plateau pressure (estimate of average alveolar pressure) is selected as the initial *P* high (typically less than 35 cmH_2O based on ARDSnet criteria).[3] The *P* low is set at 0 cmH_2O to create a maximum difference between *P* high and *P* low to allow for a rapid peak expiratory phase and maximize expiratory flow so that adequate emptying of the lungs occurs with each pressure decrease and CO_2 removal is maximal. T high should be set for a minimum of 4.0 seconds to allow sufficient time for alveolar recruitment and gas diffusion. Usually, *T* low is set between 0.5 and 1.0 seconds to prevent collapse of recruited alveoli. An excessively long T low can lead to alveolar derecruitment that will manifest as a decreased PaO_2. As the patient begins to recruit alveoli, the *T* high time is increased by 0.5–1.0 seconds.[17] The longer the *T* high time that is provided, the less opportunity there is for derecruitment of alveoli with each exhalation. The patient will maintain adequate minute ventilation with spontaneous breaths taken during the periods of *T* high.

During APRV, the patient maintains the ability to breathe spontaneously during all phases of the cycle. Thus, APRV is considered a fully spontaneous ventilation mode. As such, neuromuscular blockade is not used during this mode, which has been shown to decrease length of ICU stay and medication costs.[7] Another benefit of spontaneous breathing during APRV is that in comparison to pressure-support ventilation, APRV leads to decreased intrapulmonary shunting, decreased dead space, increased PaO_2, increased oxygen delivery, and increased CO_2 elimination due to increased alveolar ventilation.[b] Although specific ventilation and perfusion studies were not performed in this case, the arterial blood gas results are consistent with improvement in oxygenation and increased CO_2 elimination (first PaO_2/FIO_2 ratio >300 since the onset of mechanical ventilation and first $PaCO_2$ value less than 45 mmHg in preceding 48 hours) within 12–24 hours of transitioning to APRV, consistent with the time to maximal benefit reported in the human medical literature.

APRV also leads to similar or improved measures of hemodynamic performance, such as stroke volume, cardiac index, and renal blood flow.[b,6,14] Although cardiac output was not measured in this case, there was no evidence of a negative effect on systemic blood pressure after the transition to APRV. The dog was previously receiving both norepinephrine and dobutamine for hemodynamic support of suspected septic shock and these drugs were weaned 24 hours prior to the initiation of APRV and her MAP remained constant while receiving APRV.

There are different techniques that can be used to increase alveolar recruitment during mechanical ventilation. Commonly employed recruitment maneuvers include sustained inflation (inspiration with sustained inflation pressures of approximately 35–50 cmH_2O for 20–40 seconds), intermittent "sighs" (multiple consecutive inspirations at elevated airway pressures of 45 cmH_2O) and intermittent, incremental increases in PEEP or peak inspiratory pressure (to greater than 60 cmH_2O) for brief periods of time.[18] These methods can cause transient alveolar recruitment, but also have unfavorable transient side effects such as hypoxemia due to decreased minute ventilation (during the maneuver) or hypotension due to decreased venous return secondary to markedly elevated peak airway pressures. Systematic review has shown that although there is short-term improvement in oxygenation with the use of recruitment maneuvers, there is little evidence that there is a positive effect on clinically important outcomes, with mortality similar to patients in which recruitment manoeuvres are not employed.[19] Additionally, if there is insufficient

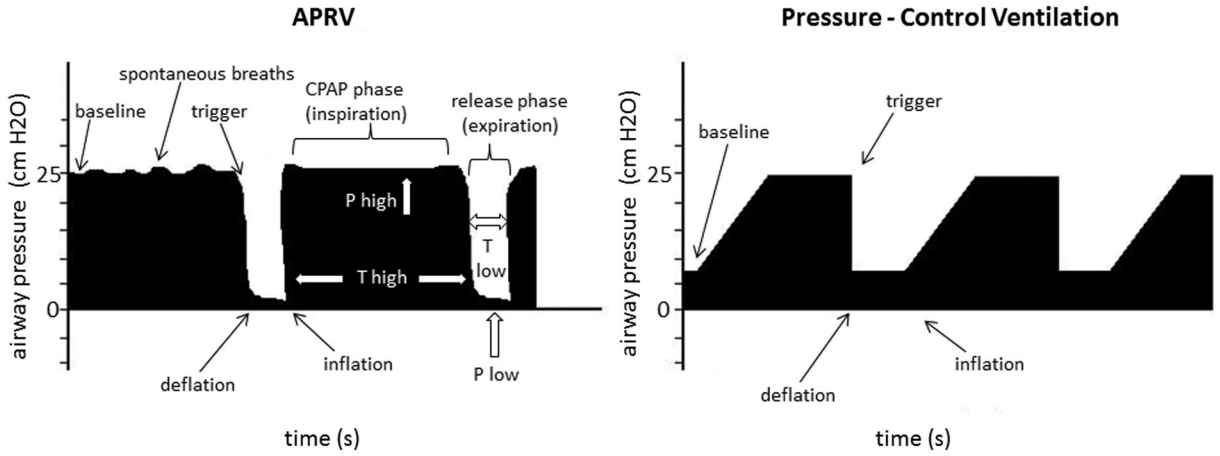

Figure 2: Example ventilator waveforms (pressure/time) in airway pressure release ventilation in comparison to conventional pressure control ventilation. The phases of airway pressure release ventilation are also illustrated.

PEEP following the maneuver, the recruited alveoli may become derecruited. Therefore, another benefit of APRV is the elevated baseline airway pressure that may produce gradual, near-complete alveolar recruitment while avoiding excessively high airway pressures. Avoiding intermittent recruitment using other high-pressure maneuvers can minimize injury due to derecruitment or overdistension of more easily recruited alveoli.[17]

Although the potential hemodynamic and pulmonary benefits of APRV make it an attractive mode of ventilation for hypoxemic patients, this mode of ventilation is not appropriate for all patients. This mode of ventilation requires spontaneous breathing, thus requiring lower levels sedation than conventional mechanical ventilation. Therefore, APRV is contraindicated in patients that require deep sedation or pharmacologic coma for management of their underlying disease, or for patients with neuromuscular disease who are unable to generate sufficient respiratory effort. APRV is also theoretically contraindicated in patients suffering from obstructive lung disease, who require a long expiratory time for sufficient expiration.

In this case report, the dog developed ARDS with refractory hypoxemia. APRV was selected in order to maximize alveolar recruitment while minimizing the potentially deleterious cardiovascular and pulmonary effects of other more commonly used recruitment maneuvers. The dog remained hemodynamically stable and showed clinical (increased P/F) and radiographic evidence of improved alveolar recruitment following initiation of APRV. Unfortunately, it is impossible to identify whether the use of APRV was the cause of this dog's improvement or whether she would have improved if conventional pressure-controlled ventilation was continued for longer. Because her peak increase in arterial oxygenation was noted 12 hours after initiation of APRV, and based on information regarding the use of APRV in human patients, it is logical to assume that APRV was helpful in this patient. However, the effect of time on her improvement is unknown and, as such, the role of APRV in her clinical improvement cannot be fully elucidated.

There is limited information regarding the positive effects of APRV on outcome in the human medical literature and most studies examining APRV are evaluating heterogenous patient populations. There are data to show that APRV decreases the length of hospital stay and ventilation requirements.[8] However, another study found no difference in these outcome-specific endpoints.[20]

Conclusions

This report describes a dog with ARDS successfully managed using APRV. Although APRV has not been shown to improve survival in human patients with ARDS, the positive effects on hemodynamic parameters, oxygen exchange, and dead space ventilation have been more clearly demonstrated. This report illustrates that APRV can be used in canine patients and may represent a feasible alternative mechanical ventilation mode for the management of canine patients with refractory hypoxemia and ARDS.

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Footnotes

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[e] Isoflurane, Abbott Laboratories, St-Laurent, PQ.
[f] Diazepam, Sandoz Canada.
[g] Ketamine Hydrochloride, Binoche Animal Health Care, Belleville, ON.
[h] Ampicillin Sodium, Novopharm, Toronto, ON.
[i] Enrofloxacin, Bayer Inc, Toronto, ON.
[j] Hallowell EMC Model 2002, Hallowell EMC, Pittsfield, MA.
[k] Moduflex Elite, Dispomed, Joliette, PQ.
[l] Hydro-Therm HME, Intersurgical Incorporated, Liverpool, NY.
[m] Fentanyl, Sandoz Canada.
[n] Dexmedetomidine, Pfizer Animal Health, Kirkland, PQ.
[o] Midazolam, Sandoz Canada.
[p] Evita XL, Draeger Medical Canada, Richmond Hill, ON.
[q] Plasma-lyte A, Baxter Corporation, Mississauga, ON.
[r] Voluven, Fresenius Kabi, Germany.
[s] Dobutamine, Hospira, Montreal, PQ.
[t] Norepinephrine, Sandoz Canada.
[u] Clindamycin, Sandoz Canada.
[v] Ceftazidime, Pharmaceutical Partners of Canada.
[w] BiPAP, Respironics, Murrysville, PA.

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