

The Impact of Endotracheal Suctioning on Gas Exchange and Hemodynamics During Lung-Protective Ventilation in Acute Respiratory Distress Syndrome

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OBJECTIVE: To evaluate the respiratory and hemodynamic effects of open suctioning (OS) versus closed suctioning (CS) during pressure-control (PC) and volume-control (VC) ventilation, using a lung-protective ventilation strategy in an animal model of acute respiratory distress syndrome (ARDS). **SETTING:** Animal laboratory in a university hospital. **DESIGN:** Randomized cross-over evaluation. **ANIMALS:** Eight female Dorset sheep. **INTERVENTIONS:** Lung lavage was used to simulate ARDS. We applied VC and PC mechanical ventilation with a tidal volume of 6 mL/kg and positive end-expiratory pressure (PEEP), adjusted based on a table of PEEP versus fraction of inspired oxygen (F_{IO_2}). Suctioning was performed for 10 s with a suction pressure of -100 mm Hg, during both OS and CS. OS and CS were randomly performed with each animal. Hemodynamics and arterial blood gases were recorded before, during, and after endotracheal suctioning. **RESULTS:** The P_{aO_2}/F_{IO_2} ratios before suctioning were similar in all groups, as were the PEEP and F_{IO_2} . P_{aO_2}/F_{IO_2} was lower after OS than after CS/VC or CS/PC. There was no post-suctioning difference in oxygenation between CS/VC and CS/PC. P_{aCO_2} recorded 10 min after suctioning was greater than the presuctioning value, in all groups. Intrapulmonary shunt fraction increased between baseline and 10 min post-suctioning with OS and CS/VC, but did not significantly increase with CS/PC. There were no significant changes in hemodynamics pre-suctioning versus post-suctioning with OS, CS/VC, or CS/PC. **CONCLUSION:** P_{aO_2}/F_{IO_2} was better maintained during CS with both VC and PC modes during lung-protective ventilation for ARDS, as compared with OS, and shunt fraction post-suctioning changed least with PC. *Key words:* airway suctioning, closed suctioning, open suctioning, lung-protective ventilation, intensive care, ventilator. [Respir Care 2006;51(5):497–502. © 2006 Daedalus Enterprises]

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This study was funded by an unrestricted grant from Ballard Medical Products, Draper, Utah.

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Introduction

Mechanical ventilation is an essential procedure in the treatment of many severely ill patients. However, mechanical ventilation itself can cause lung injury.^{1–4} The primary variables responsible for ventilator-induced lung injury are alveolar overdistention (due to high transpulmonary pressure) and the repetitive derecruitment and reopening of unstable alveolar units (due to insufficient positive end-expiratory pressure [PEEP]).⁵ Endotracheal suctioning can cause derecruitment, which promotes collapse and the need to reopen lung units, thus potentially worsening lung injury in patients with acute respiratory distress syndrome (ARDS)/acute lung injury (ALI).

Previous animal and clinical studies have demonstrated that disconnecting the patient from the ventilator during suctioning can cause alveolar derecruitment and transient hypoxemia.^{6–8} Closed suctioning (CS) systems allow uninterrupted ventilatory support during suctioning, supplying oxygen-enriched gas and ventilation with PEEP. Thus, CS should limit or even prevent hypoventilation, alveolar derecruitment, and hypoxemia.^{7,8} Suctioning-induced lung derecruitment in ARDS/ALI can also be reversed by performing recruitment maneuvers after suctioning,^{9,10} and can be minimized by avoiding ventilator disconnection.⁸

However, performing CS can generate sub-atmospheric pressure in the patient's airway if the flow generated from the suctioning device is greater than the ventilator flow.¹¹ In this situation the advantages of CS may be negated, resulting in hypoxemia secondary to alveolar derecruitment. On the other hand, airflow from the alveoli may be minimized by airway collapse (ie, flow limitation), leaving in question the magnitude of the suctioning effect on alveolar volume. In theory, flow limitation would be more likely to occur at the low lung volumes maintained by limitation of tidal volume (V_T) and plateau pressure, which emphasizes the importance of studying suctioning with current lung-protective strategies.¹²

Three recent reports suggested that CS during ALI/ARDS^{7,8,13} better maintains gas exchange and lung volume than does open suctioning (OS), but the ventilator management of the reported patients was not provided according to the low- V_T method of the National Institutes of Health ARDS Network study.¹⁴

The aim of the present study was to evaluate the hemodynamic and gas-exchange effects of OS and CS during volume-control (VC) and pressure-control (PC) ventilation, while using a V_T of 6 mL/kg, with PEEP and F_{IO_2} adjusted according to the ARDS Network algorithm,¹⁴ in sheep with ARDS induced by lung lavage. We hypothesized that gas exchange during ARDS-Network ventilation would be better maintained during CS than during OS, with either VC or PC.

Methods

This study was approved by the Animal Care Committee of the Massachusetts General Hospital, Boston, Massachusetts.

Instrumentation

We studied 8 female Dorset sheep (25–35 kg), which were fasted for 24 hours immediately prior to the study. Orotracheal intubation (9-mm inner-diameter endotracheal tube [ETT]) was performed during deep halothane anesthesia delivered via mask. The external jugular was then cannulated and an 8 French sheath introducer was inserted. After line placement, the anesthetic was changed to total intravenous anesthesia, with a loading dose of 10 mg/kg pentobarbital, 4 mg/kg ketamine, and 0.1 mg/kg pancuronium, followed by continuous infusion of pentobarbital (4 mg/kg/h), ketamine (8 mg/kg/h), and pancuronium (0.1 mg/kg/h), providing surgical anesthesia with paralysis.

An 18-gauge catheter was then placed into the femoral artery for continuous measurement of arterial blood pressure and arterial blood gases. Arterial and mixed venous blood samples were drawn for blood gas analyses. P_{aO_2} , P_{aCO_2} , pH, oxyhemoglobin saturation, and hemoglobin content were assessed with a blood gas analyzer (model 282, Ciba Corning Diagnostics, Norwood, Massachusetts). Flow (\dot{V}) at the ETT was measured with a heated pneumotachometer (Hans Rudolph, Kansas City, Missouri) connected to a differential pressure transducer (MP-45 [± 2 cm H₂O], Validyne, Northridge, California). Volume was determined by digital integration of the flow signal. A differential pressure transducer (MP-45 [± 100 cm H₂O], Validyne, Northridge, California) was used to measure airway-opening pressure (P_{aO_2}). Cardiac output and pulmonary arterial pressure were monitored with a 7.5 French pulmonary-artery catheter (831 HF 7.5, Edwards Life Sciences, Irvine, California) inserted into the left external jugular vein and connected to a computer (9520A, Edwards Laboratory, Irvine, California). Proper position of the catheter was confirmed via pressure-waveform analysis before and after balloon occlusion. Cardiac output was determined via thermal dilution technique.

The \dot{V} , P_{aO_2} , blood pressure, and pulmonary arterial pressure signals were amplified (model 8805C, Hewlett Packard, Waltham, Massachusetts) and converted to digital signals, using an analog-to-digital converter (DI-220, Dataq Instruments, Akron, Ohio), and recorded at a sampling rate of 100 Hz, using data-acquisition software (WinDaq/200, version 1.36, Dataq Instruments, Akron, Ohio). All infusions, including the anesthetic, were given via volumetric infusion pump. A heating blanket was used to maintain a core temperature of 38–39°C. An orogastric tube was placed to empty the stomach.

Protocol

After intubation, basic ventilatory settings were VC with V_T of 10 mL/kg, decelerating-flow waveform, ratio of inspiratory time to expiratory time 1:2, fraction of inspired oxygen (F_{IO_2}) 1.0, and PEEP 5 cm H_2O . The respiratory rate was adjusted to achieve eucapnia (P_{aCO_2} 35–45 mm Hg). Ventilation throughout the protocol was provided by a model 840 ventilator (Puritan Bennett, Carlsbad, California).

After stabilization on basic ventilator settings, severe lung injury was produced with bilateral lung lavage, with instillation of 1 L of isotonic saline (warmed to 39°C), repeated every 30 min until P_{aO_2} decreased to ≤ 100 mm Hg at F_{IO_2} of 1.0 and PEEP of 5 cm H_2O . A stable lung injury was defined as a P_{aO_2} change of $< 10\%$ after 60 min. Normally, 2–4 lavages were required in each animal, necessitating 2–3 hours to establish a stable lung injury.

Following lung injury, lung-protective mechanical ventilation was provided, with a V_T of 6 mL/kg, decelerating flow pattern (VC), respiratory rate 35 breaths/min, and PEEP and F_{IO_2} set per the ARDS Network F_{IO_2} /PEEP table¹³ with VC or PC. Inspiratory time was maintained at 0.4 s. During PC the peak pressure was adjusted to maintain a V_T of 6 mL/kg. The application of PC and VC were randomized, as were the suctioning method, described below.

Continuous CS was performed during VC (CS/VC) and PC (CS/PC), using a 14 French CS catheter (TrachCare, Ballard Medical Products, Draper, Utah) connected to the ETT, with a suctioning time of 10 s and suction pressure of -100 mm Hg. OS was performed with the same catheter, the same suctioning time, and the same suctioning pressure, but with the ventilator disconnected from the animal.

All animals received one OS, CS/VC, and CS/PC, in a random sequence. After each suctioning the animal was ventilated in VC or PC for 40 min or as much time as necessary to achieve gas-exchange stability before the next suctioning. During this stabilization period, PEEP and F_{IO_2} were reset if necessary, based on the PEEP/ F_{IO_2} table.

At the end of the experiment the animals were sacrificed during deep anesthesia (10 mg/kg pentobarbital), with rapid injection of 50 mL saturated potassium chloride solution. Electrocardiogram and arterial blood pressure measurement confirmed cardiac arrest.

Airway flow, P_{aO_2} , blood pressure, pulmonary arterial pressure, and cardiac output were recorded before, during, and 10 min after suctioning, and arterial blood gas values were measured before and at 1 min, 5 min, and 10 min after suctioning. Cardiac output was not measured during suctioning.

Statistics

All data (\dot{V} , P_{aO_2} , blood pressure, pulmonary arterial pressure, cardiac output, and arterial blood gas values) are expressed as mean \pm SD. The effects of OS, CS/VC, and CS/PC on these variables were assessed with analysis of variance for repeated measures. Post-hoc pairwise comparisons were made with Tukey's test. These analyses were conducted with SPSS 10.0 (SPSS, Chicago, Illinois). The effects of OS, CS/VC, and CS/PC on shunt, P_{aCO_2} , and P_{aO_2}/F_{IO_2} were assessed with a generalized-estimating-equation analysis, which is an extension of the general linear model that accounts for clustering of the outcome variable within each animal. These analyses were conducted with Stata statistics software (StataCorp, College Station, Texas). Differences with p values < 0.05 were considered statistically significant.

Results

The PEEP (OS 18 cm H_2O , CS/VC 18 cm H_2O , CS/PC 19 cm H_2O) and F_{IO_2} (all 1.0) selected from the PEEP/ F_{IO_2} table were similar in all suctioning groups. The P_{aO_2}/F_{IO_2} recorded before OS, CS/VC, and CS/PC were low, and equivalent to one another (OS 156.7 ± 42.1 mm Hg, CS/VC 154.5 ± 49.6 mm Hg, CS/PC 161.8 ± 35.2 mm Hg, $p = 0.75$). Differences in P_{aO_2}/F_{IO_2} before and after suctioning are illustrated in Figure 1. Overall, a significant interaction was present between the groups (OS, CS/VC, and CS/PC) and over the 10-min post-suctioning evaluation time ($p < 0.001$). The analysis was repeated excluding the baseline data, and in this analysis the interaction term was not

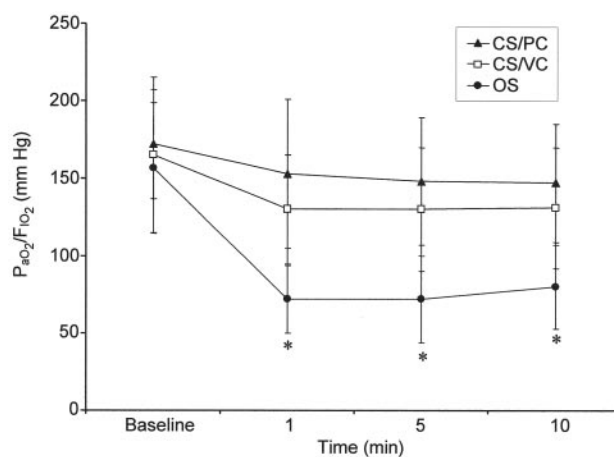


Fig. 1. Ratio of P_{aO_2} to fraction of inspired oxygen (F_{IO_2}) before (baseline) and after suctioning, with open suctioning (OS) or closed suctioning (CS) with pressure-control (PC) or volume-control (VC) ventilation. OS caused a greater P_{aO_2}/F_{IO_2} decrease than did CS, with both VC and PC. * CS/VC and CS/PC were significantly different from OS (both $p < 0.001$).

significant ($p = 0.766$), indicating that the effect of group (OS, CS/VC, and CS/PC) did not change between 1 min and 10 min. At 5 min, the CS/VC and CS/PC groups had higher P_{aO_2}/F_{IO_2} values than did the OS group (both $p < 0.001$). No difference in oxygenation was observed between VC and PC during CS ($p = 0.72$).

Figures 2 and 3 illustrate the changes in P_{aCO_2} and pulmonary shunt, respectively. Hypercapnia was greater overall after suctioning than before suctioning during OS, CS/VC, and CS/PC (all $p < 0.001$), and hypercapnia increased during the 10 min after suctioning ($p < 0.001$). There were no significant interactions between the groups (OS, CS/VC, and CS/PC) and the 10-min post-suctioning period. Pulmonary shunt was also greater 10 min after suctioning than before suctioning, with both OS and CS/VC ($p < 0.001$), but not with CS/PC ($p = 0.34$).

Table 1 shows arterial blood pressure, pulmonary arterial pressure, heart rate, and cardiac output before, during, and after suctioning. No significant changes were detected before or 10 min after suctioning with OS, CS/VC, or CS/PC. However, during OS there was a trend toward an increase in blood pressure and a decrease in pulmonary arterial pressure and HR. Throughout all phases of the study the respiratory rate was 35 breaths/min in all the animals, and V_T was maintained at 6 mL/kg of body weight (thus, V_T ranged between 150 mL and 210 mL). After suctioning in PC, the PC level did not require adjustment to maintain the V_T , and since flow was delivered with a

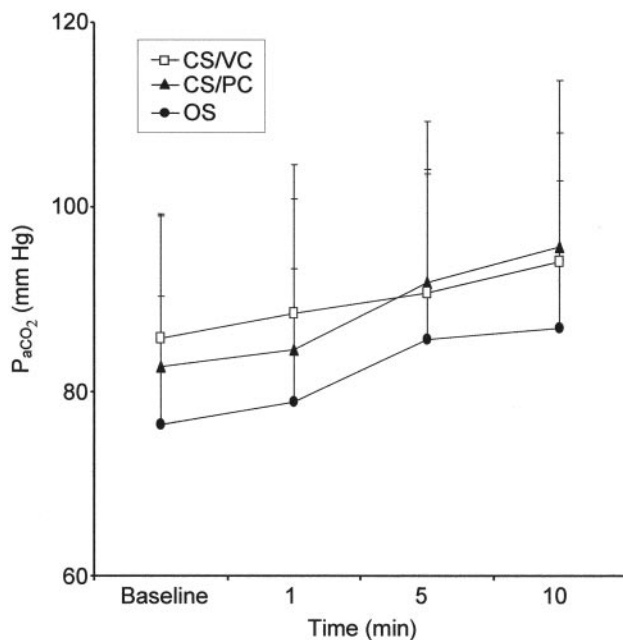


Fig. 2. P_{aCO_2} before and after suctioning, with open suctioning (OS) or closed suctioning (CS) with volume-control (VC) or pressure-control (PC) ventilation. P_{aCO_2} increased over the 10-min evaluation period ($p < 0.001$). In all settings there was a significant increase in P_{aCO_2} 10 min after suctioning ($p < 0.05$).

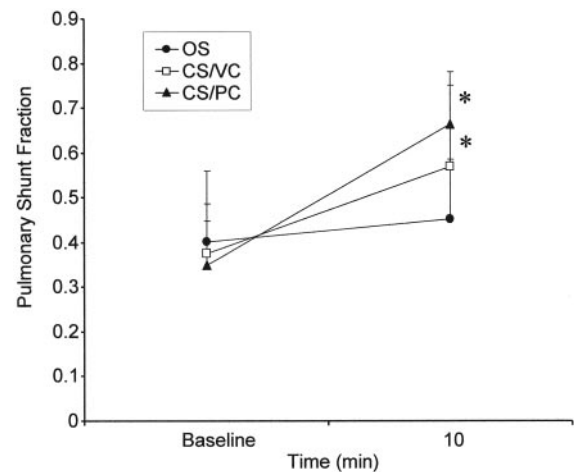


Fig. 3. Pulmonary shunt before and 10 min after endotracheal suctioning, with open suctioning (OS) or closed suctioning (CS) with volume-control (VC) or pressure-control (PC) ventilation. * Shunt was significantly higher at 10 min than at baseline with OS and CS/VC, but not with CS/PC (both $p < 0.001$).

Table 1. Hemodynamic Variables

Variable	Before, During, or After Suctioning	Type of Suctioning		
		OS	CS/VC	CS/PC
BP (mm Hg)	Before	112 ± 13	110 ± 11	108 ± 17
	During	115 ± 16	119 ± 14	121 ± 25
	After	109 ± 14	110 ± 14	108 ± 20
PAP (mm Hg)	Before	30 ± 5	31 ± 6	31 ± 3
	During	27 ± 9	33 ± 6	32 ± 5
	After	33 ± 6	33 ± 6	33 ± 4
HR (beats/min)	Before	145 ± 26	153 ± 33	157 ± 38
	During	133 ± 16	150 ± 34	160 ± 39
	After	135 ± 9	157 ± 24	163 ± 35
CO (L/min)	Before	6.53 ± 2.77	5.92 ± 2.41	6.08 ± 1.53
	During	NA	NA	NA
	After	6.56 ± 1.51	6.38 ± 2.10	6.62 ± 1.68

OS = open suctioning

CS/VC = closed suctioning with volume ventilation

CS/PC = closed suctioning with pressure ventilation

BP = mean arterial blood pressure

PAP = mean pulmonary artery pressure

HR = heart rate

CO = cardiac output

NA = not applicable

decelerating flow pattern, end-inspiratory pressure in all settings was ≤ 35 cm H₂O.

Discussion

Our findings add to the existing literature, since the study was performed during lung-protective ventilation us-

ing the ARDS Network ventilation strategy. The main findings of the present study are:

1. The P_{aO_2}/F_{IO_2} ratio was better maintained during CS than during OS, with either VC or PC.
2. Although the hypoventilation associated with OS led to marked hypercapnia, the use of CS did not prevent hypercapnia 10 min after suctioning.
3. The hemodynamic effects of suctioning did not differ for any of the experimental conditions. However, a non-significant trend suggested increased cardiovascular stress during OS.

Endotracheal suctioning and bronchoscopy are two of the most common secretion-management procedures performed in mechanically ventilated patients, although lung-volume loss, hypoxemia, and hemodynamic compromise are known risks of these procedures.^{6,7-10} Progressive atelectasis in ARDS can exacerbate hypoxemia and may produce further lung injury plus systemic injury through the release of cytokines and right-ventricular failure.¹⁵

Increased Hypercapnia

One unanticipated finding was the degree of hypercapnia we observed following only 10 seconds of suctioning. Based on prior literature regarding bronchoscopy¹⁶ and brain-death apnea testing,¹⁷ we had anticipated only modest reductions in alveolar ventilation after suctioning. However, we are unaware of prior literature that has examined the effect of suctioning on gas exchange during permissive hypercapnia. The use of 30-kg sheep instead of adult patients may account for some of the CO_2 increase. The lung of a sheep is smaller overall than that of an adult human, but the size of the trachea and main bronchi are the same as a male adult human. It may have been that the suction catheter traveled deeper into the lung of the animal, causing greater ventilation-perfusion mismatch. However, the P_{O_2} after suctioning during VC and PC would argue against that possibility. Potential mechanisms of worsening hypercapnia following brief suctioning are unknown. Increased dead space may occur because of reduced cardiac output (although this was not observed in the present study), overdistention of compliant lung units following collapse of noncompliant lung units, or CO_2 -induced bronchodilation due to poorly perfused lung units.¹⁸ Or the presence of marked hypercarbia before the initiation of suctioning may have contributed to the further increase in CO_2 levels. In addition, increased intrapulmonary shunt, especially in the setting of permissive hypercapnia, may increase CO_2 because of venous admixture. Which, if any, of these mechanisms contributed to our findings is unclear and requires further study.

Comparison With Other Studies

Our findings are similar to those of other groups that have evaluated CS.^{7,8,19-21} Other groups have found that arterial desaturation related to suctioning is greater with OS than with CS.^{7,10,19-22} Specifically, Maggiore et al⁸ studied the effect of CS and OS in 9 patients with ALI. CS was performed via the swivel adapter connected to the ETT and using a Hi-Care CS system (DAR-Tyco Healthcare Group, Mirandola, Italy). During both CS approaches the patients were ventilated with V_T of 6–8 mL/kg, respiratory rate of 18–25 breaths/min, PEEP level chosen by the managing physician, and F_{IO_2} adjusted to maintain blood oxygen saturation (measured via pulse oximetry [S_{pO_2}]) $\geq 92\%$. In addition, CS was performed with the ventilator delivering pressure support to reach 40 cm H_2O total pressure. S_{pO_2} dropped more with OS than with CS (OS $-9.2 \pm 7.6\%$, CS with swivel $-1.7 \pm 0.9\%$, CS with Hi-Care $-2.2 \pm 2.7\%$, swivel with 40 cm H_2O pressure support $-1.5 \pm 0.6\%$, Hi-Care with 40 cm H_2O pressure support $-1.3 \pm 0.6\%$, $p < 0.01$).

Cereda et al⁷ reported similar data from 10 critically ill ALI patients. They also found a greater S_{pO_2} reduction during OS than during CS (from $97.4 \pm 2.9\%$ to $97.2 \pm 2.9\%$ during CS vs from $97.7 \pm 3.0\%$ to $94.6 \pm 5.1\%$ during OS). In the Cereda et al study,⁷ ventilation was provided in VC, V_T was about 630 mL, PEEP was 10 cm H_2O , respiratory rate was 15 breaths/min, plateau pressure was 27 cm H_2O , and F_{IO_2} was set to maintain $S_{pO_2} > 95\%$.

In addition, a much greater end-expiratory lung-volume change with OS than with CS has been documented. Cereda et al⁷ found a 1.2 ± 0.7 -L volume change with CS, measured using induction plethysmography. Using computed tomography, Brochard et al⁶ observed a > 300 -mL end-expiratory lung-volume change during OS versus CS. Finally, Maggiore et al⁸ found a 123-mL end-expiratory volume change with CS versus a 1,645-mL volume change with OS in 6 ARDS patients, measured using induction plethysmography.

Limitations

This study was performed with an animal model, not with patients, and the model was of the severest form of ARDS, so these data must be cautiously applied to patients with less severe ARDS. However, the fact that our data are consistent with that of other groups^{7,8,19-21} increases the credibility of our findings. In addition, we did not directly measure lung volume (with induction plethysmography or magnetometers), nor did we measure lung compliance, and thus one could argue that the loss of lung volume induced by suctioning is somewhat speculative. Although we accept this criticism, we believe, based on the literature discussed above,⁸ that the fall in oxygen saturation ob-

served in the context of suctioning is from alveolar derecruitment.

Second, because suctioning is generally performed for secretion management, one potentially important outcome variable would be the effectiveness of secretion removal. For example, if a suctioning strategy simply led to direct removal of gas being delivered by the ventilator, then suctioning might be ineffective at removing secretions. Because PC is associated with unlimited airflow from the ventilator (as compared with VC), one might predict that CS would be less effective during PC. Since we did not observe a difference in gas exchange between PC and VC with suctioning, we do not believe that important differences in the effectiveness of suctioning were taking place. However, we do acknowledge that we have no direct quantitative measure of suctioning effectiveness for secretion removal.

Third, we measured hemodynamics at only one time point after suctioning (10 min), and therefore do not know if transient fluctuations in blood flow occurred immediately following suctioning. As a result, we cannot assess if changes in blood flow affected dead space or shunt fraction.

Despite these limitations, we believe that our findings provide an important message to clinicians treating ARDS patients—that suctioning should always be performed with a CS system in patients with ARDS/ALI, even in the context of state-of-the-art mechanical ventilation strategies.

Conclusion

In conclusion, during lung-protective ventilation, using a V_T of 6 mL/kg and the ARDS Network¹⁴ PEEP/ F_{IO_2} table, CS, with either VC or PC, avoids arterial desaturation better than does OS. With all the suctioning procedures, P_{aCO_2} increased beyond expectations 10 min after suctioning. Hemodynamic changes were similar during all suctioning procedures.

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