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Are there differential effects of endotracheal suction on respiratory mechanics and gas exchange in patients with severe sepsis using pressure- and volume-controlled ventilation?

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Abstract

The aim of the current study was to investigate the differential effects of endotracheal suction on respiratory mechanics and gas exchange in patients with severe sepsis using pressure-controlled ventilation (PCV) and volume-controlled ventilation (VCV). We prospectively included 76 patients with severe sepsis receiving mechanical ventilation due to acute respiratory failure (ARF). The effects of endotracheal suction on respiratory mechanics and gas exchange during VCV and PCV were compared. PCV used an inspiratory pressure that caused the same tidal volume (VT) as that of VCV; a VT of 9 mL/kg predicted body weight and an 8.0 mm inner-diameter endotracheal tube were used. Patients underwent suction for 15 s using an open suction system with 12F catheters connected to a 150 mmHg vacuum. VT was decreased by 28.9% and 27.8% at 1 min and 10 min after suction compared with the baseline when using PCV, and compliance decreased by 31.4% and 30.3% (P<0.05), respectively. When using VCV, the airway peak pressure (Ppeak) was increased by 31.5% and 28.0% at 1 and 10 min, respectively, after suction compared with the baseline; airway plateau pressure (Pplat) increased by 22.0% and 22.9%, and compliance decreased by 34.4% and 33.2% (P<0.05). In PCV, PaO_2 was increased by 6.8% and 12.4% at 3 min and 10 min, respectively, after suctioning, compared to respective increases of 18.9% and 29.6% observed using VCV (P<0.05). In PCV, the difference in PaO_2 at 10 min after suctioning compared with the baseline was significant (72.5 ± 16.9 *vs.* 87.5 ± 17.2 mmHg; P<0.05). In VCV, no significant difference was observed (84.5 ± 17.1 *vs.* 86.1 ± 14.7 mmHg; P>0.05). Endotracheal suction may impair gas exchange and respiratory mechanics in patients with severe sepsis under both PCV and VCV, but the effects on gas exchange recover quickly under VCV.

Keywords: Endotracheal suction, Sepsis, Pressure-controlled ventilation, Volume-controlled ventilation, Gas exchange, Respiratory mechanics.

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Introduction

Mechanical ventilation (MV) is increasingly used as a life-saving tool in the treatment of acute and chronic respiratory failure, especially in potentially reversible cases. It effectively improves gas exchange while reducing dyspnea and inspiratory effort. However, it also impairs spontaneous clearing of airway secretions in critical patients, as coughing is less effective or impossible [1,2]. Endotracheal suction (ES) is a periodic procedure that removes mucus from the airways in MV patients with artificial airways, and is reportedly essential to maintain airway patency and cleanliness [3,4]. Recent recommendations for ES use advocate several key points of the procedure [5] including: 1) advancing the suction catheter until resistance is met; 2) not applying suctioning routinely, but only as needed; 3) not using saline instillation; 4) using the shallow suctioning method; and 5) not applying suction pressure for longer than 15 s. However, ES is not free from hazards and may be associated with physiological complications, such as lung derecruitment and resultant hypoxemia. Nurses should work meticulously and carefully, as many side effects develop despite the use of appropriate suctioning methods and judicious indication of the invasive procedure.

Different endotracheal suction systems are used; the most commonly used technique in developing countries is the open suction system (OSS) [6]. When suctioning with an OSS, the patient should first be detached from the ventilator; airway suctioning should be performed using a disposable sterile catheter connected to a vacuum system. The OSS may create negative pressure in the trachea and affect gas exchange and respiratory mechanics, leading to serious and even life-threatening complications such as infection, alterations in hemodynamic parameters, bronchoconstriction, atelectasis, and pain. An experimental study comparing the effects of open and closed suctioning showed that VCV could be used to rapidly restore lung aeration and oxygenation after lung collapse induced by an OSS. However, few studies have investigated

the effects of endotracheal suction on respiratory mechanics and gas exchange in patients. Acute organ dysfunction induced by sepsis most commonly affects the respiratory system. Respiratory compromise is classically manifested as acute respiratory distress syndrome (ARDS), which requires mechanical ventilation.

In recent years, pressure-controlled ventilation (PCV) has been considered a protective ventilator method, as it prevents the uncontrolled increase of alveolar pressure and may reduce the risk of lung injury during MV [7-9]. Compared with volume-controlled ventilation (VCV), PCV has been reported to favor gas distribution and shown to reduce breathing effort [10-12], but few studies have compared the effect of ES on patients using both PCV and VCV. We hypothesized that ES might have different side effects depending on the ventilator mode. This study investigated whether ES has different effects on respiratory mechanics and gas exchange in patients with severe sepsis who use either PCV or VCV. The findings may contribute to future meta-analyses and guide the use of suction in patients.

Materials and Methods

Ethics statement

This was a single-center prospective cohort study. The study was approved by the Ethics Committee of the First Affiliated Hospital of China Medical University (Liaoning, People's Republic of China). Prior to the study, we explained the study process to the patients' families and they provided written informed consent. We also observed all ethical criteria in accordance with the most recent version of the Declaration of Helsinki.

Study design and subjects

The study was conducted from February 2011 to March 2014 in the 16-bed emergency intensive care unit (EICU) at the First Affiliated Hospital of China Medical University. The hospital was chosen for the following reasons: OSS was used in patients receiving MV and the ventilator used can collect and continuously store data including VT, peak airway pressure (Ppeak), and plateau airway pressure (Pplat). All patients were connected to a monitor to obtain heart rate (HR) as beats per minute (bpm) and mean arterial pressure (MAP). Pulse oximetry was performed on all patients and arterial blood gas was measured locally within the unit. Consecutive subjects with severe sepsis receiving mechanical ventilation due to acute respiratory failure (ARF) were screened for participation in the study. According to international recommendations [13], sepsis was defined as infection plus at least satisfying two systemic inflammatory response criteria. Severe sepsis was defined as sepsis plus sepsis-induced organ dysfunction or tissue hypoperfusion. The criteria for the diagnosis of ARF were: 1) type I: PaO_2<60 mmHg, $PaCO_2$<50 mmHg; 2) type II: PaO_2<60 mmHg, $PaCO_2 \geq 50$ mmHg. Exclusion criteria were: age<18 years, pregnancy, preexisting lung disease, hemodynamic instability, a contraindication of sedation, and the need for repeated suctioning during conversion from one ventilation mode to another.

Patients were ventilated *via* an 8.0 mm inner-diameter endotracheal tube using either PCV or VCV (Maquet Servo V. 2.0, Germany). Sedation was performed with a continuous infusion of propofol (5-10 mg/h). The infusion rate was adjusted so that the patient exhibited no spontaneous breathing efforts during the study. The patient was also administered an intravenous bolus of 3 mg propofol before ES; muscle relaxants were not used. Patients were monitored using electrocardiography, continuous invasive blood pressure monitoring, and pulse oximetry (Phillips Intellivue MP60 monitor, Medizinsysteme, Germany). After MV, the severity of disease was assessed using a Sequential Organ Failure Assessment (SOFA) score [14]. The presence of dysfunction in an organ was defined when the degree of dysfunction was ≥ 1. The most abnormal value for each clinical and laboratory parameter included in the SOFA system was recorded after MV and then transformed into the dysfunction score, which was graded from 0 to 4. The SOFA score was calculated by summing the worst scores for each of the six organ systems (cardiovascular, neurological, respiratory, renal, hepatic, and coagulation).

Protocol

A crossover design was used. Before the start of the study, patients were randomized to one of two sequences of the two ventilator modes: PCV or VCV. In the first sequence, patients were first subjected to PCV followed by OSS; after 30 min they were subjected to VCV followed by OSS. In the second sequence, patients were first subjected to VCV followed by OSS; after 30 min they were subjected to PCV followed by OSS. When changing the ventilation mode, the original inhaled gas oxygen concentration (FiO_2), end-expiratory positive pressure (PEEP), VT (VCV, 9 mL/kg predicted body weight; PCV, inspiration pressure to reach an equal VT to VCV), and respiratory rate were unchanged. The inspiratory/expiratory ratio was adjusted to 1:1.5-2.0.

Suction

A standardized recruitment method was used to standardize lung volume after MV. After fully suctioning oral, nasal, and airway secretions, the VT of VCV was increased to increase the Pplat by 10 cm H_2O for 20 s. In PCV, the inspiration pressure was increased by 10 cm H_2O and maintained for 20 s. OSS was performed when the alarm for Ppeak (≥ 40 cm H_2O) in VCV or for VT (≤ 6 mL/kg) in PCV sounded, or when SpO_2 decreased by >5%. No supplementary hyperventilation or oxygenation maneuvers were performed before or after suction. During the suction procedure, a 12F catheter was inserted into the endotracheal tube followed by disconnection of the endotracheal tube from the Y-shaped tube of the ventilator's circuit. The catheter was introduced until resistance was met; suctioning was performed for 15 s using a 150 mmHg vacuum. The catheter was gently rotated and removed

gradually. The endotracheal tube was reconnected to the ventilator for MV immediately after suctioning.

Measurements

Measurements were taken 15 min after standardizing the lung volume (baseline), immediately after disconnection (0 min), and 1, 3, 5, and 10 mins after suctioning. The HR, MAP, VT, Ppeak, and Pplat were recorded. The total respiratory system compliance (Crs) was calculated with the equation

$Crs=VT/(Ppeak-PEEP) \rightarrow (1)$

Values of VT, Ppeak, and Pplat were calculated by averaging five respiratory cycles. Dynamic changes in SpO_2 were continuously monitored. Arterial blood gas analysis was performed at baseline, 0, 3, and 10 mins after suction.

Statistical analysis

A power analysis assumed that the difference in a change in compliance at 1 min after suctioning would be 15 ± 10% and indicated that 64 patients should be included (α=0.05, 1-β=0.90; crossover design). The data are presented as mean ± standard deviation SD) if not otherwise indicated. A χ^2 test was used to compare frequencies. The one-way analysis of variance (ANOVA) and the Student's t-test were used to analyze normally distributed variables; the Mann-Whitney U-test was used to analyze non-normally distributed variables. Comparisons between nominal variables of the two groups used Fisher's exact test. All tests of statistical significance were two-sided; P<0.05 was considered significant. All statistical analyses were performed using PASW Statistics for Windows Version 18.0 (SPSS, Inc., Chicago, IL, USA).

Results

Baseline characteristics of patients

The baseline characteristics of all patients in the cohort are presented in Table 1. The study included 76 patients (35 males, 41 females) aged 19 to 76 years with a mean age of 57.5 ± 11.2 years, including 40 patients in the PCV-first group and 36 patients in the VCV-first group. The source of sepsis was pulmonary in 31 cases (40.8%), urinary in 12 cases (15.8%), abdominal/gastrointestinal in 13 cases (17.1%), skin/soft tissue in 7 cases (9.2%), other identified source in 6 cases (7.9%); and unidentified in 7 cases (9.2%). The most common existing comorbidities were diabetes mellitus (23.7%) and ischemic heart failure (17.1%). The severity of the illness was recorded as the average SOFA after MV. There were no significant differences in the characteristics of the two groups (all, P>0.05).

Table 1. *Baseline characteristics of patients.*

Characteristics	Overall population (N=76)	PCV Group (N=40)	VCV Group (N=36)
Gender, Male/Female (n/n)	35/41	17/23	18/18
Age (yr) (mean ± SD)	57.5 ± 11.2	56.9 ± 10.1	58.2 ± 9.7
SOFA after MV (mean ± SD)	5.7 ± 1.8	5.8 ± 1.4	5.6 ± 1.4
Sepsis origin (n)			
Pulmonary	31	17	14
Urinary	12	5	7
Abdominal/gastrointestinal	13	7	6
Skin/soft tissue	7	3	4
Others/unknown	13	8	5
Comorbid diseases			
Hypertension	10	4	6
Diabetes mellitus	18	10	8
Ischemic heart failure	13	7	6
Congestive heart failure	6	3	3
Chronic neurologic disorder	8	3	5
Cancer	3	2	1

Chronic neurologic disorder including stroke with residual neurologic deficit, multiple sclerosis, Parkinson's disease and severe dementia. SOFA: Sequential Organ Failure Assessment.

Changes in hemodynamic parameters

Changes in HR and MAP before and at different time points after suctioning using PCV and VCV are shown in Table 2. At 5 min after suctioning, HR and MAP were respectively increased by 12.0% and 12.9% when using PCV compared with the baseline (HR: 103 ± 16 *vs.* 92 ± 17 bpm; MAP: 96 ± 17 *vs.* 85 ± 15 mmHg; P<0.05). HR and MAP were respectively increased by 13.3% and 12.2% when using VCV (HR: 102 ± 19 *vs.* 90 ± 16 bpm; MAP: 92 ± 15 *vs.* 82 ± 13 mmHg; P<0.05). There were no significant differences in HR and MAP changes at 10 min after suctioning compared with the baseline using PCV or VCV (all, P>0.05). Furthermore, there were no significant differences in HR or MAP between PCV and VCV at different time points after suctioning (all, P>0.05).

Table 2. *The changes of heart rate and mean arterial pressure under different ventilation modes for patients before and after suction.*

		PCV	VCV
HR (beat/min)	Baseline	92 ± 17	90 ± 16
	0 min	112 ± 19	115 ± 21
	1 min	120 ± 22*	113 ± 18*
	3 min	113 ± 26*	110 ± 23*
	5 min	103 ± 16*	102 ± 19*
	10 min	91 ± 15	89 ± 18
MAP (mmHg)	Baseline	85 ± 15	82 ± 13

0 min	91 ± 16	88 ± 18
1 min	93 ± 14*	90 ± 15*
3 min	95 ± 19*	91 ± 17*
5 min	96 ± 17*	92 ± 15*
10 min	89 ± 16	85 ± 14

*Compared with the baseline, P<0.05; HR: Heart Rate; MAP: Mean Arterial Pressure; PCV: Pressure Controlled Ventilation; VCV: Volume Controlled Ventilation

Changes in respiratory mechanics

Changes in VT, Crs, and airway pressure before and after suctioning at different time points are shown in Table 3.

In PCV, VT and Crs had respectively decreased by 28.9% and 31.4% compared with baseline values (VT: 6.4 ± 1.7 *vs.* 9.0 ± 0.2 mL/kg; Crs: 18.1 ± 5.7 *vs.* 26.4 ± 5.6 mL/cmH_2O; P<0.05). VT and Crs had respectively decreased by 27.8% and 30.3% at 10 min after suctioning (VT: 6.5 ± 1.9 *vs.* 9.0 ± 0.2 mL/kg; Crs: 18.4 ± 4.3 *vs.* 26.4 ± 5.6 mL/cmH_2O; P<0.05). No significant differences in the changes in VT or Crs at 10 min after suctioning were found compared with parameters at 0 min (all, P>0.05).

In VCV, Ppeak and Pplat showed respective increases of 31.5% and 22.0% at 1 min after suctioning compared with baseline values (Ppeak: 33.8 ± 7.4 *vs.* 25.7 ± 6.8 cm H_2O; Pplat: 27.7 ± 6.3 *vs.* 22.7 ± 5.1 cm H_2O; P<0.05). Crs decreased by 34.4% (16.8 ± 3.8 *vs.* 25.6 ± 4.3 mL/cm H_2O; P<0.05). At 10 min after suctioning, the Ppeak and Pplat showed respective increases of 28.0% and 22.9%, respectively (Ppeak: 32.9 ± 2.8 *vs.* 25.7 ± 6.8 cm H_2O, Pplat: 27.9 ± 4.8 *vs.* 22.7 ± 4.8 cm H_2O; P<0.05). Crs decreased by 33.2% (17.1 ± 5.7 *vs.* 25.6 ± 4.3 mL/cm H_2O; P<0.05).There were no significant differences in the Ppeak and Crs changes at 10 min after suctioning compared with 0 min (all, P>0.05).

Table 3. *The changes of tidal volume, compliance and airway pressure under different ventilation modes for patients before and after suction.*

Point-in-time	PCV		VCV		
	VT (ml/kg)	Crs (ml/cm H_2O)	Ppeak (cm H_2O)	Crs (ml/ cm H_2O)	Pplat (cm H_2O)
Baseline	9.0 ± 0.2	26.4 ± 5.6	25.7 ± 6.8	25.6 ± 4.3	22.7 ± 5.1
0 min	7.4 ± 2.1	21.2 ± 6.8*	36.4 ± 7.5*	16.1 ± 3.9*	26.4 ± 5.7
1 min	6.4 ± 1.7*	18.1 ± 5.7*	33.8 ± 7.4*	16.8 ± 3.8*	27.7 ± 6.3*
3 min	6.6 ± 1.5*	18.9 ± 4.8*	33.6 ± 5.7*	16.7 ± 4.6*	27.6 ± 5.2*
5 min	6.6 ± 1.3*	18.8 ± 3.9*	33.7 ± 6.2*	16.8 ± 5.5*	27.2 ± 5.1*
10 min	6.5 ± 1.9*	18.4 ± 4.3*	32.9 ± 5.8*	17.1 ± 5.7*	27.9 ± 4.8*

*Compared with the baseline, P<0.05; PCV: Pressure Controlled Ventilation; VCV: Volume Controlled Ventilation; VT: Tidal volume; Ppeak: Airway peak pressure; Crs: Compliance; Pplat: Airway plat pressure.

Changes in oxygenation

Compared with 0 min, PaO_2 had increased by 6.8% at 3 min after suction using PCV (68.9 ± 14.2 *vs.* 64.5 ± 15.2 mmHg; P>0.05) and by 12.4% at 10 min (72.5 ± 16.9 *vs.* 64.5 ± 15.2 mmHg; P>0.05). There was also a significant difference in PaO_2 at 10 min after suctioning compared with the baseline (72.5 ± 16.9 *vs.* 87.5 ± 17.2 mmHg; P<0.05).

PaO_2 had increased by 18.9% at 3 min after suctioning when using VCV compared with 0 min (77.5 ± 16.4 *vs.* 65.2 ± 13.6 mmHg; P<0.05), and by 29.6% at 10 min (84.5 ± 17.1 *vs.* 65.2 ± 13.6 mmHg; P<0.05). There was no significant difference in PaO_2 at 10 min after suctioning compared with the baseline at 1 min after suction (84.5 ± 17.1 *vs.* 86.1 ± 14.7 mmHg; P>0.05).

When using PCV, PaO_2 increased by 6.7 ± 1.8% and 12.3 ± 4.0% at 3 min and 10 min after suctioning, compared with the value at 0 min. This increase was lower than that seen when using VCV (18.8 ± 5.6% and 29.6 ± 7.3%; P<0.05). The difference in $PaCO_2$ using different ventilation modes was not statistically significant (P>0.05).

Discussion

MV is a method that mechanically assists or replaces spontaneous breathing. MV should be considered when clinical or laboratory signs indicate the patient cannot maintain adequate oxygenation or ventilation [15,16]. Periodic endotracheal suctioning is a frequently used procedure for intubated patients receiving MV due to their inability to spontaneously clear the airway. It can help avoid accumulation of secretions and tracheal occlusion, thereby guaranteeing optimal oxygenation or ventilation. However, ES may also cause adverse effects such as hypoxemia, disturbances in cardiac rhythm, and the development of ventilator-associated pneumonia (VAP) [17]. The most commonly used technique is an OSS that entails disconnecting the patient from the ventilator and suctioning the airway. OSS may lead to different effects on gas exchange and respiratory mechanics in patients under VCV and PCV. However, there are few studies on the differential effects of OSS in patients with severe sepsis using PCV and VCV.

This study evaluated the effects of OSS on gas exchange and respiratory mechanics in 76 severe sepsis patients using PCV and VCV, using a crossover control method. We standardized lung volumes in the different modes to ensure patients were at the same basal state before suctioning, and then began suctioning according to clinical indications. During OSS, there was an initial drop in lung volume immediately following the patient's disconnection from the ventilator, followed by a second drop after initiation of suctioning. Therefore, OSS may lead to alveolar collapse, resulting in reduced compliance; that is followed by a decrease in airway pressure and loss of lung

volume, which can be ascribed to the disconnection and to the suction generating negative pressure in the airway [18].

The VT was not changed during VCV. Airway pressure increases in response to reduced compliance and increased resistance, and overexpansion of the open alveolar may also cause an increase in Pplat. In PCV, alveolar collapse leads to an increase of respiratory resistance. Stabilizing the inspiratory pressure to a fixed point and limiting the maximum airway pressure delivered to the lung may reduce VT. During lung volume standardization, the secretions in the oral and nasal cavities and in the airway were fully removed by suctioning; therefore, each suction procedure in this study removed a smaller volume of secretion than was typically observed. This indicates that suctioning removed more gas, which caused a greater loss of lung volume during the procedure. An open suctioning procedure, where negative pressure is applied without any contact with mucus within the airway, may cause more deleterious effects on lung volume. In contrast, when the suction catheter directly contacts the mucus, it may have a less deleterious effect on lung volume. For patients with greater secretion removal, less gas is removed and the side effects may not be as significant as in the present study.

During MV, sputum accumulation in the airway can lead to an increased airway resistance and airway pressure. However, we did not observe a decrease in respiratory pressure below the baseline after OSS when using VCV. A possible explanation is that OSS also evokes a transient bronchoconstrictor response, and therefore does not reduce respiratory resistance below the presuction value. This may be related to the fact that the catheter usually reaches the carina or the main bronchi and is thus effective only in cleaning mucus from the proximal airways. However, the leading factors that influence airway resistance are the medium and small bronchi up to the seventh generation, which are the main sites of respiratory resistance [19]. Due to bronchial spasms resulting from stimulation caused by the suction tube and other factors such as alveolar collapse and atelectasis, airway resistance after suction was not lower than the baseline, which presented as an increased Ppeak in VCV.

Our results also suggest that PaO_2 gradually increased after suctioning, but returned to baseline relatively quickly in VCV patients. PaO_2 respectively increased by 6.8% and 12.4% at 3 and 10 min after suctioning when using PCV compared with presuction values; there was an 18.9% and a 29.6% increase using VCV. We demonstrated that the increase in PaO_2 was greater in patients using VCV than in those using PCV at 3 and 10 min after suctioning. In PCV, respiratory resistance increased as a result of alveolar collapse; VT declined because of the preset inspiratory pressure, which resulted in a PaO_2 decrease. In VCV, a fixed VT causes increased airway pressure followed by relative overexpansion of those parts of the lung that remain open due to the increased Pplat. To a certain extent, the fixed VT affected lung recruitment, which may partially explain the rapid recovery of gas exchange in VCV. PCV is a time-cycled mode in which approximately square waves of pressure are applied and released by a decelerating flow that results in a more even distribution of ventilation than VCV [20]. We speculate that after suctioning, the increased respiratory pressure when using PCV to maintain a consistent VT, may be more effective in reducing adverse effects during suctioning; however, this speculation requires further study.

The most common clinical methods used to prevent hypoxia and promote recovery before and after suctioning were to provide a high concentration of oxygen or increase VT [21]. Before suctioning, inhalation of high concentrations of oxygen can increase the alveolar-lung capillary oxygen concentration gradient and increase the oxygen reserve, while inhalation of high concentrations of oxygen after suctioning contributes to rapid recovery from the hypoxic state. However, this method cannot prevent the lung volume reduction induced by suction, and absorption atelectasis may occur after inhalation of high-concentration oxygen [22]. Hyperventilation may cause excessive traction on the alveolar epithelium and vascular endothelium, resulting in lung injury. It may also cause pneumothorax or have an adverse effect on hemodynamics. This study evaluated the effect of OSS on HR and MAP in patients with severe sepsis who used PCV and VCV. The results of this study showed that ES can increase HR in both PCV and VCV patients who underwent OSS. HR significantly increased immediately after the procedure terminated, and mean HR decreased to the baseline level by 10 min after suctioning. The decrease may be independent of the mode of MV and may be attributed, but not limited, to mechanical stimulation from the catheter, hypoxia, and the pain and stress experienced, despite the use of propofol.

Several important issues should be considered when interpreting the results of our study. It was a monocentric study, and institution-specific variables may have influenced the present results. In addition, our conclusions may not be applicable to patients with severe sepsis with preexisting lung disease due to the exclusion criteria used in this study. Furthermore, although this was a prospective study, the enrolled subjects did not have the same causes or courses of severe sepsis, and therefore, there may have been some bias towards patients in different phases of severe sepsis. Another potential problem with the study is the short monitoring time of the indicators of respiratory mechanics after suctioning. Nevertheless, it should be emphasized that the study was conducted at an academic tertiary referral emergency department, which is included in a referral system that prioritizes severe cases. The results may not be generally applicable to other clinical environments.

In conclusion, our study provides comprehensive and reliable evidence that OSS for patients with severe sepsis receiving MV may impair gas exchange and decrease lung compliance under both PCV and VCV, but the effects on gas exchange recover quickly when using VCV. The present study confirmed that OSS was associated with reduced compliance; therefore, we suggest avoiding OSS unless absolutely necessary. In the presence of a clear indication for suction, such as bubbling secretions in the trachea, OSS may be the method of choice after conversion to the VCV ventilation mode.

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