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Gas exchange impairment induced by open suctioning in acute respiratory distress syndrome: Impact of permissive hypercapnia*

Maria Paula Caramenz, MD, PhD, Eriko Miyoshi, MD, R. Scott Harris, MD, Robert M. Kacmarek, PhD, RRT, and Atul Malhotra, MD

Department of Anesthesia and Critical Care, and Respiratory Care, Massachusetts General Hospital and Harvard Medical School, Boston, MA (MPC, EM); Harvard Medical School, Department of Medicine, Massachusetts General Hospital, Boston, MA (RSH); Harvard Medical School, Respiratory Care, Massachusetts General Hospital, Boston, MA (RMK); and Harvard Medical School, Department of Medicine (Pulmonary and Critical Care and Sleep), Brigham and Women's Hospital, and Beth Israel Deaconess Medical Center, Boston, MA (AM)

Abstract

Objective—To determine whether hypercarbia occurs following the use of open suctioning in lung lavage injured sheep and whether the baseline PaCO₂ and duration of suctioning affect gas exchange.

Design—Prospective laboratory evaluation.

Setting—Animal laboratory in a university hospital.

Subjects—Seven female Dorset sheep.

Interventions—Lung lavage was used to create acute respiratory distress syndrome (ARDS). Mechanical ventilation was provided to produce different baseline PaCO₂ levels.

Measurements and Main Results—Lung injury was developed by isotonic saline lavage until the PaO₂ decreased to 100–150 mm Hg on an FIO₂ of 1.0, positive end-expiratory pressure (PEEP) 5 cm H₂O, and tidal volume 10 mL/kg. Then tidal volume was decreased to 6 mL/kg. Open suctioning was performed on each animal. Each animal experienced four experimental conditions in random order (PaCO₂ 40 and 80 mm Hg and duration of suctioning 10 and 30 secs). Before each of the four experimental conditions, animals underwent lung recruitment continuous positive airway pressure 40 cm H₂O for 40 secs to normalize volume history followed by ventilation for 15 mins where FIO₂ and PEEP were set based on the ARDSNet FIO₂/PEEP. Mean arterial blood pressure, heart rate, pulmonary artery pressure, pulmonary artery occlusion pressure, cardiac output, and arterial blood gases were measured before, 1 min after, and then every 2 mins after open suctioning for 30 mins. Neither the duration of suctioning nor the baseline level of CO₂ had an important influence on the magnitude of the desaturation and the recovery of PO₂ following suctioning ($p < .05$). Level of PEEP did influence the recovery of PaO₂ following suctioning.

*See also p. 644.

Conclusions—While neither baseline CO₂ nor duration of suctioning affected the gas exchange alterations induced by endotracheal suction, high levels of PEEP can help to avoid the associated gas exchange abnormalities in ARDS.

Keywords

airway suctioning; closed suctioning; open suctioning; lung-protective ventilation; intensive care unit ventilators

Mechanical ventilation is frequently required to maintain gas exchange in patients with severe acute respiratory failure. Evidence suggests that lung heterogeneity may contribute to major shear forces within the lung parenchyma, leading many intensivists to use the strategy of open lung protective ventilation (i.e., “open the lung and keep it open”) to promote homogeneity. Thus, there has been considerable interest in the impact of various interventions on lung recruitment and alveolar collapse, particularly when surface tension is high.

Disconnecting the endotracheal tube from the ventilator circuit may cause substantial loss of lung volume, which is further exacerbated by suctioning. In-line closed suction systems have the advantage over open suctioning in maintaining lung volume. However, there are risks associated with closed suction: generation of large negative airway pressures and auto-cycling of the ventilator (1). Fernandez et al. (2) demonstrated reductions in lung volume during both quasi-closed and closed system suctioning but significantly greater loss of lung volume with the open system. In addition, open suctioning may lead to a marked decrease in arterial oxygen tension and increase in arterial carbon dioxide tension (3). During open suctioning, there is still controversy over the impact of the duration of suctioning on gas exchange.

During bronchoscopy, suctioning is frequently applied to remove secretions and lavage fluid. In addition, gas exchange is relatively ineffective during the procedure (4). Ongoing blood flow to the lung further diminishes lung volume due to oxygen absorption. During such a “breath hold,” the majority of CO₂ is buffered in the periphery and not removed by the lung. As a result, end-expiratory lung volume would be predicted to diminish substantially as a result of gas suctioning through the bronchoscope as well as gas absorption through the circulation during the procedure. However, minimal data are available regarding how the duration of bronchoscopy influences the resulting gas exchange abnormalities. Moreover, the majority of studies were not performed using protective mechanical ventilator strategies, such as permissive hypercapnia. Previously, we have shown (5) marked increases of PaCO₂ 10 mins following suctioning during lung-protective ventilation. The goal of this physiologic study was to determine whether baseline PaCO₂ or duration of suctioning influenced the degree of gas exchange abnormality that occurred following open suctioning in acute respiratory distress syndrome (ARDS).

METHODS

Every effort was made to minimize suffering by the animals. The protocol was approved by the Massachusetts General Hospital animal research committee. Seven fasting female sheep (25–30 kg) underwent orotracheal intubation during deep halothane anesthesia by mask. After pulmonary arterial catheter placement (to assess mixed venous blood, cardiac output, and pulmonary artery pressure), 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/hr), ketamine (8 mg/kg/hr), and pancuronium (0.1 mg/kg/hr) providing surgical anesthesia with paralysis.

A carotid arterial catheter was placed to allow continuous blood pressure and arterial blood gas assessment (analyzed immediately using 282, Ciba Corning Diagnostics, Norwood, MA). Flow ($\dot{V}$) at the endotracheal tube was measured by a heated pneumotachometer (Hans Rudolph, Kansas City, KS) connected to a differential pressure transducer ($MP-45 \pm 2$ cm H_2O , Validyne, Northridge, CA). Volume was determined by digital integration of the flow signal. A differential pressure transducer ($MP-45 \pm 100$ cm H_2O , Validyne) was used to measure airway opening pressure (P_{ao}).

All signals— $\dot{V}$, P_{ao} , arterial blood pressure, and pulmonary arterial pressure—were amplified (8805C, Hewlett Packard, Waltham, MA) and converted to digital signals using an analog to digital converter (DI-220, Dataq Instruments, Akron, OH) and recorded at a sampling rate of 100 Hz, using data acquisition software (Chart 5.1.i, Powerlab, ADInstruments). All infusions, including the anesthetic, were given via volumetric infusion pumps. An orogastric tube was placed to empty the stomach, and an esophageal balloon was placed to monitor spontaneous inspiratory efforts.

Protocol

After intubation, basic ventilatory settings were volume control ventilation at a tidal volume of 10 mL/kg, inspiratory-expiratory ratio 1:2, FIO_2 1.0, and positive end-expiratory pressure (PEEP) 5 cm H_2O . Respiratory rate was adjusted to achieve eucapnia ($PaCO_2$ 35–45 cm H_2O).

Lung Injury

Severe lung injury was produced by bilateral lung lavage with a 1-L instillation of isotonic saline warmed to 39°C, repeated every 30 mins until the PaO_2 decreased to 100–150 mm Hg on an FIO_2 of 1.0, PEEP 5 cm H_2O and tidal volume 10 mL/kg. A stable lung injury was defined as a PaO_2 change <10% after 30 mins.

Randomization—Via sealed envelopes, we randomized each animal twice (first baseline CO_2 value and first open suctioning time).

Ventilator Settings

Following injury, tidal volume was decreased to 6 mL/kg. Respiratory rate was set to establish target $PaCO_2$ (40 or 80 mm Hg according to randomization order). Open suctioning was performed on each animal. $PaCO_2$ was adjusted to 40 or 80 mm Hg and suction time 10 or 30 secs (random orders). To normalize lung volume history before each of the four experimental conditions, animals received recruitment maneuvers of continuous positive airway pressure 40 cm H_2O for 40 secs, followed by baseline ventilation for 15 mins where FIO_2 and PEEP were set based on the ARDSNet FIO_2 /PEEP (6).

Airway Suctioning

Open suctioning (10 or 30 secs) was performed using a 14-Fr, TrachCare catheter at a pressure of –100 cm H_2O .

Measurements

Mean arterial blood pressure, heart rate, pulmonary arterial pressure, pulmonary artery occlusion pressure, cardiac output, and arterial blood gases were measured before, 1 min after, and then every 2 mins after open suctioning for 30 mins. During the collecting time, an inspiratory pause was performed to measure pulmonary compliance and resistance.

At the end of the experiment (end of observation period), euthanasia using deep anesthesia (10 mg/kg pentobarbital) was performed by rapid injection of 50 mL of saturated potassium chloride solution. Electrocardiogram and arterial blood pressure confirmed cardiac arrest.

Statistics

All data ($\dot{V}$, Pao, arterial blood pressure, pulmonary arterial pressure, cardiac output, and arterial blood gases) were expressed as median (25th to 75th percentiles). Variables were compared by two-way analysis of variance for repeated measures, considering $p < .05$ as statistically significant.

Using cardiac index, PEEP, arterial PCO_2 , and PO_2 as dependent variables, we performed multiple linear regressions to assess their associations with the level of PaCO_2 or suctioning time.

RESULTS

Figure 1 shows the increment in PaCO_2 that occurs over time following suctioning in the various different groups. There was no difference in the increment in PaCO_2 based on baseline CO_2 , although there was a trend for severe elevations under conditions of baseline hypercapnia ($p = .06$), based on marked elevations in some of the animals. The duration of suctioning had no significant impact on the magnitude of the PaCO_2 increase that occurred.

The level of PEEP, as set based on the ARDSNet criteria, had an important influence on the magnitude of desaturation and recovery of gas exchange following suctioning ($p < .05$). As seen in Figure 2, the trials with PEEP set < 10 cm H_2O led to marked decrements in PaO_2 (55% decrease from the baseline) with delayed and incomplete recovery to baseline PaO_2 . In contrast, the trials with PEEP > 15 cm H_2O led to modest reductions in PaO_2 (13%) with recovery to baseline PaO_2 within 3 mins.

The change in respiratory system compliance with suctioning was not different in the various groups over time. Furthermore, there were no significant effects of suctioning on the hemodynamic variables (heart rate, cardiac output, mean arterial pressure, pulmonary arterial pressure, and pulmonary artery occlusion pressure, which were assessed before suctioning and 1 min, 5 mins, and 30 mins following suctioning). As can be seen in Table 1, there were no major changes in the hemodynamic variables assessed. Although minor elevations in occlusion pressure were occasionally observed, these were in the setting of elevated PEEP, and therefore the transmural left ventricular pressures were likely to be essentially unchanged.

DISCUSSION

The findings of this study are important for a number of reasons. First, we have observed highly variable elevations in carbon dioxide tension following suctioning in some animals, when analyzed individually, despite no significant increase in average CO_2 . In some cases, the PaCO_2 increased by as much as 40 mm Hg following brief suctioning. However, in other cases, the observed elevations in PaCO_2 following suctioning were rather trivial. Second, we found no consistent effect of the duration of suctioning on the subsequent elevations in PaCO_2 experienced by the animal. Thus, even 10 secs of suctioning was enough to cause substantial perturbations in gas exchange in some cases. Third, we found no consistent effect of baseline carbon dioxide values on the subsequent increase of PaCO_2 following suctioning. Thus, elevations in carbon dioxide tension were occasionally quite marked in animals with and without baseline hypercapnia. However, the trend toward significance would make us more cautious in the setting of baseline hypercapnia. Fourth, there were no

statistically significant hemodynamic effects of suctioning. Thus, decreases in cardiac output leading to increased pulmonary deadspace were not likely to be responsible for the carbon dioxide elevation.

Our results are consistent with prior literature in this area and extend the findings. Although substantial literature exists on the effects of suctioning and bronchoscopy on gas exchange, minimal data are available in the literature in the setting of permissive hypercapnia in ARDS. One could argue that low levels of minute ventilation would be more susceptible to carbon dioxide elevations if the mechanism of deterioration involved increases in deadspace. On the other hand, high levels of PCO_2 could have important bronchodilator properties that could minimize the impact of suctioning on air flow resistance and flow limitation. Thus, depending on the prevailing mechanics, the effect of base-line PCO_2 could be quite variable.

Our results demonstrate no consistent effect of baseline PCO_2 on the CO_2 elevation experienced after suctioning. However, because these elevations were highly variable, clinicians should be aware of the possibility of marked deterioration in gas exchange in some patients who undergo even brief suctioning. Since the degree of elevation is relatively unpredictable, measurement of an arterial blood gas may be necessary to quantify the degree of abnormality. Also of note, the most serious changes in blood gas tensions were within minutes of suctioning. Therefore, a blood gas would need to be drawn relatively soon after suctioning or bronchoscopy to quantify the degree of abnormality.

Another interpretation of our findings is that transient fluctuations in PCO_2 are relatively well tolerated. Both in the sheep in the present study and in our clinical experience, the transient fluctuations in gas exchange were unlikely to cause major clinical sequelae. However, in particular patients (e.g., patients with increased intracranial pressure), these gas exchange abnormalities may be more poorly tolerated.

A number of strategies have been suggested to minimize the potential impact of suctioning or bronchoscopy on gas exchange. A recent study (7) suggested that hyperoxygenation with 100% oxygen for a minimum of 1 min (or 20 breaths) should be the method of choice to avoid a decrease in PaO_2 following suctioning. In the same study, the authors showed an increase in heart rate, systolic blood pressure, and PaO_2 ; however, we found no significant hemodynamic adverse effects in the present study. In addition, we would be cautious about the potential for hyperoxia to worsen hypercapnia (8). Given our data, the application of PEEP may be a preferable method to avoid the oxygenation abnormalities induced by suctioning.

Our study had the following limitations. First, we did not quantify dead-space or regional pulmonary perfusion. Therefore, we can only speculate as to the mechanisms underlying the observed changes in gas exchange. However, we did quantify lung mechanics and hemodynamics, as summarized here. Second, because our study used saline lavage lung injury in sheep, our findings may not generalize to other clinical settings. Therefore, other forms of lung injury or ARDS in humans may behave somewhat differently. For example, an oleic acid injury may have substantial airway secretions, which could affect regional ventilation and thus could have different gas exchange properties. In addition, we protocolized mechanical ventilation using the ARDSNet PEEP/ FIO_2 criteria; therefore, findings may be different if other ventilator strategies were used. Third, we used open suctioning in the present study, whereas many investigators have advocated the routine use of closed suctioning to minimize gas exchange abnormalities. However, in a prior study using a similar model (5), we observed similar changes in PCO_2 in open vs. closed suctioning. Thus, we do not believe that the mode of suctioning had a major impact on our findings. Despite these limitations, we believe that our findings are accurate and that our

conclusions are relevant to the treating clinicians. Moreover, the mechanisms underlying our findings may be of interest to researchers in this field (7).

CONCLUSION

Open suctioning is generally well tolerated during lung-protective ventilation with or without permissive hypercapnia. Even brief procedures can occasionally lead to profound abnormalities in gas exchange. Increased PEEP may protect against desaturation induced by suctioning in ARDS.

Acknowledgments

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Figure 1.

Changes in PaCO₂ that occurred over time in the various groups. The values shown are for individual animals. *A*, PaCO₂ of 80 mm Hg with suction time of 10 secs; *B*, PaCO₂ of 80 mm Hg with suction time of 30 secs; *C*, PaCO₂ of 40 mm Hg with suction time of 10 secs; *D*, PaCO₂ of 40 mm Hg with suction time of 30 secs; conditions represented all seven animals. Note the individual variability in the PaCO₂ values for the animals in different experimental conditions.

Figure 2.

Percentage of PaO₂ change stratified by positive end-expiratory pressure (*PEEP*) levels. The animals were divided into three groups based on their baseline PEEP settings: >15 cm H₂O, 10–15 cm H₂O, and <10 cm H₂O. Animals with PEEP >15 cm H₂O recovered the PaO₂ 3 mins after suctioning, while animals with PEEP <10 cm H₂O did not recover the PaO₂ even 31 mins after the suctioning. The rapid recovery of PaO₂ for the highest PEEP group occurred despite the fact that these were the sickest animals, as PEEP was determined based on the ARDSNet criteria.

Table 1

Values for hemodynamic variables and lung mechanics during each situation of the protocol

	Baseline	1	5	30
80_10				
MAP, mm Hg	129 ± 12	130 ± 12	129 ± 10	135 ± 13
MPAP, mm Hg	33 ± 9	35 ± 8	35 ± 8	38 ± 9
PAOP, mm Hg	17 ± 4	17 ± 4	19 ± 5	23 ± 11
HR, bpm	141 ± 22	133 ± 26	125 ± 33	140 ± 18
CO, L/mL/m ²	5 ± 1	6 ± 1	6 ± 1	6 ± 1
Compl, L/cm H ₂ O	0.015 ± 0.004	0.011 ± 0.003	0.012 ± 0.002	0.014 ± 0.002
Peak pressure, cm H ₂ O	26 ± 1	30 ± 6	29 ± 6	28 ± 5
PEEP, cm H ₂ O	13 ± 4	13 ± 5	13 ± 5	13 ± 5
80_30				
MAP, mm Hg	121 ± 15	127 ± 14	128 ± 15	129 ± 10
MAP, mm Hg	17 ± 4	19 ± 6	18 ± 5	21 ± 6
PAOP, mm Hg	17 ± 4	19 ± 6	18 ± 5	21 ± 6
HR, bpm	128 ± 29	139 ± 30	137 ± 30	144 ± 23
CO, L/mL/m ²	5 ± 1	6 ± 1	6 ± 1	6 ± 1
Compl, L/cm H ₂ O	0.014 ± 0.003	0.012 ± 0.003	0.013 ± 0.003	0.012 ± 0.002
Peak pressure, cm H ₂ O	26 ± 5	30 ± 4	30 ± 4	28 ± 3
PEEP, cm H ₂ O	13 ± 5	13 ± 5	13 ± 5	13 ± 5
40_10				
MAP, mm Hg	115 ± 24	121 ± 23	121 ± 24	119 ± 24
MPAP, mm Hg	35 ± 6	33 ± 9	36 ± 8	37 ± 8
PAOP, mm Hg	20 ± 5	19 ± 5	19 ± 3	22 ± 7
HR, bpm	20 ± 5	19 ± 5	19 ± 3	22 ± 7
CO, L/mL/m ²	5 ± 2	5 ± 2	5 ± 2	5 ± 2
Compl, L/cm H ₂ O	0.023 ± 0.006	0.016 ± 0.003	0.019 ± 0.004	0.022 ± 0.005
Peak pressure, cm H ₂ O	26 ± 5	30 ± 4	30 ± 4	28 ± 3
PEEP, cm H ₂ O	13 ± 5	13 ± 4	13 ± 6	13 ± 5
40_30				
MAP, mm Hg	118 ± 32	120 ± 36	116 ± 31	112 ± 32
MPAP, mm Hg	34 ± 9	36 ± 7	35 ± 9	40 ± 6
PAOP, mm Hg	18 ± 3	18 ± 3	19 ± 4	21 ± 4
HR, bpm	18 ± 3	18 ± 3	19 ± 4	21 ± 4
CO, L/mL/m ²	4 ± 2	5 ± 2	5 ± 2	5 ± 2
Compl, L/cm H ₂ O	0.021 ± 0.006	0.017 ± 0.006	0.017 ± 0.006	0.019 ± 0.005
Peak pressure, cm H ₂ O	27 ± 4	30 ± 5	29 ± 5	30 ± 4
PEEP, cm H ₂ O	14 ± 4	13 ± 4	14 ± 5	13 ± 5

MAP, mean arterial pressure; MPAP, mean pulmonary artery pressure; PAOP, pulmonary artery occlusion pressure; HR, heart rate; bpm, beats per min; CO, cardiac output; Compl, compliance; PEEP, positive end-expiratory pressure; 80 mm Hg PaCO₂ 10 secs of suctioning (80_10); and 80

mm Hg, PaCO₂ 30 secs of suctioning (80_30); and 40 mm Hg PaCO₂ 10 secs of suctioning (40_10); 40 mm Hg PaCO₂ and 30 secs of suctioning (40_30).

Values are mean $\pm$ SD.

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