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ORIGINAL

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Closed system endotracheal suctioning maintains lung volume during volume-controlled mechanical ventilation

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Abstract *Objective*: A closed suction system (CS) maintains connection with the mechanical ventilator during tracheal suctioning and is claimed to limit loss in lung volume and oxygenation. We compared changes in lung volume, oxygenation, airway pressure and hemodynamics during endotracheal suctioning performed with CS and with an open suction system (OS).
Design: Prospective, randomized study.
Setting: Intensive care unit in a university hospital.
Patients: We enrolled ten patients, volume-controlled (VC) ventilated with a Siemens Servo 900 ventilator (PaO_2/FIO_2 192 ± 70, PEEP 10.7 ± 3.9 cmH_2O).
Interventions: We performed four consecutive tracheal suction maneuvers, two with CS and two with OS, at 20-min intervals. During the suction maneuvers continuous suction was applied for 20 s.
Measurements and main results: We measured end-expiratory lung volume changes (ΔV_L), tidal volume ($V_T$rt), respiratory rate (RR) and minute volume ($V_E$rt) by respiratory inductive plethysmography; arterial oxygen saturation (SpO_2), airway pressure and arterial pressure (PA). Loss in lung volume during OS (ΔV_L 1.2 ± 0.7 l) was significantly higher than during CS (ΔV_L 0.14 ± 0.1 l). During OS we observed a marked drop in SpO_2, while during CS the change was only minor. During CS ventilation was not interrupted and we observed an immediate increase in RR (due to the activation of the ventilator's trigger), while $V_T$rt decreased, $V_E$rt was maintained.
Conclusions: Avoiding suction-related lung volume loss can be helpful in patients with an increased tendency to alveolar collapse; CS allows suctioning while avoiding dramatic drops in lung volumes and seems to be safe during the VC ventilation setting that we used.

Key words Mechanical ventilation · Suction/instrumentation · Gas exchange · Alveolar recruitment

Introduction

Current clinical suggestions for the management of acute lung injury (ALI) emphasize the importance of optimizing alveolar recruitment and of maintaining lung volume [1]. Alveolar collapse seems to be harmful to the lung and should be carefully avoided by a targeted ventilation strategy including appropriate levels of positive end-expiratory pressure (PEEP) [2].

The intubated patient needs periodic suctioning of secretions due to the inability to clear the airway spontaneously [3]. The most commonly used technique entails the patient's disconnection from the ventilator and suctioning of the airways. Disconnection itself results in airway pressure drop and loss of lung volume, but a fur-

ther volume decrease is observed during suctioning [4] due to the generation of negative pressure in the airway. Therefore, open suctioning (OS) may lead to alveolar collapse and potentially hinders efforts aimed at maintaining lung volume. Hyperoxygenation and hyperinflation maneuvers are often used before and after OS to limit hypoxemia [5, 6, 7, 8] but do not directly prevent lung collapse.

Closed suctioning (CS) systems, with a catheter positioned between the endotracheal tube and the Y piece of the ventilator's circuit, can be used as an alternative to OS [9]. These systems allow non-interrupted ventilatory support during suctioning and have been shown to limit or avoid gas exchange impairment and hemodynamic disturbances due to the maneuver [10, 11]. However, direct evidence of lung volume preservation during CS has not been entirely demonstrated. Negative airway pressure generation with consequent lung volume loss is possible with CS if the flow output from the ventilator is lower than the suction flow. In bench tests and animal studies, large negative airway pressures was observed when CS was applied during volume-controlled (VC) ventilation [12, 13, 14].

The effects of CS on lung volume have not been investigated in mechanically ventilated humans. The aim of this study was to assess changes in lung volumes, airway pressures, arterial oxygenation and hemodynamics measured during CS, compared to OS, in VC ventilated ALI patients.

Methods

Patients

We recruited ten consecutive adult patients with ALI [15] undergoing controlled mechanical ventilation at PEEP higher or equal to 5 cmH_2O. Exclusion criteria were: bronchospasm or a positive history for chronic obstructive pulmonary disease (COPD), elevation in intracranial pressure and hemodynamic instability. Demographic and clinical data of the study population are shown in Table 1. As is routine for controlled ventilation in our department, all patients were VC ventilated, sedated by continuous infusion of propofol or fentanyl and paralyzed with hourly boluses of pancuronium bromide 0.06 mg/kg [16]. The ventilator used was a Servo 900C (Siemens Elema, Solna, Sweden) in all patients. The patient's ventilatory status, and type and size of the artificial airway are shown in Table 2.

Measurements

At the time of the study all patients already had an arterial catheter in place. Arterial blood gases were measured by an ABL 300 gas analyzer (Radiometer, Copenhagen, Denmark). The ECG tracing, obtained by bipolar leads, and the systemic arterial pressure signal were continuously recorded using a hemodynamic monitor (Kontron Systems, Watford, England). The arterial oxygen saturation (SpO_2) was monitored by pulse oximetry (Ohmeda, Louisville, Colo., USA). Airway pressure and flow were measured using the built-in pressure transducer and pneumotachometer of the ventilator. Pulmonary volume changes were measured by inductive plethysmography (NIMS, Miami Beach, Fla., USA). The analog output port of each monitor and transducer used was connected to a data acquisition system (Colligo, Elekton, Agliano Te, Italy) that allowed analog to digital conversion of signals sampled at 200 Hz. The system was implemented with an anti-aliasing low-pass filter. All tracings were calibrated and stored for subsequent off-line analysis. We assumed that the respiratory system of paralyzed patients has a single degree of freedom and therefore we measured lung volume changes only from the rib cage (RC) signal of the inductive plethysmograph [4]. The RC signal was calibrated, both for increasing and decreasing volume steps, using the V_T (tidal volume) obtained from the ventilator display as a reference value. The pneumotachometer of the ventilator was first calibrated with a water-sealed spirometer within an error of ± 5 % of the measured value.

Table 1 Age, diagnosis and ratio between arterial oxygen tension and inspired oxygen tension (PaO_2/FIO_2) of the patients included in the protocol (*ALI* acute lung injury, *ARDS* acute respiratory distress syndrome)

Patient	Age	Diagnosis	PaO2/ FIO_2	Days of intubation
1	66	Gastric inhalation	129	8
2	62	ALI, cardiac arrest	193	5
3	70	Pneumonia	266	13
4	73	Sepsis	69	4
5	65	Postoperative pneumonia	197	8
6	73	Sepsis	264	13
7	55	ARDS, lymphoma	224	17
8	52	ALI, cardiac arrest	121	6
9	20	ARDS	282	13
10	50	ARDS	170	12
mean	58.6		192	
SD	15.9		70.1	

Protocol

The protocol was approved by the local ethics committee in conformity with the Helsinki declaration on human research. Informed consent was obtained from the patient's next of kin. After a patient's recruitment, we set an inspiratory time of 25 % on the ventilator (with the exception of one patient, for whom it was 33 %), an inspiratory plateau time of 10 % and a trigger sensitivity of –2 cmH_2O, keeping all remaining ventilatory settings unchanged. We then connected all patients to a commercially available CS system (Mallinckrodt Medical, Mirandola, Italy) provided with a 12 Fr-sized suction catheter. The CS system was positioned between the endotracheal tube and the Y piece of the ventilator's circuit and was left in place throughout the study. The suction catheter was initially in the locked position while the water irrigation port was kept closed all the time.

After an adaptation period of 20 min, we performed both a CS and a OS maneuver twice, in an alternate randomized (CS-OS or OS-CS) sequence for a total of four steps. We performed each suctioning maneuver after a time interval of 20 min or longer from the previous one, to achieve stability in SpO_2 values. During each step measurements and recordings were performed at three different times: VC ventilation before suctioning (VCpre, Table 3); suction-

Table 2 Ventilatory status of the patients (*RR* respiratory rate, *VTi* inspiratory tidal volume *VE* minute ventilation, *PEEP* positive end-expiratory pressure, *PIP* peak inspiratory pressure, *Plateau* inspiratory plateau pressure, *Paw* mean airways pressure, *Ti %* inspiratory time %, *Tube* internal diameter, *O/T* oro-tracheal, *TLT* translaryngeal tracheostomy)

Patient No.	RR	VTi/kg	VE (l)	PEEP (cmH_2O)	PIP (cmH_2O)	Plateau (cmH_2O)	Paw (cmH_2O)	Ti %	Tube mm
1	15	10.0	11.5	12	30.1	26.1	18.6	33	O/T 7.5
2	13	9.9	8.3	10	26.5	23.2	14.2	25	TLT 8
3	12	8.0	8.6	15	37.4	35.2	21.9	25	TLT 7.5
4	10	6.4	6	6	23	20.2	10	25	O/T 7.5
5	17	8.8	7.8	6	36.8	30.1	13.6	25	O/T 7.5
6	17	8.7	9.3	10	33.5	25.8	15.6	25	O/T 7
7	16	7.0	9.2	13	42.3	36	21	25	O/T 7.5
8	12	9.3	8.1	5	25.4	20	11	25	O/T 8
9	14	10.0	8.8	15	40	35.1	21	25	TLT 8.5
10	26	10.4	14.6	15	46.3	44.6	20.9	25	TLT 8
Mean	15.1	8.8	9.2	10.7	34.1	29.6	16.8		
SD	4.5	1.3	2.3	3.9	7.8	8.0	4.5		

Table 3 Lung volumes measured by respiratory inductive plethysmography, arterial oxygen saturation, airway pressures, arterial blood gases, heart rate and arterial pressure, obtained during closed system *(CS)* and open system *(OS)* suction (*VCpre* volume-controlled ventilation before suctioning, *VCpost* volume-controlled ventilation after suctioning, ΔVL drop in lung volume, SpO_2 pulse oximetry, *VTrt* tidal volume obtained by respitrace, *VErt* minute ventilation obtained by respitrace, *PIP* peak inspiratory pressure, *PEEP* positive end-expiratory pressure, *Plateau* inspiratory plateau pressure, *Paw* mean airway pressure, *RR* respiratory rate, *MAP* mean arterial pressure, *HR* heart rate, PaO_2 arterial oxygen tension, $PaCO_2$ arterial carbon dioxide tension, HbO_2 hemoglobin oxygen saturation)

	CS			OS		
	VCpre	Suction	VCpost	VCpre	Suction	VCpost
DVL (ml)[a]	/	–133.2 ± 129.9	/	/	–1231.5 ± 858.3	/
SpO_2%[b,c,d]	97.4 ± 2.9	97.2 ± 2.9	97.6 ± 3.0	97.7 ± 3.0	94.6 ± 5.1	97.0 ± 3.8
VTrt (ml)[b,c,d,e]	617.1 ± 78.3	228.4 ± 86.3	618.5 ± 88.6	630.4 ± 71.0	/	636 ± 82.4
VErt (l/min)[b,c,d]	9.1 ± 2.6	9.0 ± 3.8	9.1 ± 2.5	9.4 ± 2.6	/	9.4 ± 2.5
PIP (cmH_2O)[b,c,d,e]	32.5 ± 9.1	26.1 ± 9.6	32.2 ± 8.5	32.3 ± 8.2	/	32.2 ± 8.4
PEEP (cmH_2O)[b,c,d,e]	10.2 ± 4.3	7.9 ± 4.4	10.2 ± 4.3	10.2 ± 4.4	/	10.1 ± 4.4
Plateau (cmH_2O)[b,c,d]	27.4 ± 8.4	/	27.4 ± 8.3	27.5 ± 8.2	/	27.5 ± 8.1
Paw (cmH_2O)[b,c,d,e]	15.9 ± 5.1	18.0 ± 5.5	15.9 ± 5.1	16 ± 5.1	/	15.9 ± 5.1
RR[b,c,d,e]	15.1 ± 4.5	39.8 ± 6.6	15.1 ± 5.4	15.1 ± 4.3	/	15.1 ± 4.3
MAP(mmHg)[b,c,d,f,g]	79.4 ± 11.7	81.2 ± 11.9	80.0 ± 11.6	78.1 ± 10.2	83.2 ± 14.7	84.5 ± 13.6
HR	97.1 ± 21.9	100.2 ± 20.0	97.6 ± 21.3	98.7 ± 22.3	97.5 ± 21.4	98.1 ± 22.5
PaO_2 (mmHg)	123.5 ± 26.1	/	123.2 ± 25.7	122.6 ± 26.0	/	117.3 ± 31.1
$PaCO_2$ (mmHg)	48.1 ± 14.3	/	47.9 ± 14.0	47.4 ± 14.0	/	49.2 ± 14.3
HbO_2 (%)	95.6 ± 2.6	/	95.7 ± 2.6	96.6 ± 2.7	/	95.2 ± 3.3

[a] $p < 0.01$ versus suction OS
[b] $p < 0.05$ CS versus OS
[c] $p < 0.05$ suction versus (VCpre + VCpost)
[d] Interaction b*c: $p < 0.05$ the effect of suctioning is different with the two systems
[e] $p < 0.05$ suction CS versus VC (preCS + postCS)
[f] $p < 0.05$ VCpre versus VCpost
[g] Interaction b*f: $p < 0.05$ the difference between VCpre and VCpost is affected by the system

ing (suction) and VC ventilation after suctioning (VCpost). At the beginning of each step, we withdrew a first arterial blood sample (VCpre sample, Table 3), following which we started continuous data acquisition. After 30 s of recording, we started the suctioning procedure.

With the OS, the endotracheal tube was disconnected from the ventilator, a 12 Fr suction catheter (Medicoplast, Illingen, Germany) was inserted in the endotracheal tube and advanced until resistance was met, then it was withdrawn 2–3 cm. Negative pressure was then applied continuously for 20 s, during which time the catheter was gently rotated. Then, suctioning was stopped, the catheter was withdrawn and the patient was reconnected to the ventilator.

With the CS system, the suction catheter was unlocked and inserted in the endotracheal tube while maintaining connection with the ventilator. Similarly to OS, the catheter was advanced and vacuum was continuously applied for 20 s. Then suction was stopped, the catheter was withdrawn and locked. With both OS and CS, data recording was continued for a further 2 min following the end of suctioning, then a second arterial blood gas sample was collected (VCpost sample, Table 3). In all patients we applied negative pressure of –100 mmHg [5] with both systems. No hyperoxygenation or hyperinflation maneuvers were performed before or after suctioning.

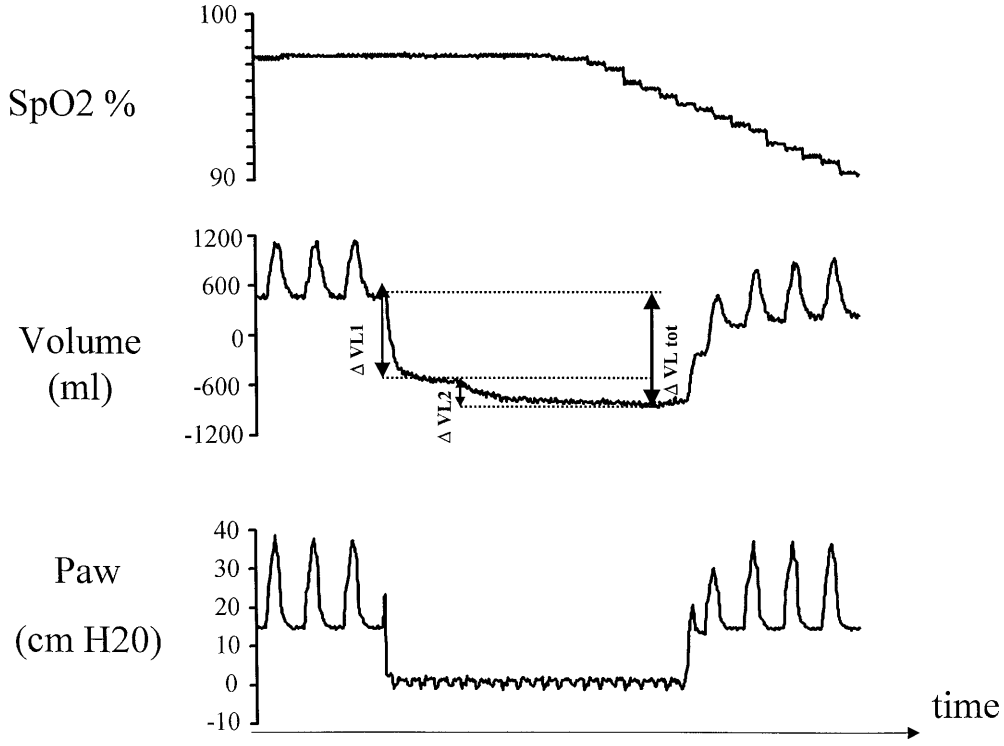

Fig. 1 Representative recordings of pulse oximetry (SpO_2), lung volume (VT) and airway pressure (Paw) obtained from single patient during open suctioning. During open suctioning we observed a marked decrease in SpO_2 and Paw; an immediate drop in lung volume (ΔVL_1) is followed by an additional decrease after the start of suctioning (ΔVL_2). The total drop in lung volume is computed as $\Delta VL_1+\Delta VL_2$

Data analysis

All recordings were analyzed using a dedicated software (Computo, Elekton). For each step, we analyzed the last three consecutive breaths recorded before the beginning of the endotracheal suctioning maneuver (VCpre, Table 3) and the last three consecutive breaths recorded in the 2 min following the end of suctioning (VCpost, Table 3). From each breath, we obtained the peak inspiratory pressure (PIP), the inspiratory plateau pressure (Ppl), the mean airway pressure (Paw), the end-expiratory pressure (PEEP) and the respiratory rate (RR). RR was obtained as 60 s divided by the length in seconds of a respiratory cycle, from the start of one inspiration to the start of the next one. We obtained VT as the difference between the plethysmographic RC signal value at end inspiration and at end expiration (VTrt) and the minute ventilation (VErt) as VTrt*RR. We obtained heart rate (HR) from the ECG signal and mean arterial pressure (MAP) as the average arterial pressure signal during the time corresponding to the three breaths analyzed. SpO_2 was measured as the average pulse oximetry during the same three breaths.

We then analyzed the tracings obtained during the suctioning maneuver (suction, Table 3). With the OS we identified, on the RC signal (Fig. 1), a first drop in lung volume immediately following a patient's disconnection from the ventilator (ΔVL_1), followed by a second drop after the start of suctioning (ΔVL_2). We measured ΔVL_1 as the difference in RC signal between the end-expiratory value of the last breath before disconnection and the value immediately before the start of suctioning. We measured ΔVL_2 as the change in RC signal from the start to the end of suctioning. The total change in lung volume due to the suctioning maneuver (ΔVL_t) was computed as $\Delta VL_1+\Delta VL_2$. With OS, we could not measure values of airway pressure and flow, due to the patient's disconnection from the ventilator circuit. During CS, ventilator assistance was also continued during suctioning. We therefore analyzed the last three consecutive breaths at the end of the suctioning period and obtained VTrt, PIP, Paw and PEEP. We measured ΔVL_t as the difference in RC signal between the end-expiratory value of the last breath before insertion of the suction catheter and the last breath at the end of the suctioning period.

With both systems, MAP and HR were obtained from the analysis of the last 5 s of suctioning. We recorded the minimum SpO_2 value reached following the start of the suctioning maneuver and the time when this value was reached (suction, Table 3). In Table 3 data are presented as the mean of the two CS maneuvers and the mean of the two OS maneuvers, thus organized in a six-column table.

With regard to statistical analysis, all data are expressed as means ± standard deviation. Drop in lung volumes (ΔVL) were compared using a paired *t*-test analysis (Table 3). Data for which measurements were taken during all of the six times of the study (Table 3) were processed using an ANOVA two-way analysis with five orthogonal contrasts, in a factorial experimental design with two systems (CS; OS) and three levels per system (VCpre; suction; VCpost) [17]. This analysis was designed to compare: (a) the two systems, (b) VC ventilation versus suction (VCpre+VCpost vs suction) and (c) VCpre versus VCpost. The study of the interactions assessed: (d) whether the effect of suction was different between the two systems and (e) whether the difference between pre- and post-suction was affected by the system used.

Measurements of VT, VE and airway pressures were available during five times of the study and were first processed with a general ANOVA two-way analysis, then data obtained during CS were analyzed by two orthogonal contrasts; in this way we could assess whether VCpre and VCpost values were different and whether CS suction was different from VC (preCS + postCS).

Blood gas values (Table 3) were processed using ANOVA two-way analysis, in a factorial, experimental design with two systems (CS; OS) and two levels per system (VCpre; VCpost), with three

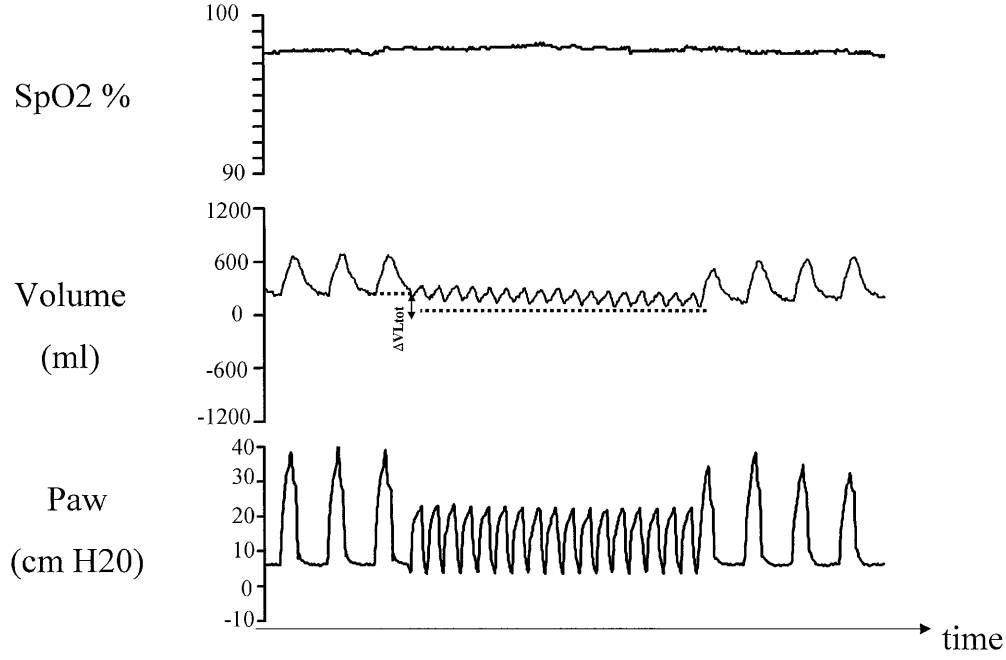

Fig. 2 Representative recordings of pulse oximetry (SpO_2), lung volume (VT) and airway pressure (Paw) obtained from a single patient during closed suctioning. During closed suctioning loss in VT, Paw and SpO_2 is only minor; during CS suctioning the ventilator's trigger is activated and respiratory rate (RR) is increased

orthogonal contrasts. We compared the two systems, VCpre versus VCpost, and the interaction assessed whether the difference between pre- and post-suction was affected by the system used [17]. A p value less than 0.05 was considered significant.

Results

Figures 1 and 2 show tracings of airway pressure, SpO_2 and lung volume obtained in a representative patient during endotracheal suctioning. During OS, an immediate drop in lung volume (ΔV_{L_1}) due to disconnection from the ventilator was followed by an additional decrease in volume (ΔV_{L_2}) when suctioning was started (Fig. 1). During CS we observed only a relatively slight decrease in the end-expiratory lung volume (Fig. 2) and $\Delta V_{L_{tot}}$ was significantly smaller than values measured during OS (Table 3).

No significant difference in SpO_2 was detected between VCpre and VCpost; VCpre and VCpost values were not affected by the two different aspiration systems. Table 3 shows a marked drop in SpO_2 during OS suctioning, while the change in SpO_2 during CS is only minor: the interaction analysis showed, indeed, that the effect of suctioning during OS is significantly different than that during CS. Figure 1 shows how, during OS, SpO_2 started to decrease and continued to decrease after reconnection to the ventilator. The minimum value was reached at an average time of 77 ± 29 s from the beginning of the maneuver. In seven patients SpO_2 returned to the baseline value within 120 s. In three of the five patients in whom PEEP was higher than 10 cmH_2O, SpO_2 did not return to baseline within the 120 s recording time. The SpO2 signal was stable during the whole CS procedure (Fig. 2). In the 2 min after OS we observed a significant increase in MAP (Table 3) but no significant changes in HR. We observed no significant change in MAP or in HR during and after CS. Blood gas values collected before and after OS and CS showed no statistically significant differences (Table 3).

In conclusion, with OS we observed: (1) a major drop in lung volume during suction compared to suction with CS, (2) compared to VC values, SpO_2 started to decrease significantly during suction and returned to baseline before the end of the recording and (3) an increase in MAP after suction. During CS, mechanical ventilation was not interrupted during suctioning (Fig. 2). Compared to VC during CS suctioning we observed (Table 3): an immediate increase in RR and a decrease in VTrt; inspiratory flow and time were unchanged, but expiratory time was decreased with a reduction or a total disappearance of expiratory flow (Figure 1B). PEEP and PIP were smaller, while Paw increased.

Discussion

We compared OS and CS in VC ventilated ALI patients and obtained the following main results: CS allowed the maintenance of lung volumes, of ventilation and of positive airway pressure during suctioning. Compared to OS, CS avoided decreases in SpO_2 and changes in MAP.

Effects of open suctioning

In our patients, OS resulted in substantial lung volume loss, compatible with the fact that PEEP was applied in all patients. However, a further volume decrease occurred during the application of negative pressure, due to the creation of negative pressure in the airways [18]. Possibly lung volumes below functional residual capacity were reached. Similar volume losses have been observed by Brochard et al. with thoracic CT scan analysis in patients undergoing OS [4]. In our study the lung volume decrease during suctioning might have been increased by the continuous application of negative pressure for a relatively long time. However, stability of the respitrace RC signal was reached before the end of the suctioning maneuver in all of our patients (Fig. 1) and it was already stable from 15 s after the start of suctioning. Limitation of the suctioning time to shorter than 15 s and intermittent aspiration are usually recommended to limit arterial oxygen desaturation [3, 5] and mucosal trauma [19]. We chose to apply negative pressure continuously for 20 s to verify the ability of CS to maintain the lung volume stable, even during sustained aspiration, and to compare its effects to OS in a similar time period.

The effects of OS on oxygenation were similar to data reported in other studies [4, 11]. Hypoxemia during OS has been ascribed to interruption of ventilation, loss of positive airway pressure, decrease in lung volume, atelectasis formation and reduction of alveolar oxygen concentration [3]. Noticeably, all patients in whom the drop in SpO_2 persisted more than 120 s were ventilated with a PEEP higher than 10 cmH_2O [20]. SpO_2 did not reach life threatening values in any of our patients, although we did not try to hyperoxygenate or to hyperventilate our patients before or after the OS maneuver. Increases in mean systemic arterial pressure due to OS have already been described and could result from hypoxemia, airway manipulation and tracheal stimulation [10].

Effects of closed suctioning

Closed suctioning is intended to preserve airway pressures, ventilation and FIO_2 during suctioning [3] and is therefore used in patients with severe gas exchange impairment. The stability of SpO_2 during CS in our study is not surprising and is in line with other authors' results [11]. However, several aspects of patient-ventilator interaction during CS application are not entirely clear. When the ventilator cannot replace the amount of gas suctioned by the CS system, the potential arises for reduced or negative pressures in the airways [11, 12]. In certain conditions, the use of a CS system may not adequately prevent, or may even enhance, the loss in lung volume during tracheal suctioning [21].

Ventilator type and setup seem to affect the response to CS. Largely negative airway pressures have been observed with volume-control ventilatory modes [14] and with relatively low inspiratory flow settings [12]. In a lung simulator study, CS applied during VC ventilation with a Servo 900C ventilator resulted in more negative airway pressures compared to pressure-controlled ventilation and to the use of a Siemens 300 [13].

We designed this study with the aim of assessing the safety of CS and its efficacy in maintaining lung volumes stable with settings commonly used in our clinical practice. In addition, we decided to set the trigger sensitivity at –2 cmH_2O to allow ventilator triggered-cycling during the application of negative pressure. With this setup, the ventilator responded to gas aspiration by triggered-cycling and by increasing RR. Breaths delivered by the ventilator had unchanged inspiratory flow and time but reduced expiratory time, compared to VC ventilation. The resulting increase in total delivered volume was adequate to compensate for the gas withdrawn from the ventilator circuit by the CS system. In fact, we observed only a slight decrease of end-expiratory lung volume and airway pressure, and we did not detect a negative airway pressure in any of our patients during CS. V_T measured by plethysmography was lower than during VC ventilation, due to aspiration of most of the gas delivered by the ventilator. However, the higher RR during CS resulted in a comparable V_E.

The higher RR was associated with an increase in Paw. An alternative explanation for the increased Paw could be the possible development of dynamic hyperinflation and intrinsic PEEP; we may exclude this mechanism because no increase in lung volume was detected by the plethysmograph [22]. However, patients with a positive history for bronchospasm or COPD were excluded from our study. The possible development of intrinsic PEEP can therefore not be excluded under these conditions.

Other ventilator settings might result in different findings. In particular, a lower trigger sensitivity or trigger inactivation might lead to highly negative airway pressure during CS [12]. A higher trigger sensitivity may allow an exaggerated self-cycling response by the ventilator. A lower inspiratory flow might make compensation of suctioned gas more difficult and cause lung volume loss. We used a relatively small size of catheter and a vacuum pressure of –100 mmHg based on clinical guidelines [5]. In clinical practice more negative pressure and a bigger catheter size (14 Fr) are often used to obtain higher aspiration flows and a satisfactory secretion removal; these may lead to lower airway pressures and significant volume decreases during suctioning [18].

Clinical implications

Various techniques have been introduced to prevent oxygenation impairment during suctioning or to speed the return to optimal values. The most commonly used methods include administration of a high FIO_2 and of high VTs before and after the suction maneuver [3, 5]. These techniques are probably adequate to avoid dangerous drops in oxygenation and make CS unnecessary in most patients. However, hyperinflation and hyperoxygenation do not preserve lung volumes during suctioning. There has been increasing attention towards ventilatory strategies aimed at optimizing alveolar recruitment [1]. In this context, avoiding suction-related lung volume loss can be helpful in patients with an increased tendency to alveolar collapse.

Constant-flow oxygen insufflation has been shown to be effective in preserving lung volume during suctioning [4], but the technique requires the use of specially designed endotracheal tubes. CS using commercially available systems allows suctioning while avoiding dramatic drops in airway pressure and lung volumes and seems to be safe during VC with the ventilatory strategy that we used.

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