RESPIRATORY CARE Paper in Press. Published on July 15, 2014 as DOI: 10.4187/respcare.02892

Maintenance of Airway Pressure During Filter Exchange Due to Auto-Triggering

Joakim Engström MSc CCRN, Henrik Reinius MD, Camilla Fröjd PhD CCRN, Hans Jonsson MSc CCRN, Göran Hedenstierna MD PhD, and Anders Larsson MD PhD

BACKGROUND: Daily routine ventilator-filter exchange interrupts the integrity of the ventilator circuit. We hypothesized that this might reduce positive airway pressure in mechanically ventilated ICU patients, inducing alveolar collapse and causing impaired oxygenation and compliance of the respiratory system. METHODS: We studied 40 consecutive ICU subjects (P_{aO_2}/F_{IO_2} ratio ≤ 300 mm Hg), mechanically ventilated with pressure-regulated volume control or pressure support and PEEP ≥ 5 cm H_2O. Before the filter exchange, (baseline) tidal volume, breathing frequency, end-inspiratory plateau pressure, and PEEP were recorded. Compliance of the respiratory system was calculated; F_{IO_2}, blood pressure, and pulse rate were registered; and P_{aO_2}, P_{aCO_2}, pH, and base excess were measured. Measurements were repeated 15 and 60 min after the filter exchange. In addition, a bench test was performed with a precision test lung with similar compliance and resistance as in the clinical study. RESULTS: The exchange of the filter took 3.5 ± 1.2 s (mean ± SD). There was no significant change in P_{aO_2} (89 ± 16 mm Hg at baseline vs 86 ± 16 mm Hg at 15 min and 88 ± 18 mm Hg at 60 min, *P* = .24) or in compliance of the respiratory system (41 ± 11 mL/cm H_2O at baseline vs 40 ± 12 mL/cm H_2O at 15 min and 40 ± 12 mL/cm H_2O at 60 min, *P* = .32). The bench study showed that auto-triggering by the ventilator when disconnecting from the expiratory circuit kept the tracheal pressure above PEEP for at least 3 s with pressure controlled ventilation. CONCLUSIONS: This study showed that a short disconnection of the expiratory ventilator circuit from the ventilator during filter exchange was not associated with any significant deterioration in lung function 15 and 60 min later. This result may be explained by auto-triggering of the ventilator with high inspiratory flows during the filter exchange, maintaining airway pressure. (ISRCTN.org registration ISRCTN76631800.) *Key words: acute lung injury; positive-pressure respiration; positive end-expiratory pressure; air filters; intensive care units.* [Respir Care 2014;59(8):1210–1217. © 2014 Daedalus Enterprises]

Introduction

PEEP is used to prevent alveolar de-recruitment and maintain oxygenation in mechanically ventilated patients with lungs prone to atelectasis, eg, in ARDS.[1,2] It has been found in experimental ARDS models that lung collapse occurs within seconds after discontinuation of positive airway pressure.[3] In fact, Neumann et al[3] showed that the time constant for development of a major collapse was 0.6 s. It is well known that oxygenation deteriorates when the endotracheal tube is disconnected from the ventilator circuit in mechanically ventilated ICU patients, particu-

Mr Engström, Dr Reinius, Dr Fröjd, Mr Jonsson, and Dr Larsson are affiliated with Anesthesiology and Intensive Care, Department of Surgical Sciences, Uppsala University; Dr Hedenstierna is affiliated with the Department of Medical Sciences, Uppsala University, Uppsala, Sweden.

This research was supported by the Swedish Heart-Lung Foundation, by Swedish Research Council Uppsala county grant 5315, and by an institutional grant from the Department of Surgical Sciences, Anesthesiology and Intensive Care, Uppsala University. The authors have disclosed no conflicts of interest.

Correspondence: Joakim Engström MSc CCRN, Department of Surgical Sciences, Uppsala University, SE-751 85 Uppsala, Sweden. E-mail: joakim.engstrom@surgsci.uu.se.

DOI: 10.4187/respcare.02892

RESPIRATORY CARE Paper in Press. Published on July 15, 2014 as DOI: 10.4187/respcare.02892

larly in connection with endotracheal suctioning.[4-6] Moreover, the sole removal of positive airway pressure causes a marked decrease in lung volume.[5,7] In our ICU, we routinely exchange ventilator filters every 24 h for hygienic reasons, in accordance with the recommendations by the manufacturer.[8] These filters are placed between the expiratory limb of the ventilatory tubing and the ventilator. Because this procedure breaks the integrity of the ventilatory circuit, we hypothesized that this could compromise lung function, something that is not recognized or discussed either in the clinic or in studies of different ventilatory strategies. Therefore, the aim of this study was to assess whether the daily routine exchange of ventilator filters would lead to deterioration of oxygenation or compliance of the respiratory system in mechanically ventilated ICU patients. To further explore the mechanisms, we assessed the airway pressure change proximal to the tip of the endotracheal tube in a bench test after a simulated filter exchange.

QUICK LOOK

Current knowledge

Breathing circuit filters in the expiratory limb of the ventilator circuit protect ventilator components from moisture and contamination. Daily routine changes of breathing circuit filters are recommended to prevent increased expiratory resistance and untoward events. Changing the filter requires breaking the circuit, loss of airway pressure, and the potential for lung de-recruitment.

What this paper contributes to our knowledge

In a group of mechanically ventilated patients with hypoxemia, a short disconnection of the expiratory ventilator circuit during filter exchange was not associated with any significant deterioration in lung function. This may be explained by auto-triggering of the ventilator with high inspiratory flow, maintaining airway pressure.

Methods

The study was divided into 2 parts: (1) a clinical study in 40 mechanically ventilated subjects (Fig. 1) and (2) a bench test using different ventilatory modes to estimate the pressure change distal to the endotracheal tube at a simulated ventilator filter exchange (Fig. 2).

The Clinical Study

The study was performed in Anesthesiology and Intensive Care, Department of Surgical Sciences, Uppsala University, Uppsala, Sweden. The study was approved by the university ethics committee (ISRCTN.org registration ISRCTN76631800). Informed consent was obtained from the subjects' next of kin before inclusion.

Mechanically ventilated subjects were included consecutively if: P_{aO_2}/F_{IO_2} ratio was $\leq$ 300 mm Hg, PEEP was $\geq$ 5 cm H_2O, patient had an arterial cannula, patient was $\geq$ 18 y, and patient was not pregnant.

Protocol

The subjects were mechanically ventilated with pressure-regulated volume control (PRVC), pressure controlled ventilation, or pressure support ventilation using a Servo-i ventilator (Maquet, Wayne, New Jersey). Flow triggering was used and set at 1 L/min in all subjects. The inspiratory rise time was set at 5%. The ventilator tubing circuit set (A4VXXXXX, Vital Signs, Totowa, New Jersey) had an inner diameter of 22 mm and was 275 cm in length (137.5 cm inspiratory and 137.5 cm expiratory limb). The size of the endotracheal tube (ETT) (Portex Blue Line Sacett, Smiths Medical, Hythe, Kent, United Kingdom) or tracheostomy tube (Shiley Evac tracheostomy tube cuffed system, Covidien, Mansfield, Ohio) was recorded, as well as whether a heat-moisture exchanger (HME, Pharma Systems, Knivsta, Sweden) or an active humidifier (RT430, Fisher & Paykel Healthcare, Auckland, New Zealand) was used.

Before the exchange of the high-efficiency particulate air filter (Servo Duo Gard, Maquet), placed between the expiratory limb of the ventilatory circuit and the ventilator, tidal volume, breathing frequency, end-inspiratory plateau pressure (EIP), and PEEP were recorded (baseline). In the subjects with controlled ventilation without any subject-triggered breaths (n = 32), compliance was calculated as tidal volume/(EIP − PEEP). Both EIP and PEEP were measured after a prolonged pause of 10 s. F_{IO_2}, arterial blood pressure, and pulse rate were recorded, and arterial blood was sampled for determination of P_{aO_2}, P_{aCO_2}, pH, and base excess (ABL800 Flex, Radiometer, Brondby, Denmark).

The subject remained connected to the ventilator during the whole filter exchange procedure. The expiratory tubing was disconnected from the old filter, which was then removed from the ventilator inlet and exchanged, and the expiratory tubing was reconnected to the new filter. Measurements were repeated 15 and 60 min after the filter exchange. In addition, the duration of the exchange procedure was recorded. Finally, in 4 subjects, airway pressure (P_{aw}) was measured in the Y-piece connected to the ETT and 1 cm below the ETT tip via a 15 cm, 16 gauge catheter (Arrow, Limerick, Pennsylvania). Endotracheal

RESPIRATORY CARE Paper in Press. Published on July 15, 2014 as DOI: 10.4187/respcare.02892

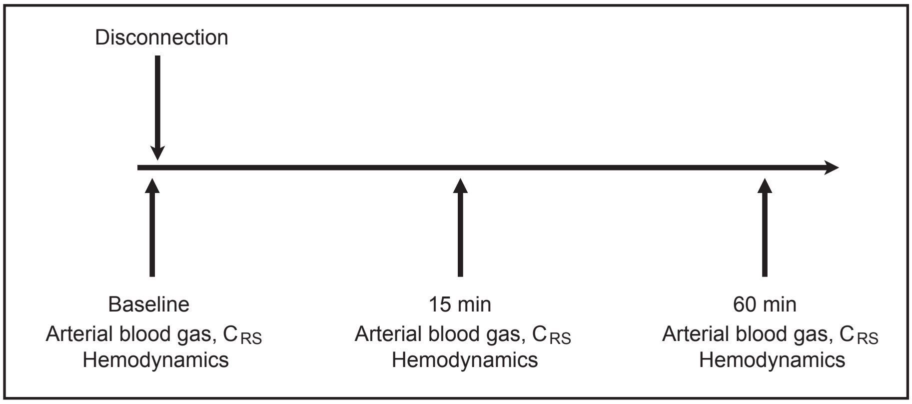

Fig. 1. Outline of the study. The arrows above the horizontal line indicate interventions, whereas the arrows below the line indicate measurements. C_{RS} = compliance of the respiratory system.

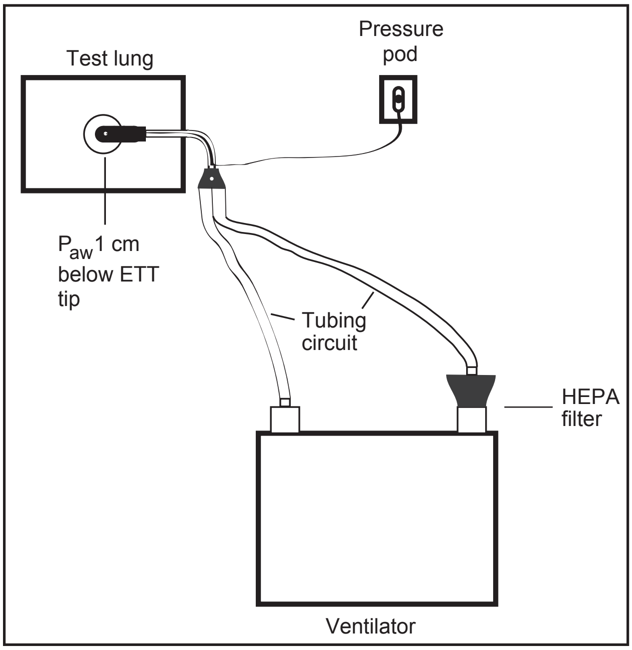

Fig. 2. Experimental setup of the bench test. The high-efficiency particulate air filter was placed in the expiratory limb of ventilator. P_{aw} = airway pressure. ETT = endotracheal tube.

disconnection and suctioning were not performed in any subject for at least 4 h before study inclusion. No changes were made in subject position or ventilator settings during the study protocol.

Bench Test

The "tracheal" airway pressure decrease was measured in a lung model (Accu Lung precision test lung, Fluke Biomedical, Everett, Washington).

The test lung was set at compliance values 10 or 20 mL/cm H_2O, resistance 5 cm H_2O/L/s (the resistance setting was chosen to avoid inadvertent auto-PEEP), and was connected through an inner diameter 6 or 8 mm ETT (Portex Blue Line Sacett, Smiths Medical International)

Table 1. Subject Characteristics

Subject Characteristics (*N* = 40)	Values
Age (y)	64 ± 15
Female sex (*n*, %)	12 (30)
SAPS 3	67 ± 14
Duration of mechanical ventilation (d)	8.6 ± 9.9
ICU stay (d)	10 ± 11
Hospital stay (d)	31 ± 38
ICU mortality (*n*, %)	5 (13)
30-d mortality (*n*, %)	9 (23)
ARDS	
Mild (*n*, %)	13 (32.5)
Moderate (*n*, %)	25 (62.5)
Severe (*n*, %)	2 (5)
Mechanical ventilation settings	
Tidal volume (mL/kg)	7.2 ± 1.6
Breathing frequency (breaths/min)	12 ± 5
F_{IO_2}	0.5 ± 0.1
EIP (cm H_2O)	24 ± 5
PEEP (cm H_2O)	12 ± 4
Gas exchange	
Arterial pH	7.39 ± 0.07
P_{aCO_2} (mm Hg)	45 ± 14
P_{aO_2} (mm Hg)	89 ± 16
C_{RS} (mL/cm H_2O)	41 ± 11
BE (mmol/L)	1.0 ± 4.9
Circulatory parameters	
Mean arterial pressure (mm Hg)	77 ± 14
Pulse rate (beats/min)	88 ± 23

Values are mean ± SD unless otherwise specified.
SAPS 3 = simplified acute physiology score 3
EIP = end-inspiratory plateau pressure
C_{RS} = compliance of the respiratory system
BE = base excess

RESPIRATORY CARE Paper in Press. Published on July 15, 2014 as DOI: 10.4187/respcare.02892

Table 2. Individual ARDS, P_{aO_2}/F_{IO_2}, PEEP, P_{aO_2}, and C_{RS} Before the High-Efficiency Particulate Air Filter Change and 15 and 60 Min Afterward

Subject No.	ARDS*	P_{aO_2}/F_{IO_2} (mm Hg)	PEEP (cm H_2O)	P_{aO_2} (mm Hg)			C_{RS} (mL/cm H_2O)		
				Baseline	15 min	60 min	Baseline	15 min	60 min
1	2	156	17	94	95	80	53	53	60
2	3	231	15	116	116	95	40	37	37
3	3	200	12	120	83	81	NA†	NA	NA
4	3	222	11	122	106	96	37	34	37
5	1	82	13	66	56	57	48	44	44
6	2	194	16	107	121	145	40	30	35
7	2	131	17	72	62	61	38	30	28
8	2	122	17	73	71	70	22	27	26
9	2	108	13	76	74	75	43	42	43
10	2	166	17	92	81	85	43	45	45
11	2	142	17	71	67	69	25	25	22
12	1	89	12	67	64	73	53	50	55
13	2	113	16	79	79	86	33	29	30
14	2	139	12	83	80	85	35	35	42
15	2	155	16	93	92	87	40	30	35
16	3	231	19	116	116	113	47	43	43
17	2	126	14	95	95	97	59	58	59
18	2	164	11	90	98	114	60	60	60
19	2	156	10	78	89	98	26	27	26
20	2	158	5	79	71	69	15	16	17
21	3	207	5	93	83	87	50	50	49
22	2	197	8	98	93	92	NA	NA	NA
23	3	208	10	73	70	68	NA	NA	NA
24	3	218	10	98	102	107	NA	NA	NA
25	2	186	5	93	85	86	NA	NA	NA
26	2	189	12	76	72	77	NA	NA	NA
27	2	143	13	93	85	86	37	34	35
28	2	153	12	69	67	65	NA	NA	NA
29	2	134	8	80	83	78	41	41	41
30	3	257	18	116	116	107	26	26	28
31	2	191	15	105	108	123	30	20	30
32	3	230	12	69	78	80	NA	NA	NA
33	3	237	8	107	103	105	52	52	NA
34	2	185	11	74	76	80	44	64	54
35	2	162	15	89	95	104	40	38	40
36	2	133	9	86	87	77	46	45	38
37	3	229	6	92	83	83	NA	NA	NA
38	3	219	9	77	72	69	52	53	54
39	3	289	5	101	85	96	50	49	49
40	2	198	7	89	95	98	45	46	47
Mean ± SD		176 ± 47	12 ± 4	89 ± 16	86 ± 16	88 ± 18	41 ± 11	40 ± 12	40 ± 12

* ARDS severity is divided in three classes: (1) severe, (2) moderate, and (3) mild.
† NA = missing value due to spontaneous breathing
C_{RS} = compliance of the respiratory system

and a 275-cm, inner diameter 22 mm tubing circuit (A4VXXXXX, Vital Signs, the same as used in the clinic) to a Servo-i ventilator (Maquet) set at either pressure controlled ventilation (EIP 25 cm H_2O, 10 cm H_2O PEEP, or volume controlled ventilation with the same EIP and PEEP as during pressure controlled ventilation. The ratio of inspiratory time to expiratory time (I-E ratio) was 1:2 and the rate 15 or 25/min. The inspiratory rise time was set at 5% (similar to subject values), P_{aw} was measured 1 cm below the ETT tip in the test lung via a 15 cm, 16 gauge catheter (Arrow). At each of the above combinations, the expiratory circuit was disconnected from the ventilator during 2, 3, 4, 5, 6, and 10 s to simulate filter exchange. The filter was disconnected from the tubing. Flow trigger

RESPIRATORY CARE Paper in Press. Published on July 15, 2014 as DOI: 10.4187/respcare.02892

set at 1 L/min and pressure trigger set at −20 cm H_2O were used at every step. In addition, the suctioning support function was activated at the end of each sequence. During all the procedures, inspiratory flow (obtained from the ventilator) and tracheal pressure were registered.

Statistics

The primary outcome variables were changes in P_{aO_2}. A power analysis indicated that for a clinically important decrease in P_{aO_2} (10 ± 15 mm Hg [mean ± SD]) with a $P < .05$ and a power of 0.95, 32 subjects would be needed. We therefore enrolled 40 subjects in this study. The data were analyzed by one-way analysis of variance.

The data from the bench test were analyzed with the *t* test.

For the statistical analyses, the Prism 6.0 statistical program (GraphPad Software, La Jolla, California) was used. $P < .05$ was considered a priori as statistically significant.

Results

Clinical Study

Twelve women and 28 men (2 with severe, 25 with moderate, and 13 with mild ARDS[9]) were enrolled (Table 1); 8 were ventilated with pressure support ventilation, 12 with pressure controlled ventilation, and 20 with PRVC; 39 of the subjects were orally intubated, and one had a tracheal cannula. PEEP was 12.0 ± 4.0 cm H_2O, F_{IO_2} was 0.5 ± 0.1, and the P_{aO_2}/F_{IO_2} ratio was 176 ± 47 mm Hg. The mean time on the ventilator was 8.6 ± 9.9 d. The tube sizes used in the studied subjects were inner diameter 7 mm for women ($n = 12$) and inner diameter 8 mm for men ($n = 28$). The gas was humidified with a heat-moisture exchanger in 20 subjects and with an active humidifier for the remaining subjects ($n = 20$).

The mean duration of the filter exchange was 3.5 ± 1.2 s. There were no significant changes in P_{aO_2} (89 ± 16 mm Hg at baseline vs 86 ± 16 mm Hg at 15 min and 88 ± 18 mm Hg at 60 min, $P = .24$; Table 2, Fig. 3) or in compliance of the respiratory system (41 ± 11 mL/cm H_2O at baseline vs 40 ± 12 mL/cm H_2O at 15 min and 40 ± 12 mL/cm H_2O at 60 min, $P = .32$; Table 2, Fig. 3). Arterial pH (7.39 ± 0.07 at baseline vs 7.39 ± 0.08 at 15 min and 7.39 ± 0.08 at 60 min) and P_{aCO_2} (43 ± 10 mm Hg at baseline vs 43 ± 10 mm Hg at 15 min and 44 ± 11 mm Hg at 60 min) as well as hemodynamics (pulse rate 88 ± 23 beats/min at baseline vs 88 ± 21 beats/min at 15 min and 87 ± 20 beats/min at 60 min [MAP 77 ± 14 mm Hg at baseline vs 75 ± 15 m Hg at 15 min and 75 ± 10 mm Hg at 60 min]) did not change during the study period.

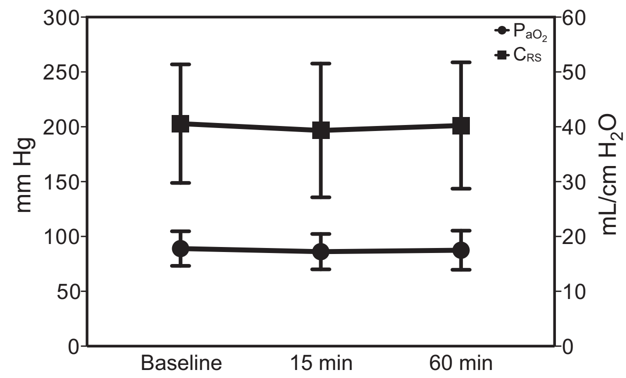

Fig. 3. Mean P_{aO_2} (mm Hg) and compliance of the respiratory system (C_{RS}; mL/cm H_2O) with SD before the high-efficiency particulate air filter change and 15 min and 60 min after.

Table 3. Airway Pressure (P_{aw}) During the High-Efficiency Particulate Air Filter Change

Subject No.	Disconnection Time (s)	ETT Size (mm, inner diameter)	PEEP (cm H_2O)	P_{aw} (cm H_2O)
17	3.0	7	14	14
35	3.0	8	15	14
38	3.0	8	9	8
39	3.5	8	5	5

P_{aw} = airway pressure
ETT = endotracheal tube

In the 4 subjects (Nos. 17, 35, 38, and 39, all ventilated with PRVC) where the pressure below the ETT was measured, the airway pressure was maintained above PEEP in all subjects during the 3–3.5-s disconnection period (Table 3).

Bench Test

After disconnection of the ventilator circuit, the ventilator delivered 4 auto-triggered inspirations with a total duration of 3–10 s, depending on the I-E ratio and the set breathing frequency. The inspiratory flow pattern differed between the 2 ventilation modes. In the pressure controlled ventilation mode, the inspiratory flow reached a maximum rate of 3,300 mL/s in 0.3 s in all auto-triggered inspirations. In the volume controlled ventilation mode, flow of the first triggered inspiration was the same as with the pressure controlled ventilation (3,300 mL/s) mode, but flow took 1.2 s to reach its maximum rate. Flow in the volume controlled ventilation mode decreased in inspiration numbers 2, 3, and 4 to 2,500 mL/s. With pressure controlled-ventilation, P_{aw} was maintained above the set PEEP of 10 cm H_2O in all cases. The lowest P_{aw} (12 ± 1.2 cm H_2O) was independent of other settings and tube size.

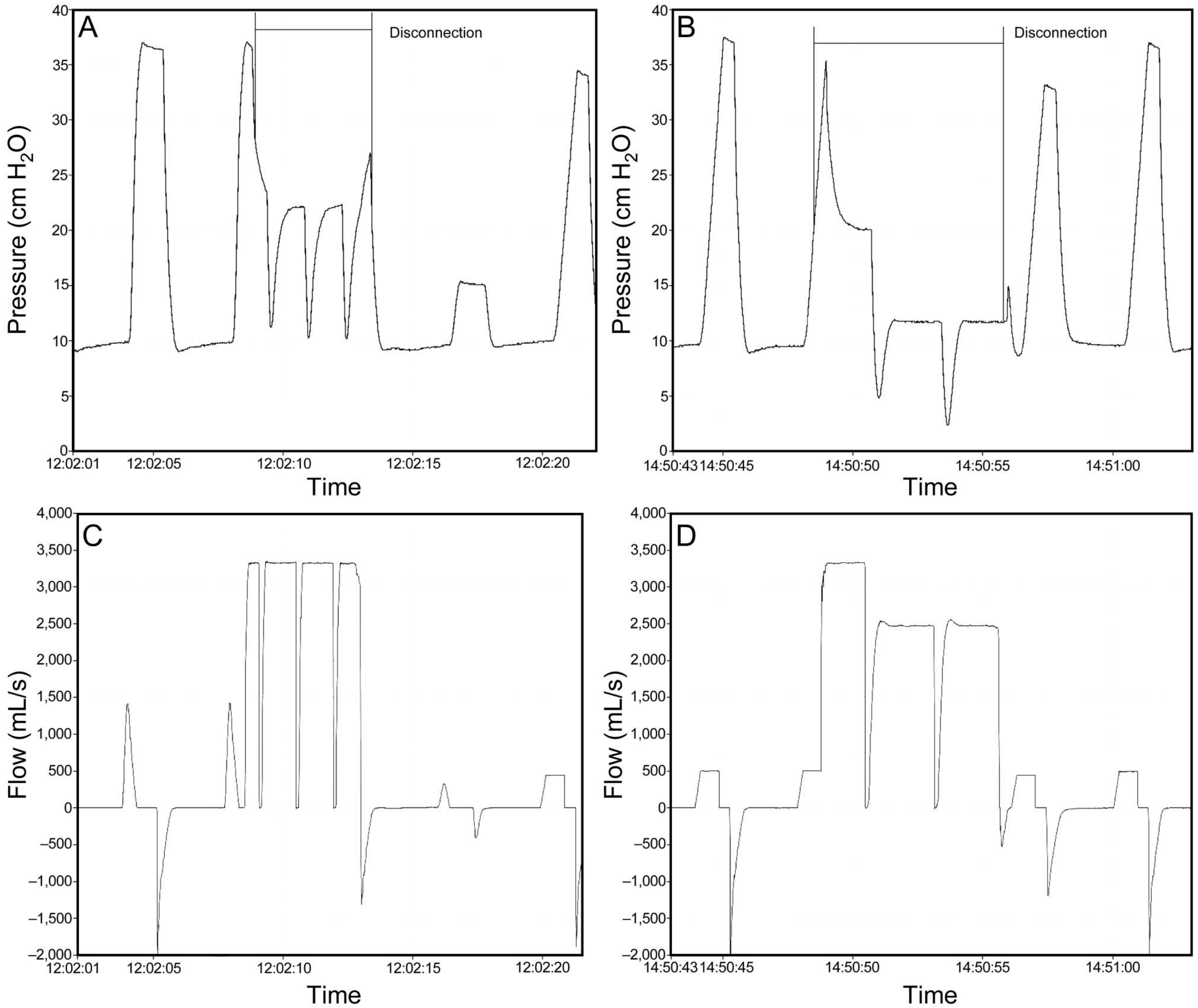

Fig. 4. Airway pressure 1 cm below an inner diameter 8 mm endotracheal tube tip during experimental high-efficiency particulate air filter change in the bench model. A and C: the ventilator was set at pressure controlled ventilation (end-inspiratory plateau pressure 25 cm H_2O), breathing frequency of 15 breaths/min, ratio of inspiratory time to expiratory time 1:2, flow triggering 1 L/min. B and D: for volume controlled ventilation, the settings were the same as during pressure controlled ventilation. The test lung was set to compliance 10 mL/cm H_2O.

However, with volume controlled ventilation, P_{aw} decreased to a minimum of 4.3 ± 1.2 cm H_2O ($P < .001$ compared with pressure controlled ventilation) (Fig. 4). In both pressure controlled ventilation and volume controlled ventilation, P_{aw} decreased to 0 cm H_2O 0.7 ± 0.2 s after the auto-triggered inspirations discontinued. With the suction support function activated, P_{aw} decreased to 0 cm H_2O within 1.7 ± 0.4 s after disconnection (Fig. 5), and the same pattern occurred with the −20 cm H_2O trigger setting.

Discussion

This study shows that exchange of ventilator filters placed between the expiratory limb of the ventilatory circuit and the ventilator did not deteriorate lung function, as assessed by arterial oxygenation and respiratory system compliance in subjects mechanically ventilated with a Servo-i ventilator with PRVC or pressure support ventilation with low flow triggering settings. Tracheal pressure monitoring in 4 subjects indicated that the pressure did not decrease during the short disconnection procedure. The bench test demonstrated that disconnection during pressure controlled ventilation induced auto-triggering of the ventilator, thus maintaining the airway pressure. However, in the bench test, with volume controlled ventilation using a 5% rise time similar to that with pressure controlled ventilation, there was a delay in achieving maximal flow at auto-triggering, causing an initial drop in airway pressure. When turning off the trigger, the airway pressure

RESPIRATORY CARE Paper in Press. Published on July 15, 2014 as DOI: 10.4187/respcare.02892

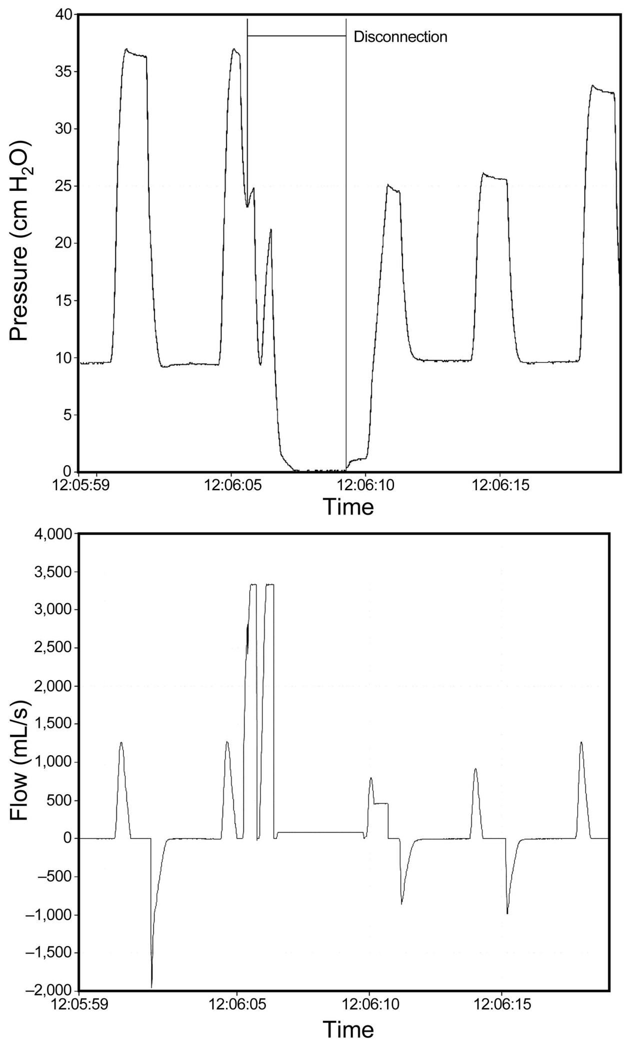

Fig. 5. Airway pressure 1 cm below inner diameter 8 mm endotracheal tube tip during experimental high-efficiency particulate air filter change in the bench model with suction support activated. The ventilator was set for pressure controlled ventilation (end-inspiratory plateau pressure 25 cm H_2O), breathing frequency of 15 breaths/min, ratio of inspiratory time to expiratory time 1:2, flow triggering 1 L/min. The test lung was set to compliance 10 mL/cm H_2O.

immediately decreased to zero in both volume controlled ventilation and pressure controlled ventilation mode.

Two factors are important to explain the clinical results in the subjects. First, the trigger was set at low triggering effort, causing the ventilator to easily sense a loss of airway pressure, which the ventilator software interpreted as a trigger effort, inducing a new inspiration with a duration as set by the I-E ratio and ventilatory rate. Without any trigger, the disconnection of the ventilator circuit would not induce any new breaths. Second, in both PRVC and pressure support ventilation, the ventilator delivers a pressure-regulated breath, and therefore the upper flow rate is only limited by the capacity of the ventilator to deliver gas flow and the resistance in the ventilator tubings. Thus, as shown in the bench test and in the 4 subjects in whom we measured tracheal pressure, the high flow combined with the resistance in the expiratory tubing generated an airway pressure higher or equal to the set PEEP. However, one cannot exclude the possibility that, in our group of subjects with mainly mild to moderate ARDS, oxygenation recovered fast, explaining our results. Theoretically, with volume controlled ventilation, which delivers a specific volume with a limited square wave flow pattern, the flow should not be high enough to maintain an adequate airway pressure level. However, unexpectedly, in the bench test the flow with volume controlled ventilation increased to approximately 3 L/s after a short delay, corresponding to the set rise time and a flow trigger delay of 100 ms, inducing only a short drop in airway pressure (Fig. 4). This is because the Servo-i has temporary pressure support in the volume controlled ventilation mode that is switched on when the inspiratory pressure drops below 3 cm H_2O the expected pressure value during inspiration, indicating that the flow demand of the patient is higher than the ventilator is set to deliver. However, this temporary pressure support feature is model-dependent and can be deactivated in newer models for the United States market of the Servo-i (Åke Larsson, Maquet Critical Care, Solna, Sweden, personal communication).

The clinical implication of this study is that, during exchange of ventilator filters, auto-triggering can maintain airway pressure above PEEP, but absence of auto-triggering may allow PEEP to be lost. The ventilator should therefore preferably be set to pressure controlled or supported mode, with a low triggering threshold. By doing so, the tracheal pressure is maintained. Furthermore, the suction support should not be activated.

Limitations

In the power analysis a priori, we estimated the number of subjects using a high power (0.95). In fact, a power analysis a posteriori showed 0.99, indicating that it is not likely that filter exchange in the patient category studied leads to a deterioration in lung function. However, the clinical portion of the study was performed in subjects without extremely high PEEP levels or F_{IO_2}. With very high PEEP, even during pressure control, the flow and resistance in the ventilator tubing might not be adequate to maintain a sufficient airway pressure. In patients needing very high PEEP and/or F_{IO_2}, we still believe that ventilator filters should be exchanged with caution to avoid inadvertent lung collapse. Moreover, other brands of ventilators than the one used in this study may have other features, eg, the auto-triggering will be discontinued earlier, which needs to be considered. In particular, the finding in the bench test that only a short reduction of pressure occurred with volume controlled ventilation is model- and brand-dependent. Furthermore, other tubing sets with other lengths and diameters could give other resistance patterns, which may influence the obtained airway pressure.

Conclusions

This prospective observational study in ICU subjects, mechanically ventilated in pressure modes with low triggering threshold settings, demonstrated that a short disconnection of the expiratory ventilator circuit from the ventilator during filter exchange was not associated with any significant deterioration in lung function. A bench test suggests that this result is explained by auto-triggering with high inspiratory flows during the filter exchange, maintaining the airway pressure.

ACKNOWLEDGMENTS

We express our gratitude to Ing-Marie Larsson, Ewa Wallin, and the ICU personnel at the University Hospital in Uppsala, Sweden for excellent help and dedication.

REFERENCES

1. Gattinoni L, D'Andrea L, Pelosi P, Vitale G, Pesenti A, Fumagalli R. Regional effects and mechanism of positive end-expiratory pressure in early adult respiratory distress syndrome. JAMA 1993;269(16):2122-2127.
2. Briel M, Meade M, Mercat A, Brower RG, Talmor D, Walter SD, et al. Higher vs lower positive end-expiratory pressure in patients with acute lung injury and acute respiratory distress syndrome: systematic review and meta-analysis. JAMA 2010;303(9):865-873.
3. Neumann P, Berglund JE, Fernández Mondéjar E, Magnusson A, Hedenstierna G. Dynamics of lung collapse and recruitment during prolonged breathing in porcine lung injury. J Appl Physiol 1998;85(4):1533-1543.
4. Dyhr T, Laursen N, Larsson A. Effects of lung recruitment maneuver and positive end-expiratory pressure on lung volume, respiratory mechanics and alveolar gas mixing in patients ventilated after cardiac surgery. Acta Anaesthesiol Scand 2002;46(6):717-725.
5. Dyhr T, Bonde J, Larsson A. Lung recruitment manoeuvres are effective in regaining lung volume and oxygenation after open endotracheal suctioning in acute respiratory distress syndrome. Crit Care 2003;7(1):55-62.
6. Lindgren S, Odenstedt H, Olegård C, Söndergaard S, Lundin S, Stenqvist O. Regional lung derecruitment after endotracheal suction during volume-or pressure-controlled ventilation: a study using electric impedance tomography. Intensive Care Med 2007;33(1):172-180.
7. De Campo T, Civetta JM. The effect of short-term discontinuation of high-level PEEP in patients with acute respiratory failure. Crit Care Med 1979;7(2):47-49.
8. Maquet. Servo Duo Guard data sheet. http://www.maquet.com/content/documents/datasheet_optionaccessory/servo_data_duoguard_rev01_en_all.pdf. *Accessed August 15, 2013.*
9. ARDS Definition Task Force, Ranieri VM, Rubenfeld GD, Thompson BT, Ferguson ND, Caldwell E, et al. Acute respiratory distress syndrome: the Berlin Definition. JAMA 2012;307(23):2526-2533.