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Sigh Improves Gas Exchange and Lung Volume in Patients with Acute Respiratory Distress Syndrome Undergoing Pressure Support Ventilation

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Background: **The aim of our study was to assess the effect of periodic hyperinflations (sighs) during pressure support ventilation (PSV) on lung volume, gas exchange, and respiratory pattern in patients with early acute respiratory distress syndrome (ARDS).**

Methods: **Thirteen patients undergoing PSV were enrolled. The study comprised 3 steps: baseline 1, sigh, and baseline 2, of 1 h each. During baseline 1 and baseline 2, patients underwent PSV. Sighs were administered once per minute by adding to baseline PSV a 3- to 5-s continuous positive airway pressure (CPAP) period, set at a level 20% higher than the peak airway pressure of the PSV breaths or at least 35 cm H_2O. Mean airway pressure was kept constant by reducing the positive end-expiratory pressure (PEEP) during the sigh period as required. At the end of each study period, arterial blood gas tensions, air flow and pressures traces, end-expiratory lung volume (EELV), compliance of respiratory system (Crs), and ventilatory parameters were recorded.**

Results: **Pao_2 improved ($P < 0.001$) from baseline 1 (91.4 ± 27.4 mmHg) to sigh (133 ± 42.5 mmHg), without changes of $Paco_2$. EELV increased ($P < 0.01$) from baseline 1 (1,242 ± 507 ml) to sigh (1,377 ± 484 ml). Crs improved ($P < 0.01$) from baseline 1 (40.2 ± 12.5 ml/cm H_2O) to sigh (45.1 ± 15.3 ml/cm H_2O). Tidal volume of pressure-supported breaths and the airway occlusion pressure ($P_{0.1}$) decreased ($P < 0.01$) during the sigh period. There were no significant differences between baselines 1 and 2 for all parameters.**

Conclusions: **The addition of 1 sigh per minute during PSV in patients with early ARDS improved gas exchange and lung volume and decreased the respiratory drive.**

This article is accompanied by an Editorial View. Please see Suter PM: Does the advent of (new) low tidal volumes bring the (old) sigh back to the intensive care unit? ANESTHESIOLOGY 2002; 96:783–4.

CURRENT ventilatory approaches to the acute respiratory distress syndrome (ARDS) suggest the use of low tidal volumes (V_T)[1] to limit alveolar distending pressure[2,3] and relatively high positive end-expiratory pressure (PEEP)[4] to prevent alveolar collapse and cyclical opening and closing.[5] In a recent large multicenter trial, ventilation with V_T as low as 6 ml/kg of predicted body weight reduced mortality compared with traditional ventilation with larger V_T.[1] Although it is effective in limiting injury associated with alveolar stretching,[2,3] ventilation with low V_T may lead to progressive atelectasis and consequent hypoxia.[6] Although the use of high PEEP levels may partially counteract this tendency and stabilize alveoli, minimize cyclical inspiratory opening, and prevent further lung collapse,[7,8] atelectatic lung regions may persist.

Lung recruitment maneuvers (RMs) such as sighs have been successfully used in patients during general anesthesia to restore the decreased respiratory system compliance (Crs) and arterial oxygenation commonly associated with use of low V_T.[9] This result has led investigators to test the use of RMs in patients with ARDS during ventilation with low V_T and high PEEP, improving lung function[10] and outcome.[4] Cyclically delivered RMs have been effective in ARDS patients managed with continuous positive pressure ventilation (CPPV), to induce alveolar recruitment[11] and allow the use of lower PEEP and mean airway pressure (Paw_m), while preserving gas exchange and lung volumes.[12] Recently, many clinicians and investigators underscored the potential role and benefits of maintaining spontaneous breathing by using modes of partial ventilatory support such as pressure support ventilation (PSV).

The ability of PSV to permit diaphragmatic activity[13,14] and reduce the need for sedative drugs[15] might be of benefit to patients with acute respiratory failure. In a recent study,[16] PSV was utilized effectively in patients with acute lung injury, with no significant gas exchange differences in comparison with CPPV. In that study, PSV was more likely to fail in patients with a high minute ventilation (V_E) and low Crs, in agreement with previous studies that showed decreased efficacy of PSV in sicker patients.[17,18] The beneficial effect of sighs during CPPV may possibly extend to modes of partial ventilatory support, such as PSV. Sighs may counteract the tendency of lung collapse associated with low V_T and thus improve gas exchange.

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Table 1. Patient Characteristics and Outcome

Patient No.	Diagnosis	Sex (M/F)	Age (yr)	Days of Intubation	F_{IO_2}	PSV (cm H_2O)	P_{AO_2}/F_{IO_2} (mmHg)	Days of Intensive Care Unit Stay	Outcome
1	Polytrauma	M	48	2	0.55	14	165.2	9	S
2	Pneumonia	F	69	5	0.6	12	103.1	37	D
3	Pneumonia	M	51	7	0.5	10	150	12	S
4	Polytrauma	F	68	2	0.5	14	170.8	21	S
5	Pneumonia	M	75	2	1	10	167.3	9	S
6	Pancreatitis	M	72	3	0.6	10	123.3	4	D
7	Pneumonia	M	67	4	0.6	8	181.5	11	D
8	Peritonitis	M	86	7	0.5	10	143.2	24	D
9	Pneumonia	F	63	6	0.7	17	127.7	17	S
10	Pneumonia	M	80	1	1	14	107.8	8	S
11	Polytrauma	M	53	7	0.4	18	165.7	20	S
12	Pneumonia	M	60	8	0.6	15	171.3	23	S
13	Pneumonia	M	65	2	0.5	10	173.2	17	S
	Mean ± SD	10M/3F	66 ± 11	4.2 ± 2.6	0.6 ± 0.2	12.4 ± 31	150 ± 26	16.3 ± 8.9	4D/9S

Data are expressed as mean ± SD.

D = deceased; S = survived; days of intubation = days elapsed between the time of intubation to the time of the study; F_{IO_2} = inspired fraction of oxygen; PSV = pressure support ventilation; P_{AO_2}/F_{IO_2} = arterial tension of oxygen to inspired fraction of oxygen ratio.

The aim of our study was to investigate the effects of sighs, administered at a frequency of one per minute, on gas exchange, lung volume, and respiratory pattern during PSV in patients with early ARDS.

Materials and Methods

Subjects

We enrolled 13 patients (table 1) who met the criteria for ARDS defined by the American European Consensus Conference[19] and had no history of chronic obstructive pulmonary disease. All patients were studied within 7 days after the diagnosis of ARDS. At the time of enrollment, all subjects were intubated and had an arterial catheter in place. The level of sedation, obtained with benzodiazepines, propofol, and fentanyl, allowed patient arousal and coordinated motor responses to verbal commands. Subjects were studied during clinical and hemodynamic stability in a supine or semirecumbent position. On the day of the study, patients were already ventilated with PSV set by the attending physician according to empirical criteria.[16]

Protocol

The protocol was approved by the institutional ethics committee, and informed consent was obtained from the patient's next of kin. The investigation was conducted according to the Helsinki Declaration. After enrollment, all patients were connected to an Evita-4 ventilator (Dräger, Lübeck, Germany), set in PSV with fraction of inspired oxygen (F_{IO_2}), inspiratory pressure, and PEEP levels previously selected by the attending physician. The time of inspiratory pressure rise was set at 0.2 s, and PSV parameters were kept unchanged throughout the study. The protocol consisted of three consecutive study periods: baseline period 1 (Base 1), sigh period (Sigh), and baseline period 2 (Base 2), each one lasting a minimum of 1 h.

During Base 1 and Base 2, patients were ventilated with PSV alone. During Sigh the ventilator was set in biphasic positive airway pressure (BIPAP) + pressure support ventilation (BIPAP+ASB on a Dräger Evita-4 ventilator), and sighs were performed by means of the higher BIPAP level. During BIPAP,[20] ventilation is assisted by alternating between a PEEP high level and a PEEP low level. Spontaneous breaths are possible but not supported during the PEEP high level (CPAP mode), but they are supported by the set level of PSV during the PEEP low level. BIPAP parameters were initially set as follows: PEEP high level ($Plat_S$), 20% higher than the peak airway pressure during PSV or 35 cm H_2O, whichever was higher; respiratory rate, 1 breath/min; and length of the sigh (Ti_S), individually set between 3 and 5 s, according to the patient's tolerability (mainly limited by coughing reflex and hemodynamics; fig. 1).

Because of the higher inspiratory pressure and the longer Ti_S obtained by design during Sigh, the Paw_m could have increased. To avoid the possible influence of changes in Paw_m on oxygenation, Paw_m (averaged over a 1-min period) was always kept constant by adjusting the PEEP level.

Gas Exchange and Hemodynamics

We determined all measurements at the end of each study period. We collected arterial blood samples to measure Pa_{O_2}, Pa_{CO_2}, pH, and hemoglobin concentration and saturation (HbO_2) (ABL 330; Radiometer, Copenhagen, Denmark). Blood pressures were measured with pressure transducers zeroed at the mid-axillary line. During Sigh, all hemodynamic parameters were measured between two sighs.

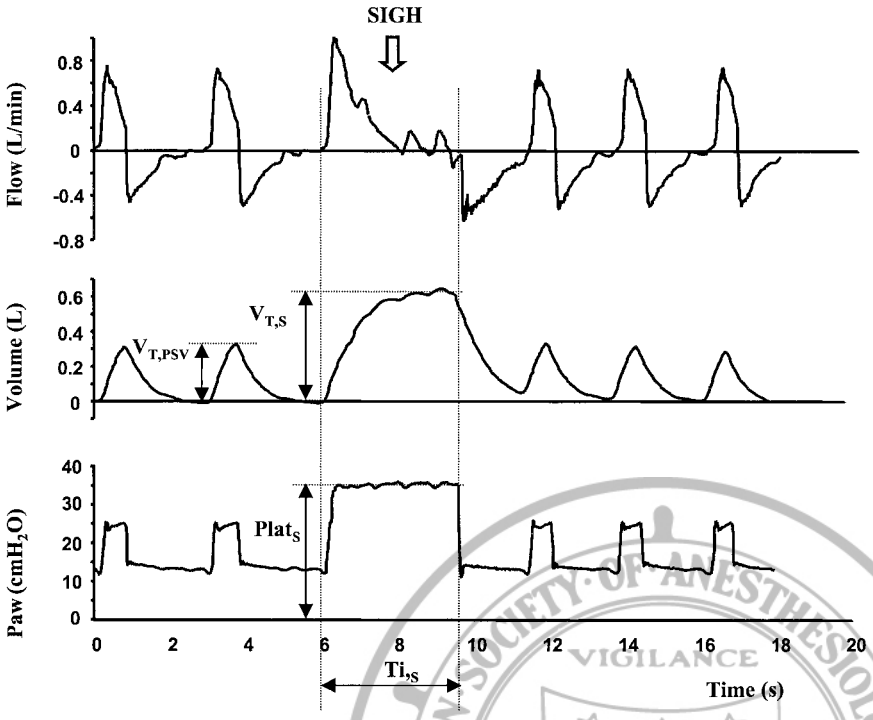

Fig. 1. Recorded traces of air flow (*top*), volume (*middle*), and airway pressure (*bottom*), showing a biphasic positive airway pressure (BIPAP)–sigh breath (white arrow). $V_{T,PSV}$ = tidal volume of pressure support ventilation; $Plat_S$ = higher positive end-expiratory pressure (PEEP) level during BIPAP breath; $V_{T,S}$= tidal volume of sigh; Ti_S = inspiratory time of sigh.

Respiratory Mechanics

Airway pressure and flow were measured with a CP100 Pulmonary Monitor (Bicore Monitoring Systems, Irvine, CA), through a disposable flowmeter (VarFlex flow transducer; Bear Medical Systems, Bilthoven, The Netherlands) inserted between the Y-piece of the ventilator circuit and the proximal end of the endotracheal tube. The analog output of the pulmonary monitor was connected to a personal computer and processed *via* an analog-to-digital converter (Colligo, Elekton, Italy) at a sample rate of 100 Hz.

A 5-min-long sample was recorded at the end of each study period and stored for subsequent computer analysis. After the first 3 min of recording, we performed a minimum of three end-expiratory and three end-inspiratory airway occlusions, by pressing for at least 3 s the appropriate button on the ventilator panel. During Sigh, the occlusion maneuvers were performed between two sighs. The airway pressure drop in the first 100 ms of an occluded inspiration ($P_{0.1}$) was measured as an index of the neuromuscular drive.[21] We performed the end-expiratory occlusion first, to avoid interference with the measurement of $P_{0.1}$. Data were then analyzed with dedicated software (Computo; Elekton, Agliano Terme, Italy). Tidal volume was obtained by integration of the flow signal.

From the analysis of traces with nonoccluded breaths we obtained (1) respiratory rate, by counting the number of breaths in 1 min; (2) inspiratory time (Ti_{PSV}) and expiratory time (Te_{PSV}) and inspiratory to expiratory time ratio ($I:E_{PSV}$) by averaging at least 10 PSV breaths; (3) mean tidal volume in 1 min ($V_{T,mean}$); (4) V_E, calculated as $V_{T,mean} \times$ respiratory rate; (5) Paw_m, measured as the average of the Paw_m signal during a 60 s period; and (6) V_T of PSV breaths ($V_{T,PSV}$), determined by averaging at least 10 breaths. From sigh breaths we determined (1) tidal volume ($V_{T,S}$); (2) inspiratory time ($T_{I,S}$); (3) inspiratory plateau pressure ($Plat_S$), corresponding to the higher BIPAP level applied to perform sighs; (4) and fraction of minute ventilation supplied by the sigh breaths ($V_{E,S}$), calculated as $V_{T,S}/V_E$.

From the analysis of occluded breaths we computed (1) end-inspiratory elastic recoil pressure (Pel,rsi), measured as the plateau airway pressure (Paw) at relaxation of respiratory muscles, identified by visual inspection during an inspiratory occlusion; (2) end-expiratory elastic recoil pressure (Pel,rse) measured as the plateau Paw at relaxation of the respiratory muscles, identified by visual inspection during an end-expiratory occlusion (all the occlusions that did not reach an identifiable relaxation were discarded); and (3) $P_{0.1}$, measured during the first end-expiratory pause as the deflection in Paw in the first 100 ms after the start of patient inspiratory effort. We discarded by inspection the initial 5 ms to avoid uncertainties in recognizing the actual start of the inspiratory effort; the Paw drop in the following 100 ms was taken as the $P_{0.1}$ value.[22] We computed the Crs as $Crs = V_{T,PSV}/(Pel,rsi - Pel,rse)$.

Lung Volume

To assess the effects of sighs on lung volumes, we measured the end-expiratory lung volume (EELV), which represents the total gas volume at end expiration when PEEP is applied. A 5-cm clampable tube was inserted

between the endotracheal tube and the Y-piece of the ventilator. Immediately after an end-expiratory occlusion maneuver, performed as described above, we clamped the endotracheal tube. We then disconnected the patient from the ventilator and measured the lung volume by a simplified closed-circuit helium dilution technique.[23] Measurements were performed in duplicate. During Sigh, the EELV was measured one or two breaths immediately before a new sigh.

Statistical Analysis

Values are expressed as mean ± SD. We used one-way analysis of variance for repeated measurements to compare the three study steps. Individual comparisons were performed with use of paired *t* tests, with application of the Bonferroni correction for multiple comparisons. We specifically planned to compare Sigh with Base 1 and Base 2 with Base 1. For all the tests used, $P < 0.05$ was considered significant.

Results

Relevant characteristics of the patients at the time of enrollment are shown in table 1. The main study results are shown in table 2. Time elapsed from the diagnosis of ARDS was 3.7 ± 1.8 days. Hemodynamic parameters did not show any significant change throughout the study.

Sigh Profile

$Plat_S$ was 38 ± 3.2 cm H_2O, corresponding to a $V_{T,S}$ of 1,148 ± 301 ml; Ti_S was 3.6 ± 0.7 s.

Gas Exchange

The introduction of one sigh per minute was associated with an increase in Pao_2 (41.6 ± 33.9 mmHg [29.8 ± 16.2%]; $P < 0.001$; fig. 2A), accompanied by an increase in HbO_2 (1.5 ± 1.5% [1.5 ± 1.5%]; $P < 0.01$). Returning to Base 2 led to a reversal of the gas exchange improvement, and Pao_2 and Hbo_2 were not significantly different during Base 1 and Base 2. $Paco_2$ and pH did not show any significant change throughout the protocol.

Lung Volume and Respiratory Mechanics

PEEP was significantly lower during Sigh with respect to Base 1 (average decrease, 1.1 ± 0.7 cm H_2O). V_E did not show any significant difference among the three steps. EELV increased significantly from Base 1 to Sigh (average increase, 136 ± 140 ml [12.3 ± 10.8%]; range, −82–392 ml; $P < 0.01$; fig. 2B) and returned to baseline value after discontinuation of the sigh. Crs increased significantly during Sigh with respect to Base 1 (4.9 ± 4.9 ml × cm H_2O^{-1} [10.4 ± 9.4%]; $P < 0.01$), whereas Crs Base 2 was not significantly different from Crs Base 1 (fig. 2C).

Table 2. Gas Exchange, Hemodynamics, and Ventilatory Parameters

Values	Baseline 1	Sigh	Baseline 2
P_{AO_2} (mmHg)	91.4 ± 27.4	133 ± 42.5†	93.3 ± 21.6
P_{ACO_2} (mmHg)	45.8 ± 7.6	47.3 ± 10.7	47.9 ± 10.3
Hbo_2 (%)	94.9 ± 2.4	96.4 ± 2.2‡	94.9 ± 3.1
pH	7.43 ± 0.05	7.43 ± 0.05	7.43 ± 0.06
HR (b · m^{-1})	89 ± 17	90 ± 16	91 ± 18
PAs (mmHg)	143 ± 19	148 ± 29	143 ± 43
PAd (mmHg)	65 ± 12	63 ± 14	64 ± 8
V_E (L · min^{-1})	9.2 ± 2.5	9.9 ± 2.8	9.5 ± 2.3
Paw_m (cm H_2O)	14.9 ± 4.7	14.9 ± 4.9	14.9 ± 4.4
PEEP (cm H_2O)	11 ± 3	10 ± 4‡	11 ± 3
EELV (ml)	1242 ± 507	1378 ± 484‡	1260 ± 547
Crs (ml · cm H_2O^{-1})	40 ± 12	45 ± 15‡	38 ± 13
$V_{T,PSV}$ (ml)	418 ± 57	370 ± 81‡	405 ± 61
$T_{i,PSV}$ (s)	0.8 ± 0.2	0.7 ± 0.2‡	0.9 ± 0.3
$I:E_{PSV}$	0.5 ± 0.1	0.5 ± 0.3	0.5 ± 0.2
RR (b · m^{-1})	23 ± 7	24 ± 8	25 ± 11
$P_{0.1}$ (cm H_2O)	2.1 ± 1.5	1.2 ± 0.7‡	1.7 ± 0.8
$Plat_s$ (cm H_2O)	—	38 ± 3.2	—
$V_{T,S}$ (ml)	—	1148 ± 301	—
Ti_s (s)	—	3.6 ± 0.7	—
$\%V_{E,S}$	—	11.2 ± 3	—

Data are expressed as mean ± SD; † Sigh *vs.* Baseline 1, $P < 0.001$; ‡ Sigh *vs.* Baseline 1, $P < 0.01$.

P_{AO_2} = arterial tension of oxygen; P_{ACO_2} = arterial tension of carbon dioxide; Hbo_2 = oxygenated hemoglobin (percent); HR = heart rate; PAs = systolic arterial pressure; PAd = diastolic arterial pressure; V_E = minute ventilation; Paw = mean airway pressure; PEEP = positive end expiratory pressure; EELV = end expiratory lung volume; Crs = compliance of respiratory system; $V_{T,PSV}$ = tidal volume of pressure support ventilation; $T_{i,PSV}$ = inspiratory time of pressure support ventilation; $I:E_{PSV}$ = inspiratory to expiratory ratio; RR = respiratory rate; $Plat_{,S}$ = higher PEEP level during BIPAP breath; $Vt_{,s}$ = tidal volume of sigh; $Ti_{,S}$ = inspiratory time of sigh; $\%V_{E,S}$ = fraction of minute ventilation supplied by the sigh breath.

Respiratory Pattern

During Sigh, there was a significant decrease of $V_{T,PSV}$ ($P < 0.01$; fig. 3A), Ti_{PSV} ($P < 0.01$; fig. 3B), and $P_{0.1}$ ($P < 0.01$; fig. 3C). All three parameters returned to baseline after discontinuation of sighs, and no significant differences were found between Base 1 and Base 2. There was no significant change in $I:E_{PSV}$ during the three study periods.

Discussion

The main result of this study was that the addition of one sigh per minute during PSV in patients with early ARDS improved arterial oxygenation and likely promoted alveolar recruitment.

Effect of Ventilatory Strategy on Gas Exchange and Lung Volume

The use of sigh with $Plat_S$ higher than 35 cm H_2O improved arterial oxygenation in all studied patients, in spite of a slightly lower PEEP and similar Paw_m (table 2). This positive effect on gas exchange was associated with

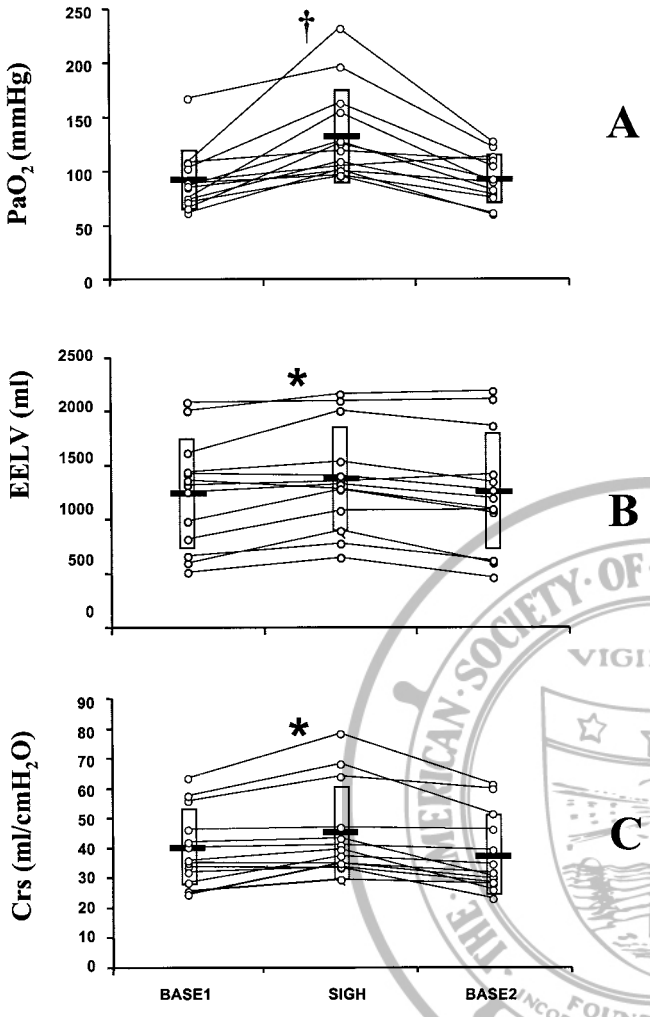

Fig. 2. Changes in arterial tension of oxygen (Pao_2) (*A*), end-expiratory lung volume (EELV) (*B*), and compliance of respiratory system (Crs) (*C*) during baseline 1 (BASE1), sigh (SIGH), and baseline 2 (BASE2). Solid lines represent changes in each patient between different steps. Solid horizontal bars represent the mean values, and open vertical columns indicate SD (†SIGH *versus* BASE1, $P < 0.001$; *SIGH *versus* BASE1, $P < 0.01$).

an increase in EELV and Crs, suggesting that sighs possibly promoted alveolar recruitment. These results are in agreement with the reported beneficial effect of various types of RMs that have been applied in fully ventilated and paralyzed patients. These RMs included the administration of a sustained inflation lasting several seconds,[10,24] a cyclical higher inflation pressure,[11] and a cyclical higher PEEP.[12]

We computed Crs by applying the airway occlusion technique during PSV breaths, recognizing the presence of muscle relaxation by visual inspection. In spite of the many possible limitations of this method, we showed in a previous study[25] that during PSV the airway pressure plateau during inspiratory occlusion is a reliable measurement of the relaxed elastic recoil pressure of the respiratory system in patients with acute lung injury or ARDS.

The average $Plat_S$ used in our study was 38 cm H_2O. Using higher inflation pressure during a sigh might have resulted in more effective recruitment and further improvement in arterial oxygenation but also in increased risk of barotrauma. The $Plat_S$ values that we used are similar to the inflation pressures that have been effective in improving gas exchange in other studies in which sighs were cyclically delivered.[11,12] Moreover, in spontaneously breathing patients, inspiratory pressure levels may also be limited by subjective tolerability and stimulation of the cough reflex.

The modality used in our study to administer sigh differs in some ways from other studies in which cyclically delivered sighs successfully improved gas exchange.[11,12]

First, it was our goal to preserve spontaneous respiratory activity. During CPPV in paralyzed patients, gas distribution in the lung is determined mainly by regional

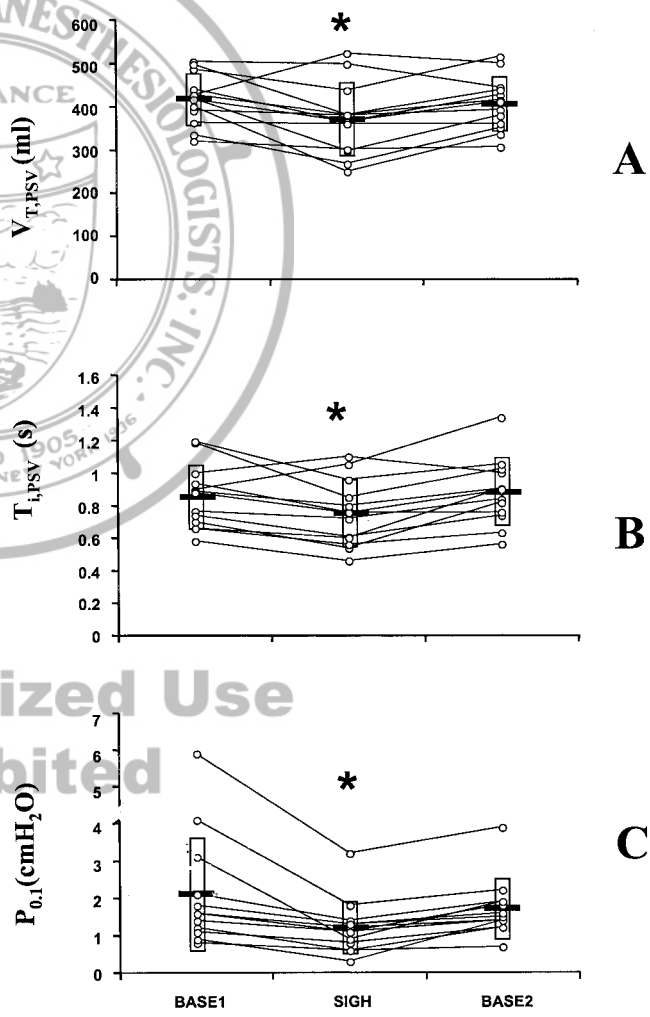

Fig. 3. Changes in respiratory pattern of pressure support ventilation, during baseline 1 (BASE1), sigh (SIGH), and baseline 2 (BASE2): $V_{T,PSV}$ = mean tidal volume during pressure support ventilation (*A*); $T_{I,PSV}$ = inspiratory time of pressure support breaths (*B*); and $P_{0.1}$ = airway occlusion pressure (*C*). Solid lines represent changes in each patient between different steps. Solid horizontal bars represent the mean values, and open vertical columns indicate SD (*SIGH *versus* BASE1, $P < 0.01$).

compliance. In supine position, anterior (nondependent) regions of the lung are the most compliant, and ventilation in the posterior (dependent) regions may be compromised, leading to progressive atelectasis. It has been shown that preserving spontaneous ventilatory effort may improve gas exchange, possibly by enhancing ventilation of the dependent parts of the lung and preventing atelectasis.[14,26]

Second, in our study, sighs were pressure-controlled rather than volume-controlled. During pressure control ventilation, inspiratory flow stops when alveolar pressure reaches the set inspiratory pressure. If alveolar recruitment takes place, more gas is delivered to maintain plateau pressure, facilitating the filling and stabilization of newly opened alveoli.[27]

Third, regardless of the ventilatory mode, sigh pressure was maintained for a relatively long time, possibly favoring the filling of recruited alveoli also by gas redistribution.

Finally, compared to the study of Pelosi *et al.*,[11] who used three consecutive sighs per minute, and the study of Foti *et al.*,[12] who increased the PEEP for two consecutive breaths every 30 s, in our study a single sigh per minute proved beneficial. Identifying the lowest effective sigh rate is of primary importance because of the known drawbacks on lung parenchyma due to high distending volumes or pressures. However, Davies at al.[28] showed that sighs delivered at a rate of two breaths every 10 min were of no benefit during PSV, despite the use of inflation pressures comparable with those of our study. The role of frequency on sigh effectiveness remains to be formally explored.

It is worth noting that Crs was slightly higher in our patients than in those studied by Pelosi *et al.*[11] Hence, the tendency to develop alveolar collapse was possibly lower in our patients. However, the higher level of PEEP used in the study of Pelosi *et al.*[11] should have prevented derecruitment more efficiently[29] than in our study, where level of PEEP was limited also by the conscious patient's tolerability.

Effects on Respiratory Pattern

The introduction of sighs was associated with a decrease in respiratory drive, as shown by a limited but significant decrease in $P_{0.1}$, $V_{T,PSV}$, and $T_{I,PSV}$. It has been shown that with propofol general anesthesia a sigh reduces the V_T of the ensuing breaths, possibly due to a post-sigh inhibition of peripheral chemoreceptors and lung mechanoreceptors.[30] A similar mechanism may have been at play in our patients.

Several factors may have determined these changes. It has been shown that during PSV both V_E and $P_{0.1}$ decrease when oxygenation is improved.[13] The additional V_T provided by the sigh supplied between 10 and 12% of baseline V_E, decreasing the amount of ventilation that patients had to provide.

The observed effect of sighs on the $P_{0.1}$ may also be explained in part by the changes in EELV,[31] which may directly affect the $P_{0.1}$ measurement. However, we did not find any significant correlation between the effect of sigh on $P_{0.1}$ and that on EELV.

Clinical Implications

Partial ventilatory techniques prevent paralysis and reduce the use of sedative drugs. It was our goal to learn whether by adding sigh it was possible to extend the indication for the use of partial ventilatory techniques to patients with early ARDS. In a recent study, acute lung injury was successfully managed with PSV in 38 of 48 patients,[11] suggesting the possibility of safe use of PSV in the early phases of ARDS. PSV was more likely to fail for patients with high ventilatory needs and low Crs. By decreasing the respiratory drive, as in this study, sigh may increase the efficacy of PSV in this patient population.

Moreover, the achievement of a better Crs and alveolar recruitment may play a relevant role in increasing the success rate of PSV in ARDS patients. The response to PSV is also directly affected by pulmonary edema[17] and hypoxia.[18] The increase in arterial oxygenation associated with the introduction of the sigh may increase PSV efficacy in this patient population.

It is worth considering that we selected patients in the earlier phase of acute respiratory failure, when alveolar collapse and dependent atelectasis, mainly related to increased lung weight and superimposed pressure, have shown good recruitability.[5] The same results may not be achieved in patients with late-stage ARDS. In addition, sighs may have a less pronounced effect in patients who are recovering from the acute phase of respiratory failure,[28] where a fibroproliferative process has started and alveolar collapse is driven by an increased pulmonary elastance. Early addition of sighs may have the function of opening previously collapsed lung and preventing lung collapse associated with low V_T ventilation.

Our goal was to investigate the acute short-term physiologic effects of sigh administration. Indeed, no conclusion could be drawn about the outcomes for the patients. A different study design will be necessary to investigate the long-term effects of this approach.

In conclusion, the addiction of one sigh per minute, delivered as pressure-controlled breath to PSV in ARDS patients, improved oxygenation and lung mechanics. These findings suggest a possible role for clinical evaluation of periodic recruitment maneuvers during assisted breathing.

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