Effects of inspiratory pause on CO₂ elimination and arterial PₐCO₂ in acute lung injury

Jérôme Devaquet,¹ Björn Jonson,² Lisbet Niklason,² Anne-Gaëlle Si Larbi,¹ Leif Uttman,² Jérôme Aboab,¹ and Laurent Brochard¹

¹Medical Intensive Care Unit, AP-HP, INSERM Unit 841, Centre Hospitalier Albert Chenevier-Henri Mondor, Créteil, France; ²Department of Clinical Physiology, University Hospital, Lund, Sweden

Submitted 22 May 2008; accepted in final form 17 September 2008

Devaquet J, Jonson B, Niklason L, Si Larbi A-G, Uttman L, Aboab J, Brochard L. Effects of inspiratory pause on CO₂ elimination and arterial PₐCO₂ in acute lung injury. J Appl Physiol 105: 1944–1949, 2008. First published 18 September 2008; doi:10.1152/japplphysiol.90682.2008.—A high respiratory rate associated with the use of small tidal volumes, recommended for acute lung injury (ALI), shortens time for gas diffusion in the alveoli. This may decrease CO₂ elimination. We hypothesized that a postinspiratory pause could enhance CO₂ elimination and reduce PₐCO₂, by reducing dead space in ALI. In 15 mechanically ventilated patients with ALI and hypercapnia, a 20% postinspiratory pause (Tp20) was applied during a period of 30 min between two ventilation periods without postinspiratory pause (Tp0). Other parameters were kept unchanged. The single breath test for CO₂ was recorded every 5 min without postinspiratory pause (Tp0). Other parameters were kept unchanged. The single breath test for CO₂ was recorded every 5 min to measure tidal CO₂ elimination (VtCO₂), arterial dead space (VₐDaw), and of the alveolar plateau. PₐCO₂, PₐCO₂ and physiological and alveolar dead space (VₐDphys, VₐDalv) were determined at the end of each 30-min period. The postinspiratory pause, 0.7 ± 0.2 s, induced on average <0.5 cmH₂O of intrinsic positive end-expiratory pressure (PEEP). During Tp20, VtCO₂ increased immediately by 28 ± 10% (14 ± 5 ml per breath compared with 11 ± 4 for Tp0) and then decreased without reaching the initial value within 30 min. The addition of a postinspiratory pause significantly decreased VₐDaw, by 14% and VₐDphys by 11% with no change in VₐDalv. During Tp20, the slope of the alveolar plateau initially fell to 65 ± 10% of baseline value and continued to decrease. Tp20 induced a 10 ± 3% decrease in PₐCO₂ at 30 min (from 55 ± 10 to 49 ± 9 mmHg, P < 0.001) with no significant variation in PₐO₂. Postinspiratory pause has a significant influence on CO₂ elimination when small tidal volumes are used during mechanical ventilation for ALI.

gas exchange; dead space; mechanical ventilation; ARDS

AFTER TRANSPORT of inspired gas through conducting airways, gas mixing in the respiratory zone by diffusion is time dependent. Therefore, a pause following gas insufflation may enhance gas exchange. Mechanical ventilators allow setting of a postinspiratory pause time (Tp), often in percent of the breathing cycle.

During mechanical ventilation, prolonged Tp has been shown to enhance CO₂ elimination (8, 10–12, 14, 21). In healthy pigs, a prolonged Tp increases CO₂ elimination per tidal breath (VtCO₂) by decreasing airway dead space (VₐDaw) (20). It was suggested that a prolonged Tp increased the mean distribution time (MDT) of inspired gas, so as to allow more time for diffusion of CO₂ towards more central airways (7). MDT, further explained below, expresses the time available for enhanced diffusion between inhaled tidal volume and resident alveolar gas (2).

In pigs at health and with acute lung injury (ALI), Aström et al. recently found that a certain prolongation of MDT achieved with a longer Tp or with a longer inspiratory insufflation time had similar positive effects on CO₂ exchange (4). They noted that a prolonged Tp had a larger effect on MDT than a similar prolongation of inspiratory insufflation time.

Recently Aboab et al. showed that a longer Tp enhances CO₂ exchange evaluated from volumetric capnography in ALI and acute respiratory distress syndrome (ARDS) (2). Positive effects were observed both with regards to a reduced VₐDaw and an elevated alveolar plateau. In their study Tp was only changed for one breath at a time, and PₐCO₂ was not measured. The question was left open if the beneficial effect of Tp was of temporary nature. In pigs, Aström et al. showed that a shortened MDT led to an increase in PₐCO₂, that was nearly stable after 30 min. The study by Lessard et al. assessing CO₂ elimination in patients with ARDS was not controlled for the eventual increase in auto-positive end-expiratory pressure (PEEP) and hemodynamic effects induced by the extension of inspiratory time (12). Mercat et al. did so and found that in ARDS, an extended end-inspiratory pause led to lower PₐCO₂, by reducing physiological dead space but did not lead to improved PₐO₂ (14).

In this study, we hypothesized that in patients with ALI or ARDS ventilated with small tidal volume, a postinspiratory pause applied over a sufficient period of time would enhance CO₂ elimination and reduce PₐCO₂ by reducing airway dead space and ventilation/perfusion nonhomogeneity at the alveolar level. By applying volumetric capnography during the whole study, we aimed at increased understanding about mechanisms behind effects of a prolonged Tp and dynamics of CO₂ exchange in ARDS.

PATIENTS AND METHODS

Materials. Fifteen consecutive hypercapnic mechanically ventilated patients (PₐCO₂ ≥ 45 mmHg, Table 1), were studied within 48 h after they fulfilled criteria for ALI or ARDS (5).

Sedation was achieved by continuous infusion. Neuromuscular blockade was used in three patients. The level of sedation (modified Ramsay score ≥ 5) and the absence of respiratory effort on the flow-time curve during an end-expiratory pause of 6 s were checked before the beginning of the study. The patients were studied in semirecumbent position when stable with respect to ventilation, he-
Tp0init, Tp20, and Tp0late, respectively. Referring to Fig. 3, at 35 min, Tp was set to 0%. Tp was changed to 20% for the second period.

If needed, endotracheal suction was performed well before the study and was not repeated during data collection. Clinically applied humidification/warming of inspired gas was maintained (heat/moisture exchanger in 10 patients, heated humidifier in 5). All patients had an arterial line.

Exclusion criteria were: age < 18 years, presence of a chest tube, intracranial disease, PaO2/FiO2 < 75 mmHg, [HCO3−] < 18 mmol/L, known severe Chronic Obstructive Pulmonary Disease (FEV1 < 50% predicted) or chronic respiratory insufficiency with long-term oxygen therapy. High age of the patients and, as will be shown, high physiological dead space (VDphys%) indicate a group of patients with a poor prognosis, in accordance with a 53% mortality rate (15).

Patients were ventilated in volume-controlled mode with a constant dead space (Table 2). The ventilator/computer system used for data recording has previously been described (16, 19). Signals representing airway flow, pressure, and CO2 were fed to the A/D converter of a personal computer and sampled at 100 Hz. Compliance of the ventilator tubing was 1.7 ml/cmH2O. Tracheal tube, CO2 analyzer, and heat and moisture exchanger dead space were measured in vitro (apparatus dead space volume was 86 ml if a heat and moisture exchanger was used, 41 ml for a heated humidifier). The Ethics Committee of French Intensive Care Society approved the protocol. Patients' next of kin were informed of the study protocol and gave their consent.

Procedure. Before the study, it was ensured that the extended Tp from 0% to 20% of total breathing cycle duration did not increase intrinsic PEEP by more than 1 cmH2O. Insufflation time (20%), inspiratory flow, respiratory rate, VT, FiO2, and PEEP were kept constant throughout the study.

Three periods were recorded. During the initial period of 30 min, Tp was set to 0%. Tp was changed to 20% for the second period. During the last period Tp was reset to 0%. The periods were denoted Tp0init, Tp20, and Tp0late, respectively. Referring to Fig. 3, at 35 min, the knob on the ventilator panel for setting Tp was reset from 0 to 20%. A postinspiratory pause was thereby introduced already for the ensuing inspiration. At 70 min the knob was turned back to 0% for recording of data for a further 30 min. Signal recordings were performed during this period every 5 min. At 35 and 70 min, the very first breaths during Tp20 and Tp0late were recorded. Ten breaths were analyzed during each recording. Blood gas analysis was performed with GEM Premier 3000 (Instrumentation Laboratory, Barcelona, Spain). Measurements during Tp0init are denoted baseline values.

Data analysis. Data for airway flow, pressure, and CO2 were transferred to a spreadsheet for Excel 2002 (Microsoft, Redmond, WA, USA) and analyzed according to Utman and Jonson (19). The expiratory flow signal was normalized by a correction factor so that expired tidal volume equaled the inspired measured at normal breaths.

Table 2. Characteristics of patients with acute lung injury/acute respiratory distress syndrome

<table>
<thead>
<tr>
<th>No</th>
<th>Age, y</th>
<th>SAPS II</th>
<th>Cause of ALI/ARDS</th>
<th>Underlying Disease</th>
<th>PaO2/FiO2, mmHg</th>
<th>Outcome</th>
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</thead>
<tbody>
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<td>78</td>
<td>37</td>
<td>Pneumonia</td>
<td>Ischemic cardiopathy</td>
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<td>alive</td>
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<td>22</td>
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<td>Pneumonia</td>
<td>Chestx</td>
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<td>6</td>
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<td>Ischemic cardiopathy</td>
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<td>Epilepsy</td>
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<td>Pneumonia</td>
<td>Hypertension</td>
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<td>Hypertension</td>
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<tr>
<td>10</td>
<td>39</td>
<td>55</td>
<td>Inhalation</td>
<td>Chestx</td>
<td>193</td>
<td>dead</td>
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<tr>
<td>11</td>
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<td>57</td>
<td>Inhalation</td>
<td>Cirrhosis</td>
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<tr>
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<td>41</td>
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<td>40</td>
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<tr>
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<td>60</td>
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<tr>
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<td>86</td>
<td>33</td>
<td>Pneumonia</td>
<td>Atrial fibrillation</td>
<td>196</td>
<td>dead</td>
</tr>
</tbody>
</table>

Mean ± SD 67±18 51±18 --- --- 148±25 ---

SAPS, simplified acute physiology score; ALI, acute lung injury; ARDS, acute respiratory distress syndrome.

Table 2. Experimental parameters

<table>
<thead>
<tr>
<th>No</th>
<th>Vt, ml</th>
<th>Ti, s</th>
<th>Peack, cmH2O</th>
<th>Pplat, cmH2O</th>
<th>PEEPe, cmH2O</th>
<th>PEEPot, cmH2O</th>
<th>Vdpot%, % Vt</th>
<th>Vaw%, % Vt</th>
<th>Vaw, ml</th>
<th>VDal%, % Vt</th>
<th>pH</th>
<th>PETco2, mmHg</th>
<th>Pao2, mmHg</th>
<th>Pao2/Fio2, mmHg</th>
<th>MAP, mmHg</th>
<th>HR, min−1</th>
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<tbody>
<tr>
<td>1</td>
<td>489±63</td>
<td>1.4±0.3</td>
<td>31.5±5.7</td>
<td>21.7±4.5</td>
<td>8.8±2.5</td>
<td>9.1±2.5</td>
<td>68.2±7.4</td>
<td>40.3±8.8</td>
<td>194±40</td>
<td>23.8±9.1</td>
<td>7.27±0.07</td>
<td>55±10</td>
<td>113±32</td>
<td>148±45</td>
<td>69±7</td>
<td>85±20</td>
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<td>502±63*</td>
<td>1.4±0.3</td>
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<td>21.7±4.5</td>
<td>8.8±2.5</td>
<td>9.1±2.5</td>
<td>60.8±8.8</td>
<td>33.4±8.8</td>
<td>166±41*</td>
<td>22.8±10.1</td>
<td>7.27±0.07</td>
<td>55±10</td>
<td>113±32</td>
<td>148±45</td>
<td>73±11</td>
<td>87±18</td>
</tr>
<tr>
<td>3</td>
<td>489±63</td>
<td>1.4±0.3</td>
<td>31.3±5.4</td>
<td>21.7±4.5</td>
<td>8.8±2.5</td>
<td>9.1±2.5</td>
<td>67.6±7.6</td>
<td>40.4±8.7</td>
<td>195±39</td>
<td>22.9±8.6</td>
<td>7.27±0.07</td>
<td>55±10</td>
<td>113±32</td>
<td>148±45</td>
<td>70±6</td>
<td>89±18</td>
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</table>

Vvent, ml 489±63 502±63* 489±63 <0.0001
RR, min−1 18±4 18±4 18±4 0.7±0.2
Te, s 3.0±0.6 2.3±0.5 3.0±0.6
(Ti+Tp)/Tot%, % 20 40 20
Ppeak, cmH2O 31.5±5.7 31.3±5.4 31.3±5.6 0.4
Pplat, cmH2O 21.7±4.5 21.7±4.5 21.7±4.5
PEEPe, cmH2O 8.8±2.5 8.8±2.5 8.8±2.5
PEEPot, cmH2O 9.1±2.5 9.1±2.5 9.1±2.5 <0.0001
Vdpot%, % Vt 68.2±7.4 60.8±8.8 67.6±7.6 <0.0001
Vaw%, % Vt 40.3±8.8 33.4±8.8 40.4±8.7 <0.0001
Vaw, ml 194±40 166±41* 195±39 <0.0001
VDal%, % Vt 23.8±9.1 22.8±10.1 22.9±8.6 0.4
PETco2, mmHg 0.8±0.2 0.8±0.2 0.8±0.2
pH 7.27±0.07 7.31±0.08* 7.27±0.08 <0.0001
Pao2, mmHg 55±10 49±9 55±10 <0.0001
Pao2, mmHg 113±32 110±32 116±34 0.2
Pao2/Fio2, mmHg 148±45 145±48 151±45 0.2
MAP, mmHg 69±7 73±11 70±6 0.6
HR, min−1 85±20 87±18 89±18 0.2

Ventilatory and respiratory mechanical parameters, dead spaces, blood gas and hemodynamic parameters were taken at the end of each study period. Values are mean ± SD. Vt, tidal volume; RR, respiratory rate; Ti, inspiratory time including postinspiratory pause; Te, expiratory time; (Ti+Tp)/tot, insufflation and postinspiratory pause as percent of the breathing cycle; PEEPe, external positive end expiratory pressure; PEEPot, external positive end expiratory pressure + intrinsic positive end expiratory pressure; Vdpot%, physiological dead space in percent of Vt; Vaw%, alveolar dead space in percent of Vt; FiO2, fraction of inspired oxygen; MAP, mean arterial pressure; HR, heart rate. *P = 0.001

J Appl Physiol • VOL 105 • DECEMBER 2008 • www.jap.org
Flow rate and its integral volume were then corrected for gas compression in tubing and adjusted to body temperature and pressure, saturated with water vapor (BTSPD). Accordingly, tidal volume was measured as the volume really delivered to the patient, corrected for gas compression in tubing. An unforeseen problem, further discussed below, was that during Tp20, the pressure drop at the end of inspiration caused redistribution from tubing to the lung. Therefore, \( V_T \) increased by about 13 ml or 2.6%. Partial pressure of CO\(_2\) at airway opening was calculated at actual barometric pressure.

Distribution and diffusion of tidal gas in the lung periphery starts at the moment when the interface between “fresh” inspired gas and “used” alveolar gas reaches the respiratory zone of the lung. It ends abruptly when the interface is pushed back into the airways at the start of expiration. Therefore, MDT is the interval from mean time of arrival of partitions of tidal volume in the respiratory zone until start of expiration. It expresses the time available for enhanced diffusion between inhaled tidal volume and resident alveolar gas, as mathematically described by Aboab et al. (2).

Further analysis was based upon the complete single breath test for CO\(_2\) (SBT-CO\(_2\)), which is a loop comprising an expiratory and an inspiratory limb (6). VT was corrected for gas composition in tubing and represents proximal airway dead space (\( V_{Daw,prox} \)). Area \( B \) between alveolar plateau and \( P_{CO2} \) illustrates alveolar dead space. Alveolar plateau was characterized by equation \( P_{CO2} = b + m(ln(V)) \). Its slope was determined at its midpoint.

Upon adding the postinspiratory pause, the change during the very first eight breaths in VT\( _{CO2} \), Slope, MDT, and \( V_{Daw} \) are denoted in \( \Delta VT_{CO2} \), \( \Delta \text{Slope} \), \( \Delta \text{MDT} \), and \( \Delta V_{Daw} \), respectively. The change of \( PaCO_2 \) between the ends of Tp\(_{0init}\) and Tp20 periods is denoted \( \Delta PaCO_2 \).

**Statistical methods.** Data are presented as mean and SD or SE as specified. Friedman’s nonparametric test was used to study the relationship between the quantitative variables at different periods. Differences at the level of \( P < 0.05 \) were considered statistically significant. For statistically significant differences, a Wilcoxon matched pairs signed-rank test was done to compare variables at Tp\(_{0init}\) or Tp\(_{0late}\) and Tp20. Spearman correlation coefficient was used to compare \( \Delta VT_{CO2} \), \( \Delta \text{Slope} \), \( \Delta \text{MDT} \), \( \Delta V_{Daw} \), and \( \Delta PaCO_2 \). Correlation coefficients were considered statistically significant at the 0.05 level, and linear regression was used to establish the relationship between \( \Delta V_{Daw} \) and \( \Delta PaCO_2 \).

**RESULTS**

**Ventilation and hemodynamics.** \( V_T \) was 489 ± 63 ml, corresponding to 7 ± 1 ml/kg of predicted body weight. Peak inspiratory flow rate was 0.7 ± 0.2 l/s. During Tp20, airway pressure fell by 9.6 cmH\(_2O\) during the pause (from peak pressure to plateau pressure, Table 2). Then tube decompression led to an increase of \( V_T \), on average by 13 ± 6 ml (2.6%) (Table 2). MDT changed from 0.21 ± 0.07 s during Tp\(_{0init}\) and Tp\(_{0late}\) to 0.88 ± 0.24 s during Tp20.

Mean airway pressure was 12.4 ± 2.7 cmH\(_2O\) at Tp0 and 15.1 ± 2.9 cmH\(_2O\) at Tp20 (\( P < 0.01 \)). Total PEEP increased by 0.4 cmH\(_2O\) on average during Tp20 because of a slight but significant increase in intrinsic PEEP (Table 2). Resistance and compliance of the respiratory system at Tp20 was 13.5 ± 2.8 cmH\(_2O\) l\(^{-1}\) s\(^{-1}\) and 44.0 ± 11.4 ml/cmH\(_2O\), respectively, yielding an average time constant of 0.59 s. The expiratory time for those 7 subjects who had a respiratory rate above 18 was 1.8 ± 0.2 s at Tp20, i.e., about three time constants. Accordingly, expiration was long enough to allow near-cessation of expiratory flow and low values of auto-PEEP also during Tp20 (Fig. 2). Hemodynamics remained constant during all periods (Table 2).

**CO\(_2\) elimination and dead space.** During Tp20, VT\( _{CO2} \) increased immediately by 28% (14 ± 5 ml) compared with baseline during Tp\(_{0init}\), and then decreased without reaching baseline within the Tp20 period (Fig. 3). During Tp\(_{0late}\), VT\( _{CO2} \) first fell suddenly to below baseline and then returned to baseline. During the whole Tp20, VT\(_{Daw} \) was reduced by 28 ml (\( P < 0.0001 \), Table 2, Fig. 3). The decrease was 2.2 times higher than the increase in \( V_T \). During Tp\(_{0late}\), VT\(_{Daw} \) equaled that during Tp\(_{0init}\) (Fig. 3). At the end of Tp20, VT\(_{Dphys} \) was 11 ± 4% lower than at Tp0, while VT\(_{Daly} \) was unchanged (Table 2).

During Tp20, the slope of the alveolar plateau fell immediately to 65 ± 10% of baseline value (35 ± 19 vs. 53 ± 23 mmHg/l) and then slowly decreased further to 32 ± 17 mmHg/l (Figs. 3 and 4). During Tp\(_{0late}\), the slope returned to baseline.

**Arterial blood gases.** During Tp20, \( PaCO_2 \), fell by 10 ± 3% (from 55 ± 10 to 49 ± 9 mmHg; \( P < 0.0001 \)) (Fig. 5, Table 2). During Tp\(_{0late}\), it returned to baseline. No significant change in \( PaCO_2 \) was observed.

The change in \( PaCO_2 \) from baseline until end of Tp20 (\( \Delta PaCO_2 \), mmHg) correlated with the immediate change in VT\(_{Daw} \) when the pause was instituted (\( \Delta V_{Daw, \text{ml}} \) (\( p = 0.60, P < 0.02 \)).
No correlations were found between $\Delta PaCO_2$ and $\Delta V_{Daw}$, $\Delta$Slope, or $\Delta MDT$.

A comparison was made between 8 patients with respiratory rate $\leq 18$ and 7 patients with rates $> 18$. During Tp20, in both groups, total PEEP increased by 0.4 cmH$_2$O, $PaCO_2$ decreased by 6 mmHg and $V_{Daw}$ by 28 ml.

**DISCUSSION**

During volume-controlled ventilation with small tidal volume and constant inspiratory flow in hypercapnic patients with ALI or ARDS, this study shows that a 20% postinspiratory pause time leads to a 10% decrease in $PaCO_2$ after 30 min, secondary to enhanced CO$_2$ elimination. The main explanation of increased CO$_2$ elimination is lower $V_{Daw}$.

Our results agree with previous findings that a postinspiratory pause enhances CO$_2$ elimination in healthy or surfactant-depleted animals (4, 10, 11, 20) and with those obtained in surfactant-depleted pigs in which inspiration at pressure-controlled ventilation was prolonged (13). They also agree with results in patients with ALI/ARDS (2, 8, 12, 14).

The main effect of the postinspiratory pause on CO$_2$ elimination reflects an instantaneous and continuous reduction of $V_{Daw}$ that was immediately abolished during Tp$_{0late}$. According to the MDT concept, this reflects enhanced diffusion of CO$_2$ from alveoli towards airways during the pause. As the decrease of $V_{Daw}$ was more than two times larger than the small increase in VT, the effect on $PaCO_2$ was about two-thirds caused by the change in $V_{Daw}$. As further discussed below, the ultimate changes in $PaCO_2$ could not be estimated during the test periods of 30 min. During Tp20, decompression of gas in the tubing during the pause reflects the product between (P$_{peak} - P_{plat}$) and tube compliance and was calculated to 16 ml. The increase in VT during Tp20 can accordingly be fully explained by gas decompression. The lower slope of the alveolar plateau during Tp20 indicates that a more even alveolar ventilation-perfusion relationship may have contributed to enhanced CO$_2$ elimination. The slope of the alveolar plateau mainly reflects nonsynchronous emptying of lung compartments with different ventilation/perfusion ratios (7). The immediate and nearly stable drop in slope during Tp20 (Fig. 3) suggests more even alveolar ventilation due to diffusion within...
At completely steady state, the change in PaCO2 should equal the change in initial effect on rate of CO2 elimination, in this reasonable explanation is that CO2 stores in the body of this study represented by change in VtCO2 by 28%. That VtCO2 fell rapidly and then very slowly. The reasonable explanation is that CO2 stores in the body of this group of patients are large and distributed among compartments with very different time constants for equilibration of CO2. Severe cardiopulmonary disease, increased extravascular liquid space, and poor circulation in peripheral edematous regions are likely causes of slow equilibration of CO2. Similar conclusions have previously been drawn by Henneberg et al. (9).

During Tp20, the fall in PaCO2 of 10% is the result of enhanced CO2 elimination as expressed by enhanced VtCO2. However, the full effect on PaCO2 of a change in efficient alveolar ventilation cannot be observed until CO2 stores in the body have been equilibrated, as discussed by Taskar et al. (17). At completely steady state, the change in PaCO2 should equal the change in initial effect on rate of CO2 elimination, in this study represented by change in VtCO2 by 28%. That VtCO2 did not return to baseline indicates that a steady state was not achieved during Tp20, as discussed. Accordingly, the observed change in PaCO2 probably underestimates the full effect of a postinspiratory pause of 20%. To limit confounding factors due to spontaneous changes of metabolism and hemodynamic and other physiological parameters, the study periods were limited to 30 min as a compromise against a desirable steady state during each period. Aström et al. observed in pigs at health and with a model of ALI that after 30 min of ventilation with a different MDT, PaCO2 changed by 0.75 of what was expected on the basis of immediate change in VtCO2 (4). In the present study this fraction was only 10/28, i.e., 0.36. The difference may represent a much slower equilibration time in the gravely sick patient population. In the study of Mercat et al. PaCO2 changed by only 5% when the pause was prolonged, although an equilibration time of 1 h left more time available for equilibration of CO2 stores in the body (14). In their study we estimate that MDT doubled from about 0.6 s to 1.3 s at prolonged Tp, while MDT in ours increased more than fourfold, from 0.21 to 0.88. The lower change in PaCO2 in Mercat’s study can be explained by the lower relative change in MDT, considering that the effect of variable MDT according to Aström et al. is nonlinear, i.e., more important towards lower MDT values.

At Tp20, shortening of expiration time from 80% to 60% led to a small but significant increase in intrinsic PEEP (<0.5 cmH2O). PEEP tends to increase VDaw and may hamper CO2 elimination through hemodynamic effects. Increased PEEP increases in itself VDaw due to airway distension (6, 18). Accordingly, the small increase in total PEEP might have attenuated the observed decrease in VDaw and enhanced CO2 elimination at Tp20. In spite of higher total PEEP at Tp20, peak pressure remained unchanged. This might indicate that some recruitment took place during Tp20. However, PaO2 did not increase as was also observed in the study of Mercat et al. (14). One may speculate that positive effects of recruitment were balanced by negative effects caused by diversion of blood flow to collapsed lung, thereby increasing shunt. The two groups with respiratory rate (RR) ≤ 18 and RR > 18 showed equal response to a prolonged pause, which finding may reflect a low variation in RR in the present material.

In this experimental study, we aimed at unchanged tidal volume and used CO2 turnover PaCO2 as indicators of more efficient ventilation caused by a postinspiratory pause. In clinical practice, the utility of more efficient gas exchange would rather be to decrease tidal volume for enhanced lung protection. This could, depending on circumstances, be applied so as to either reduce peak pressure to limit barotrauma or to increase PEEP to stabilize lung recruitment. Notably, a postinspiratory pause is one of several options to lower VT. Another is an increase in respiratory rate. At high rates, MDT becomes shorter. Aström showed a steep decline in CO2 exchange at...
low MDT values (4). At high respiratory rates, it may therefore be important to use a pause to adequately prolong MDT.

Conclusion. Postinspiratory pause has a great influence on CO₂ exchange not only for breaths immediately following resetting. A prolongation of Tp leads to a decrease of PaCO₂, as hypothesized. The effects of Tp are mostly due to diffusion of CO₂ into airways but also to a more homogenous ventilation/hypothesized. The effects of Tp are mostly due to diffusion of CO₂ into airways but also to a more homogenous ventilation/perfusion at the alveolar level. The mode of inspiratory gas delivery should be taken into account, e.g., in the context of low tidal volume ventilation (1). The ancillary observation of a long equilibrium time for PaCO₂ in ALI, indicating that 30 min is too short for full appreciation of the effect on PaCO₂ of a change in ventilation, merits further studies.

GRANTS

This study was supported by the Swedish Heart Lung Foundation and was financed by Chancellerie des Universités de Paris.

REFERENCES


