Prone Position in Artificially Ventilated Chronic Obstructive Pulmonary Disease Patients Assessment of Lung Mechanics

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Abstract

The purpose of this study was to compare the effect of prone position on the lung mechanics and blood gases exchange in mechanically ventilated chronic obstructive pulmonary disease (COPD) patients relative to the supine position.

Methods: Thirty mechanically ventilated patients with acute respiratory failure type [1] and on top of chronic obstructive pulmonary disease with a volume controlled mode were put in prone position for one hour then turned back to supine position. Arterial samples for blood gases analysis "ABG" and Lung mechanics parameters were taken before turning, 30 and 60 minutes after turning the patient to prone position and 30 minutes after returning the patient to supine position.

Results: After one hour of pronation the PIP increased from 33.23 ± 6.8 to 36.4 ± 6.5, the Pmean increased from 10.1 ± 2.3 to 11.74 ± 2.8, Cdyn decreased from 22.47 ± 3.9 to 20.77 ± 3.2, the Pplate decreased from 24.08 ± 4.1 to 21.8 ± 3.9, the Cstat increased from 33.36 ± 5.7 to 42.04 ± 8.9, the PaO2 increased from 94.83 ± 38.8 to 105 ± 39.4 and the PaCO2 decreased from 55.1 ± 12.02 to 50.6 ± 11.08.

Conclusion: Prone position showed significant decrease in plateau pressure and increase in static compliance, it also improves oxygenation and CO2 wash relative to supine position. The peak inspiratory pressure, mean airway pressure, and intrinsic positive end expiratory pressure increase in prone position. The effect of prone position on oxygenation and CO2 wash lasts for at least one hour after turning the patients to the supine position.

Key Words: Mechanical ventilation – COPD – Prone position – Lung mechanics – Gas exchange.

Introduction

CHRONIC obstructive pulmonary disease (COPD) is now recognized as an inflammatory disease of the airways. The new American Thoracic Society (ATS)/European Respiratory Society (ERS) definition reflects these scientific advances: "Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and is associated with an abnormal inflammatory response of the lungs to noxious particles or gases, primarily caused by cigarette smoking". The diagnosis of COPD is suggested by findings on history and/or physical examination and is confirmed by laboratory tests, usually with a supportive risk factor (eg, of familial COPD and/or of cigarette exposure). Spirometry is indispensable in establishing the diagnosis because it is a standardized and reproducible test that objectively confirms the presence of airflow obstruction [1].

In the presence of high resistance to expiratory flows and short expiratory times, the respiratory system is unable to return to its resting volume at the end of exhalation. The positive pressure within regions of hyper inflated lung raises the mean intrathoracic pressure and causes the inspiratory muscles to operate at a higher than resting lung volume. Thus, dynamic hyperinflation places the respiratory muscles at a considerable mechanical disadvantage [2] and further impairs respiratory muscle function. Intrinsic positional end expiratory pressure (PEEP) also imposes a substantial inspiratory threshold load.

Several factors contribute to development of gas exchange abnormalities in patients with obstructive lung disease. Airway obstruction produces regional hypoventilation that produces ventilation/perfusion mismatch and hypoxemia. Loss of the capillary bed due to emphysema or compression of pulmonary capillaries by over inflated alveoli also tends to increase dead space, and this wasted ventilation further compromises the ability of the respiratory muscles to provide adequate ventilation [3].

Turning and positioning of critically ill patients are well-accepted nursing activities, with the
primary purpose being to relieve pressure, improve patients’ comfort and aid pulmonary secretions drainage [4]. However, body positioning of critically ill patients may have a profound effect on arterial oxygenation, which is reflected by the oxygen saturation level in the blood [8]. Optimal oxygenation depends on the match of ventilation (V) to perfusion (Q) ratio, with optimal oxygenation, occurring when the best ventilated areas are best perfused [6].

The use of the prone position (PP) was first advocated over two decades ago as a strategy for improving oxygenation in patients with acute bilateral lung injury disease, pneumonia and adult respiratory distress disease (ARDS) [7-9]. Despite the numerous studies demonstrating a significant improvement in oxygenation, prone positioning is still underused [10].

Altering the patient’s position to prone improve oxygenation by reducing the ventilation/perfusion (V/Q) mismatch and decreases the shunt [11,12]. The Prone position also maintains perfusion, as it does not decrease blood flow to the recruited regions. In other words, the PP improves the V/Q match and decreases the shunt [11]. Changes in pleural pressures favoring alveolar recruitment in the dorsal areas in the prone position may also contribute to the increased oxygenation, however, this is not yet fully understood. It is also suggested that the prone position aids pulmonary secretions to be mobilized and drained aiding ventilation.

Decreased atelectasis and more uniform inflation should result in more homogenous and increased average alveolar septal tension the latter is transmitted to airway walls via connective tissue cables [13], resulting in outward wall traction and airway caliber increase [14].

In this study we tried to compare the effects of prone position on the lung mechanics and blood gases exchange in mechanically ventilated chronic obstructive pulmonary disease (COPD) patients relative to the supine position.

Material and Methods

Thirty patients with acute respiratory failure type II "hypercapnic respiratory failure" due to chronic obstructive pulmonary disease (COPD) admitted to the intensive care unit (ICU) Cairo University (Egypt) were included in the study.

All Patients were orotracheally intubated and mechanically ventilated because of acute respiratory failure on top of COPD, with chest radiographs excluding other etiologies e.g. pneumothorax.

Patients with left ventricular failure, lobar atelectasis, pulmonary embolism, pulmonary edema, haemodynamic instability and new or additional administration of inotrops, vasodilators or anti-arrhythmic treatment.

All patients were subjected to clinical examination with special consideration to haemodynamic parameters, specially blood pressure, wheezing, bronchial breathing, prolonged expiration, barrel chest, use of accessory muscles of breathing, cyanosis, evidence of right heart failure, and peripheral edema.

Laboratory investigations were done with special emphasis on complete blood count (CBC), electrolytes levels "sodium, potassium, calcium, and magnesium", urea, creatinine, SGOT, SGPT, billirubin "total and direct", albumin and serial blood gases analysis was done every 12 hours and whenever needed.

Admission and follow-up ECG was done with Special consideration to the presence or development of new arrhythmia.

Chest X-ray was done to assist the diagnosis of COPD like signs of hyperinflation flattened diaphragm on the lateral chest film, and to detect any exclusion criteria.

Anesthesia and neuromuscular blockade were induced and maintained throughout the study with propofol and cisatracurim if needed respectively.

Baseline ventilation settings: Volume control mode with Tidal volume 7ml/Kg L, respiratory rate 12, inspiratory time to total respiratory cycle length ratio 0.25, inspiratory flow 40 L/M, plateau pressure time 0.2 s, FIo2 0.5, Positive End Expiratory Pressure (PEEP) 0cm.

Patients were put in supine position, and then be turned to the prone position for 60 min. The patients then were turned back to supine position again.

Arterial samples for blood gases analysis "ABG" and lung mechanics parameters were taken before turning, 30 and 60 minutes after turning the patient to prone position and 30 minutes after returning to supine position. Parameters included Peak airway pressure, Plateau pressure, Mean airway pressure, Static lung compliance (C_stat), Dynamic compliance (Cdyn) and Intrinsic positive end expiratory pressure (Auto-PEEP).
Data analysis:

All data were collected retrospectively as part of the ICU database.

Categorical data are displayed as percentages. Continuous data are reported as mean value ± standard deviation (SD) and range. Comparisons were performed with unpaired student, t test for continuous. A probability value <0.05 was considered as significant. Professional statistical package for social science (SPSS version 13) computer software was used for data analysis.

Results

Our study was carried out on 30 patients with acute on top of chronic type II respiratory failure due to COPD and all were subjected to mechanical ventilation.

Patients characteristics:

The age of the patients included in the study ranged from 40 to 85 years with a mean age (64.13±11.132) years.

The sex distribution was 80% (24 patient) males and 20% (6 patients) females.

Data collected for the 30 patients from the ventilator readings and the results of the ABG were statistically managed in the form of mean and standard deviation for each parameter in both positions. The four readings "supine before pronation (supine 1), prone position after 30 minutes of pronation (prone 1), prone position after 60 minutes of pronation (prone 2), and supine position after 30 minutes turning back supine" are included in table (1), where: PIP (Peak inspiratory pressure), P mean (mean airway pressure), P pl ate (Plateau pressure), C stat (Static lung compliance), C dyn (Dynamic compliance), PEEP i (Intrinsic positive end expiratory pressure), (PaO 2 ) and (PaCO 2 ).

Comparing prone (1) to prone (2):

There was no statistically significant difference in pulmonary pressures and mechanics in patients in prone (1) and prone (2), but there was a statistically significant increase in oxygen pressure and decrease in Carbon dioxide pressure in prone (2) relative to prone (1), (Table 2), (Figs. 1,2).

Comparing supine (1) to prone (2):

Regarding pulmonary pressures and mechanics, there was statistically significant increase in PIP, P mean , P pl ate and C stat in prone (2) relative to supine (1). It also shows that there is statistically significant decrease in P pl ate and C dyn in prone (2) relative to supine (1).

Comparing supine (1) to prone (2):

Regarding pulmonary ventilation, there was a statistically significant increase in oxygen pressure and decrease in Carbon dioxide pressure in prone (2) relative to supine (1), (Table 3 & Fig. 3).

Comparing prone (2) to supine (2):

There was statistically significant increase in PIP, P mean , P pl ate and C stat in prone (2) relative to supine (2). It also shows that there is statistically significant decrease in P pl ate and C dyn in prone (2) relative to supine (2).

Comparing supine (1) to supine (2):

Pulmonary pressures and mechanics showed statistically significant decrease in P pl ate in supine (2) relative to supine (1). There was statistically non-significant increase in PIP, P mean , P pl ate and C stat in supine (2) relative to supine (1) and statistically non-significant decrease in C dyn in supine (2) relative to supine (1).

Comparing prone (2) to supine (2):

Regarding pulmonary ventilation showed a statistically significant increase in oxygen pressure and statistically significant decrease in Carbon dioxide pressure in supine (2) relative to supine (1), (Table 5).

<table>
<thead>
<tr>
<th>Table (1): Collected data in the 4 positions of the study.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine (1)</td>
</tr>
<tr>
<td>PIP</td>
</tr>
<tr>
<td>P mean</td>
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<tr>
<td>P plate</td>
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<td>C stat</td>
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<td>C dyn</td>
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<tr>
<td>PEEP i</td>
</tr>
<tr>
<td>PaO 2</td>
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<tr>
<td>PaCO 2</td>
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</tbody>
</table>

Table (2): Comparison between prone (1) and prone (2) regarding pulmonary pressures, mechanics, and ventilation parameters.

<table>
<thead>
<tr>
<th>Prone (1)</th>
<th>Prone (2)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PIP</td>
<td>36.36±6.5</td>
<td>36.4±6.5</td>
</tr>
<tr>
<td>P mean</td>
<td>11.66±2.7</td>
<td>11.742.8</td>
</tr>
<tr>
<td>P plate</td>
<td>22±4.1</td>
<td>22±3.8</td>
</tr>
<tr>
<td>C stat</td>
<td>41.3±8.9</td>
<td>42.04±8.9</td>
</tr>
<tr>
<td>C dyn</td>
<td>20.7±3.4</td>
<td>20.8±3.2</td>
</tr>
<tr>
<td>PEEP i</td>
<td>6.67±2.3</td>
<td>6.77±2.5</td>
</tr>
<tr>
<td>PaO 2</td>
<td>102.67±39.35</td>
<td>105±39.4</td>
</tr>
<tr>
<td>PaCO 2</td>
<td>55.1±11.38</td>
<td>50.63±11.08</td>
</tr>
</tbody>
</table>

NS: Non significant. *: Significant. **: Highly significant.
Figure 1: Comparing the mean values of pulmonary pressures and mechanics between prone (1) and prone (2).

Table 3: Comparison between supine (1) and prone (2) regarding pulmonary pressures, mechanics, and ventilation parameters.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Supine (1)</th>
<th>Prone (2)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PIP</td>
<td>33.23±6.8</td>
<td>36.4±6.4</td>
<td>0.02*</td>
</tr>
<tr>
<td>PeO2</td>
<td>24.08±4.1</td>
<td>21.8±3.9</td>
<td>&lt;0.01**</td>
</tr>
<tr>
<td>Pstat</td>
<td>33.36±5.7</td>
<td>42.04±8.9</td>
<td>&lt;0.01**</td>
</tr>
<tr>
<td>Pdyn</td>
<td>22.47±3.9</td>
<td>20.77±3.2</td>
<td>0.01*</td>
</tr>
<tr>
<td>PEEPi</td>
<td>5.57±1.8</td>
<td>6.77±2.4</td>
<td>0.02*</td>
</tr>
<tr>
<td>PaO2</td>
<td>94.8±3.8</td>
<td>105±39.4</td>
<td>&lt;0.01**</td>
</tr>
<tr>
<td>PaCO2</td>
<td>55.1±12.02</td>
<td>50.6±11.08</td>
<td>&lt;0.01**</td>
</tr>
</tbody>
</table>

NS: Non significant. *: Significant. **: Highly significant.

Figure 2: Comparing the mean values of ventilation parameters between prone (1) and prone (2).

Figure 3: Comparing the mean values of pulmonary pressures and mechanics between supine (1) and prone (2).

Figure 4: Comparing the mean values of pulmonary pressures and mechanics between prone (2) and supine (2).

Table 4: Comparison between prone (2) and supine (2) regarding pulmonary pressures, mechanics, and ventilation parameters.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Prone (2)</th>
<th>Supine (1)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
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<td>PIP</td>
<td>36.4±6.5</td>
<td>33.6±6.8</td>
<td>0.02*</td>
</tr>
<tr>
<td>PeO2</td>
<td>11.7±2.3</td>
<td>10.36±2.4</td>
<td>0.03*</td>
</tr>
<tr>
<td>Pstat</td>
<td>22.3±3.8</td>
<td>23.8±3.9</td>
<td>0.01*</td>
</tr>
<tr>
<td>Pdyn</td>
<td>42.04±8.9</td>
<td>33.8±4.9</td>
<td>&lt;0.01**</td>
</tr>
<tr>
<td>PEEPi</td>
<td>20.8±3.2</td>
<td>22.28±3.7</td>
<td>0.02*</td>
</tr>
<tr>
<td>PaO2</td>
<td>105±39.4</td>
<td>96.8±35.11</td>
<td>&lt;0.01**</td>
</tr>
<tr>
<td>PaCO2</td>
<td>50.6±11.08</td>
<td>52.57±11</td>
<td>0.02*</td>
</tr>
</tbody>
</table>

NS: Non significant. *: Significant. **: Highly significant.

Table 5: Comparison between supine (1) and supine (2) regarding pulmonary pressures, mechanics, and ventilation parameters.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Supine (1)</th>
<th>Supine (2)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PIP</td>
<td>33.23±6.8</td>
<td>33.6±6.8</td>
<td>0.2 (NS)</td>
</tr>
<tr>
<td>PeO2</td>
<td>10.14±2.3</td>
<td>10.36±2.4</td>
<td>0.3 (NS)</td>
</tr>
<tr>
<td>Pstat</td>
<td>24.08±4.1</td>
<td>23.8±3.9</td>
<td>0.03*</td>
</tr>
<tr>
<td>Pdyn</td>
<td>33.36±5.7</td>
<td>33.8±4.9</td>
<td>0.1 (NS)</td>
</tr>
<tr>
<td>PEEPi</td>
<td>22.47±3.9</td>
<td>22.28±3.7</td>
<td>0.2 (NS)</td>
</tr>
<tr>
<td>PaO2</td>
<td>94.8±3.8</td>
<td>96.8±35.11</td>
<td>0.02*</td>
</tr>
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<td>52.57±11</td>
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</tr>
</tbody>
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Discussion

Chronic obstructive pulmonary disease (COPD) exacerbations is associated with increased bronchoconstriction, which increases the work of breathing and can precipitate respiratory failure. This is especially likely in those with more severe underlying disease and a poor respiratory reserve [15].

Treatment with mechanical ventilation is performed in an attempt to correct the deteriorating gas exchange that is unresponsive to conservative measures by:

a- Improving hypoxaemia that has not been corrected with supplemental oxygen delivered by nasal cannula or face mask.
b- Reversing respiratory acidosis that has resulted from the underlying disease or from uncrirical application of oxygen.

c- Relieving respiratory distress until the primary disease process reverses or improves. Further targets are the improvement of clinical manifestations of respiratory failure e.g. dyspnoea, tachypnoea, accessory muscle recruitment, and paradoxical motion of the ribcage and abdomen [16].

Ventilation of patients with Adult Respiratory Distress Syndrome (ARDS) in the prone position has repeatedly been shown to improve the arterial oxygenation and to have few unwanted side effects [17].

The alveolar dimensions depend on the transpulmonary pressure (alveolar pressure-pleural pressure) [18]. Previous studies [19,20] have demonstrated that:

I- The pleural and transpulmonary pressure gradient becomes more homogeneous in the prone position than in the supine, so there is less difference between the non-dependent and dependent regions.

II- The prone position may allow the lungs to fit more uniformly into the thorax such that pleural pressure becomes less positive in the lung regions near to the vertebra than in the supine position.

III- A nongravitational gradient in perfusion has been observed in the prone position, i.e., more perfusion in the lung regions near the vertebra.

IV- Moving into the prone position may reverse the distribution of the lung weight, i.e., more weight is superimposed on the alveoli in ventral regions, while less weight is superimposed on the alveoli in the dorsal regions. These are believed to be important mechanisms of alveolar recruitment in the prone position [21].

Prone position also, reduces lobar interdependence and promotes the recruitment of juxtadiaphragmatic lung regions by decreasing the chest wall compliance through a limitation of the expansion of the cephalic parts of the thorax [22]. The re-aeration of these lung areas involves an increase in alveolar volume [23]. Cardiac and abdominal compression appears to be the main factors causing loss of dorsal and caudal lung aeration [24]. Decreased of intra-abdominal pressure and relief of abdominal compression in the prone position could possibly be an additional factor that increases the alveolar volume [25]. Previous mechanisms help lung recruitment, making the lung able to tolerate larger volumes of air at the same pressure level, and so increasing the lung static compliance.

Chiumello, et al. in a study on 11 patients with ARDS ventilated in prone position, they demonstrated that prone position increases respiratory system compliance and mean airway pressure, but the compliance decreases when thoraco-pelvic support is applied [26].

Similar results were obtained by Galiatsou, et al. who conducted a study on 22 patients with lobar pneumonia or Adult Respiratory Distress Syndrome. They showed that the prone position increases respiratory system compliance relative to supine position in lobar pneumonia, but there was no change in compliance in ARDS. They suggested that PP reduces lobar interdependence and promotes the recruitment of juxtadiaphragmatic lung regions.

On the other hand Pelosi, et al. in a study on 16 patients with ARDS concluded that there was no significant change between prone position and supine position regarding respiratory system static compliance. He refers to the fact that limited movement in chest wall secondary to limitation of the sternal movement decreases the lung compliance.

Regarding lung mechanics, our results showed that prone positioning increased the static compliance and decreased the plateau pressure relative to supine position, while there was no significant difference in compliance between supine (1) and supine (2).

The difference between our results and the previous results can be explained by noticing that Pelosi’s study was conducted on patients with ARDS while our study was conducted on chronic obstructive pulmonary disease patients. The chest wall in emphysematous COPD patients becomes nearly in neutral position, as loss of lung recoil pressure increases the neutral position of respiratory system, and this may be enhanced by accompanying reduction in chest wall recoil pressure so the sternal movement is limited, especially during COPD.

Mentzelopoulos, et al. conducted a study on 10 chronic obstructive pulmonary disease patients with acute respiratory failure on mechanical ventilation in prone position. They reported that prone position increased static compliance, and they suggested that probable contributory factors include increased ribcage elastance and elimination of lung compression by the heart, resulting in increased static lung compliance, attenuated lung inflation.
gradient, and more homogenous regional and increased total effective alveolar ventilation versus supine [26,27].

However, the results of our study do not match those of Masoud, et al. who conducted a study on 30 patients with acute on top of chronic respiratory failure due to chronic obstructive pulmonary disease. Patients were subjected to volume controlled, pressure controlled, and pressure support in three different positions: Supine, semi setting and prone, they concluded that there was no significant difference in static compliance between prone and supine position [29]. The difference between our results and these results can be explained by noticing that the study was conducted while the patients were awake, and so the effect of voluntary breathing and straining can not be excluded. Also only 10 patients were put on volume controlled mode of ventilation during prone positioning, the small study number may also affect the results.

Our results showed also that there was an increase in peak airway pressure and decrease in dynamic compliance in prone position relative to supine position. Shifting from the supine to the prone position resulted in a significant reduction in the compliance of the thoracoabdominal cage. Indeed, the compliance of the rib cage component of the chest wall is known to be nonhomogeneous, the ventral part (sternal) presenting a "larger freedom to move" than the dorsal part (vertebral). The dishomogeneity is further enhanced in the supine position where movement remains unimpeded in only the ventral part (the dorsal part of the chest lies on the bed). In the prone position, the stiffer component of the rib cage (dorsal part) is free to move, whereas movement of the more compliant (ventral part) becomes impeded by lying on the bed. This results in a decrease in net rib cage compliance. Previous mechanisms can result in the increase in peak airway pressure and so the increase or decrease in dynamic compliance. Our results regarding peak airway pressure matched those of Masoud, et al.

Mentzelopoulos, et al. found that there was insignificant decrease in dynamic compliance of the respiratory system [26,27], this may be due to the relatively small number of patients in the study.

Regarding gas exchange, our results showed that prone positioning improves arterial oxygenation and carbon dioxide washing relative to supine position. Since the distribution of perfusion is largely gravity independent and the largest proportion of the perfusion goes through the dorsal lung regions in both supine and prone position. Positive pressure ventilation, regardless of the position, reduces the vertical perfusion gradient with the patient in the prone position and amplifies the gradient in the supine position [30], and so a larger proportion of perfusion distributes to the well ventilated dorsal regions in the prone position and a smaller amount of desaturated blood perfuses the poorly and/or non aerated ventral lung regions in prone position and so better ventilation perfusion matching.

Our results matched with Mentzelopoulos, et al. who reported that prone positioning improves arterial oxygenation, and augment CO₂ elimination versus supine position, as pronation augments ventilation perfusion (V/Q) matching and CO₂ elimination versus supine position [26,27].

Galiatsou, et al. found that the prone position improves both oxygenation and CO₂ elimination as compared to supine position significantly in lobar pneumonia while in Adult Respiratory Distress Syndrome there was significant improvement in oxygenation, and non-significant improvement in CO₂ elimination, this occurs due to the re-aeration of the injured lung involves first an increase in alveolar volume and displacement of the gas-liquid interface from the alveolar ducts to the alveolus, and subsequent translocation of the edematous fluid to the interstitium [30].

Chatte, et al. conducted a study on 32 patients with non-cardiogenic respiratory failure, they reported that prone position improves arterial oxygenation, and augments CO₂ elimination versus supine position [31]. Masoud, et al. in the study on 30 COPD patients reported a significant difference in CO₂ elimination with prone position but no significant change in oxygenation [29].

Chiumello, et al. reported that prone position improve arterial oxygenation, but does not affect the CO₂ elimination [32]. PELOSI, et al. also reported that prone positioning improves arterial oxygenation, but does not affect the CO₂ elimination [28]. Both previous studies were conducted on patients with Adult Respiratory Distress Syndrome and patient's PaCO₂ in both was nearly in the normal range, this fact may explained why the change in the PaCO₂ was not statistically significant, while with high PaCO₂ levels in COPD patients, changes may be more significant.

In our study, the prone positioning was associated with improved gas exchange when compared with the supine position, this is in line with the results of previous studies. Whereas all these studies...
consistently reported an improvement of PaO₂ and a decrease in global shunt fraction in the prone position, our results also showed an improvement of PaCO₂.

Because body temperature was not different between the two positions and tidal volume and respiratory rate were kept constant, the improvement of PaCO₂ suggests that ventilation was more efficient in the prone position. This may be due to that regional specific alveolar ventilation was more uniform in the prone position, indicating a more homogeneous turnover of alveolar gas of perfused units in this position, and so improved ventilation-to-perfusion matching in the prone position [33].

The effect of one hour prone positioning (on PIP, PEEP₁, P_mean, C_dyn, and C_stat) was not lasting after turning the patient back to supine position relative to readings during supine position before turning, while the effect on P_plat oxygenation and carbon dioxide washing lasted after turning the patient back to supine position relative to readings during supine position before turning.

The improvement in ventilation/perfusion matching occurs with prone positioning that we pointed to, the recruitments of the lung happens during prone positioning, and the drainage of retained secretions can explain the maintained decrease in plateau pressure and the improvement in gas exchange in the form of increased oxygenation and CO₂ elimination after repositioning in the supine position.

In this regard Mentzelopoulos, et al. (2003) concluded that regarding arterial oxygenation, pronation-related benefits versus supine seem to be maintained for at least 30-65min, whereas the reverse is true for CO₂ elimination and static lung compliance that was explained by indicating that partial reversal of pronation augmented alveolar homogeneity and V/Q matching, this is consistent with enhanced dorsal lung region recruitment during tidal lung inflation versus supine because of the preceding pronation effects [26]. Again the relatively small number of patients in the study can explain that the persistent decrease in PaCO₂ was insignificant.

Chatte, et al. found that in 10 of the 23 respondents (43%) who completed the 4 h prone trial, the PaO₂/FI(O)2 returned to its starting value when patients were repositioned supine while in 13 of the 23 (57%) improvement persisted [31].

Pelosi, et al. concluded that when returning to the supine position oxygenation slightly declined, but no significant differences were observed when compared with baseline or the prone position; but they also commented "because of the small population, we cannot exclude a type II error. Once again, these average values were derived from variable individual responses. Different oxygenation responses when returning to the supine position have been previously described, including both a reversal of oxygenation improvement and a maintenance of improved oxygenation" [28].

Summary and conclusion:

Prone positioning increases: The PIP, PEEP₁, C_stat, and P_mean decreases: P_plat, and improves arterial oxygenation and carbon dioxide washing relative to supine position. The effect of one hour prone position on P_plat, oxygenation and carbon dioxide washing lasts after turning the patient back to supine position relative to readings during supine position before turning.

Different mechanisms share in the effect of the prone position, but we can consider that the lung recruitment is the main mechanism causing the prone position effects.

References


