

Prone Position in Management of Covid 19 Patients

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Abstract: Coronavirus disease 2019 (COVID-2019) is caused by a novel coronavirus known as Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) and was identified as a pandemic by the World Health Organization (WHO) on March 11, 2020. This virus was first identified in the respiratory tract of patients with pneumonia in Wuhan, Hubei China, in December 2019 which was then indicated as a newly identified β -coronavirus (nCoV). Recent observations suggest that respiratory failure in COVID-19 is not driven by the development of the acute respiratory distress syndrome (ARDS) alone, but that (microvascular) thrombotic processes may play a role as well. During prone positioning, ventilation is improved due to changes in pleural pressure (PPL) and the amount of lung atelectasis present. When a patient with ARDS is placed prone, the dorsal lung is no longer subject to high PPL and dorsal lung atelectasis decreases. Although there is limitation of data but recent studies have showed that with correct patient selection, timely initiation and duration of patient's placement in this position can all affect the effectiveness of this treatment method.

Keywords: COVID 19 (Coronavirus disease 2019), SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2), ARDS (acute respiratory distress syndrome), Atelectasis, PPL(Lung pleural pressure).

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I. Introduction

Coronavirus disease 2019 (COVID-2019) is caused by a novel coronavirus known as Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) and was identified as a pandemic by the World Health Organization (WHO) on March 11, 2020 [1]. As of April 12, 2020, more than 1.8 million people were confirmed to have been infected and tested positive for COVID-19, with over 114,000 deaths worldwide [2]. This virus was first identified in the respiratory tract of patients with pneumonia in Wuhan, Hubei China, in December 2019 which was then indicated as a newly identified β -coronavirus (nCoV) [3,4]. Recent observations suggest that respiratory failure in COVID-19 is not driven by the development of the acute respiratory distress syndrome (ARDS) alone [5], but that (microvascular) thrombotic processes may play a role as well. There is a strong association between D-dimer levels, disease progression and chest CT features suggesting venous thrombosis [6].

Table 1. ARDS Berlin definition.

The Berlin definition of acute respiratory distress syndrome

Timing	Within 1 week of a known clinical insult or new or worsening respiratory symptoms
Chest imaging ^a	Bilateral opacities — not fully explained by effusions, lobar/lung collapse, or nodules
Origin of edema	Respiratory failure not fully explained by cardiac failure or fluid overload. Need objective assessment (e.g., echocardiography) to exclude hydrostatic edema if no risk factor present
Oxygenation ^b	
Mild	200 mmHg < PaO ₂ /FIO ₂ ≤ 300 mmHg with PEEP or CPAP ≥ 5 cmH ₂ O ^c
Moderate	100 mmHg < PaO ₂ /FIO ₂ ≤ 200 mmHg with PEEP ≥ 5 cmH ₂ O
Severe	PaO ₂ /FIO ₂ ≤ 100 mmHg with PEEP ≥ 5 cmH ₂ O

Abbreviations: CPAP, continuous positive airway pressure; F_IO₂, fraction of inspired oxygen; PaO₂, partial pressure of arterial oxygen; PEEP, positive end-expiratory pressure; ^aChest radiograph or computed tomography scan; ^bIf altitude is higher than 1,000 m, the correction factor should be calculated as follows: [PaO₂/FIO₂ (barometric pressure/760)]; ^cThis may be delivered noninvasively in the mild acute respiratory distress syndrome group.

During prone positioning, ventilation is improved due to changes in pleural pressure (PPL) and the amount of lung atelectasis present. PPL is the sum of all forces acting to compress the alveolus and includes the weight of tissue above the alveolus and the transmitted pressure across the diaphragm from the abdomen. Simplistically, an alveolus will remain open when the intra-alveolar pressure exceeds PPL. When a patient with ARDS is placed prone, the dorsal lung is no longer subject to high PPL and dorsal lung atelectasis decreases. Conversely, the ventral lung units are exposed to a higher PPL and are more likely to collapse. This 'sponge model' was first described by Gattinoni et al [7] and provides a satisfying explanation for the rapid radiographic changes in the distribution of atelectasis apparent with prone positioning in ARDS.

The lungs are not symmetrical between the two positions due to both the position of the heart (and other ventral intrathoracic structures) and its compression of the subjacent lung parenchyma. Furthermore, in patients who have lost diaphragmatic tone (due to sedation or paralysis), abdominal contents displace the diaphragm caudally, causing compression of the posterior-caudal lung parenchyma [8]. All of these factors are reversed in the prone position, creating a situation in which more recruited lung is available in the prone position than in the supine position and the vertical gradient of PPL is decreased.

Elharrar et al [9] reported a single-center before-after study that included 24 patients with acute hypoxemic respiratory failure and infiltrates on chest computed tomographic scans. Prone positioning was started without changing the system for oxygen supply or fraction of inspired oxygen (FIO₂). Four patients did not tolerate the prone position for more than an hour (requiring later intubation); 6 of 15 patients who tolerated prone position showed a mean (SD) increase in PaO₂ of more than 20% from baseline (74 [16] to 95 [28] mm Hg; *P* = .006) but 3 patients returned to baseline PaO₂ after supination.

Sartini et al [10] performed a 1-day cross-sectional before-after study that included 15 awake patients with mild and moderate ARDS. The estimated mean (SD) PaO₂:FIO₂ was 157 (43). Patients received NIV with sessions of prone positioning after poor response to continuous positive airway pressure (CPAP) of 10 cm H₂O. On the day of the study, the patients had a median of 2 sessions (interquartile range [IQR], 1-3) of prone positioning for 3 hours (IQR, 1-6 hours). Compared with before receiving NIV, oxygenation and respiratory rate improved during NIV while prone (estimated PaO₂:FIO₂, 100 [IQR, 60-112] to 122 [IQR, 118-122] and respiratory rate 28 breaths/min [IQR, 27-30] to 24 [21-25] breaths/min), and remained improved 1 hour after NIV session in prone position in most patients (12 of 15). At 14 days, 1 patient was intubated and another died.

The abovementioned studies on the effectiveness of prone position in Covid patients clearly point out that correct patient selection, timely initiation and duration of patient's placement in this position can all affect the effectiveness of this treatment method. The prone position during spontaneous and assisted breathing in covid patients with acute hypoxemic respiratory failure may become a therapeutic intervention in the near future.

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