REVIEW

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Mechanical Ventilation Strategies in Patients with Intra-Abdominal Hypertension and Open Abdomen

Fatma YILDIRIM[1], **Irem KARAMAN**[2], **Mehmet YILDIRIM**[3]

[1]University of Health Sciences, Dışkapı Yıldırım Beyazıt Research and Education Hospital, Department of Chest Diseases, Pulmonary Intensive Care Unit, Ankara, Turkey

[2]Bahçeşehir University Faculty of Medicine, Istanbul, Turkey

[3]University of Health Sciences, Dışkapı Yıldırım Beyazıt Research and Education Hospital, Department of Internal Medicine, Medical Intensive Care Unit, Ankara, Turkey

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Corresponding Author: Fatma Yildirim
E mail: ftagassi@hotmail.com

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ABSTRACT

Intraabdominal hypertension (IAH) is an important cause of intraabdominal organ dysfunction and has adverse effects on chest mechanics including an increase in intrathoracic pressure, a decrease in lung volumes, and chest wall compliance due to cephalic shift of diaphragm, and collapse in alveoli leading to hypoxemia. Management with mechanical ventilation (MV) strategies has a pivotal role to reduce these adverse effects and worsening lung injury.

There is a lack of studies and evidence on MV strategies in patients with IAH. This study analyzes the recent literature.

The definition of IAH in critically ill patients, risk factors, intra-abdominal pressure (IAP) measurement, IAH treatment, MV management in patients with respiratory failure, modes of MV, the role of assisted breathing, the management of airway pressures, optimal driving pressure, targeted plateau pressure, positive end-expiratory pressure (PEEP) and usage of the prone position are discussed.

Elevated IAP decreases lung compliance. Therefore, MV management should be performed with the guidance of transpulmonary pressure (Ptp) and IAP measurements in patients with IAH. Tidal volüme (TV) should be kept equal to or lower than 6-8 ml/kg and lung-protective MV strategies should be applied. The patient's need for PEEP over 10 cm H_20 during MV is a predictor of increased IAP. Since a PEEP requirement higher than 10 cm H_2O may be a predictor of increased IAP, the lowest PEEP level suitable to patients' condition should be set.

Key words: Intraabdominal hypertension, mechanical ventilation, open abdomen

Introduction

I. Definition of Intraabdominal Hypertension, Risk Factors and Classification

Intraabdominal hypertension (IAH) expresses stable pressure of abdominal cavity. In a healthy individual, intraabdominal pressure (IAP) is about 5-6 mmHg, while in cases of obesity and morbid obesity, its normal value may rise up to 12-14 mmHg, respectively. In critically ill patients, IAP is between 5-7 mmHg. IAH is defined as the persistent measurement of IAP above 12 mmHg. Staging of IAH is made based on IAP values. Grade 1,2,3 and 4 IAH are defined as IAP between 12-15 mmHg, 16-20 mmHg, 21-25 mmHg and higher than 25 mmHg respectively. Abdominal compartment syndrome (ACS) is characterized with a new intraabdominal organ dysfunction with IAH and sustained IAP > 20 mmHg (1).

IAH and ACS are mainly caused by too much intra-abdominal volume within the abdominal cavity. Main risk factors for increased intraabdominal pressure (IAP) include major abdominal surgery or trauma, major burns, prone positioning, ileus or intestinal obstruction, acute pancreatitis, decompensated cirrhosis with large volume ascites, hemoperitoneum, intraabdominal infections, large volume crystalloid infusions, and blood transfusion of greater than 10 units. Compartment syndrome threatens the viability of the tissue within this compartment. Excessive intra-compartmental hypertension leads to devastating abnormalities in diverse organs and systems, many of which are readily discoverable with routine monitoring in the critical care unit, and all of which are related to decreased preload, increased afterload, and extrinsic compression, with decreased end-organ oxygen delivery and utilization. Increased IAP leads to organ dysfunction of all abdominal organs due

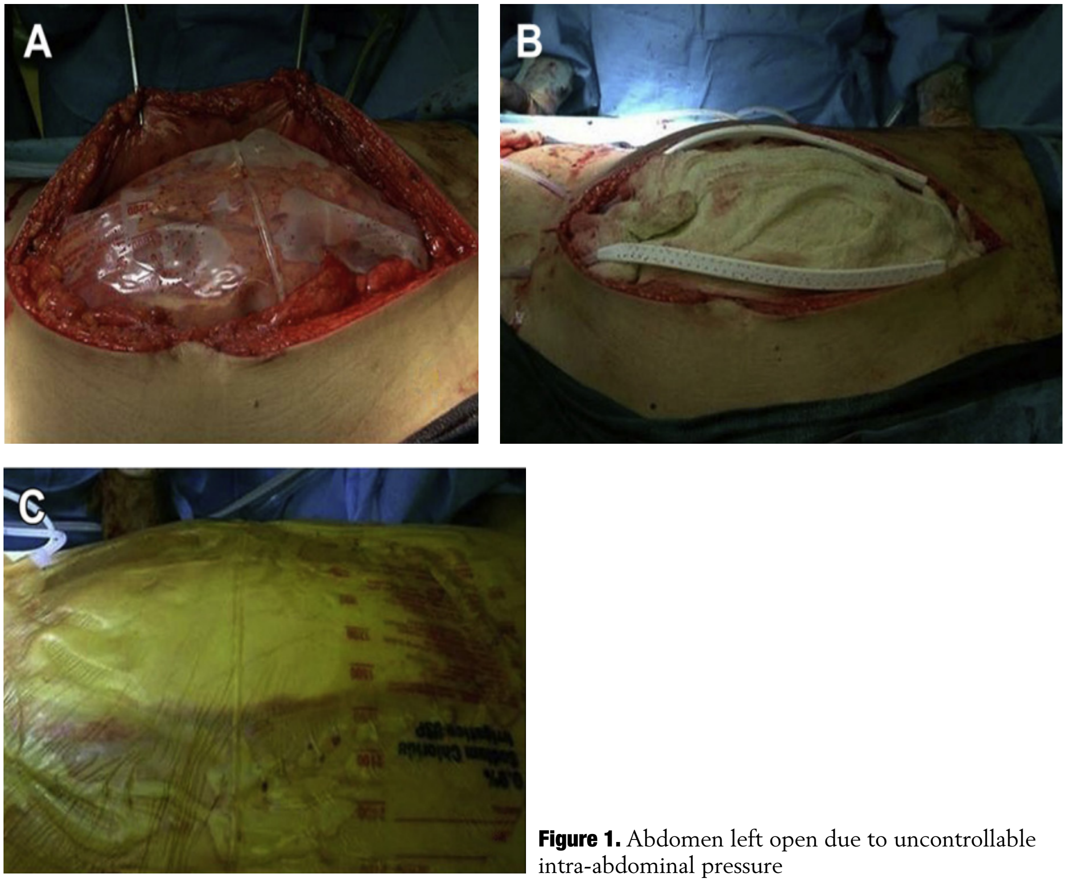

Figure 1. Abdomen left open due to uncontrollable intra-abdominal pressure

to compromised arterial blood flow, venous outflow obstruction and impaired microcirculatory flow. Kidney failure is the most often and consistently described organ failure associated with IAH, but hepatic, adrenal and gastrointestinal dysfunction has also been reported repeatedly. IAH can also lead to increased bacterial translocation (1).

Intra-abdominal hypertension also has a serious impact on the function of respiratory as well as peripheral organs. In the presence of alveolar capillary damage, intra-abdominal hypertension promotes lung injury as well as edema, impedes the pulmonary lymphatic drainage, and increases intra-thoracic pressures, leading to atelectasis, airway closure, and deterioration of respiratory mechanics and gas exchange (2,3).

To prevent IAH and ACS abdomen may not be closed in the presence of an uncontrolled intraabdominal infection or an incomplete first damage control surgery with a planned second look operation or in the presence of disruption of the abdominal wall (Figure 1). The purpose of leaving the abdomen open in trauma or non-traumatic situations is to prevent organ damage and to improve the deteriorated physiology in critically injured patients. In post-operative trauma patients, damage control resuscitation may lead to IAH and ACS if abdomen is closed, which can result in severe hemodynamic disorders and multiple organ failure.

However, this is a non-anatomical condition with potential serious complications such as fluid and electrolyte loss, malnutrition, entero-atmospheric fistula, loss of abdominal wall structures, as well as negative consequences such as prolonged hospital and intensive care unit (ICU) length of stay and increased hospital costs. Therefore, it is essential to detect appropriate patients who would benefit from open abdomen strategy. Nonetheless, abdominal fascia-to-fascia closure should be performed as soon as possible when the patient's condition has improved (2). There are many criteria that reflect high risk for ACS development and suggest open abdomen strategy such as pH<7.20, lactate ≥5 mmol/L, age ≥55 years, base excess (BE)≥-6, age<55 years when BE ≥-15, body temperature ≤ 34°C, systolic blood pressure <70 mmHg, predictive blood loss >4 L, a need of ≥10 L erythrocyte replacement, and >1.5 fold increase in International normalized ratio/prothrombin time. Clinicians should also be aware of risk factors such as obesity, acute pancreatitis, liver failure, respiratory failure, acute respiratory distress syndrome (ARDS) and need for positive end expiratory pressure levels (PEEP) higher than 10 cm H_2O.

Some procedures that should be performed before surgical decompression to prevent IAH and to reduce IAP include nasogastric or colonic decompression, use of prokinetic agents, appropriate patient position, avoidance of tight dressings and bandages, escharotomy, percutaneous decompression, appropriate mechanical ventilation (MV) strategies, appropriate analgesia, sedation and vasoactive agents, administration of neuromuscular blockers, and if negative fluid balance is required, negative fluid balance using diuretics or renal replacement therapy if needed (3).

In medical ICUs, IAH is also frequently seen (38-80%) and in a recent systematic review, 25 risk factors for IAH and 16 risk factors for ACS development are defined (4). These include high volume crystalloid fluid resuscitation, sepsis, respiratory failure, shock/hypotension, obesity, sepsis, abdominal surgery, ileus, metabolic disorder/organ failure, acute kidney injury, oliguria, acute pancreatitis, high disease severity and high organ failure scores. Many of the critically-ill patients in medical ICUs have these risk factors, however IAP is not routinely measured as the measurement technique is a relatively invasive and challenging procedure. IAP measurement is recommended in critically ill patients with 2 or more of the risk factors for the development of IAH (4).

Ia. Intra-abdominal pressure measurement in critically-Ill and mechanically ventilated patients

Early serial measurements are crucial both for the diagnosis of IAH and initiation of early treatment. IAP can be measured directly or indirectly and intermittently or continuously. Direct intraperitoneal measurement is the gold standard, however it is not always possible. Indirect measurement may be performed from the bladder, stomach, via femoral vein, inferior vena cava and rectal catheter.

Transvesical IAP measurement method is the most frequently preferred way to determine IAP, as it is a low-cost and a simple procedure with lower risk of complications. Foley manometer and AbViser cover are used in transvesical IAP measurement method. The most adopted form of IAP monitoring involves intermittent IAP measurement through the bladder every 4–6 h. The bladder is filled with 5 to 10 cc of isotonic saline via Foley (no more than 25 mL of instillation volume). A 16-gauge needle is pushed forward through the aspiration port by clamping the drainage tube. Then the tube is connected to the water manometer or pressure gauge. Although triple lumen urinary catheters are also used, single lumen catheters are more suitable for measurement. The patient should be put in the supine position for IAP measurement and IAP should be measured at the end of expiration without presence of abdominal muscle contractions. The measurement should be performed in the supine position and the manometer should be kept at the mid-axillary line and at the level of the iliac crest. Injecting a minimum instillation volume of liquid during each measurement was found to be sufficient and effective (5).

In sedatized mechanically ventilated patients, IAP measurement has the best accuracy. However, mechanically ventilated patients may have spontaneous respiratory effort during the weaning period, may experience patient-ventilator asynchrony and may be in pain and distress. Under these conditions, abdominal contractions may increase IAP. Nevertheless, it is not recommended to use neuromuscular blockers or sedation during IAP measurement owing to the negative effects of these agents; and the IAP measurement can be performed when the lowest end expiratory pressure is seen. The patients on noninvasive mechanical ventilation (NIMV) may have increased work of breath which may lead to abnormal abdominal contractions. This may cause an increase in measured IAP, however this does not accurately reflect the increase in the intra-abdominal volume. For mechanically ventilated patients with PEEP level above 12 cm H_20, it is recommended to decrease the level of measured IAP 1-2 mmHg to determine the accurate IAP (6).

II. How Should Mechanical Ventilation Be Applied in Patients with Open Abdomen?

In critically-ill patients, ACS creates a serious co-morbidity with a mortality rate of 40-80% (1). The two most common important organ dysfunctions in ACS are acute kidney dysfunction and insufficient ventilation. In cases where the intra-abdominal pressure cannot be controlled, the abdomen can be left open. The World Society of Emergency Surgery (WSES) has released a guideline on open abdomen management in trauma and non-traumatic cases (2). It is recommended that critically-ill patients with open abdomen should be evaluated by a multidisciplinary team, and regular IAP measurements should be performed to decide the timing of closing the abdomen. Inotropic and vasoactive agents should be adjusted based on the physiological condition of the patient, and the negative fluid balance should be maintained if necessary. Hypothermia should be avoided, and coagulation disorders should be corrected. Abdominal fascia-to-fascia closure should be performed as soon as possible when the patient's physiological condition permits. However, in this guideline, there isn't any recommendation regarding MV management in patients with open abdomen. Lung protective MV strategies (low tidal volume, low plateau and low driving pressure), high PEEP levels (>10 cm H_20) and awake light sedation which provides optimum adaption to MV are suggested (2).

III. How should Mechanical Ventilation Management be Applied in Patients with Intra-abdominal Hypertension?

Subclinical organ dysfunction begins when the IAP reaches 12-15 mmHg. Thoracic and abdominal pressures have been shown to affect each other in many experimental and clinical studies (7,8). A previous study showed that MV causes a 6.8-fold increase in the risk of IAH (9), suggesting the importance of MV management in patients with IAH. In the next part of this review, tidal volume (TV), PEEP, airway pressures, driving pressure, prone position will be discussed in patients with IAH and/or ACS.

IIIa. Tidal Volume

In the literature, there is no study which investigates optimal TV in patients with IAH. A previous study (10) evaluated the patients undergoing major abdominal surgery and found that the application of lower TV [6-8 ml/kg ideal body weight (IBW)] in the perioperative period compared to higher TV (10-12 ml/IBW) was associated with lower respiratory complications. Santos et al. (11) used rat models with ARDS and IAP and compared low (6 ml/kg) vs high TV (10 ml/kg) application. They reported that high TV application was associated with increase in inflammation in pulmonary ARDS group (10). Other studies also showed that lower TV and PEEP levels showed protection against postoperative pulmonary complications (12,13).

Although, there is lack of information on optimum TV application in patients with IAH, lung protective MV strategy with low TV of 6–8 mL/kg should be performed based on the current evidence.

IIIb. Airway Pressures

Approximately 40-60% of the pressure in the abdomen is transmitted to the thorax. IAP shows respiratory variation as an indicator of abdominal wall compliance. The transfer of abdominal pressure causes rigidity in the chest wall, reducing compliance, compressing the lung, and increasing airway pressures. In addition, the increase in IAP causes a decrease in functional residual capacity even in healthy lungs, and higher pressures are needed to open the airways. Elevated IAP leads to an increase in plateau pressure (Pplat). Lung protective MV strategies suggest a target plateau pressure of 30 cm H_20 or less (14). However, this recommendation does not consider patients with IAP. The purpose of limiting Pplat is to prevent increased trans-pulmonary pressure (Ptp) and prevent alveolar overstrain and ventilator-associated lung injury (15).

The optimum way to prevent alveolar overdistension in mechanically ventilated patients is to measure esophageal pressure. The Ptp catheter is a 10 cm esophageal catheter which is inflated at the lower 1/3 of the esophagus. The catheter is connected to a monitor that measures chest mechanics. Even if there is no change in lung volumes, a decrease in esophageal pressure indicates a decrease in airway pressure. Static airway pressure minus esophageal pressure is equal to Ptp. By using esophageal catheter, chest wall (C_{cw}) compliance and lung compliance (C_L) can be estimated since it reflects intrathoracic pressure. Respiratory system compliance (C_{RS}) can be predicted by dividing TV by the difference between Pplat and PEEP (driving pressure). One of the main determinants of ventilator induced lung injury is Ptp. Inspiratory Ptp should be kept under 25 cm H_2O to prevent ventilator induced lung injury. In critically-ill patients who have normal C_{cw} and have not IAH, half of the abdominal pressure is transmitted to thoracic pressure. In the presence of IAH, abdominal-thoracic transmission (ATT) affects the esophageal and airway pressures at a similar rate and C_L/C_{RS} ratio decreases from 0.85 to 0.5 and a 30 cm H_20 Ptp is required to open atelectasis (16-19). Therefore, for patients with IAH, it is recommended to correct and calculate airway pressures using ATT. Targeted Pplat in these patients is calculated using the formulas

Targeted Pplat = Measured Pplat (cm H_2O) + [(IAP (mmHg) x 1.36)-13.36]/ 2

Targeted Pplat = (Measured Pplat- 7) + 0.7 x IAP (in mmHg)

In lung protective MV strategy, driving pressure (Pplat-PEEP) is used to optimize TV and protect against alveolar overdistension. In patients with ARDS, driving pressure levels higher than 14 cm H_2O are associated with higher mortality (20). Although there is no study which evaluates the effect of driving pressure on outcomes of patients with IAH and ARDS, it seems reasonable to keep driving pressure under 14 cm H_2O.

IIIc. Positive end-expiratory pressure adjustment

The optimal PEEP level in patients with IAP is unclear. As mentioned above, in the case of IAH, alveolar collapse occurs at higher closing pressures during expiration. Existing lung injury may deteriorate due to increased atelectasis formation and atelectotrauma due to inadequate PEEP administration in IAH (21). Therefore, high PEEP levels may be necessary to keep lungs open. However, high PEEP levels may also cause further lung injury due to alveolar overdistension as well as adverse hemodynamic effects. In animal models, several PEEP levels have been investigated and PEEP application with the same levels of IAP is associated with decrease in inflection point, improved lung compliance, declining of alveolar arterial gradient and shunt formation (22, 23).

In the study of Krebs et al. (24), PEEP titration according to the ARDSNetwork table was compared with minimum static elastance of the respiratory system (PEEP Estat,RS) in 13 patients with moderate and severe ARDS. Negative end-expiratory transpulmonary pressure was detected in seven patients. In both methods, negative Ptp was associated with higher intra-abdominal pressure and lower end-expiratory lung volume. Positive end-expiratory transpulmonary pressure was associated with lower IAP (19.4 ± 3.7 cm H2O vs. 13.0 ± 4.7 cm H2O, p = .021, Cohen's d = 1.49), higher end-expiratory lung volumes (1859 ± 656 ml vs. 2756 ± 740 ml). Mean IAP in the study population was 16.3 ± 5.3 cm H2O. They observed very large effects for IAP and end-expiratory lung volume between Ptp – and Ptp + patients at PEEP ARDSNetwork, showing that the abdominal pressure is lower and the end-expiratory lung volume is higher in Ptp + patients. There was no difference between the two groups in terms of oxygenation (PaO_2/$FİO_2$ 157.0±70.4 vs 212.4± 69.0; p=0.215) and applied PEEP values (17.4±3.2 vs 18.0±2.8; p=0.759) (24).

Gattinoni et al (25) applied different PEEP levels (5, 10, 15 and 20 cm H_2O), and they showed that in patients with extrapulmonary ARDS and IAH, higher PEEP application improved C_{RS} due to decreased C_{cw}. Conversely, higher PEEP levels in patients with pulmonary ARDS without IAH caused worsening of C_{RS} due to decreased lung compliance. In another pilot study (26) with 15 patients PEEP levels were adjusted toIAP levels as 50% of IAP or 100% of IAP. However, applied PEEP levels equal to 100% of IAP were not tolerated due to hypoxemia, hypotension, or endotracheal tube cuff leak. PEEP levels equal to 50% of IAP were better tolerated while they did not improve oxygenation (26).

The effect of PEEP levels on IAP have also been investigated. In a previous study (27), it was shown that PEEP pressure does not cause any change in IAP as long as it does not exceed 10 cm H_2O.

In the second part of the TIRIFIC which is a pair-matched retrospective study, Boehm et al. (28) investigated whether MV could be an indicator of the development of ACS in 38 patients with severe burns. Each patient with ACS was matched with a patient without ACS who had a similar burn rate, similar age, and similar MV mode [asisted spontan ventilation (ASV), pressure-controlled ventilation (PCV) and continuous positive airway pressure (CPAP)]. MV indications were defined as upper respiratory tract edema due to inhalation injury, facial burn, or excessive burn area. In patients with ACS, maximum and mean PEEP, and peak inspiratory pressure (PIP) were higher than others. Therefore, IAP monitorization and measurement is recommended in mechanically ventilated patients with burn (28). The previous study by Yang et al (29) showed that in patients with ARDS and IAH, PEEP titration guiding by Ptp improves oxygenation and chest mechanics.

IIId. Mechanical ventilation mode and assisted spontaneous ventilation

There is no clear data on which MV mode superior in patients with IAH. In some previous experimental studies with ARDS patients without IAH, assisted spontaneous ventilation was associated with reduction in lung injury (30). Prevention of atelectasis and more homogeneous distribution of TV are possible mechanisms of benefit of assisted spontaneous breathing. In addition, a recent study demonstrated that assisted spontaneous ventilation improves oxygenation and reduces lung injury in the animal models with ARDS and IAH (31). In another experimental study, it was shown that adding assisted breaths to bilevel positive airway pressure (BIPAP) did not improve hemodynamic and respiratory parameters in severe IAH, and it even contributed to histopathological damage in the lungs. Therefore, using assisted spontaneous ventilation in severe IAH may be a serious concern. On the other hand, Santos et al (32) hypothesized that the preservation of spontaneous breathing during pressure support ventilation (PSV) in rats with IAH and mild extrapulmonary ARDS would improve respiratory functions and protect them from ventilatory induced lung injury when compared to PCV. In 30 Wistar rats, mild extrapulmonary ARDS was induced via intraperitoneal injection of *Escherichia coli* lipopolysaccharide. Then, rats were divided into two groups (with IAH and without IAH). After 24 hours, invasive MV and PSV or PCV were performed. Independent from presence of IAH, rat models that were ventilated with PSV had lower airway pressures, lower IL-6 mRNA, surfactant protein B and type 3 procollagen expressions. PSV was also associated with improved oxygenation, reduced alveolar collapse, interstitial edema, and diffuse lung injury (32).

Human studies examining MV modes in patients with IAH are limited. Puiac et al (33) investigated the effects of MV parameters on IAP in 16 MV patients (6 female and 10 male and mean age 72.0±8.2). A total of 61 measurements were performed in this observational study, where the mean TV was 489 ml (5.18 ml/kg). Positive correlation between PEEP and IAP (R=0.429, p=0.006) and between TV and IAP (R=0.5471, p<0.0001), positive correlation between TV/kg and IAP (R=0.3841, p=0.0022) and between body mass index and IAP (R=0.3558, p=0.0049) was found. However, selected ventilator mode [n=22 for CPAP, n=47 for BIPAP, n=18 for synchronized intermittent mandatory ventilation (SIMV), n=2 for intermittent positive pressure ventilation (IPPV)] did not have an effect on PIP and Pplat.

The pressure-regulated volume-controlled mode (PRVC) is a dual-controlled mode that avoids high peak airway pressures in volume-controlled ventilation by calculating the compliance of the thorax and lung during the first breaths, and regulates the air flow for the set tidal volume to keep the airways pressures below the set level. Yin et al (34) randomized 40 patients with ACS into two groups in terms of ventilator modes (PCV vs PRVC). In PCV mode; PIP, inspiratory time, FiO_2 and PEEP were set between 8-18 mmHg, 0.8-1.2 seconds, 30-60% and 6-12 mmHg, respectively whereas in PRVC mode TV, respiratory rate, inspiratory to expiratory time ratio, FiO_2 and PEEP were adjusted 5-12 ml/kg, 12-18 breath/min, ½, 30-60% and 6-12 mmHg, respectively. After 6 hours of ventilation, arterial blood gas analysis, chest mechanics and hemodynamic parameters were noted, and sequential organ failure assessment score (SOFA) was calculated. In PRVC mode compared to PCV, significant reduction in $PaCO_2$ levels, PIP, mean inspiratory pressure, central venous pressure, heart rate and extravascular lung water index were observed. In addition, PRVC mode was associated with improved pH and PaO_2, oxygenation index and C_{RS}. There was no difference in mean arterial pressure, SOFA score and IAP between two groups. Investigators have also stated that PEEP levels between 6-12 mmHg lead to less IAP fluctuation (34).

IIIe. Prone positioning

Prone positioning improves respiratory mechanics and oxygenation and prevents overdistention. In patients with IAH, prone positioning may be beneficial by suspending the abdomen. In an animal model, Mure et al (35) showed that in the presence of IAH, prone positioning resulted in an improvement in pulmonary gas exchange, oxygenation, and ventilation/perfusion ratio with a decrease in IAP and Pplat. On the other hand, in patients with ARDS, prone positioning is associated with a minimal increase in IAP (from 15 mmHg to 18 mmHg) (36,37). Therefore, further studies are needed to reveal effects of prone positioning on IAP.

As a summary, elevated IAP decreases lung compliance. Therefore, MV management should be performed by the guidance of Ptp and IAP measurements in patients with IAH. TV should be kept lower than 6-8 ml/kg and lung protective MV strategies should be applied. The patient's need for PEEP over 10 cm H_20 in MV is a predictor of increased IAP. Since PEEP requirement higher than 10 cm H_2O may be a predictor of increased IAP, the lowest PEEP level which patients' condition allows should be set.

AUTHOR CONTRIBUTIONS:

Concept: FY, MY; **Design:** FY, IK, MY; **Supervision:** FY; **Data Collection and/or Processing:** IK, MY; **Analysis and/or Interpretation**: FY, IK, MY; **Literature Search:** FY, IK, MY; **Writing Manuscript**: FY, IK, MY; **Critical Review:** FY.

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