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Effect of continuous positive airway pressure on the respiratory system: a comprehensive review

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Abstract

Background: CPAP is characterized by the application of a constant and continuous positive pressure into the patient's airway. By delivering a constant pressure during both inspiration and expiration, CPAP increases functional residual capacity and opens collapsed or under ventilated alveoli, thus decreasing right to left intrapulmonary shunt and improving oxygenation in obese individuals.

Main body of abstract: Obesity is characterized by several alterations in the mechanics of the respiratory system that tend to further exaggerate impairment of gas exchange rendering these patients prone to perioperative complications, such as hypoxemia, hypercapnia, and atelectasis. Interestingly, CPAP has been advocated as an efficacious modality for prevention and treatment of postoperative atelectasis considered to be the most common postoperative respiratory complication. In OSA, the CPAP device works to splint the airway open and prevent the collapse of the upper airway that is the cardinal event of OSA leading improvement of sleep, quality of life and the reduction of the risks of the cardiovascular and neurocognitive side effects associated with the disease. Besides such a beneficial effect, there are other physiological benefits to CPAP: greater end-expiratory lung volume and consequent increase in oxygen stores, increased tracheal traction to improve upper airway patency and decrease in cardiac after load.

Conclusion: Due to various physiological benefits on the respiratory system CPAP therapy is crucial for the prevention postoperative complications particularly related to obesity and the cornerstone for the treatment of moderate to severe obstructive sleep apnea.

Keywords: Obesity, OSA, CPAP, Hypoxemia, Atelectasis

Background

The expression "continuous positive pressure breathing" (CPPB) was first used by Barach in 1938 who employed the principle in the treatment of patients with lung edema during spontaneous respiration. In the middle of the nineteen-sixties, Jørgensen and his co-workers introduced continuous positive pressure ventilation (CPPV) as a means of depressing cardiac output for hypotensive anesthesia [1].

In 1971, continuous positive airway pressure (CPAP) was introduced into pediatric practice and showed a remarkable improvement in arterial oxygen tension in the idiopathic respiratory distress syndrome of the newborn; The positive pressure in the final phase of expiration has been termed positive expiratory pressure plateau (PEPP). Thereafter, the term PEEP (positive end-expiratory pressure) was introduced. CPAP, PEPP and PEEP are synonymous terms [1, 2].

CPAP is characterized by the application of a constant and continuous positive pressure into the patient's airway. In the majority of CPAP systems expiratory retard and spontaneous ventilation are employed, during which,

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the airway pressure towards the end of the expiratory phases has not reached atmospheric level [3].

Effect on lung mechanics and V/Q ratio

The increase in body mass index (BMI) has detrimental effects on pulmonary functions leading to small airway dysfunction, expiratory flow limitation, alterations in respiratory mechanics with reduced chest wall and lung compliance [4].

Notably, relatively small increases in BMI remarkably reduce functional residual capacity (FRC) and expiratory reserve volume (ERV), consequently increasing areas of atelectasis and closing of peripheral lung units harming the V/Q mismatch and leading to hypoxemia in obese patients [5, 6].

Application of PAP, whether in the form of constant PAP or PEEP, has been used to increase the FRC, decrease right-to-left shunting, and improve oxygenation [7].

This can be explained by a progressive shift in blood flow or ventilation. It is possible that blood flow is diverted away from areas that were initially well ventilated but poorly perfused. Likewise, ventilation is shifted away from areas of functional dead space to previously under-ventilated, well-perfused alveoli (i.e., alveolar recruitment). Alveolar recruitment describes re-inflation of previously collapsed alveoli [8].

An alternative explanation is the increase in lung volume. The increase of the lung volume is the result of three separate effects. First, PEEP increases lung volume as a result of distension of already patent airways and alveoli by an amount dependent on system compliance. Second, the application of PEEP prevents alveolar collapse during expiration. Third, PEEP levels above 10 cmH_2O recruit collapsed alveoli in acute lung injury [8].

It was previously demonstrated that the application of a PEEP level of 10 cmH2O during one lung anesthesia to patients with low PaO_2 increased the FRC to normal values, thereby reducing the pulmonary vascular resistance, improving the V/Q ratio and increasing PaO_2 [9].

In mechanically ventilated normal subjects, the application of PEEP increases lung volume and reopens atelectatic lung regions, PEEP would prevent complete alveolar collapse and improve oxygenation by increasing FRC, probably by preventing airway closure and recruiting previously unventilated alveoli [10].

PEEP have been generally applied to reverse hypoxemia and to improve oxygenation. Improvement in oxygenation is usually not observed unless there is a concomitant increase in FRC. The application of PEEP is expected to increase PaO_2 and decrease intrapulmonary shunt, alveolar-arterial O_2 pressure difference, and arterial-venous O_2 content difference as it recruits lung volume [8].

Four mechanisms have been proposed to explain the improved pulmonary function and gas exchange with PEEP: [8].

a) Increased FRC.
b) Alveolar recruitment.
c) Redistribution of extra vascular lung water; and
d) Improved ventilation-perfusion matching

Dantzker and colleagues found that the application of PEEP is accompanied by decreased blood flow to poorly ventilated regions. In addition, PEEP abolished regions of shunt and redistributed blood flow from regions with high shunt to regions of very low V/Q. PEEP reduces shunt mainly by alveolar recruitment and redistribution of pulmonary perfusion [11].

Previous work in supine animals has found a similar effect of PEEP. Petersson et al studied the effect of positive end expiratory pressure (PEEP) in 12 healthy volunteers and concluded that addition of PEEP at 10 cmH_2O causes redistribution of both lung blood flow and ventilation, in accordance with results given by Richard et al. in their experimental study conducted on six female piglets [12, 13].

Speculative suggestions regarding the mechanisms causing redistribution of lung blood flow at addition of PEEP are: reduction of hypoxic vasoconstriction in atelectatic or poorly ventilated dependent regions, hyperinflation of nondependent regions increasing regional vascular resistance, reduced cardiac output and pulmonary arterial pressure, and increased airway pressures [13].

By delivering a constant pressure during both inspiration and expiration (Fig. 1), CPAP increases FRC and opens collapsed or under ventilated alveoli, thus decreasing right to left intrapulmonary shunt and improving oxygenation. The increase in FRC may also improve lung compliance, decreasing the work of breathing [14].

Likewise, the use of positive airway pressure on left ventricle (LV) function has been investigated in patients admitted to the ICU with hypoxemic cardiogenic pulmonary edema (CPE). In those patients, the hemodynamic benefit of CPAP on CPE resulted from a decrease in left ventricle end-diastolic volume (preload) following a reduced venous return [8].

Earlier studies and systemic reviews concluded that either CPAP or BiPAP have similar effects on the main outcomes in the treatment of CPE. They both showed earlier resolution of dyspnea, respiratory distress, and metabolic disturbances when compared to conventional oxygen therapy, reducing the need for intubation and hospital mortality [12–14].

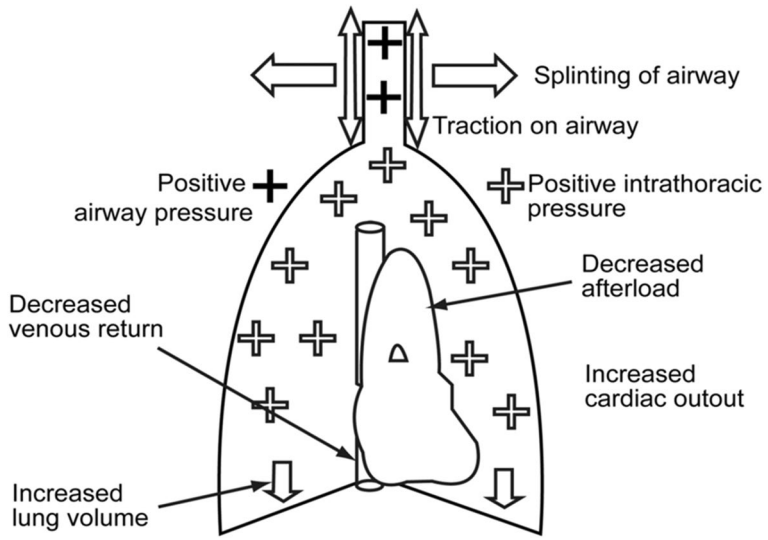

Fig. 1 Physiological effects of positive airway pressure (PAP) therapy

Effect of CPAP during anesthesia and surgery

With the induction of general anesthesia, atelectasis develops, leading to a reduction in both V/Q ratio and pulmonary compliance, even in non-obese patients. Many studies concluded that with the use of CPAP, these adverse effects can be significantly reduced as CPAP prevents the postoperative closure of airways decreasing atelectasis formation and rapidly normalizing FRC improving the intrapulmonary shunt effect [15, 16].

General anesthesia, even in the lung-healthy subject, causes an increase in intrapulmonary shunt, which may impair oxygenation. Previous studies have shown that the magnitude of shunt is correlated with the formation of pulmonary atelectasis [17]. It was also noted that atelectasis can appear within minutes after the induction of anesthesia in 85–90% of all patients [18, 16].

Up to 15% of the entire lung may be atelectatic, particularly in the basal region, resulting in a true pulmonary shunt of approximately 5% of cardiac output, leading to a reduction in both V/Q ratio and pulmonary compliance [19].

Pulmonary atelectasis is also the most common postoperative respiratory complication. In most patients, the development of postoperative atelectasis is attributable to the absence of periodic deep breaths. This occurs in association with a reduction in lung volumes and expiratory flow rates. Absence of periodic lung expansion also results in decreased surfactant activity which contributes to a reduction in FRC and to early airway closure [20].

Also, the frequent incidence of atelectasis is a major concern after cardiac surgery because it contributes to the deterioration of pulmonary function and oxygenation [21, 22].

Multiple factors, such as pleural opening, postoperative diaphragmatic dysfunction, pain, immobilization, and bed rest, in addition to possible preexisting respiratory disease, are involved in the development of atelectasis in this clinical situation [23, 22].

The use of PAP for therapy of atelectasis has gained widespread use. In spontaneously breathing, non-intubated patients, CPAP has been advocated as an efficacious modality for prevention and treatment of atelectasis [24, 25].

Altmay et al have evaluated the effects of CPAP at 10 cmH_2O during cardiopulmonary bypass (CPB) on pulmonary gas exchange after open heart surgery. They reported lesser values of intrapulmonary shunt 20 min after CPB and just after sternum closure in those patients that have used CPAP during CPB, when compared to those whose airways were kept open to atmosphere. The authors concluded that CPAP use during CPB is an effective maneuver to reduce intrapulmonary shunt just after surgery ending [26].

Loekinger et al. studied the effect of CPAP at 10 cm H_2O during CPB on postoperative pulmonary gas exchange. They observed that this maneuver resulted in a significantly improved postoperative gas exchange variables and suggested that CPAP at 10 cmH_2O may avoid dispersed alveolar collapse throughout the lungs [15].

Recovery from abdominal surgery is usually fast and uncomplicated, but postoperative hypoxemia complicates between 30% and 50% of cases, even among those undergoing uneventful procedures [27].

Loss of functioning alveolar units has been recognized as the underlying mechanism responsible for postoperative hypoxemia. Pulmonary atelectasis after abdominal surgery is, in fact, common. It may exceed 25% of the total lung volume and is seen several days after surgery [28].

Several studies have shown that, in patients with postoperative hypoxemic respiratory failure, CPAP improves gas exchange, minimizes atelectasis formation and increases FRC [29, 16].

The application of CPAP at an airway pressure of 7.5 cm H_20 at two hourly intervals during the daytime during the first three days following upper abdominal surgery has also been shown to lead to a more rapid normalization of the FRC and to a reduction in the incidence of atelectasis [21].

In a study by Ricksten et al. CPAP (10 to 15 cm H_2O) applied by a face mask for 30 breaths every waking hour, for three postoperative days after upper abdominal surgery, was more effective than deep breathing exercises in terms of improving pulmonary function and gas exchange and reducing atelectasis. The authors described that the prophylactic intermittent increase of FRC, as performed with periodic CPAP prevents postoperative closure of small airways and suggesting that CPAP can effectively reopen collapsed alveoli through collateral channels [24].

Furthermore, in a study on the prophylactic use of nasal CPAP following cardiac surgeries, patients were randomized to receive either intermittent nasal CPAP at 10 cm H_2O every 4 h or prophylactic nasal CPAP at 10 cm H_2O for at least 6 h. The authors reported that the use of CPAP had a positive impact on arterial oxygenation and reduced respiratory complications [30].

Effects of CPAP on obese individuals

CPAP successfully improved respiratory mechanics and gas exchange abnormalities in the obese patients; CPAP readily recruits collapsed alveoli matching ventilation with perfusion and improving oxygenation. CPAP was also shown to abolish intrinsic PEEP that imposes an additional mechanical load on the inspiratory muscles, thereby increasing work of breathing [10, 31].

Notably The number of obese patients undergoing surgery, either bariatric or non-bariatric, is steadily increasing. These patients have a priori healthy lungs. However, the pathophysiological changes induced by obesity make these patients prone to perioperative complications, such as hypoxemia, hypercapnia, and atelectasis [32, 33].

It is likely that the mechanisms leading to the deterioration of gas exchange in normal subjects are operating to a higher degree in obese patients. In fact, in the obese patients, the lower lung volume should enhance the small-airways collapse, with higher degree of V/Q mismatch, as compared with normal subjects [10].

It has also been demonstrated that in anaesthetized patients, PaO_2 is inversely related to BMI. The major cause of this decrease is likely related to the reduction in FRC. Moderate to severe hypoxemia has been reported in supine obese subjects during both spontaneous breathing and anesthesia and paralysis [33].

In addition, V/Q mismatch has been reported even in awake, seated, obese subjects. The lung bases are well perfused, but they are under ventilated because of airway closure and alveolar collapse. This effect is likely more pronounced and enhanced in obese subjects in the supine position during anesthesia and paralysis [33].

Furthermore, it has been shown that with increasing BMI, an increase in abdominal mass and intra-abdominal pressure is expected. Consequently, the gravitational intra-abdominal pressure gradient is likely increased, with an increased load particularly on the most dependent lung regions and a consequent more important reduction in the passive movements of the dependent part of the diaphragm. This preferential alteration of the diaphragm likely favors the development of more atelectasis in the dependent lung regions [34].

It is verisimilar that atelectasis and true shunt are higher in obese patients compared with normal subjects and several reports indicate that nasal continuous positive airway pressure may be an effective modality for therapy of pulmonary atelectasis and could be used to improve hypoxemia in obese patient. Indeed, studies have shown a good correlation between oxygenation and the amount of atelectasis [25, 35, 36].

Pelosi et al. found remarkable differences at PEEP 10 cmH_2O in lung volumes, respiratory mechanics, and gas exchange in the early postoperative period, during anesthesia and paralysis, between normal and morbidly obese patients; the authors concluded that increasing PEEP to 10 cmH_2O led to a significant improvement of respiratory mechanics, gas exchange and increase in oxygenation in the obese patients but not in normal subjects, and the oxygenation improvement is related to the amount of alveolar recruitment [10].

Despite the use of 100% oxygen, application of PEEP throughout the induction period nearly completely prevents atelectasis formation in morbidly obese patients. In one study, PaO_2 after intubation in the PEEP group was higher than in the control group, probably caused directly by the prevention of atelectasis formation [37].

Application of CPAP at 10 cm H2O for five minutes during the administration of oxygen followed by ventilation via face mask with PEEP at 10 cm H2O for five minutes was effective to prevent, or largely decrease,

atelectasis formation at the induction of general anesthesia in morbidly obese patients [37].

Effects of CPAP on OSA and obesity hypoventilation syndrome (OHS)

CPAP was also introduced for the treatment of Obstructive sleep apnea. It is believed to act by pneumatically splinting the pharyngeal airway, thereby preventing its collapse during sleep [38].

This theory was first suggested by Sullivan et al. in 1981 and further studies compared the relationship between oesophageal pressure swings and inspiratory airflow during breathing without and with CPAP in normal awake subjects. The investigators observed that, at a given oesophageal pressure, inspiratory airflow was greater during CPAP administration despite the presence of decreased genioglossus muscle activation compared with baseline [39, 40].

PAP provides a pneumatic splint by delivering an intraluminal pressure that is positive with reference to the atmospheric pressure. Upper-airway muscle tone either remains the same or decreases with the application of CPAP. Imaging studies demonstrate that PAP increases upper-airway cross sectional area and volume in awake normal subjects and OSA patients with the largest change in the lateral dimensions [41].

CPAP not only prevented the collapse of upper airway in OSA but also improved arterial oxygen values in these patients. Randomized controlled trials reported that CPAP can effectively reduce the apnoea-hypopnea index with improvement in subjective and objective sleepiness, sleep quality, and quality of life [42].

Other observational studies have shown a reduction in the risk of of metabolic cardiovascular events in OSA patients treated with PAP compared to untreated patients. In a recent study, the use of CPAP for more than 3 months was associated with marked improvement in HbA1c levels reflecting adequate control of blood glucose levels in patients with OSA compliant and adherent to CPAP treatment [43].

Accordingly in another study, the carotid intima–media thickness (CIMT), which is an early marker of atherosclerosis was found to be significantly increased in patients with OSA. Administration of CPAP for 6 months was not only beneficial for improving sleep efficiency but also for reducing cardiovascular complications associated with the disease after significant decrease in the CIMT was observed in the studied patients [44].

Various studies also demonstrated reduction in the risk for motor vehicle accidents, or increased mortality associated with sleep apnoea as well as reduction of risk for neurocognitive degeneration [45].

Not surprisingly, Gad et al. evaluated the effect of CPAP therapy on neuropsychiatric complications of OSA including depression and anxiety, neurocognitive disorders, and sleep-related quality of life. After 3 months of treatment, a remarkable improvement was evident in sleep related quality of life, depression and anxiety underlining the value of CPAP use of 6 h or more for vulnerable patients with reduced neurocognitive functions in the context of aging and comorbidities [46].

The positive airway pressure titration task force of the American Academy of Sleep Medicine has recommended that all patients should receive adequate PAP education, hands-on demonstration, careful mask fitting, and acclimatization prior to titration. The latter should be started at 4 cmH2O and continued at $\geq$ 1 cmH_2O upward incrementation over $\geq$ 5 min until $\geq$ 30 min without respiratory events is achieved. The recommended maximum CPAP pressure is 20 cmH_2O however the patient may be shifted to BiPAP if there is persistence of the apneas and hypopneas at 15 cmH_2O [47].

Most patients with OSA and significant obesity can maintain normal daytime carbon dioxide levels. However, a small proportion of patients will develop hypercapnia in the absence of lung or neuromuscular disease and are diagnosed with obesity hypoventilation syndrome [48, 49].

During sleep, eucapnic patients with OSA hyperventilate between periods of apnoea to maintain acid-base homeostasis. In addition, during wakefulness in daytime, acute hypercapnia is corrected and the excess HCO_3 is excreted. Patients with OHS have a reduced duration of ventilation between periods of apnoea while sleeping [50].

It was shown that eucapnic obese OSA patients had normal hypercapnic and hypoxic ventilatory drives while awake before nasal CPAP treatment, and there was no change in ventilatory drive following nasal CPAP treatment. In contrast, both hypercapnic and hypoxic ventilator drives during wakefulness were significantly impaired in OHS subjects before nasal CPAP treatment and improved after 2 weeks of nasal CPAP therapy [51].

Verbraecken et al. evaluated the arterial blood gases and lung function pattern in OSA patients with chronic CPAP therapy compared to matched untreated OSA controls. Their most important finding was the improvement of PaO_2 in OSA patients with normal lung function, with the greatest improvement in those patients who were the most hypoxemic before treatment. CPAP decreased the P (A-a) O_2 by 6 mmHg in OSA patients with normal lung function, expressing an improved V/Q mismatch [52].

Indeed, many studies also investigated the response of daytime hypercapnia in subjects with OHS to CPAP or BiPAP therapy. These showed that both can improve

$PaCO_2$ (Fig. 2). PAP treatment can result in a reduction in daytime $PaCO_2$ in some patients with OSA and OHS. PAP was also an effective treatment in OSA patients presenting with decompensated hypercapnic respiratory failure and avoided the need for endotracheal intubation [41].

A European group studied the effects of more than 1-year CPAP treatment on daytime lung function and hemodynamics in patients with OSA and found a slight but significant improvement in daytime blood gases [53].

Another study from the same group extended the CPAP follow-up to 5 years in 65 unselected patients with severe OSA. A significant improvement in daytime PaO_2 was reported in a subgroup of hypoxemic patients with OSA, with a slight increase in $PaCO_2$ over the 5-year period [54].

A large retrospective study looked at the effect of CPAP adherence on respiratory failure in patients with OSA. This study found that good compliance with CPAP leads to an improvement in respiratory failure, and there was a reduction in the need for oxygen supplementation with time [55].

In a prospective randomized study performed in patients with obesity hypoventilation, the authors allocated patients to receive either CPAP or BiPAP and followed them for 3 months. The results showed that daytime $PaCO_2$ levels decreased in both groups and no difference noted in the compliance between the two treatment groups suggesting that both CPAP and BiPAP can equally effective in improving daytime hypercapnia in patients with OHS without severe nocturnal hypoxemia. However, subjective sleep quality and psychomotor vigilance were significantly better in patients receiving BiPAP [56].

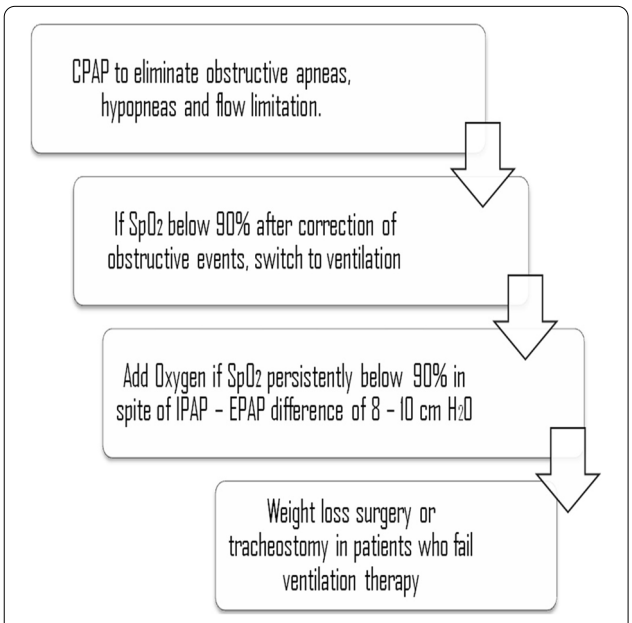

Fig. 2 Suggested algorithm in patients with OHS

Conclusion

In summary, CPAP is characterized by the application of a constant and continuous positive pressure into the patient's airway. CPAP therapy reduces the expiratory collapse of small airways and recruit collapsed alveoli in obese individuals, thereby improving the lung's functional residual capacity and gas exchange.

Application of CPAP decreases shunt, improves matching of alveolar ventilation and perfusion, reduces the requirement for supplemental oxygen, and reduces the need for mechanical ventilatory support, without any significant impairment in cardiovascular function.

Today, CPAP remains the mainstay of treatment for moderate to severe OSA in adults, resulting in improvement of sleep, quality of life and the reduction of the risks of the cardiovascular and neurocognitive side effects associated with the disease.

In OSA, the CPAP device works to splint the airway open and prevent the collapse of the upper airway that is the cardinal event of OSA. Besides such a beneficial effect, there are other physiological benefits to CPAP: greater end-expiratory lung volume and consequent increase in oxygen stores and increased tracheal traction to improve upper airway patency and lower cardiac after load and consequent increase in cardiac output. However, in OSA, lack of acceptance and inadequate adherence to PAP therapy are major obstacles leading treatment failure. A focused approach including education, objective monitoring, and clinic support is essential to optimize adherence.

Abbreviations

BMI: Body mass index; BiPAP: Bilevel Positive Airway Pressure; CIMT: Carotid intima–media thickness; CPAP: Continuous positive airway pressures; CPB: Cardiopulmonary bypass; CPE: Cardiogenic pulmonary edema; CPPB: Continuous positive pressure breathing; CPPV: Continuous positive pressure ventilation; ERV: Expiratory reserve volume; FRC: Functional residual capacity; LV: Left ventricle; OHS: Obesity hypoventilation syndrome; OSA: Obstructive sleep apnea; P (A-a) O2: Alveolar arterial oxygen gradient; PaCO2: Arterial partial pressure of carbon dioxide; PaO2: Arterial partial pressure of oxygen; PAP: Positive airway pressure; PEEP: Positive end-expiratory pressure; PEPP: Positive expiratory pressure plateau; RV: Residual volume; V/Q: Ventilation/perfusion.

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Author's contributions

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