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Perioperative Ventilation of Obstructive Lung Disease: A Physiology-Based Narrative Review

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Abstract

Obstructive lung disease encompasses a group of pulmonary disorders characterized by airflow limitation that impairs complete exhalation, resulting in air trapping and dynamic lung hyperinflation. In these conditions, airway resistance is markedly increased, often reaching 5-15 $cmH_2O/L/s$ or higher, significantly altering normal respiratory mechanics. Although lung elastance during quiet breathing may be normal or reduced-particularly due to emphysematous loss of elastic recoil-patients experience a substantial increase in the work of breathing (WOB). This elevated WOB is primarily driven by increased airway resistance, requiring greater pressure generation to move air through narrowed airways, as well as increased dependence on respiratory muscle effort to facilitate exhalation in the setting of reduced elastic recoil. Mechanical ventilation in patients with obstructive lung disease presents unique challenges, particularly in the presence of expiratory flow limitation. Inadequate expiratory time may prevent full lung emptying before the initiation of subsequent breaths, leading to breathe stacking and the development of intrinsic positive end-expiratory pressure (PEEPi or auto-PEEP). Progressive air trapping and hyperinflation reduce the available lung volume for subsequent inspiration, further increasing inspiratory effort and exacerbating ventilatory inefficiency. The choice of ventilatory mode, including volume-controlled ventilation (VCV) or pressure-controlled ventilation (PCV), is critical in optimizing respiratory mechanics and minimizing complications. Regardless of mode, the central principle of ventilatory management is to prolong expiratory time and reduce tidal volume to limit dynamic hyperinflation. In assisted ventilation, appropriately applied external PEEP may improve patient-ventilator synchrony by offsetting PEEPi, enhancing trigger sensitivity, and reducing inspiratory threshold load; however, excessive external PEEP may worsen hyperinflation by further elevating alveolar pressure. Noninvasive positive pressure ventilation (NIPPV), including continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP), has emerged as an effective strategy for respiratory support, particularly in the immediate postoperative period. When judiciously applied, NIPPV can reduce WOB, improve gas exchange, and decrease the need for invasive mechanical ventilation, thereby improving clinical outcomes. This physiology-based approach is consistent with the principles of personalized medicine, whereby perioperative respiratory care is adapted to patient-specific pathophysiological characteristics to optimize gas exchange while minimizing ventilator-induced and hemodynamic complications.

Categories: Pulmonology, Anesthesiology
Keywords: astma, lung mechanics, obstructive lung disease, perioperative ventilation, personalized medicine

Introduction And Background

Obstructive lung disease constitutes a classification of pulmonary disorders distinguished by a limitation in airflow that hinders the capacity for complete exhalation, resulting in air retention and lung hyperinflation [1]. The predominant medical conditions encompassed within this classification include chronic obstructive pulmonary disease (COPD), which integrates chronic bronchitis and emphysema, as well as asthma. These conditions share common features of airway obstruction, although the underlying mechanisms and progression may differ.

Obstructive lung diseases are a significant global health concern, with COPD being the third leading cause of death worldwide, causing 3.23 million deaths in 2019 [2], while asthma affects 262 million people in 2019 and caused 455.000 deaths [3]. The prevalence of these diseases varies by region, influenced by environmental, genetic, and lifestyle factors. The etiology of obstructive lung diseases includes exposure to risk factors such as tobacco smoke, air pollution, and occupational hazards, as well as a history of recurrent respiratory infections or allergies [4]. Inherited factors, such as alpha-1 antitrypsin deficiency, also play a role, particularly in early-onset emphysema [5]. While asthma is often linked to immune-mediated inflammation and environmental allergens, COPD results from chronic inflammation caused by noxious stimuli.

Mechanical ventilation in patients with obstructive lung diseases presents significant challenges due to the unique pathophysiological characteristics of these conditions. Prolonged expiration from airflow limitation can cause air trapping and dynamic hyperinflation. This increases intrathoracic pressure, reducing venous return and cardiac output [6]. The heterogeneous airway obstruction in conditions like COPD and asthma

complicates ventilation, with some lung regions poorly ventilated and others overdistended [7]. Elevated airway resistance and reduced lung compliance further complicate ventilation [8].

As a consequence of the aforementioned factors, these patients are often at risk of ventilator-induced lung injury (VILI) due to their fragile airways and altered lung mechanics. The anesthesiologist must carefully adjust the ventilator settings such as tidal volume, respiratory rate, and inspiratory-to-expiratory ratio in order to avoid barotrauma, volutrauma, or atelectotrauma [9]. In addition, the role of Noninvasive mechanical ventilation (NIV) is critical in the perioperative management of patients with obstructive lung diseases. NIV can help improve gas exchange and prevent perioperative pulmonary complications, i.e., hypoxemia and atelectasis [10].

Despite the availability of general guidelines for mechanical ventilation, specific recommendations for the perioperative management of patients with obstructive lung diseases remain challenging due to the heterogeneous pathophysiological mechanisms of COPD and asthma. This narrative review aims to provide a comprehensive, physiology-based approach to perioperative ventilation in these patients, integrating the underlying respiratory mechanics with practical ventilatory strategies to support individualized clinical decision-making.

Literature search and selection

A literature search was conducted using the PubMed and Scopus databases between January 2026 and September 2026 to identify relevant publications addressing the perioperative management and mechanical ventilation of patients with obstructive lung diseases. The search included combinations of the following terms: “chronic obstructive pulmonary disease,” “COPD,” “asthma,” “perioperative ventilation,” “mechanical ventilation,” “airway obstruction,” “expiratory flow limitation,” “dynamic hyperinflation,” “intrinsic positive end-expiratory pressure,” “air trapping,” and “lung-protective ventilation.” Relevant publications addressing respiratory pathophysiology, anesthetic management, ventilatory strategies, and perioperative pulmonary complications were considered. The literature was critically reviewed to identify clinically relevant evidence and physiological principles guiding individualized ventilatory management in patients with COPD and asthma. Particular emphasis was placed on the differences in the underlying pathophysiology of these conditions and their implications for intraoperative and postoperative respiratory management.

Review

Overview of obstructive lung diseases

Chronic Obstructive Pulmonary Disease

COPD is a term given to patients who have emphysema, chronic bronchitis, or both [11]. Emphysema is characterized by the dilation of airspaces distal to the terminal bronchioles, accompanied by the destruction of their walls. It is classified into two main types: centriacinar emphysema and panacinar emphysema [12]. Centriacinar emphysema primarily affects the central portion of the acinus, while the peripheral alveoli are often preserved. It is most prominent in the upper lobes, particularly at the apex, and progressively extends downward [13]. In panacinar emphysema, the entire acinus, including all alveoli, is uniformly involved. It has no specific regional preference but is often more prevalent in the lower lobes [14]. Additionally, a distinct form of emphysema occurs in individuals with α1-antitrypsin deficiency, which predisposes them to diffuse and severe lung damage, often with a lower lobe predominance [5].

Chronic bronchitis is defined by excessive mucus production in the bronchial tree, leading to increased sputum output [15]. A hallmark feature is the hypertrophy and hyperplasia of the large bronchial glands [16]. Additionally, chronic inflammatory changes are evident in the small airways, which become narrowed due to cellular infiltration and wall thickening. These inflammatory lesions contribute to obstruction and impaired airflow, further exacerbating respiratory symptoms [16].

The term emphysema is an anatomical diagnosis, based on the structural changes observed in the lungs, such as airspace enlargement and wall destruction. As such, it is often inferred rather than definitively diagnosed in living patients [12]. In contrast, chronic bronchitis is a clinical diagnosis, defined by symptoms such as persistent cough and sputum production for at least three months over two consecutive years [15]. Conclusively, τhe term COPD remains a valuable umbrella term encompassing the spectrum of obstructive pulmonary conditions. COPD is characterized by irreversible airflow obstruction and persistent inflammation [11].

Within this spectrum, two clinical phenotypes are traditionally recognized: type A and type B [17]. In type A, clinical features include a productive cough, leukopenia, sputum production, weight loss, and hyperinflated chest appearance. Auscultation reveals diminished breath sounds and occasional crackles, typically without additional adventitious sounds. Chest X-ray findings show evidence of hyperinflation with low, flattened diaphragms and reduced peripheral pulmonary vascular markings. These patients are colloquially referred to as “pink puffers” due to their characteristic appearance [18]. In type B, patients typically present with a

progressive onset of dyspnea, episodes of copious sputum production during exacerbations, and signs of airway obstruction. Auscultation reveals scattered wheezes and crackles, with both musical and non-musical adventitious sounds. Chest X-rays often show cardiac enlargement and consistent lung field changes due to chronic inflammation and airway narrowing. These individuals are commonly referred to as “blue bloaters”, reflecting their clinical presentation [19].

Asthma

Asthma is characterized by increased airway responsiveness to various stimuli, resulting in inflammation and diffuse airway narrowing. The severity of airway obstruction varies and may resolve spontaneously or in response to treatment [20].

The hypertrophied mucosa constricts, leading to wheezing. In severe cases, the airways may become obstructed by mucus plugs. Unlike other obstructive diseases, asthma does not involve alveolar wall destruction or excessive secretion of fluids [21]. Subclinical studies suggest that inflammation underpins all major features of asthma, including increased airway reactivity and narrowing [22]. However, the exact trigger initiating inflammation often remains unidentified. Asthma involves the activation of cells, which release various cytokines that contribute to inflammation and airway hyperresponsiveness. Notable cytokines include IL-4, IL-5, IL-9, and IL-13, which are linked to eosinophil activation, mucus production, and airway remodeling [23].

In some patients, asthma presents with generalized airway hyperreactivity to specific triggers, leading to episodic exacerbations. During an asthma attack, the patient may experience severe dyspnea, often accompanied by orthopnea and anxiety. The accessory respiratory muscles are visibly engaged, reflecting the increased effort required to breathe, and the lungs appear hyperinflated due to air trapping. Musical wheezing can be heard diffusely across all lung fields, caused by the narrowing of the airways [24]. The patient’s pulse is typically rapid, and pulsus paradoxus may be present, characterized by a significant drop in systolic blood pressure (greater than 10 mmHg) during inspiration [25]. A chest X-ray commonly reveals hyperinflation with increased lung volumes and flattened diaphragms, though it otherwise appears normal without evidence of consolidation or structural abnormalities [26].

Status asthmaticus refers to a severe, prolonged asthma attack lasting hours or days that does not respond to bronchodilator therapy [27]. This condition is life-threatening and often accompanied by exhaustion, dehydration, and tachycardia. Immediate and intensive medical intervention is critical in such cases.

Pathophysiological Changes Affecting Ventilation

Respiratory mechanics: In COPD, several spirometric and lung function measurements demonstrate the presence of airway obstruction. Forced expiratory volume in the first second (FEV_1), vital capacity (VC), FEV_1/forced vital capacity (FVC) ratio, and dynamic expiratory flow rates (e.g., peak expiratory flow at 50% and 75% of the VC) are reduced, indicating impaired airflow and reflecting significant airway obstruction [28]. These measurements reflect airway obstruction caused by factors such as excess mucus within the lumen or thickening of the airway walls due to inflammation. Despite airway narrowing and obstruction, early airway closure occurs at abnormally high lung volumes. This leads to an increase in residual volume (RV) [29]. Forced expiratory volumes are significantly reduced, particularly during dynamic expiration. Expiratory time is greatly prolonged, reflecting both increased airway resistance and reduced elastic recoil of the lungs. Functional residual capacity (FRC) and total lung capacity (TLC) are also elevated due to air trapping and lung hyperinflation [28]. In advanced stages of COPD, the FEV_1 can decline to less than 0.8 liters, severely limiting expiratory airflow [30]. Low flow rates during most of dynamic expiration further highlight the diminished elastic recoil of the hyperinflated lung [31].

During an asthmatic paroxysm, all measures of expiratory flow are significantly reduced, reflecting the severity of airflow limitation. These include FEV_1, FEV_1/FVC ratio, forced expiratory flow at 25-75% of FVC (FEF25-75%), maximum expiratory flow at 50% and 75% of FVC (Vmax50% and Vmax75%) [32]. Additionally, FVC is reduced due to early airway closure at the end of expiration. Static volumes are increased. More specifically, RV is increased as a result of early airway closure during full expiration. Increased smooth muscle tone causes bronchoconstriction, whereas edema and inflammation of the airway walls further narrow the lumen [33].

Gas exchange: Ventilation-perfusion mismatch (V/Q mismatch) is a fundamental characteristic of the alveolar system, often leading to decreased oxygen saturation with or without augmentation of the arterial carbon dioxide pressure (PCO_2) [34]. The alveolar-arterial gradient of oxygen is always increased [35]. The dead space is increased, especially in emphysema. The underlying causes of this V/Q mismatch are primarily attributed to alterations in lung architecture and changes in airway structures, particularly in conditions such as chronic bronchitis.

Heterogeneity in ventilation can be detected through tests, such as the single-breath washout test, which reveals uneven distribution of ventilation across the lungs [36]. Additionally, perfusion imbalances are

significantly influenced by the destruction of portions of the pulmonary capillary bed, which compromises blood flow to certain areas of the lung. Despite these discrepancies in ventilation and perfusion, arterial carbon dioxide levels often remain within normal limits [37]. This is due to compensatory mechanisms in which increased alveolar ventilation helps maintain appropriate gas exchange.

In patients with mild to moderate COPD, arterial PCO2 typically remains within the normal range. This is due to the body's compensatory mechanisms, where an increase in PCO2 stimulates the chemoreceptors, leading to enhanced alveolar ventilation. However, in more severe cases, PCO2 levels may rise. As PCO2 increases, pH decreases, resulting in respiratory acidosis [37]. In contrast, in asthma patients, arterial PCO2 is usually normal or may be slightly reduced. This is because an increase in ventilation helps prevent a rise in PCO2, despite the V/Q mismatch. In severe asthma, however, PCO2 levels may rise, leading to a reduction in pH and the development of respiratory acidosis [38].

For an accurate assessment of pulmonary gas exchange, it is essential to measure the Diffusion Lung Capacity for Carbon Monoxide (DLCO), as it provides a direct indication of the lung's capacity to transfer gases across the alveolar-capillary membrane [39]. In patients with severe emphysema, DLCO is typically reduced due to the destruction of alveolar structures and a compromised surface area for gas exchange [40]. Conversely, in asthma, DLCO is often increased, reflecting the increased pulmonary volumes associated with airway obstruction and hyperinflation [41].

Physiological considerations

Changes in Lung Mechanics

Increased airway resistance: The definition of airway resistance is the change in transpulmonary pressure required to produce airflow through the airways of the lung [42]. In simple terms, it is the ratio of the pressure difference between the oral cavity and the alveoli to the airflow: Raw = ΔP/F = Paw-Pplat/peak inspiratory flow rate, where Raw = airway resistance; ΔP = pressure change; Paw = airway pressure; Pplat = equilibrium end-expiratory pressure; and F = flow. The normal range of resistance in mechanically ventilated patients is 1-5 cmH2O/L/s [43].

In the context of obstructive diseases, the resistance within the pulmonary system may escalate to levels ranging from 5 to 15 cmH2O/L/s, or potentially exceed these values [44]. The mechanisms of airway obstruction include either partial obstruction of the lumen, for example, by increased secretions, or thickening of the wall, for example, by edema or muscle hypertrophy. Moreover, the pathology may lie outside the airways, for example, when the lung parenchyma has been partially destroyed and the airways narrow due to loss of their radial traction.

Increased compliance of the respiratory system: The compliance of a system is defined as the change in volume that occurs per unit change in pressure in the system. Compliance is the ease with which an elastic structure changes shape [45]. Therefore, compliance is a measure of the elastic resistance of a system: C = ΔV/ΔP, where C = compliance, ΔV = change in tidal volume, and ΔP = change in pressure.

Pulmonary compliance (C) is the total compliance of both lungs, measuring how much the lungs will expand (change in volume) for each unit increase in transpulmonary pressure (when there is sufficient time for the system to reach equilibrium). The normal value of compliance in mechanically ventilated patients is 50-100 mL/cmH2O [43].

The level of compliance is increased in obstructive pulmonary diseases like pulmonary emphysema, relatively diminished in asthma, and even less so in chronic bronchitis. In cases of emphysema, there is a significant decrease in elastic recoil [8]. This occurrence can be explained by the deterioration of elastic tissue due to the destruction of alveolar walls. As a result, the pulmonary structures in emphysema are considered to demonstrate a high degree of compliance. Chronic airway obstruction increases lung volumes (e.g., residual volume and total lung capacity), which places the lungs in a more inflated state. At higher lung volumes, the lung becomes mechanically "looser," further increasing compliance [46].

Decreased elastance of the respiratory system: The elastance of a system is defined as the change in pressure that occurs per unit change in the volume of the system. Elastance is the ability of a structure to return to its equilibrium position after a change it has undergone [47]: El = ΔP/ΔV, where El = elasticity, ΔP = change in pressure, ΔV = change in tidal volume

In patients with obstructive lung disease, during quiet breathing, the lung elastance is normal or reduced due to the emphysema-induced reduction of elastic recoil pressure. Dynamic lung elastance may also become significantly elevated in obstructive diseases, particularly during physical exertion or at increased respiratory rates. At higher breathing frequencies, a larger proportion of each breath is distributed to lung units with lower resistance and compliance (i.e., shorter time constants), leading to greater tidal expansion of these rapidly ventilated lung regions and an overall increase in dynamic elastance. Under conditions of tachypnea, the dynamic elastance of the lungs can increase by a factor of ten or more.

Elastic recoil pressure is significantly reduced in patients diagnosed with emphysema, in contrast to individuals suffering from asthma or chronic bronchitis, who typically demonstrate a more normative level of elastic recoil pressure. In the context of emphysema, the reduction of elastic recoil pressure is considered to result from the microscopic destruction of lung parenchyma [48]. On the contrary, in the context of asthma, the reduction in elastic recoil may manifest acutely and can be ameliorated through the administration of bronchodilator therapy. These more acute reductions in elastic recoil observed in certain asthmatic patients may be associated with changes in surface tension due to the extravasation of plasma proteins stemming from inflammatory processes [49].

Hyperinflation and air trapping: Mechanical ventilation in patients with inefficient exhalation can be challenging as patients may not achieve full exhalation prior to the triggering of another breath, a phenomenon known as breath stacking [50]. In the setting of breath stacking, because full exhalation has not occurred, additional air remains within the alveoli at the end of expiration, a phenomenon known as gas trapping. Increased air within the alveoli at the end of expiration increases the end-expiratory pressure, a phenomenon known as intrinsic positive end-expiratory pressure (PEEPi or auto-PEEP) [51]. Breath stacking and gas trapping increase end-expiratory alveolar pressure and volume. Therefore, inspiration begins at a higher-than-normal lung volume and pressure [52].

Elevated plateau pressure can signify hyperinflation in the setting of obstructive disease. Evaluation of the expiratory flow curve on the ventilator can indicate breath stacking under normal conditions. The expiratory portion of the flow curve should return to zero before the end of expiration, indicating that expiratory flow has ceased [53]. If a breath is triggered and administered before expiratory flow has reached zero, breath stacking is present.

The magnitude of auto-PEEP can also be assessed by employing an expiratory pause maneuver on the ventilator. With this maneuver, the expiratory valve is closed at the end of expiration, not allowing any additional air to leave the respiratory system [54]. If, at the end of expiration, alveolar pressure is still higher than proximal airway pressure, air will continue to flow from the alveoli to the ventilator, raising proximal airway pressure. The amount that proximal airway pressure rises above PEEP applied at the ventilator is referred to as auto-PEEP or intrinsic PEEP.

Increased work of breathing (WOB): WOB is the amount of energy or O_2 consumption needed by the respiratory muscles to produce enough ventilation and respiration to meet the metabolic demands of the body [55]. Specifically, WOB represents the energy expenditure necessary to overcome the elastic and resistive components of the respiratory system and facilitate the movement of air into and out of the lungs during spontaneous breathing. It is quantified as the total product of the distending pressure and the volume displaced during inhalation and exhalation. Mathematically, WOB is expressed as: $WOB = \int PpldV$, where Ppl denotes pleural pressure over time relative to the resting pleural pressure, and dV represents the volume over time relative to the resting thoracic volume. In the presence of obstructive pulmonary diseases, WOB is augmented due to heightened resistance within the airways. There exists a substantial augmentation in elastic work required to counteract PEEPi or auto-PEEP as well as the expiratory work of respiration [56].

The increased WOB in obstructive diseases results from increased airway resistance, as more pressure needs to be generated in order to move air through the narrowed airways, leading to greater effort and increased WOB [57]. Moreover, because of decreased elastance, the patient must rely more on muscular effort to expel air, which adds to the WOB. Air trapping and lung hyperinflation reduce the available lung volume for the next breath [31]. As a result, the patient has to work harder to inhale fresh air because the lungs are already overinflated, which increases the WOB. V/Q mismatch can increase the dead space (areas of the lung that are ventilated but not perfused with blood). This reduces the efficiency of each breath, as more energy is used to ventilate parts of the lung that do not contribute to gas exchange, further increasing the WOB [58]. Over time, the increased effort required for breathing can lead to fatigue of respiratory muscles, including the diaphragm and accessory muscles. This can make breathing even more difficult, compounding the work required to move air in and out of the lungs. These factors necessitate an increased expenditure of energy by the body to maintain adequate ventilation, thereby making the act of breathing more demanding.

Hemodynamic Implications

Due to the close anatomical and functional interconnections between the pulmonary and cardiovascular systems, dysfunctions in one system often have repercussions on the other. Specifically, right heart abnormalities are commonly observed in patients with COPD [59]. Significant lung disease through pulmonary vascular obstruction or pulmonary vasoconstriction secondary to hypoxia causes increased pulmonary vascular resistance (PVR). Pulmonary artery pressure is increased in patients with obstructive lung disease [60]. Mechanical factors, including the reduction in the network of blood vessels, particularly the small pulmonary capillaries, that are responsible for facilitating gas exchange in the lungs, thrombosis, and lung hyperinflation, also play a significant role in elevating pulmonary artery pressure. Patients with chronic obstructive pulmonary disease are most likely to develop pulmonary hypertension (PH), with up to 20% of patients showing signs of PH [61]. When pulmonary vascular pressure increases gradually and persistently, the increased pressure can strain the right ventricle (RV). The primary response of the RV is

hypertrophy, which characterizes the early stages of chronic cor pulmonale (CCP) [62].

While the primary impact of pulmonary disease is on the RV, the left ventricle may also be affected. In the earlier stages of obstructive lung disease, cardiac output is typically preserved or slightly elevated, as the heart operates at a high point on the Frank-Starling curve [63]. This positioning allows for an effective increase in cardiac output during exercise or periods of heightened demand. In more advanced cases, cardiac output often becomes reduced due to multiple factors. Increased PVR increases the workload on the RV, which may struggle to maintain right heart output over time. Consequently, the volume of blood returning to the left ventricle is diminished, leading to a subsequent reduction in systemic cardiac output [63]. More precisely, marked dynamic hyperinflation combined with elevated intrinsic PEEPi severely restricts venous return, raises PVR, lowers right ventricular preload, and may quickly trigger acute intraoperative hypotension or a state of obstructive shock, making prompt recognition essential and, if needed, brief ventilator disconnections [64].

Impact of surgery and anesthesia

Influence of General Anesthesia on Respiratory Mechanics

In patients with obstructive lung disease, the induction of general anesthesia in the supine position leads to a significant decrease in FRC, similar to what is observed in healthy individuals [65]. However, in individuals with obstructive lung disease, the reduction in FRC can exacerbate pre-existing air trapping and hyperinflation, leading to further V/Q mismatch. For example, the mean FRC in the awake state is typically around 2.7 ± 0.07 L, whereas during anesthesia and paralysis, it decreases to 2.15 ± 0.06 L [66,67]. This reduction in FRC occurs quickly after induction of anesthesia and remains relatively stable throughout the procedure.

The mechanical behavior of the lungs is also altered during general anesthesia, and this effect is more pronounced in obstructive lung disease. Respiratory compliance generally decreases due to the reduction in lung volume, particularly in patients with obstructive conditions. In these patients, the chest wall compliance remains relatively unaffected, but lung compliance is significantly impaired. The reduction in lung compliance during anesthesia may be partly due to decreased lung volumes, which can worsen airway collapse and atelectasis in the more dependent areas of the lung [68]. Additionally, anesthesia leads to an increase in respiratory resistance, driven by both the airway resistance and the "additional" resistance resulting from changes in the elastic properties of the lung and airway function. This increase in resistance further complicates ventilation in patients with obstructive lung disease, as it increases the WOB and contributes to a lower oxygenation status during surgery [69].

V/Q *Dynamics During Intraoperative General Anesthesia*

The physiological dynamics of V/Q balance are critically important during general anesthesia, particularly when patients are placed in the supine position and administered anesthesia. These factors significantly affect gas exchange and overall respiratory and cardiovascular function.

V/Q mismatch refers to the imbalance between the amount of air reaching the alveoli and the blood flow in the pulmonary capillaries, which is a key determinant of effective gas exchange. In the supine position, gravity alters the distribution of ventilation and perfusion in the lungs, potentially worsening the V/Q mismatch, especially in patients with pre-existing pulmonary conditions. When a patient is in the supine position, the posterior (dependent) parts of the lungs receive more blood flow due to gravity [70]. However, ventilation in these regions may be limited due to atelectasis (collapse of alveolar units) or reduced chest wall compliance, leading to poor V/Q matching in these areas. Conversely, the anterior (nondependent) lung regions may receive less perfusion, but may still have more adequate ventilation [71]. This can lead to wasted ventilation in parts of the lung with poor perfusion, further contributing to V/Q mismatch.

In the supine position, the diaphragm is displaced upward by abdominal contents, which reduces FRC and lung volumes. This reduction in lung volumes can predispose to atelectasis, especially in the posterior lung zones where ventilation is already reduced due to the gravitational effects. The decreased FRC and increased airway closure during exhalation (due to hyperinflation or loss of elastic recoil) can further exacerbate V/Q mismatch, leading to reduced oxygenation and ventilation. In patients with obstructive lung disease (e.g., COPD), the supine position can worsen air trapping and hyperinflation, thus further impairing ventilation and increasing the WOB [72].

Under general anesthesia, the loss of respiratory drive and muscle tone significantly affects the ventilatory response. This leads to hypoventilation, reduced tidal volume, and respiratory depression, which can exacerbate V/Q mismatch and hypoxia [73]. Certain intravenous anesthetic agents, such as opioids, can increase airway resistance, particularly in the upper airways, and promote airway collapse and atelectasis, especially in the supine position. This not only compromises ventilation but also reduces the efficiency of gas exchange, further worsening V/Q mismatch [74].

Anesthetic agents, particularly muscle relaxants, can reduce chest wall compliance and impair diaphragmatic movement, reducing the effectiveness of ventilation and further compromising lung function [73]. This can lead to a reduction in FRC and an increased likelihood of hypoxemia during general anesthesia. Anesthesia can impair hypoxic pulmonary vasoconstriction (HPV), a physiological mechanism that directs blood flow away from poorly ventilated regions of the lung [75]. In patients under general anesthesia, impaired HPV can lead to further V/Q mismatch by failing to properly match blood flow to ventilated lung regions, thus increasing the risk of hypoxemia.

The supine position alters the distribution of cardiac output in the lungs, shifting more blood to the dependent regions [71]. In normal lungs, this might not significantly impair gas exchange, but in patients with lung disease or pre-existing V/Q mismatch, this redistribution can worsen the imbalance, especially if ventilation to these regions is insufficient [76]. The increased venous return associated with the supine position may also increase the workload of the RV. In patients with right heart failure or PH, this can contribute to right ventricular strain and worsen V/Q mismatch.

Risks Associated With General Anesthesia and Mechanical Ventilation

General anesthesia presents multiple hazards for individuals afflicted with obstructive lung diseases, attributable to the modified physiology of their respiratory mechanisms. General anesthesia frequently induces relaxation of the airway smooth muscles, resulting in heightened airway resistance [73]. In patients with obstructive lung conditions, this phenomenon may aggravate pre-existing airway constriction, thereby increasing the probability of airflow obstruction and complicating the process of ventilation. Individuals suffering from obstructive lung diseases inherently encounter compromised gas exchange as a consequence of airway collapse, mucus obstruction, and inflammatory processes. The ramifications of anesthesia, including diminished FRC and atelectasis, further exacerbate the disparity between ventilation and perfusion, culminating in hypoxemia [74].

The administration of general anesthesia, particularly when patients are positioned supine, markedly reduces FRC. In subjects with obstructive lung disease, this decrement may facilitate dynamic hyperinflation, air trapping, and an elevated risk of alveolar collapse. Patients with obstructive lung diseases frequently exhibit impaired carbon dioxide clearance due to increased airway resistance and diminished expiratory flow [77]. Anesthesia may further compromise ventilation, resulting in hypercapnia, which has the potential to induce respiratory acidosis if not managed with sufficient vigilance.

Patients diagnosed with asthma or COPD exhibit an elevated susceptibility to bronchospasm during the processes of anesthesia induction, maintenance, or emergence. This phenomenon is particularly concerning in individuals with asthma characterized by airway hyperresponsiveness, wherein exposure to specific anesthetic agents, airway manipulation, or alternative triggers can incite significant bronchoconstriction [78]. The presence of obstructive lung disease frequently correlates with excessive mucus secretion and diminished mucociliary clearance. General anesthesia may suppress the cough reflex and impair the clearance of respiratory secretions, thereby increasing the risk of airway obstruction, atelectasis, and postoperative pulmonary infections.

Patients suffering from obstructive lung disease possess an increased vulnerability to barotrauma attributable to air trapping and hyperinflation, particularly during mechanical ventilation while under anesthesia. The application of excessive pressure may inflict barotrauma upon alveolar structures, resulting in complications such as pneumothorax or subcutaneous emphysema [79]. Individuals with obstructive lung conditions are predisposed to postoperative complications, including pneumonia, atelectasis, or extended periods of mechanical ventilation following surgical procedures. This predisposition is largely a consequence of their underlying compromised lung function, inadequate gas exchange, and diminished capacity to effectively clear airway secretions [80].

Ventilation strategies

Preoperative Assessment and Optimization

The preoperative assessment of patients with obstructive lung disease should focus on determining disease severity, recent clinical progression, and whether the patient's respiratory condition is adequately optimized before surgery. Chest radiography can help identify pulmonary air trapping and hyperinflation, which may be indicated by diaphragmatic flattening, a relatively small cardiac silhouette, and increased lung-field lucency. The presence of bullous changes should also be documented because of their potential clinical significance [81].

Pulmonary function tests are essential for evaluating the degree of airflow obstruction and its reversibility following bronchodilator administration. Important parameters include forced expiratory volume in one second (FEV_1), the FEV_1/FVC ratio, and peak expiratory flow rate. Patients with preoperative pulmonary function values below 50% of the predicted value may be at increased risk of postoperative pulmonary complications [82].

Arterial blood gas analysis will provide additional information regarding disease severity. Hypoxemia and hypercapnia are commonly observed in patients with moderate to severe obstructive lung disease, while elevated carbon dioxide levels may indicate significant airflow limitation and air trapping. Appropriate bronchodilator therapy should be continued until the time of surgery to optimize respiratory function. Depending on the patient's condition, treatment may include β_2-agonists, inhaled corticosteroids, leukotriene modifiers, mast cell stabilizers, theophylline, and anticholinergic agents [83].

Intraoperative Ventilation Approaches

During the initial phase of mechanical ventilation in patients with obstructive lung diseases, the primary objective is not to normalize arterial blood gas values but rather to minimize complications associated with pulmonary hyperinflation while maintaining an acceptable arterial pH of approximately 7.25-7.30 [84]. Although dynamic hyperinflation and auto-PEEP are common concerns in both severe asthma and COPD, ventilatory management should be tailored to the underlying pathophysiology of each condition. In acute asthma, particular emphasis should be placed on reversing bronchospasm and minimizing airway smooth muscle hyperreactivity, whereas in patients with emphysema and severe COPD, optimizing expiratory time is essential to reduce air trapping and prevent excessive dynamic hyperinflation.

Volume-controlled ventilation (VCV) vs. pressure-controlled ventilation (PCV): In patients with obstructive lung diseases, the selection between VCV and PCV is essential for optimizing respiratory mechanics and minimizing complications. VCV delivers a predetermined tidal volume, helping maintain consistent ventilation and reducing the risk of inadequate ventilation. However, increased airway resistance and reduced lung compliance may lead to elevated airway pressures. In volume assist-control ventilation, a square inspiratory flow waveform can be selected to facilitate the assessment of respiratory mechanics. Nevertheless, because the ventilator delivers a fixed tidal volume regardless of changes in airway resistance, peak airway pressure may increase, raising the risk of barotrauma and volutrauma, particularly in patients with severe airflow obstruction [85]. Another limitation of VCV is the potential development or worsening of dynamic hyperinflation and auto-PEEP when expiratory time is insufficient. This may increase intrathoracic pressure, impair venous return, and contribute to hemodynamic instability. Furthermore, the fixed tidal volume may not accommodate regional variations in lung compliance, potentially resulting in uneven gas distribution and regional overdistension [86]. Therefore, VCV requires careful adjustment in patients with COPD and asthma, in whom airway resistance may fluctuate considerably.

In contrast, PCV delivers gas at a predetermined inspiratory pressure, allowing tidal volume to vary according to changes in airway resistance and lung compliance. This may help limit peak airway pressures and reduce the risk of barotrauma. However, the delivered tidal volume can be inconsistent, particularly in patients with severe airflow obstruction. Evidence supporting the use of PCV in obstructive lung diseases remains limited, and the lack of sufficient clinical studies makes its advantages in this population uncertain [87]. A major concern with PCV is the influence of increased inspiratory resistance and auto-PEEP on the delivered tidal volume. As airway resistance increases, inspiratory flow and the pressure gradient between the ventilator circuit and the alveoli may decrease, resulting in unpredictable tidal volumes and potentially inadequate ventilation. Conversely, in patients with status asthmaticus, airway obstruction may improve rapidly during treatment, potentially causing an unexpected increase in tidal volume despite unchanged pressure settings. Modern ventilators provide continuous monitoring of respiratory parameters, allowing clinicians to identify changes in lung mechanics. Appropriate alarm limits can facilitate early detection of significant variations in tidal volume and minute ventilation, enabling timely adjustments to ventilator settings [88].

Overall, the choice between VCV and PCV in patients with COPD or asthma should be individualized according to the severity of airflow obstruction, respiratory mechanics, and response to mechanical ventilation. Close monitoring and appropriate adjustment of ventilator settings are necessary to maintain adequate gas exchange while minimizing dynamic hyperinflation and ventilator-induced barotrauma.

Low tidal volume strategy: Numerous studies, particularly those investigating acute respiratory distress syndrome (ARDS), have demonstrated the benefits of low tidal volume ventilation in preventing excessive lung inflation. In patients with COPD, using lower tidal volumes helps limit plateau pressure to below 30 cm H_2O, reducing the risk of ventilator-induced lung injury [89]. Lower tidal volumes can also shorten inspiratory time, thereby extending the time available for expiration and allowing the hyperinflated lungs to empty more effectively.

The protective effect of low tidal volume ventilation in COPD is closely related to the underlying pathophysiology of the disease, which is characterized by pulmonary hyperinflation. This occurs through two main mechanisms: loss of elastic recoil due to destruction of lung parenchyma, resulting in static hyperinflation, and airflow limitation caused by small airway damage, leading to dynamic hyperinflation. These changes increase the lung's relaxation volume, resulting in elevated end-expiratory lung volume and residual volume, while reducing inspiratory reserve volume. Consequently, the capacity of the lungs to accommodate additional tidal volume is limited, making them more susceptible to excessive distension and increasing the risk of barotrauma when high tidal volumes are used [90].

PEEP management: In patients with obstructive lung disease receiving assisted mechanical ventilation, the main objectives of applying PEEP are to improve ventilator triggering, reduce the WOB associated with the inspiratory threshold load imposed by PEEPi, and enhance patient-ventilator synchrony. One effective strategy for reducing or eliminating PEEPi is to decrease minute ventilation, even if this results in alveolar hypoventilation and permissive hypercapnia [91]. In patients with COPD receiving assist-control ventilation, inspiratory flow is often increased to shorten inspiratory time and provide a longer period for expiration, with the aim of reducing PEEPi. However, increasing inspiratory flow may also increase the respiratory rate [92], potentially shortening expiratory time and worsening air trapping. Interestingly, a study involving mechanically ventilated patients with COPD demonstrated that increasing inspiratory flow from 30 to 90 L/min increased respiratory rate but paradoxically reduced PEEPi [93]. This reduction was attributed to a prolongation of expiratory time, which facilitated lung deflation. The researchers suggested that shortened inspiratory time, combined with the heterogeneous time constants characteristic of COPD, allowed some lung units to remain overinflated during neural expiration. This persistent inflation may stimulate vagal receptors, prolonging expiratory time [93].

Historically, the use of external PEEP in patients with COPD was avoided because of concerns that it could worsen pulmonary hyperinflation. However, subsequent evidence has demonstrated that applying external PEEP during assisted ventilation in patients with airflow limitation can reduce the effort required to trigger the ventilator, improve patient-ventilator synchrony, and decrease the WOB. Once ventilator settings have been optimized, external PEEP can be gradually titrated to approximately 70% of the static intrinsic PEEP, with careful monitoring of plateau pressure during VCV and tidal volume during PCV. The benefits of external PEEP in mechanically ventilated patients with COPD depend on the presence of airflow limitation and patient-triggered breaths [94,95].

Because the effect of external PEEP on pulmonary hyperinflation is unpredictable, its titration should be guided by measurements of static intrinsic PEEP obtained during passive ventilation [96]. In patients with COPD who experience dynamic hyperinflation during assisted mechanical ventilation, applying low levels of external PEEP may reduce the WOB, facilitate ventilator triggering, and improve patient-ventilator interaction [97].

Adjustments for prolonged expiratory time to avoid air trapping: In patients with obstructive lung diseases and expiratory flow limitation requiring mechanical ventilation, the primary management strategy is to prolong expiratory time and reduce tidal volume to facilitate complete exhalation. Increasing expiratory time allows expiratory flow to return to zero before the next breath begins, thereby reducing air trapping, auto-PEEP, and pulmonary hyperinflation. From the ventilator's perspective, expiration is the period between the end of one inspiratory phase and the initiation of the next breath [98]. The relationship between inspiratory and expiratory durations is expressed as the inspiratory-to-expiratory (I:E) ratio. Patients with obstructive lung disease generally benefit from a lower I:E ratio, which provides more time for exhalation. Since the respiratory cycle consists of inspiration and expiration, shortening inspiratory time increases the time available for expiration [99]. Inspiratory time can be reduced by shortening the duration of each breath or decreasing the respiratory rate.

In assist-control ventilation modes, including VCV and PCV, reducing the set respiratory rate decreases the total time spent in inspiration per minute, thereby extending expiratory time. In VCV, increasing inspiratory flow allows the preset tidal volume to be delivered more rapidly, shortening inspiratory time and consequently increasing expiratory time [100]. Reducing tidal volume also shortens inspiratory duration at a constant flow rate and decreases the volume of gas that must be exhaled, facilitating more complete expiration. In PCV, airway pressure is controlled, and the end of inspiration is determined by the set inspiratory time. Therefore, directly shortening inspiratory time increases the duration of expiration. Additionally, reducing the set inspiratory pressure decreases the delivered tidal volume, reducing the amount of gas that must be exhaled and helping to limit air trapping and dynamic hyperinflation [101].

Challenges and controversies

Debates in PEEP Optimization

High PEEP is primarily used to prevent atelectotrauma by avoiding repetitive alveolar collapse and reopening; however, its physiological effects differ substantially between acute hypoxemic respiratory failure and COPD [102]. In recruitable lungs, higher PEEP stabilizes unstable alveoli and reduces cyclic shear stress. In contrast, COPD lungs are characterized by airflow limitation, dynamic hyperinflation, and auto-PEEP, rather than widespread alveolar collapse. In this setting, excessive external PEEP may not improve recruitment but instead exacerbate lung hyperinflation, increase end-expiratory lung volume, and elevate intrathoracic pressure. While a carefully titrated external PEEP may be beneficial in selected COPD patients by counterbalancing auto-PEEP and reducing inspiratory threshold load, overly high PEEP increases the risk of overdistension, barotrauma, and hemodynamic compromise, underscoring the need for disease-specific PEEP optimization.

An incremental-decremental recruitment maneuver is typically performed by stepwise increasing PEEP (e.g., in 2-5 cmH_2O increments) while using PCV with a fixed driving pressure of ~15 cmH_2O and limiting the

maximum airway pressure (Pmax) to ~40 cmH_2O, then decrementing PEEP stepwise and selecting the PEEP level at which respiratory system compliance is highest (and/or driving pressure is lowest), indicating the best trade-off between recruitment and overdistension. However, in COPD, the utility of these maneuvers is fundamentally limited [103]. The predominant mechanical abnormality in COPD is increased airway resistance and expiratory flow limitation, not alveolar collapse; therefore, incremental increases in PEEP may preferentially inflate already open alveoli rather than recruit collapsed regions. During decremental titration, compliance may falsely appear optimal at higher PEEP levels due to reduced inspiratory effort or altered chest wall mechanics, rather than true lung recruitment. As a result, optimal PEEP in COPD is often defined not by maximal compliance alone, but by the level that minimizes dynamic hyperinflation, reduces auto-PEEP, and improves patient-ventilator synchrony, frequently requiring lower PEEP levels and prolonged expiratory times rather than aggressive recruitment strategies [103].

Pressure-volume (P-V) loop analysis provides detailed insight into the interaction between applied PEEP and the altered respiratory mechanics characteristic of COPD. A slope (b) greater than 1 on the inspiratory limb reflects a disproportionate increase in airway pressure relative to tidal volume delivery, indicating reduced respiratory system compliance. In COPD, this phenomenon is predominantly driven by static and dynamic hyperinflation, resulting from expiratory flow limitation, prolonged time constants, and incomplete lung emptying. As end-expiratory lung volume progressively increases, ventilation is forced to operate on the upper, flatter portion of the lung compliance curve, where elastic recoil is reduced, and further volume expansion requires substantially higher pressure [104].

Excessive external PEEP, when added to already elevated auto-PEEP, further elevates alveolar pressure and shifts the FRC above the optimal mechanical range. This upward shift reduces the number of available recruitable units and increases regional overdistension, particularly in relatively preserved lung regions, thereby amplifying stress heterogeneity. The resulting flattening of the inspiratory limb and widening of loop hysteresis reflect increased energy dissipation, impaired elastic unloading during expiration, and worsening dynamic hyperinflation [105]. From a pathophysiological standpoint, this environment promotes volutrauma through excessive alveolar stretch, barotrauma due to high end-inspiratory pressures, and hemodynamic compromise secondary to increased intrathoracic pressure and reduced venous return.

Furthermore, excessive PEEP in COPD increases inspiratory threshold load, exacerbating patient-ventilator asynchrony and triggering ineffective efforts, particularly during assisted modes. Recognition of a $b > 1$ slope on P-V loops is therefore critical, as it signals ventilation beyond the lung's optimal mechanical range. Therapeutic adjustments-including reduction of external PEEP, limitation of driving pressure, and prolongation of expiratory time-allow deflation toward a more compliant operating zone, reducing elastic workload, improving synchrony, and minimizing ventilator-induced lung injury in this vulnerable population [104].

Use of Advanced Modes of Ventilation

High-frequency oscillatory ventilation (HFOV) delivers very small tidal volumes and very high frequencies while maintaining a relatively constant mean airway pressure, a strategy intended to limit volutrauma and atelectrauma by maintaining sustained lung recruitment. While this approach is physiologically appealing in diffuse alveolar disease, its applicability to COPD is inherently limited by the underlying mechanics of airflow obstruction. COPD is characterized by expiratory flow limitation, prolonged respiratory time constants, and a marked predisposition to dynamic hyperinflation, features that fundamentally conflict with the short expiratory times and fixed pressure baseline intrinsic to HFOV [106]. Importantly, large randomized trials evaluating HFOV in adult respiratory failure, such as OSCAR [107] and OSCILLATE [108], focused on hypoxemic ARDS populations and did not include patients with obstructive lung disease, and overall failed to demonstrate a mortality benefit.

Evidence supporting HFOV in COPD is therefore sparse and largely limited to small physiological studies. In a pilot study by Frerichs et al. [109], ten patients with acute COPD exacerbations and hypercapnic respiratory failure were briefly transitioned to HFOV using lower mean airway pressures than those typically applied in ARDS. Although short-term gas exchange was preserved without immediate adverse effects, the study highlighted persistent concerns regarding air trapping and overdistension. Even when lower oscillatory frequencies (5-6 Hz) and reduced mean airway pressures (~20-22 cmH_2O) are employed, expiratory flow limitation may prevent complete lung emptying, leading to progressive intrinsic PEEP and rising end-expiratory lung volume. This upward shift in the operating point places ventilation on the flatter, overdistended portion of the P-V curve, increasing elastic load and impairing carbon dioxide elimination, which in HFOV depends on oscillatory amplitude and gas diffusion-both compromised in obstructive physiology [109].

Given the absence of robust randomized evidence in COPD populations and the high mechanistic risk of worsening dynamic hyperinflation, intrinsic PEEP, and hemodynamic compromise, HFOV is not recommended as a routine ventilation strategy in obstructive lung disease. If considered at all, its use should be restricted to highly selected rescue scenarios under strict monitoring, with careful limitation of mean airway pressure and oscillatory amplitude, and frequent assessment of auto-PEEP and cardiovascular

effects.

In contrast, adaptive support ventilation (ASV) represents a closed-loop mode that continuously adjusts tidal volume, respiratory rate, and inspiratory pressure based on real-time measurements of respiratory system mechanics, with the goal of minimizing the WOB while maintaining adequate minute ventilation [110]. This adaptive behavior is particularly advantageous in COPD, where airway resistance and expiratory time constants are variable and prone to rapid change. By applying optimization principles derived from the Otis equation, ASV typically selects lower respiratory rates and longer expiratory times, thereby directly targeting the principal mechanism of ventilator-induced harm in obstructive disease: dynamic hyperinflation.

Clinical evidence supports this physiological rationale. In a randomized controlled trial by Kirakli et al. [111], ASV was compared with conventional pressure support ventilation (PSV) for weaning in mechanically ventilated COPD patients. ASV was associated with significantly shorter weaning duration while achieving similar weaning success rates and a trend toward reduced ICU length of stay. More recently, Sehgal et al. [112] evaluated the feasibility of ASV during noninvasive ventilation for acute COPD exacerbations and reported comparable NIV success rates and potential reductions in intubation compared with PSV. Collectively, these studies suggest that ASV's adaptive algorithms, by favoring ventilatory patterns aligned with obstructive pathophysiology, may improve patient-ventilator synchrony, reduce dynamic hyperinflation, and facilitate liberation from mechanical ventilation. Although larger multicenter trials are still required, current data indicate that ASV offers a favorable physiological and clinical profile in obstructive lung disease compared with fixed-parameter ventilation modes.

Postoperative care

Weaning Strategies

Gradual reduction in ventilatory support is a cornerstone of successful weaning from mechanical ventilation, particularly in patients with COPD, in whom respiratory muscle dysfunction, dynamic hyperinflation, and intrinsic PEEP substantially increase the WOB. Rather than abrupt withdrawal of support, ventilatory assistance is progressively reduced to allow physiological adaptation while minimizing respiratory muscle fatigue. This strategy typically involves stepwise decreases in pressure support, mandatory breath frequency, or assist levels, accompanied by close monitoring of gas exchange, respiratory rate, tidal volume, and patient comfort. In COPD, gradual unloading is especially important, as sudden increases in ventilatory demand may overwhelm weakened inspiratory muscles, exacerbate auto-PEEP, and precipitate tachypnea, ineffective triggering, and weaning failure.

From a pathophysiological standpoint, gradual weaning provides time for partial recovery of diaphragmatic function, improved coordination between neural respiratory drive and mechanical output, and reduction of patient-ventilator asynchrony. Progressive reductions in ventilatory support promote spontaneous breathing while still providing sufficient assistance to overcome increased airway resistance and PEEPi. Clinical studies have demonstrated that stepwise pressure support reduction or the use of adaptive modes facilitates smoother transitions to spontaneous breathing compared with abrupt spontaneous breathing trials, particularly in high-risk populations such as COPD patients [113]. Importantly, this approach allows early recognition of weaning intolerance, manifested by rising $PaCO_2$, increasing WOB, or hemodynamic instability, enabling timely adjustment of ventilatory settings and reducing the likelihood of reintubation.

A representative example of this strategy is given by the randomized controlled trial conducted by Kirakli et al. [111]. The study details a unit-specific PSV weaning protocol that was devised within a dedicated respiratory intensive care unit; accordingly, it should be viewed as an illustrative framework rather than a universally prescriptive algorithm. In this protocol, weaning began only after strict clinical stability criteria were satisfied, underscoring the value of individualized patient assessment and institutional expertise. Pressure support was first set at 15 cmH_2O above PEEP and then lowered in 2 cmH_2O steps at regular reassessment intervals, while keeping low external PEEP (3-5 cmH_2O) to reduce intrinsic PEEP. Readiness for extubation was evaluated at a pressure support level of 7 cmH_2O, with this cutoff chosen mainly to offset endotracheal tube and circuit resistance.

Recent recommendations from the 2024 American Association for Respiratory Care (AARC) Clinical Practice Guideline emphasize a standardized and individualized approach to ventilator liberation in critically ill adults. Readiness for a spontaneous breathing trial (SBT) should be assessed based on the improvement of the underlying cause of respiratory failure, adequate oxygenation, hemodynamic stability, and the patient's ability to initiate inspiratory effort. The guideline suggests that the rapid shallow breathing index (RSBI) is not mandatory for determining readiness for an SBT and that SBTs may be performed either with or without pressure-support ventilation. Furthermore, the fraction of inspired oxygen (FiO_2) should not be increased during the SBT. Throughout the trial, respiratory pattern, hemodynamic parameters, gas exchange, and patient comfort should be closely monitored. In patients with COPD, these general recommendations should be complemented by careful assessment of expiratory flow limitation, PEEPi, dynamic hyperinflation, and respiratory muscle workload, as these factors may contribute to weaning failure. Therefore, successful ventilator liberation requires not only the fulfillment of general readiness criteria but also an individualized

assessment of respiratory mechanics and the patient's ability to sustain spontaneous breathing without excessive respiratory effort [114].

Overall, this structured, physiology-informed approach demonstrates how stepwise withdrawal of ventilatory support can be effectively applied in COPD, while emphasizing the need for individualized adjustments rather than rigid protocol generalization. In particular, the application of external PEEP should be guided by the presence of demonstrable expiratory flow limitation and patient-triggered breaths, as its inappropriate use may worsen pulmonary hyperinflation, increase alveolar pressure, and further elevate the inspiratory threshold load.

Role of Noninvasive Ventilation

The application of noninvasive positive pressure ventilation (NIPPV), including continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP), has emerged as a key approach to respiratory support during the immediate postoperative period in the post-anesthesia care unit (PACU). Because of residual effects of anesthetics and opioids, diminished cough effectiveness, reduced FRC, and atelectasis, postoperative patients are especially prone to respiratory compromise [115]. CPAP delivers a steady positive airway pressure across the entire respiratory cycle, which enhances alveolar recruitment, increases end-expiratory lung volume, and helps prevent airway collapse [116]. Consequently, CPAP is particularly beneficial for individuals with obstructive sleep apnea, obesity, or isolated postoperative hypoxemia, since it improves oxygenation without increasing inspiratory effort or changing ventilatory drive.

BiPAP uses two separate pressure settings-higher inspiratory positive airway pressure (IPAP) and lower expiratory positive airway pressure (EPAP)-enabling it to integrate alveolar stabilization with active ventilatory assistance. This modality is especially helpful in patients with hypoventilation, hypercapnia, or increased WOB, including those with chronic obstructive pulmonary disease or residual neuromuscular dysfunction after surgery [117]. By increasing tidal volume and relieving the inspiratory muscles, BiPAP improves carbon dioxide elimination, whereas EPAP preserves airway patency and counteracts atelectasis. Compared with CPAP, BiPAP allows more individualized tailoring of support but calls for closer monitoring to prevent excessive pressure delivery, gastric insufflation, or intolerance in the patient [118].

In postoperative COPD patients managed in the PACU, initial NIPPV settings should be conservative and tailored to minimize dynamic hyperinflation, reduce inspiratory threshold load, and maintain patient-ventilator synchrony. When CPAP is selected in patients with preserved ventilatory drive and predominantly hypoxemic respiratory failure, starting pressures of 5-8 cmH_2O are commonly appropriate, with lower levels (5-6 cmH_2O) favored initially to partially offset PEEPi without excessively increasing end-expiratory lung volume [119]. Inspired oxygen should be titrated to achieve SpO_2 88-92%, avoiding unnecessary hyperoxia that may exacerbate CO_2 retention. Because CPAP does not provide ventilatory assistance, its use should be cautious in patients with marginal ventilatory reserve.

BiPAP is generally preferred when COPD patients exhibit elevated $PaCO_2$, increased respiratory effort, or residual anesthetic-related hypoventilation. Typical initial settings in the PACU include IPAP 10-12 cmH_2O and EPAP 4-5 cmH_2O, resulting in a pressure support of approximately 6-8 cmH_2O. This level is usually sufficient to enhance tidal volume and reduce inspiratory muscle load while limiting the risk of overdistension. EPAP should remain low to moderate to avoid worsening hyperinflation, while still providing partial compensation for PEEPi. Respiratory rate, tidal volume, mental status, and patient comfort should be reassessed frequently, with cautious IPAP increments only if hypercapnia or respiratory distress persists [120].

Overall, noninvasive ventilation in the PACU should be applied early, selectively, and with close clinical surveillance. In postoperative COPD patients, careful initial titration and frequent reassessment during the first 30-60 minutes are essential to ensure improvement in WOB and gas exchange [121]. Failure to demonstrate early clinical benefit or evidence of deterioration should prompt timely escalation of care rather than aggressive increases in airway pressure.

Conclusions

Perioperative ventilation of obstructive lung disease is a challenge for physicians. A physiology-based approach to perioperative ventilation provides a framework for personalized and precision respiratory medicine. Restricted airflow prevents full exhalation, leading to trapped air and progressive overinflation of the lungs. Decreased lung elastance and increased resistance in the airways augment the WOB, making mechanical ventilation especially challenging due to risks of incomplete exhalation between breaths and the development of auto-PEEP. Therefore, successful ventilator management requires approaches including prolonged expiration time, reduced breath volume, careful use of externally applied PEEP, and the use of noninvasive ventilation techniques to improve coordination between the patient and ventilator while optimizing respiratory support.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Acquisition, analysis, or interpretation of data: Panagiota Griva

Drafting of the manuscript: Panagiota Griva

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