Review Article

Postoperative Dyspnea: Most Frequent Etiologies, Initial Evaluation, and Early Clinical Management

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Abstract

Postoperative dyspnea is a frequent and clinically significant complication that arises from a complex interaction of perioperative pathophysiological mechanisms, underlying comorbidities, and surgical factors. General anesthesia plays a central role by reducing functional residual capacity, promoting small airway closure, and generating ventilation–perfusion mismatch. These alterations favor the development of dependent atelectasis, which represents the most common cause of early postoperative dyspnea. Additional contributors include postoperative pain-induced hypoventilation, diaphragmatic dysfunction, residual neuromuscular blockade, fluid overload, ventricular dysfunction, and systemic inflammatory responses that impair pulmonary gas exchange. Pulmonary etiologies predominate in the differential diagnosis, particularly atelectasis, pulmonary edema, pneumonia, and pulmonary embolism, although cardiovascular, upper airway, metabolic, and systemic causes must also be considered. Risk stratification is essential, as advanced age, preexisting pulmonary or cardiovascular disease, obesity, obstructive sleep apnea, smoking history, prolonged surgical duration, emergency surgery, and high-risk procedures significantly increase the likelihood of postoperative

respiratory complications. A structured diagnostic approach integrates arterial blood gas analysis, chest radiography, electrocardiography, cardiac biomarkers, computed tomography pulmonary angiography when indicated, and bedside lung and cardiac ultrasound. Early management prioritizes supportive measures such as supplemental oxygen, non-invasive respiratory support, semi-upright positioning, and continuous monitoring, followed by etiology-directed treatment including respiratory physiotherapy, diuretics, anticoagulation, antibiotics, bronchodilators, or reversal of neuromuscular blockade when appropriate. Preventive strategies, including preoperative optimization, lung-protective ventilation, restrictive fluid management, early mobilization, and thromboprophylaxis, are fundamental to reducing postoperative pulmonary complications, morbidity, and mortality, ultimately improving overall surgical outcomes.

Key words

Atelectasis, ventilación–perfusión, insuficiencia respiratoria, estratificación de riesgo, monitoreo continuo, ventilación no invasive.

Introduction

The incidence of postoperative dyspnea varies considerably according to the type of surgical procedure performed, with thoracic surgery representing the highest-risk category. Postoperative dyspnea is particularly prevalent after lung resections and pneumonectomies, where direct surgical manipulation of pulmonary parenchyma and alterations in respiratory mechanics predispose patients to impaired oxygenation. Impaired oxygenation, considered a marker of lung injury, has been reported in approximately 70.8% of patients following lung resection, with significant associations identified between postoperative oxygenation deficits and intraoperative variables such as driving pressure and inspired oxygen fraction [1]. Among thoracic procedures, pneumonectomy carries the greatest risk burden, as postoperative pulmonary complications occur in approximately 21% of patients, underscoring the magnitude of respiratory vulnerability in this population [2].

Upper abdominal surgery also demonstrates a substantial incidence of postoperative pulmonary complications. Although specific data focusing exclusively on dyspnea incidence are less frequently reported in comparison to thoracic surgery, these procedures are consistently associated with respiratory compromise due to diaphragmatic dysfunction, postoperative pain, and reduced lung volumes. In contrast, vascular and orthopedic surgeries generally present a lower overall incidence of postoperative dyspnea. Nevertheless, patients with significant pre-existing cardiopulmonary disease or those undergoing complex or prolonged procedures remain at meaningful risk, indicating that surgical category alone does not fully determine respiratory outcomes [1, 2].

From a clinical perspective, postoperative dyspnea holds particular importance as an early warning sign. The presence of dyspnea on postoperative day one may signal the onset of postoperative pulmonary complications, and higher scores on validated symptom inventories have been shown to correlate with an increased likelihood of adverse events [3]. This association highlights the prognostic value of early symptom recognition and structured assessment. The development of postoperative pulmonary complications has a direct and measurable impact on clinical outcomes. In patients undergoing pneumonectomy, such complications have been identified as major risk factors for in-hospital mortality [2]. Moreover, dyspnea and its underlying causes contribute to prolonged hospitalization and higher rates of intensive care unit admission [4].

The relationship between postoperative dyspnea and adverse outcomes is further reflected in its

association with increased morbidity and mortality across surgical populations. Postoperative pulmonary complications following lung cancer surgery, for example, have been linked not only to elevated mortality rates but also to significantly longer hospital stays. The prolongation of hospitalization is particularly pronounced in cases complicated by extended mechanical ventilation or septic shock, which may increase hospital stay duration by more than 17 days [2, 5]. In severe scenarios, dyspnea accompanied by significant respiratory compromise necessitates escalation of care to the intensive care unit, especially when respiratory failure develops or when non-invasive respiratory support is required [6, 7].

The objective of this article is to analyze the most frequent etiologies of postoperative dyspnea, outline a structured and systematic approach to its initial clinical evaluation, and describe evidence-based strategies for early management to facilitate timely diagnosis, reduce postoperative complications, and improve overall patient outcomes.

Methodology

This manuscript was developed as a structured narrative review aimed at providing an updated and clinically integrated analysis of postoperative dyspnea, with a particular focus on its most frequent etiologies, initial diagnostic approach, and early evidence-based management strategies. The review was conducted in accordance with the SANRA (Scale for the Assessment of Narrative Review Articles) framework and emphasized interpretative synthesis, pathophysiological–clinical integration, and practical applicability rather than systematic quantitative pooling. Special attention was given to the differentiation between pulmonary and cardiac causes, risk stratification according to type of surgery, structured bedside evaluation, and early therapeutic interventions in both immediate and early postoperative settings. The objective was to generate a clinically applicable framework to support timely recognition and management in diverse surgical populations.

A comprehensive literature search was performed in PubMed, Scopus, and Web of Science, including peer-reviewed articles published in English or Spanish between January 2020 and December 2026. This timeframe was selected to incorporate contemporary perioperative care protocols, advances in lung ultrasound and point-of-care echocardiography, updated recommendations on non-invasive ventilation strategies, and recent data on postoperative pulmonary complications across surgical specialties. Foundational studies were included when necessary to provide conceptual or pathophysiological context. The search strategy combined MeSH and free-text terms using Boolean operators related to postoperative dyspnea, postoperative pulmonary complications, atelectasis, pulmonary embolism, perioperative myocardial infarction, respiratory failure, non-invasive ventilation, lung expansion techniques, and perioperative risk assessment. The initial search yielded 184 records; after removal of duplicates and title–abstract screening, 92 articles underwent full-text evaluation, and 49 studies were included in the final synthesis. Selection and evaluation were conducted independently by two authors, with discrepancies resolved through consensus. Exclusion criteria comprised non–peer-reviewed publications, isolated case reports, editorials, purely technical descriptions without clinical outcome data, redundant datasets, and studies not directly addressing postoperative respiratory symptoms, etiologic evaluation, or early management strategies.

Eligible studies included randomized controlled trials, large observational cohorts, meta-analyses, expert consensus statements, and contemporary international guidelines from relevant anesthesiology, surgery, and critical care societies. Priority was assigned to multicenter studies, investigations with standardized definitions of postoperative pulmonary

complications, and research evaluating early diagnostic tools such as bedside ultrasound as well as comparative effectiveness of preventive and therapeutic interventions. Extracted variables included study design, surgical population characteristics, type of procedure, incidence and etiology of dyspnea, diagnostic strategies employed, respiratory support modalities, complication rates, and short-term clinical outcomes. Methodological quality and internal validity were assessed narratively, considering risk of bias, heterogeneity in definitions of dyspnea and pulmonary complications, follow-up duration, and consistency across findings. In cases of conflicting evidence, greater interpretative weight was given to higher-level evidence and guideline-supported recommendations. Given its narrative design, this review is subject to potential selection bias and does not provide pooled quantitative estimates. Artificial intelligence–based tools were used exclusively to assist in literature organization and structural coherence, whereas critical appraisal and final interpretation were conducted independently by the authors to preserve methodological rigor.

Perioperative Pathophysiological Mechanisms

General anesthesia produces significant alterations in respiratory physiology that contribute directly to the development of postoperative dyspnea. One of the primary mechanisms involved is the reduction in functional residual capacity, a common consequence of anesthetic induction. The decrease in functional residual capacity promotes small airway closure and leads to ventilation–perfusion mismatch, a key determinant of impaired gas exchange in the perioperative period. This mismatch reduces effective oxygenation and represents a major contributor to postoperative dyspnea. In addition to airway closure, general anesthesia frequently results in the formation of dependent atelectasis. The collapse of alveolar units impairs blood oxygenation and decreases lung compliance, thereby predisposing patients to postoperative respiratory complications and worsening dyspneic symptoms [8, 9].

Beyond anesthetic-related effects, postoperative mechanical factors further aggravate respiratory compromise. Pain, particularly following upper abdominal or thoracic surgery, commonly induces hypoventilation due to limited chest wall expansion and protective splinting. This reduction in effective ventilation exacerbates dyspnea by further decreasing lung volumes and impairing adequate alveolar ventilation. Additionally, surgical procedures involving the thorax or upper abdomen may compromise diaphragmatic function, restrict lung expansion and contributing to persistent respiratory difficulty in the early postoperative period. Residual neuromuscular blockade represents another important factor, as incomplete reversal of neuromuscular blocking agents can lead to generalized muscle weakness, including involvement of the respiratory musculature. This weakness diminishes ventilatory capacity and increases the risk of postoperative dyspnea [10].

Hemodynamic alterations also play a central role in the pathogenesis of postoperative respiratory symptoms. Careful perioperative fluid management is essential, since fluid overload can elevate pulmonary capillary hydrostatic pressure and promote the development of pulmonary edema. The accumulation of fluid within the pulmonary interstitium and alveolar spaces interferes with effective gas exchange, resulting in dyspnea. Acute ventricular dysfunction may occur in the postoperative setting. Both left and right ventricular impairment can contribute to hemodynamic instability, elevate pulmonary pressures, and exacerbate respiratory compromise [11].

In parallel, inflammatory and metabolic responses induced by surgical stress further influence postoperative respiratory function. Surgery triggers systemic inflammatory activation, which increases vascular permeability and promotes pulmonary edema, thereby

worsening respiratory status and predisposing to complications [8]. At the same time, the metabolic demands of the postoperative state lead to increased oxygen consumption. In certain cases, metabolic acidosis may develop, prompting compensatory tachypnea as the body attempts to maintain acid–base homeostasis. This hyperventilatory response may be perceived clinically as dyspnea, linking metabolic disturbances to postoperative respiratory symptomatology [11].

Most Frequent Etiologies and Structured Differential Diagnosis

Pulmonary causes represent the most frequent etiologies of postoperative dyspnea, with atelectasis being the predominant contributor, particularly within the first 48 hours after surgery. Atelectasis develops when alveoli collapse, resulting in impaired gas exchange and reduced lung compliance. It is highly prevalent, occurring in up to 90% of anesthetized patients, and may be further exacerbated by intraoperative mechanical ventilation strategies and specific surgical procedures such as laparoscopic interventions [8, 12]. The resulting ventilation–perfusion mismatch and decreased oxygenation make atelectasis a central mechanism underlying early postoperative respiratory symptoms. In addition to alveolar collapse, pulmonary edema must be considered, whether cardiogenic or non-cardiogenic in origin. In both forms, fluid accumulation within the pulmonary interstitium and alveolar spaces compromises oxygen diffusion and contributes significantly to dyspnea. Diagnostic tools such as lung ultrasound have demonstrated utility in identifying pulmonary edema in the perioperative setting [13].

Postoperative pneumonia constitutes another important pulmonary cause and is associated with considerable morbidity, particularly following cardiovascular surgery. Advanced age, smoking history, and prolonged mechanical ventilation are recognized risk factors, whereas preventive strategies such as preoperative respiratory physiotherapy may reduce its incidence [14]. Pulmonary embolism, although less frequent than atelectasis or pneumonia, represents a potentially life-threatening condition that requires prompt recognition and treatment. It typically presents with sudden-onset dyspnea and is confirmed through appropriate imaging modalities. Additionally, bronchospasm and aspiration pneumonitis should be considered, especially in patients with underlying obstructive lung disease. These conditions can precipitate significant postoperative respiratory distress and further complicate clinical management [15].

Cardiovascular causes also play a critical role in the differential diagnosis of postoperative dyspnea. Acute heart failure may develop secondary to fluid overload or myocardial dysfunction, leading to pulmonary congestion and respiratory symptoms. Similarly, perioperative myocardial ischemia or infarction can impair cardiac output and increase pulmonary venous pressures, thereby manifesting as dyspnea. Hemodynamically significant arrhythmias further compromise cardiac efficiency and may produce symptoms consistent with heart failure, including shortness of breath [16].

Upper airway etiologies must also be considered, particularly in the immediate postoperative period. Laryngeal edema and cervical hematoma, often related to surgical manipulation or perioperative trauma, can result in airway obstruction and acute breathing difficulty. In susceptible individuals, postoperative factors may exacerbate obstructive sleep apnea, leading to recurrent episodes of apnea and consequent dyspnea [17].

Systemic and metabolic conditions may contribute to the sensation of dyspnea. Severe anemia reduces oxygen-carrying capacity and can result in tissue hypoxia, prompting compensatory respiratory responses. Sepsis and metabolic acidosis alter respiratory drive and frequently produce tachypnea as a compensatory

mechanism. Moreover, psychological factors such as anxiety or pain-related hyperventilation may intensify or mimic respiratory distress, further complicating the clinical picture and necessitating careful diagnostic evaluation [16].

Risk Factors and Patient Stratification

Advanced age represents one of the most significant risk factors for postoperative pulmonary complications and, consequently, for postoperative dyspnea. Elderly patients are particularly vulnerable due to age-related physiological changes, including reduced pulmonary reserve, decreased chest wall compliance, and diminished immune response, often compounded by multiple comorbidities. Evidence indicates that advanced age is an independent predictor of postoperative pulmonary complications, which are associated with prolonged mechanical ventilation and increased mortality rates [18].

Preexisting pulmonary or cardiovascular disease further amplifies the risk of postoperative respiratory compromise. Patients with chronic obstructive pulmonary disease or underlying cardiovascular conditions exhibit reduced physiological reserve and are more prone to respiratory failure and other postoperative complications. These comorbidities predispose individuals to impaired gas exchange, hemodynamic instability, and delayed recovery following surgery [18, 19].

Obesity and obstructive sleep apnea constitute additional critical risk factors. Obstructive sleep apnea, in particular, is associated with an increased risk of cardiorespiratory complications in the perioperative setting, largely due to challenges in airway management and heightened sensitivity to anesthetic agents [20, 21]. Obesity compounds this risk by increasing the probability of difficult intubation and ventilation, as well as by altering respiratory mechanics through reduced lung volumes and increased work of breathing [22].

A history of smoking also plays a substantial role in postoperative risk stratification. Smoking is associated with chronic impairment of lung function and increased susceptibility to postoperative pulmonary complications. It exacerbates respiratory morbidity by promoting conditions such as pneumonia and atelectasis in the postoperative period [23].

Intraoperative factors likewise influence the development of postoperative dyspnea. Prolonged surgical duration is associated with extended exposure to anesthesia, greater likelihood of fluid shifts, and increased risk of respiratory compromise [24]. Positive fluid balance during surgery further contributes to pulmonary edema by increasing hydrostatic pressures within the pulmonary circulation, thereby predisposing patients to dyspnea in the postoperative period. Careful management of fluid administration is therefore essential to minimize respiratory complications [18].

Emergency surgery represents another important determinant of postoperative risk. The urgent nature of these procedures often precludes adequate preoperative optimization and exposes patients to greater physiological stress, resulting in higher rates of postoperative complications [24]. High-risk procedures such as thoracic, upper abdominal, and major vascular surgeries inherently increase the likelihood of postoperative dyspnea. These operations significantly affect respiratory mechanics and are frequently associated with substantial blood loss and fluid shifts, further contributing to respiratory compromise [11].

Initial Diagnostic Workup

Arterial blood gas analysis represents a fundamental component in the evaluation of postoperative dyspnea, as it provides immediate and objective information regarding oxygenation, ventilation, and acid–base status. By assessing parameters such as partial pressures of oxygen and carbon dioxide, as well as pH and bicarbonate levels, arterial blood gas testing

facilitates the identification of respiratory failure and metabolic disturbances that may contribute to dyspnea [8]. Its rapid availability makes it particularly valuable in unstable patients or in those with suspected hypoxemia or ventilatory impairment.Chest radiography remains a routinely employed imaging modality in the postoperative setting. It allows for the assessment of common pulmonary complications, including atelectasis, pleural effusion, and pneumothorax, which are frequently encountered after thoracic surgery [11]. In addition to pulmonary findings, chest X-rays can reveal cardiac enlargement or signs of pulmonary congestion suggestive of heart failure, thereby contributing to differentiation between cardiac and pulmonary causes of dyspnea [25].

Electrocardiography is equally essential in the diagnostic workup of postoperative dyspnea. An electrocardiogram facilitates the detection of arrhythmias, ischemic changes, or signs of right heart strain, which may be associated with pulmonary embolism or other cardiac conditions [26]. Its use is particularly important in patients with a history of cardiovascular disease or in those presenting with concurrent chest pain, as it aids in identifying myocardial ischemia or infarction [27]. When cardiac ischemia is suspected, the measurement of cardiac biomarkers, such as troponins, provides additional diagnostic clarification. Elevated troponin levels may indicate myocardial injury and support the diagnosis of perioperative myocardial infarction, a recognized complication in patients with cardiovascular comorbidities undergoing surgery [11].

In selected cases, D-dimer testing may be utilized to assist in ruling out pulmonary embolism, particularly when interpreted in conjunction with clinical probability scores such as the Wells score (28). However, the diagnostic utility of D-dimer in postoperative patients is limited by the high prevalence of false-positive results in this population [29].

Computed tomography pulmonary angiography remains the gold standard for diagnosing pulmonary embolism and should be performed when clinical suspicion persists based on initial assessment and laboratory findings [30]. This imaging modality provides detailed visualization of the pulmonary vasculature and enables definitive confirmation or exclusion of embolic events [31].

Bedside ultrasound, encompassing both lung ultrasound and cardiac echocardiography, has emerged as a rapid and non-invasive diagnostic tool in the evaluation of dyspnea. Lung ultrasound is particularly effective in identifying pleural effusions and pulmonary edema, while echocardiography allows assessment of cardiac function, including right ventricular performance and pulmonary artery pressures [8]. The evaluation of right ventricular function is especially critical in patients with suspected pulmonary hypertension or right heart strain [32].

Early Clinical Management

Immediate supportive measures constitute the cornerstone of early management in patients presenting with postoperative dyspnea. Supplemental oxygen is a primary intervention aimed at correcting hypoxemia and improving oxygen saturation. Non-invasive respiratory support modalities, including high-flow nasal oxygen and continuous positive airway pressure, have demonstrated effectiveness in managing dyspnea while potentially avoiding the need for invasive mechanical ventilation [6, 16].

In addition to oxygen therapy, patient positioning plays a relevant role in symptom relief. Placing the patient in a semi-upright position facilitates lung expansion and decreases the work of breathing. This positioning optimizes ventilation–perfusion matching and represents a simple yet effective intervention during the immediate postoperative period [33]. Continuous monitoring is equally essential to ensure early detection of respiratory deterioration. The use of continuous pulse oximetry and capnography

allows prompt identification of hypoxemia and respiratory depression, conditions that may otherwise go unnoticed with intermittent assessments. Continuous monitoring has proven superior to routine spot checks in recognizing desaturation events and preventing adverse outcomes [34]. In more complex or severe cases, early involvement of anesthesia or critical care teams is recommended, as specialized expertise enables advanced interventions and individualized management strategies when dyspnea does not respond adequately to initial measures [17].

Beyond supportive care, treatment should be directed toward the underlying etiology. Incentive spirometry and respiratory physiotherapy are effective interventions for preventing and managing atelectasis, as they promote lung expansion and improve pulmonary function. Nurse-guided incentive spirometry has been associated with reductions in hypoxic episodes and postoperative pulmonary complications [10, 35]. When dyspnea is attributable to cardiogenic pulmonary edema, diuretics and vasodilators are indicated to reduce fluid overload and optimize cardiac performance, thereby alleviating respiratory symptoms [17].

In cases of pulmonary embolism, prompt initiation of anticoagulation therapy is critical to prevent further thrombus formation and facilitate clot resolution [36]. If pneumonia is identified as the underlying cause, appropriate antibiotic therapy is essential to control infection and improve respiratory status [16]. Bronchospasm, particularly in patients with reactive airway disease, can be managed effectively with bronchodilators to relieve airway constriction and reduce dyspnea [37]. When residual neuromuscular blockade contributes to respiratory compromise, the administration of reversal agents is necessary to restore adequate muscle function and improve ventilation [33].

Clinical Algorithm and Escalation Criteria

Clinical stratification in patients with postoperative dyspnea should be guided primarily by hemodynamic stability and oxygenation status. Assessment of hemodynamic stability requires careful evaluation of blood pressure, heart rate, and the potential need for vasopressor support. The presence of hemodynamic instability signals a higher-risk scenario and may necessitate more aggressive interventions, including early endotracheal intubation or transfer to the intensive care unit. In parallel, oxygenation status must be objectively quantified, commonly through the PaO2/FiO2 ratio. A ratio below 150 indicates severe hypoxemia and warrants prompt intervention [38]. Individualized positive end-expiratory pressure settings have been shown to improve oxygenation and reduce postoperative pulmonary complications, highlighting the importance of tailored ventilatory strategies [39].

A structured approach also requires differentiation between immediate and delayed complications. Immediate airway-related causes, such as laryngospasm, airway obstruction, and aspiration, represent critical emergencies and are recognized contributors to perioperative cardiac arrest [40]. Rapid identification and management of these conditions are essential to prevent catastrophic outcomes. In contrast, delayed cardiopulmonary complications, including atelectasis, pneumonia, and pulmonary edema, may develop hours to days after surgery. Continuous monitoring for signs of respiratory distress and hypoxemia is therefore necessary to detect these evolving conditions in a timely manner [15].

When respiratory support is required, clear criteria should guide the use of noninvasive ventilation. Noninvasive ventilation is recommended in patients at high risk of reintubation, particularly those with chronic obstructive pulmonary disease or heart failure [38]. Nevertheless, patient tolerance remains a relevant limitation, as discomfort has been reported as a cause of treatment discontinuation

in clinical trials [7]. Furthermore, certain clinical parameters can predict failure of noninvasive ventilation. A high respiratory rate, low PaO2/FiO2 ratio, and elevated rapid shallow breathing index are associated with increased likelihood of noninvasive ventilation failure, thereby necessitating close surveillance and readiness to escalate care [38].

Endotracheal intubation becomes necessary in cases of severe hypoxemia, inability to protect the airway, or failure of noninvasive ventilation. Prompt recognition of these indications is critical to avoid further deterioration. At the same time, technical vigilance is essential, as unrecognized esophageal intubation constitutes a serious and potentially fatal error [40]. Certain patient characteristics, including a history of difficult intubation, obesity, or altered mental status, may warrant earlier consideration of definitive airway management [38].

Criteria for intensive care unit transfer must be clearly established. Persistent hemodynamic instability, severe hypoxemia, and the requirement for continuous monitoring or advanced respiratory support are key indications for escalation to intensive care [38]. Regarding extubation strategy, immediate extubation, when clinically feasible, has been associated with improved outcomes and a reduced need for prolonged intensive care stays [41].

Prevention and Prognostic Implications

Preoperative optimization of comorbidities represents a fundamental strategy in reducing the risk of postoperative dyspnea and associated pulmonary complications. Particular attention should be given to patients with chronic obstructive pulmonary disease, in whom interventions such as smoking cessation, pulmonary rehabilitation, and adequate control of concomitant conditions have been shown to decrease postoperative complications [42]. In addition to medical optimization, structured preoperative physiotherapy programs, including breathing exercises, have demonstrated a significant protective effect. In patients undergoing major abdominal surgery, preoperative respiratory physiotherapy reduced the incidence of postoperative pulmonary complications by 47%, highlighting the impact of proactive perioperative preparation [43].

Intraoperative strategies also play a decisive role in prevention. Lung-protective ventilation approaches, characterized by the use of low tidal volumes and individualized positive end-expiratory pressure, are essential in minimizing ventilator-induced lung injury and decreasing the incidence of postoperative pulmonary complications [44]. More specifically, ventilation strategies guided by driving pressure have been associated with improved pulmonary outcomes and reduced postoperative pulmonary complications in patients undergoing heart transplantation, underscoring the importance of tailored ventilatory management [45].

Fluid management constitutes another critical preventive measure. Restrictive fluid strategies during surgery have been associated with a lower risk of postoperative pulmonary complications when compared to liberal fluid administration. A meta-analysis demonstrated that higher intraoperative fluid volumes are linked to increased rates of pulmonary complications, reinforcing the need for careful hemodynamic and volume management [46].

Postoperatively, early mobilization and respiratory physiotherapy are key components of prevention and recovery. Interventions such as lung expansion techniques and incentive spirometry have been shown to reduce postoperative pulmonary complications and facilitate functional recovery. These measures are integral to Enhanced Recovery After Surgery protocols, which incorporate early mobilization and have demonstrated reductions in overall morbidity and hospital length of stay [47, 48].

Thromboprophylaxis is equally essential in preventing venous thromboembolism, a frequent

postoperative complication that can significantly worsen respiratory status if pulmonary embolism develops [49]. Effective implementation of prophylactic measures therefore contributes not only to vascular protection but also to respiratory stability. The impact of early recognition on clinical outcomes cannot be overstated. Prompt identification and management of postoperative dyspnea and its underlying causes are associated with reduced mortality and improved long-term outcomes. The use of automated continuous monitoring systems and non-invasive ventilatory support facilitates early detection of deterioration and timely intervention, thereby improving postoperative safety and prognosis [47].

Conclusions

Postoperative dyspnea arises from a complex interplay of perioperative pathophysiological mechanisms, most prominently anesthesia-induced reductions in functional residual capacity, atelectasis, ventilation–perfusion mismatch, hemodynamic instability, and inflammatory responses, with pulmonary causes - particularly atelectasis - being the most frequent etiologies. Its multifactorial nature requires systematic risk stratification, especially in elderly patients and those with preexisting cardiopulmonary disease, obesity, smoking history, or exposure to high-risk or prolonged surgical procedures.

Early recognition through structured diagnostic evaluation and prompt, etiology-directed management - including supportive oxygen strategies, targeted imaging, ventilatory support, and escalation based on hemodynamic and oxygenation status - is essential to reduce morbidity, mortality, intensive care utilization, and hospital length of stay. Preventive strategies such as preoperative optimization, lung-protective ventilation, restrictive fluid management, early mobilization, and thromboprophylaxis play a critical role in improving postoperative respiratory outcomes and long-term prognosis.

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