Intensive Care Med
https://doi.org/10.1007/s00134-026-08397-3

NARRATIVE REVIEW

Mechanical ventilation for ICU patient with obesity: current best practices and future directions

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Abstract: Obesity is increasingly prevalent among critically ill patients and profoundly alters respiratory mechanics, gas exchange, and cardiopulmonary interactions, complicating ventilatory management. Excess adipose tissue increases pleural pressure, reduces functional residual capacity, and promotes airway closure and atelectasis, increasing susceptibility to hypoxemia, hypercapnia, and ventilator-induced lung injury. These physiological alterations necessitate tailored ventilatory strategies distinct from those used in patients without obesity. Lung-protective ventilation using tidal volume indexed to predicted body weight remains the cornerstone of management, as lung size does not increase proportionally with body mass. Patients with obesity often require higher positive end-expiratory pressure to counteract elevated pleural pressure and prevent airway closure although optimal titration strategies remain uncertain. Interpretation of airway pressures requires caution, as increased chest wall elastance may result in elevated plateau and driving pressures without excessive lung stress. Adjunctive monitoring tools, including esophageal pressure measurement and electrical impedance tomography, may help individualize ventilatory management by improving assessment of transpulmonary pressure and regional ventilation. Airway management is particularly challenging in patients with obesity due to rapid oxygen desaturation and increased risk of difficult intubation; positive-pressure preoxygenation and video laryngoscopy improve procedural safety. Adjunctive therapies, such as prone positioning, non-invasive ventilation, and extracorporeal membrane oxygenation, are feasible and may provide benefit when clinically indicated. Liberation from mechanical ventilation requires careful assessment, and prophylactic non-invasive ventilation may reduce extubation failure in selected patients. Despite increasing recognition of obesity-specific physiology, most current recommendations are extrapolated from general ICU populations. Dedicated clinical trials are needed to define optimal ventilatory strategies and improve outcomes in this growing population of critically ill patients with obesity.

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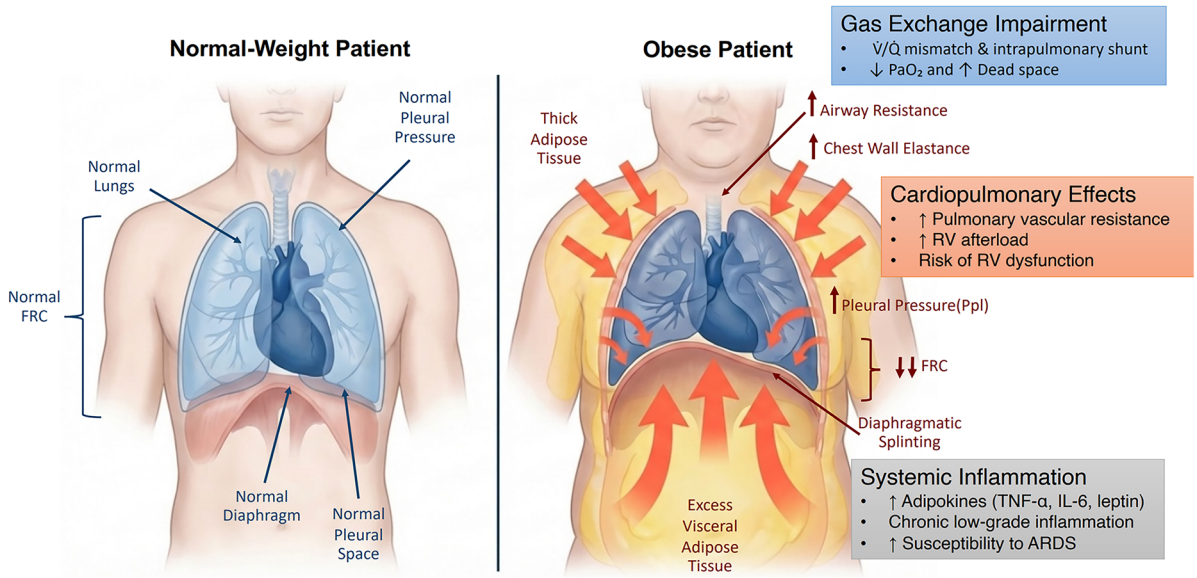
Graphical abstract:

MECHANICAL VENTILATION FOR ICU PATIENT WITH OBESITY

Current Best Practices and Future Directions

Objective

This review summarizes current best practices and highlights major gaps in evidence, outlining priorities for future trials in this rapidly growing ICU population.



Key messages

- Obesity profoundly reshapes respiratory physiology, with higher pleural pressure, reduced lung volumes, and a strong tendency toward atelectasis and hypoxemia during mechanical ventilation.
- Standard ventilatory metrics may be misleading in these patients, as elevated airway pressures often reflect chest wall mechanics rather than true lung stress.
- Key clinical questions remain unresolved, including optimal PEEP titration, the role of advanced monitoring (esophageal pressure, EIT), and the best strategies for intubation and extubation.
- Adjunctive approaches such as prone positioning, NIV, and ECMO appear feasible, but evidence specific to obesity is still limited.

Kitisin, N., Caroli, A., Carvalho Ferreira, J. *et al.* Mechanical ventilation for ICU patient with obesity: current best practices and future directions. *Intensive Care Med (2026).* https://doi.org/**10.1007/s00134-026-08397-3**

ICM Intensive Care Medicine

Keywords: Obesity, Mechanical ventilation, Positive end-expiratory pressure, Respiratory mechanics, Tidal volume, Acute respiratory distress syndrome

Introduction

Obesity, traditionally defined as a body mass index (BMI) ≥ 30 kg/m^2, is a global epidemic and is increasingly recognized as a condition reflecting excess adiposity and its associated health effects rather than BMI alone [1]. Estimates from the Global Burden of Disease study indicate that the prevalence of adult obesity has more than doubled over the past three decades, with nearly 60% of adults projected to have overweight or obesity worldwide by 2050 [2]. In the intensive care unit (ICU), patients with obesity constitute a substantial proportion of admissions [3, 4].

Among critically ill patients, obesity is associated with prolonged mechanical ventilation and longer ICU stay [4]. It is also linked to increased lung derecruitment and a higher incidence of acute respiratory distress syndrome (ARDS), further complicating ventilatory management [4, 5]. Despite this increased morbidity, several studies report similar or lower mortality in patients with moderate obesity compared with those of normal weight, an observation termed the "obesity paradox", although this apparent advantage is less consistent in severe obesity [6].

Airway management and ventilatory support are particularly challenging in patients with obesity, who have a higher risk of difficult intubation and rapid desaturation [7]. Excess adiposity alters respiratory mechanics by increasing chest wall elastance and reducing functional residual capacity (FRC), promoting airway closure, atelectasis, and impaired gas exchange [8, 9]. These physiological alterations increase susceptibility to respiratory failure and complicate the application of conventional ventilatory strategies, highlighting the need for approaches tailored to this growing ICU population [10].

Despite the increasing prevalence of obesity in the ICU, patients with severe obesity have been underrepresented in major ventilation trials, and current ventilatory practices are largely extrapolated from general ICU populations without obesity-specific validation. This review synthesizes current evidence on mechanical ventilation in critically ill patients with obesity (Table 1) and highlights priorities for clinical practice and future research.

Pathophysiology of respiratory failure in patients with obesity

Obesity profoundly alters respiratory mechanics, gas exchange, and cardiopulmonary interactions, increasing susceptibility to respiratory failure and complicating ventilatory support in critically ill patients [11, 12]. These alterations arise from reductions in lung volumes, impaired chest wall compliance, increased airway resistance, and elevated pleural pressures, which together promote airway closure, atelectasis, and impaired ventilatory efficiency [11–13]. The physiological consequences of obesity are heterogeneous and tend to be more pronounced in patients with severe obesity, such that some ventilatory considerations described may be particularly relevant in higher obesity classes; however, robust evidence stratified by obesity severity is limited. These pathophysiological alterations and their interrelationships are illustrated in Fig. 1.

Key Points

In patients with obesity, reduced lung volumes and elevated pleural pressure increase the risk of atelectasis, hypoxemia, and ventilator-induced lung injury, requiring tailored ventilatory strategies.

Tidal volume should be indexed to predicted body weight, and PEEP often needs to be individualized, as airway pressures may overestimate true lung stress due to increased chest wall elastance.

Most recommendations are extrapolated from general ICU populations, highlighting the need for obesity-specific trials.

Respiratory mechanics

Reduced lung volumes and diaphragmatic dysfunction

FRC and expiratory reserve volume are markedly reduced in patients with obesity, primarily due to elevated intra-abdominal pressure and compression of thoracic structures by excess adipose tissue. FRC decreases by approximately 5–15% for every 5 kg/m^2 increase in BMI [11]. When FRC falls below closing capacity, airway closure occurs during tidal breathing, promoting atelectasis, ventilation–perfusion mismatch, and impaired oxygenation [14]. Increased abdominal pressure also elevates the diaphragm at rest and limits its caudal excursion during inspiration, reducing inspiratory capacity [15]. Sedation, supine positioning, and respiratory muscle unloading during mechanical ventilation further exacerbate diaphragmatic dysfunction and promote dependent lung collapse [16].

Reduced chest wall compliance and increased pleural pressure

Excess adipose tissue over the thorax and abdomen increases respiratory system elastance and reduces chest wall compliance [16]. Elevated pleural pressure reduces transpulmonary pressure, particularly in dependent lung regions, promoting compression atelectasis even without intrinsic lung disease [17, 18].

Increased airway resistance and airway closure

Airway resistance is increased due to reduced lung volumes and increased external compressive forces [16]. Airway closure may occur within the tidal volume

Table 1 Summary of evidence for key ventilation strategies in patients with obesity

Domain	Key clinical question	Recommendation	Best available evidence	Bottom line	Obesity-specific considerations
Airway management and preoxygenation					
Preoxygenation	Does positive-pressure preoxygenation reduce peri-intubation hypoxemia more in patients with obesity?	Prefer NIV (or HFNC where NIV not feasible); head-up or ramped positioning	RCT in critically ill adults with obesity subgroup benefit [36]; network meta-analysis of RCTs [34]	Positive-pressure strategies reduce severe hypoxemia, and benefit appears larger as BMI increases	Rapid desaturation during apnea due to reduced FRC; NIV provides positive pressure to counteract airway closure and atelectasis
Intubation technique	How to reduce failed first-pass intubation and peri-intubation complications?	Video laryngoscopy as first-line approach	Meta-analysis of RCTs in patients with class 2–3 obesity [37]; DEVICE trial in critically ill adults [38]	VL improves glottic visualization and first-pass success, and likely reduces complications in patients with obesity	Higher rates of difficult intubation; VL particularly advantageous with increased neck circumference and high Mallampati scores
Ventilation settings					
Tidal volume	Should V_T be based on predicted body (PBW) weight in patients with obesity?	VT of 6–8 mL/kg PBW, calculated from height and sex using standard equations	ARDSNet trial [39]	PBW-based V_T minimizes VILI risk, and obesity does not justify larger V_T	Lung size does not scale with body weight; common error of using actual body weight leads to injurious volumes
PEEP	Do patients with obesity need higher PEEP, and how should it be titrated?	Start with adequate PEEP to prevent airway closure; individualize using Pes, EIT, or driving pressure	Physiological studies [40, 42], PROBESE trial [41]; ART trial subgroup data [44]	Obesity physiology supports higher PEEP needs, but outcome data are limited and largely extrapolated	Elevated pleural pressure requires higher PEEP to maintain positive transpulmonary pressure; standard PEEP (5–10 cmH_2O) may be insufficient
Recruitment maneuvers	Should recruitment be routine in patients with obesity?	Not routine; consider selectively for refractory hypoxemia; always pair with sufficient PEEP; monitor hemodynamics	Small physiological studies; perioperative RCTs [9, 43]; PROBESE trial [41]	Transient oxygenation improvement is possible but routine aggressive recruitment is not supported	Greater atelectasis burden may yield larger recruitment potential; hemodynamic compromise more likely with high intrathoracic pressures
Oxygenation targets	How to approach hypoxemia in patients with obesity?	Optimize lung recruitment and PEEP before escalating FiO_2; avoid hyperoxia	Physiological rationale	Address atelectasis as root cause before increasing FiO_2	Atelectasis is a major driver of hypoxemia; increasing FiO_2 without addressing atelectasis worsens absorption atelectasis
Respiratory mechanics monitoring					
Driving pressure	Can standard driving pressure thresholds be applied in patients with obesity?	Target ≤ 15 cmH_2O; interpret with caution, may overestimate lung stress	Amato et al. [52]; De Jong et al. [53]	Driving pressure associated with outcomes, but higher thresholds may be safe if chest wall elastance is dominant	Elevated chest wall elastance inflates airway-derived driving pressure; partitioning lung vs. chest wall mechanics improves interpretation
Esophageal pressure (Pes) and EIT	How to avoid misinterpreting airway pressures in patients with obesity?	Pes estimates transpulmonary pressure; EIT visualizes regional ventilation; both may guide individualized PEEP titration	EPVent-2 trial [56]; De Santis Santiago et al. [10]; Nestler et al. [19]	Improves physiological interpretation and personalization but outcome benefit is uncertain	Most informative in patients with obesity where chest wall contribution is high; Pes reflects regional not global pleural pressure; EIT signal affected by body habitus; best used in combination

Table 1 (continued)

Domain	Key clinical question	Recommendation	Best available evidence	Bottom line	Obesity-specific considerations
Adjunctive therapies					
Prone positioning	Is prone positioning feasible and beneficial in patients with severe obesity with ARDS?	Use with standard ARDS indications; obesity alone should not preclude proning; ensure adequate staffing and precautions	PROSEVA trial subgroup analyses [78]; observational and feasibility data [62]	Likely beneficial when indicated and feasible with appropriate planning	Technical challenges with positioning and pressure injury prevention; may increase intra-abdominal pressure; redistribution of lung stress may yield greater benefit given dorsal atelectasis burden
Neuromuscular blockade	How to dose NMB agents in patients with obesity?	Consider in early severe ARDS for synchrony and lung protection; dosing weight scalar uncertain	ATS clinical practice guideline [61]	May improve synchrony but mortality benefit is inconsistent and dosing remains challenging	Optimal weight scalar (actual, ideal, adjusted body weight) unclear; risk of under- or overdosing; monitor with train-of-four
ECMO	Can ECMO be used in patients with severe obesity?	Rescue therapy for refractory respiratory failure; obesity no longer an absolute contraindication	EOLIA trial subgroup data [63]	Feasible with experienced centers, and historical BMI restrictions are increasingly being relaxed	Vascular access and cannulation more challenging; circuit flows should account for higher metabolic demand
Liberation from mechanical ventilation					
SBT modality and liberation	What is the optimal SBT approach in patients with obesity?	T-piece SBT most closely reproduces post-extubation physiology; PSV-SBT may underestimate work of breathing	Physiological rationale: general ICU evidence extrapolated to patients with obesity [68, 69]	T-piece SBT or PSV-SBT do not alter the risk of reintubation, but PSV-SBT facilitates extubation decision-making	Increased work of breathing post-extubation due to reduced compliance and upper airway obstruction; consider adding low PEEP during SBT
Post-extubation support	Does prophylactic NIV reduce reintubation in ICU patients with obesity?	Prophylactic NIV recommended; apply ≥ 13 h within first 24 h post-extubation	EXTUB-OBESE RCT [51]; additional RCTs and meta-analyses in populations with overweight or obesity [75]	Signal toward reduced reintubation and mortality and duration of NIV application matters	NIV for ≥ 13 h post-extubation reduced reintubation and death vs HFNC in one RCT; a second RCT with shorter NIV duration showed no benefit, duration matters

Abbreviations: *ARDS* acute respiratory distress syndrome, *BMI* body mass index, *ECMO* extracorporeal membrane oxygenation, *EIT* electrical impedance tomography, FiO_2 fraction of inspired oxygen, *FRC* functional residual capacity, *HFNC* high-flow nasal cannula, *NIV* non-invasive ventilation, *NMB* neuromuscular blockade, *PBW* predicted body weight, *PEEP* positive end-expiratory pressure, *Pes* esophageal pressure, *PSV* pressure-support ventilation, *RCT* randomized controlled trial, *SBT* spontaneous breathing trial, *VILI* ventilator-induced lung injury, *VL* video laryngoscopy, *VT* tidal volume

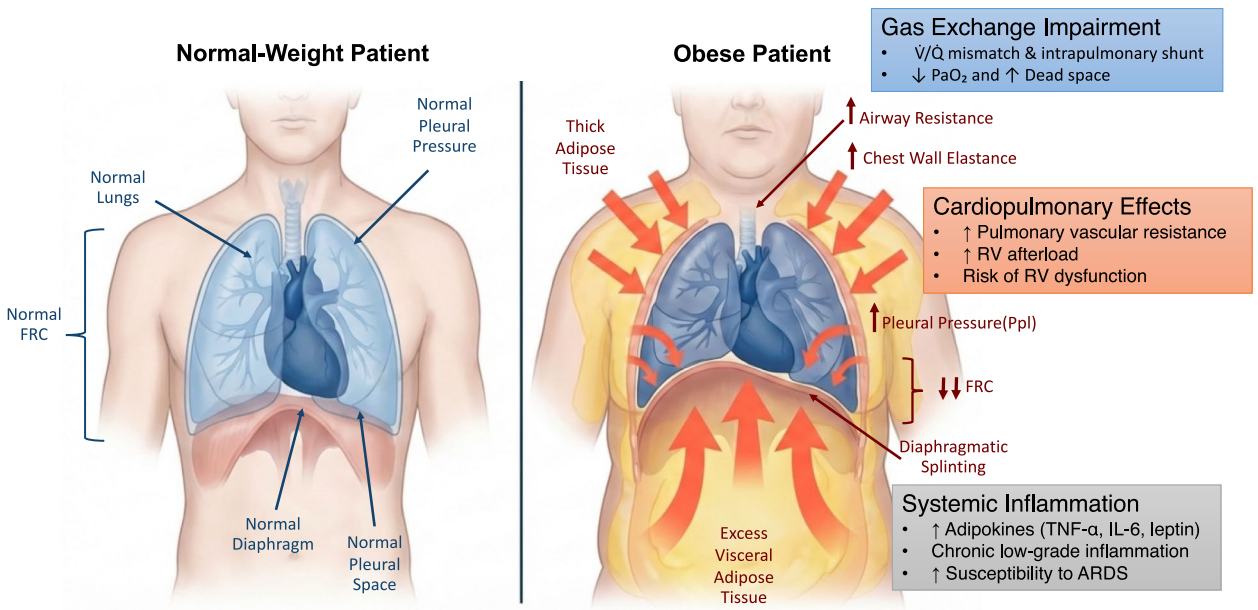


Fig. 1 Pathophysiological mechanisms of respiratory dysfunction in patients with obesity. Comparison of respiratory mechanics between normal weight and patients with obesity. Excess thoracic and visceral adipose tissue in obesity increases pleural pressure and chest wall elastance, reduces functional residual capacity, and promotes diaphragmatic splinting. These alterations lead to airway closure, ventilation–perfusion mismatch, increased driving pressure requirements, and susceptibility to atelectasis. Elevated pulmonary vascular resistance and right ventricular afterload contribute to cardiopulmonary compromise. Chronic low-grade inflammation further increases vulnerability to acute respiratory distress syndrome. FRC is functional residual capacity, Ppl is pleural pressure, and RV is right ventricle

range, requiring higher opening pressures [19]. Chronic airway inflammation further contributes to airflow limitation [20].

Impaired gas exchange and ventilatory efficiency

Mechanical alterations in patients with obesity directly impair gas exchange by promoting ventilation–perfusion (V/Q) mismatch, increasing dead space ventilation, and reducing respiratory reserve [21].

Ventilation–perfusion mismatch and shunt

Reduced ventilation in dependent lung regions, combined with relatively preserved perfusion, results in significant V/Q mismatch and increased intrapulmonary shunt [21]. Compression atelectasis, driven by reduced FRC and elevated pleural pressure, is a major contributor. These abnormalities impair oxygenation and increase vulnerability to hypoxemia during sedation, supine positioning, and critical illness.

Increased dead space and ventilatory demand

Patients with obesity often adopt rapid, shallow breathing patterns, increasing dead space ventilation and reducing effective alveolar ventilation [18]. Increased metabolic demand and CO_2 production may lead to hypercapnia when ventilatory capacity is exceeded [22].

Cardiopulmonary interactions and right ventricular function

Respiratory mechanical alterations in patients with obesity closely interact with cardiovascular physiology, particularly affecting right ventricular (RV) function [8, 12, 23]. Elevated pleural pressures and airway pressures increase pulmonary vascular resistance by compressing pulmonary vessels and promoting hypoxic pulmonary vasoconstriction [24]. Chronic hypoxemia, commonly associated with obesity hypoventilation syndrome and obstructive sleep apnea, contributes to pulmonary vascular remodeling and pulmonary hypertension.

Increased pulmonary vascular resistance raises RV afterload and may impair RV function [23, 24]. Mechanical ventilation, particularly when associated with high intrathoracic pressures, may further reduce venous return and alter cardiac output [25]. Conversely, improved lung aeration and oxygenation may reduce hypoxic vasoconstriction and improve RV performance [25]. The net hemodynamic effect varies depending on the balance between lung recruitment, intrathoracic pressure, and preload conditions. These cardiopulmonary interactions contribute to increased vulnerability to circulatory instability and RV dysfunction during respiratory failure.

Systemic and inflammatory factors

Obesity is characterized by chronic low-grade systemic inflammation, mediated by adipokines and pro-inflammatory cytokines, which may increase susceptibility to acute lung injury and ARDS [26, 27]. In addition, altered pharmacokinetics may prolong drug effects and exacerbate respiratory depression during sedation [28].

Airway management and pre-intubation considerations

The respiratory mechanics and gas exchange abnormalities associated with obesity significantly increase the risk of peri-intubation complications, particularly severe hypoxemia [29, 30]. Reduced FRC, increased oxygen consumption, and a propensity for airway closure shorten the safe apneic period and limit tolerance to repeated or prolonged intubation attempts. These physiological vulnerabilities, combined with anatomical challenges, necessitate careful airway assessment and optimization before intubation.

Predicting difficult airway

Airway difficulty in patients with obesity reflects both anatomical and physiological factors [29, 30]. Established anatomical predictors, including increased neck circumference (> 40 cm), high Mallampati score (III–IV), limited mouth opening, reduced cervical spine mobility, short thyromental distance, and a history of obstructive sleep apnea, are more prevalent in this population and increase the likelihood of difficult laryngoscopy and intubation [29, 30]. Point-of-care ultrasound (Skin-to-epiglottis distance) may improve airway risk stratification although validation in ICU populations remains limited [31].

Physiological factors further increase risk. Reduced FRC and increased metabolic demand accelerate oxygen desaturation during apnea, particularly in the presence of atelectasis or underlying respiratory failure [32, 33]. Obesity itself is an independent predictor of difficult airway management in critically ill patients [29].

Preoxygenation

Effective preoxygenation is essential to prolong safe apnea and reduce the risk of severe hypoxemia [8, 34]. Positioning strategies that reduce diaphragmatic compression, such as head-up or ramped positioning, may improve oxygenation by increasing lung volumes [35] although evidence in critically ill populations is heterogeneous [29, 35].

Positive-pressure preoxygenation strategies appear particularly beneficial [34]. Non-invasive ventilation (NIV) and high-flow nasal cannula (HFNC) improve oxygenation compared with conventional facemask oxygenation, likely by preventing airway closure and atelectasis [34, 36]. In the PREOXI trial, NIV significantly reduced severe hypoxemia compared with facemask oxygenation, with the greatest benefit observed in patients with obesity [36] (Fig. 2). HFNC may also be continued during laryngoscopy to provide apneic oxygenation and help delay desaturation, which may be particularly relevant

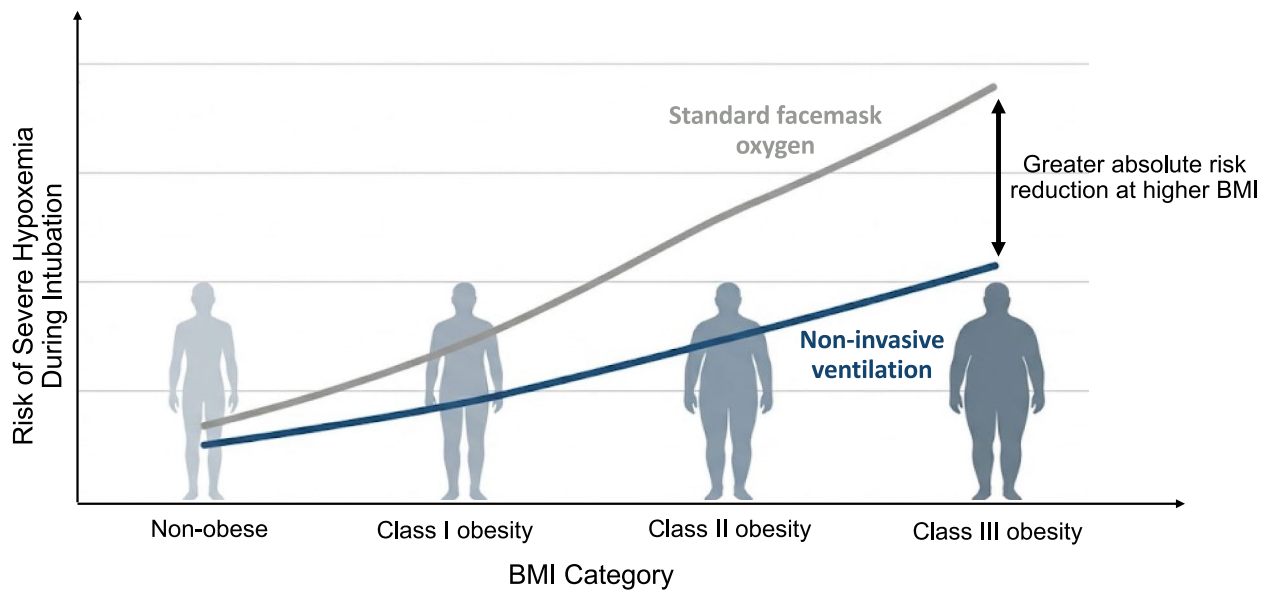


Fig. 2 Absolute risk reduction with positive-pressure preoxygenation across body mass index categories. Conceptual representation of increasing risk of severe hypoxemia during intubation with rising body mass index. As baseline risk increases, the absolute risk reduction achieved with positive-pressure preoxygenation becomes greater compared with standard facemask oxygen. Adapted from Gibbs KW et al. [36]

in patients with obesity given their reduced functional residual capacity [4].

Intubation technique

Video laryngoscopy improves glottic visualization and increases first-pass success compared with direct laryngoscopy, particularly in patients with predicted airway difficulty [37, 38]. Its use is associated with fewer failed intubations and reduced peri-intubation hypoxemia [37, 38]. Accordingly, video laryngoscopy is recommended as the preferred first-line approach for intubation in critically ill patients with obesity.

Initiation of mechanical ventilation and ventilation strategies

The altered respiratory mechanics and reduced lung volumes associated with obesity require careful adjustment of ventilatory settings to minimize ventilator-induced lung injury (VILI) and optimize gas exchange. Ventilatory management should account for reduced functional lung size, increased pleural pressure, and altered cardiopulmonary interactions [4]. A stepwise approach to ventilatory management in this population is outlined in Fig. 3.

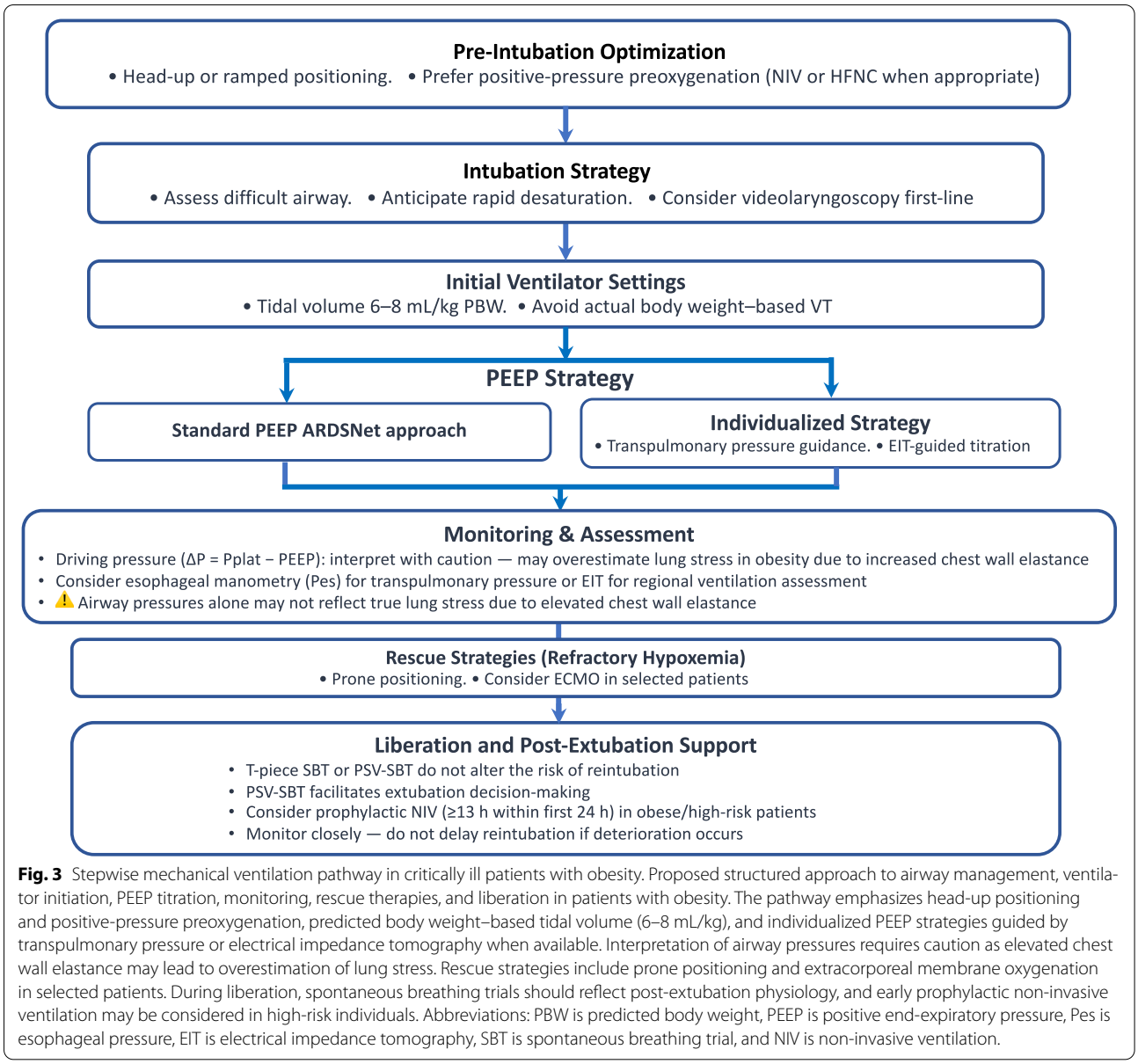


Fig. 3 Stepwise mechanical ventilation pathway in critically ill patients with obesity. Proposed structured approach to airway management, ventilator initiation, PEEP titration, monitoring, rescue therapies, and liberation in patients with obesity. The pathway emphasizes head-up positioning and positive-pressure preoxygenation, predicted body weight–based tidal volume (6–8 mL/kg), and individualized PEEP strategies guided by transpulmonary pressure or electrical impedance tomography when available. Interpretation of airway pressures requires caution as elevated chest wall elastance may lead to overestimation of lung stress. Rescue strategies include prone positioning and extracorporeal membrane oxygenation in selected patients. During liberation, spontaneous breathing trials should reflect post-extubation physiology, and early prophylactic non-invasive ventilation may be considered in high-risk individuals. Abbreviations: PBW is predicted body weight, PEEP is positive end-expiratory pressure, Pes is esophageal pressure, EIT is electrical impedance tomography, SBT is spontaneous breathing trial, and NIV is non-invasive ventilation.

Tidal volume selection

In patients with obesity, lung size does not increase proportionally with body weight. Excess adiposity restricts chest wall expansion and reduces total lung capacity, vital capacity, and end-expiratory lung volume [11]. Consequently, calculating tidal volume (V_T) based on actual body weight risks delivering excessive lung distension and increasing the risk of VILI [4]. Predicted body weight (PBW), derived from height-based equations, provides a standardized estimate of lung size and should be used to guide V_T selection [39]. Lung-protective ventilation using V_T of 6–8 mL/kg PBW remains the cornerstone of management, particularly in patients with ARDS, but is also appropriate in patients with obesity and without ARDS [39]. Reduced aerated lung volume and increased pleural pressure further support the use of low V_T to limit lung stress [4]. Current evidence does not support increasing V_T to compensate for reduced chest wall compliance as lung size determines the safe distending volume [5].

Positive end-expiratory pressure and lung recruitment

Positive end-expiratory pressure (PEEP) plays a central role in preventing airway closure and maintaining lung recruitment in patients with obesity, who are particularly prone to atelectasis due to reduced FRC and elevated pleural pressure [4, 40]. Higher PEEP improves respiratory mechanics, increases end-expiratory lung volume, and enhances oxygenation by preventing alveolar collapse [19, 40, 41]. However, the optimal PEEP level remains uncertain. Physiological studies suggest that commonly used PEEP levels may be insufficient to maintain positive transpulmonary pressure in moderate-to-severe obesity [17, 42]. Individualized PEEP strategies guided by transpulmonary pressure or imaging techniques, like CT scan, ultrasound, or electrical impedance tomography (EIT), may improve lung recruitment. Importantly, airway pressures in patients with obesity reflect both lung and chest wall mechanics; therefore, higher plateau pressures may be tolerated without excessive lung distension if transpulmonary pressures remain within safe limits [17, 42].

Recruitment maneuvers may transiently improve oxygenation and lung aeration by reopening collapsed lung regions, particularly when followed by adequate PEEP [43]. However, their use should be individualized, as recruitment maneuvers increase intrathoracic pressure and may impair venous return and cause hemodynamic instability. Current evidence does not support routine use of aggressive recruitment strategies [41, 44], but data are lacking specifically in patients with obesity.

Oxygenation targets

Oxygen therapy should aim to achieve adequate oxygenation while minimizing the risks of atelectasis and hyperoxia. Increasing PEEP and optimizing lung recruitment are preferred over escalating inspired oxygen concentration alone, as high oxygen fractions may worsen absorption atelectasis and contribute to oxygen toxicity [4, 45, 46]. In clinical practice, a pragmatic approach is to titrate oxygen therapy to maintain oxygen saturation generally above 92% and below 98% [47].

Ventilatory management in specific clinical scenarios

ARDS in patients with obesity

Patients with obesity are at increased risk of developing ARDS although mortality may be similar or lower than in patients without obesity [5, 48]. Accurate diagnosis can be challenging, as obesity-related atelectasis may mimic radiographic features of ARDS [49]. Lung-protective ventilation using low V_T indexed to PBW remains the foundation of management [39]. Higher PEEP and individualized PEEP strategies may be particularly beneficial to counteract elevated pleural pressure and optimize lung recruitment [40].

Hypercapnic respiratory failure in patients with obesity

Obesity is frequently associated with obesity hypoventilation syndrome and obstructive sleep apnea, which predispose patients to acute hypercapnic respiratory failure [50]. Management focuses on improving alveolar ventilation and reducing respiratory muscle load. NIV is an effective first-line strategy in patients with obesity with hypercapnic respiratory failure as it improves alveolar ventilation, reduces work of breathing, and corrects hypercapnia while avoiding the risks associated with invasive ventilation [8, 51]. NIV is also beneficial in postoperative patients with obesity, reducing reintubation rates and respiratory complications [51].

Early recognition of NIV failure is essential, and invasive ventilation should not be delayed in patients with worsening respiratory failure, severe hypoxemia, or organ dysfunction. When invasive ventilation is required, careful attention should be paid to positioning, ventilator settings, and airway management to minimize complications. Oxygen therapy should be used cautiously and titrated to avoid worsening hypercapnia [4].

Monitoring respiratory mechanics

Accurate assessment of respiratory mechanics is essential to guide ventilatory management in patients with obesity, especially because airway pressures may not accurately reflect lung stress due to altered chest wall mechanics.

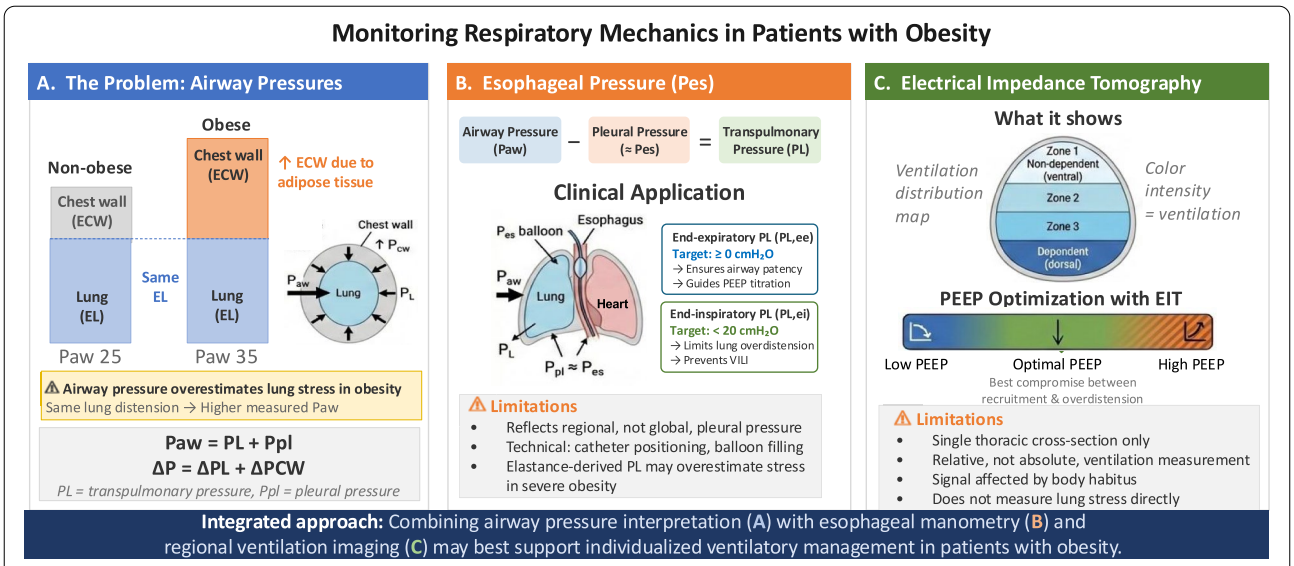


Fig. 4 Monitoring respiratory mechanics in patients with obesity. **A** In patients with obesity, increased chest wall elastance (ECW) elevates measured airway pressure (Paw) despite similar intrinsic lung elastance (EL), such that airway pressures may overestimate true lung stress. Airway pressure reflects the sum of transpulmonary pressure (PL) and pleural pressure (Ppl) (Paw = PL + Ppl), and changes in driving pressure (ΔP) reflect contributions from both lung and chest wall components (ΔP = ΔPL + ΔPCW). **B** Esophageal pressure (Pes) provides an estimate of pleural pressure and allows calculation of transpulmonary pressure (PL = Paw – Pes). Targeting a positive end-expiratory transpulmonary pressure (PL,ee) may reduce airway closure and atelectasis, whereas limiting end-inspiratory transpulmonary pressure (PL,ei) may help prevent lung overdistension. **C** Electrical impedance tomography (EIT) provides real-time regional ventilation assessment and may assist in positive end-expiratory pressure (PEEP) optimization by balancing recruitment and overdistension. Abbreviations: Paw airway pressure, Ppl pleural pressure, Pes esophageal pressure, PL transpulmonary pressure, PL,ee end-expiratory transpulmonary pressure, PL,ei end-inspiratory transpulmonary pressure, ΔP driving pressure, ΔPL lung driving pressure, ΔPCW chest wall driving pressure, ECW chest wall elastance, EL lung elastance, PEEP positive end-expiratory pressure, EIT electrical impedance tomography

Conventional indices, such as plateau pressure and driving pressure, remain useful but require cautious interpretation, and adjunctive tools, such as esophageal pressure monitoring and EIT, may provide additional physiological insights. The principles underlying respiratory mechanics monitoring in patients with obesity are summarized in Fig. 4.

Plateau pressure and driving pressure

Plateau pressure and driving pressure (defined as plateau pressure minus PEEP) are widely used to assess lung stress and guide lung-protective ventilation [52]. However, these parameters reflect the combined mechanical properties of the lung and chest wall. In patients with obesity, increased chest wall elastance and elevated intra-abdominal pressure contribute substantially to airway pressure, resulting in higher plateau and driving pressures even in the absence of lung overdistension. Elevated pleural pressure reduces transpulmonary pressure, the true distending pressure of the lung, such that airway pressure may overestimate lung stress [5]. Consistent with this physiological mechanism, the strong association between driving pressure and mortality observed in general ARDS populations appears attenuated in patients with obesity [53]. Therefore, conventional safety thresholds for plateau and driving pressure should be interpreted cautiously, and higher airway pressures may be tolerated if lung stress remains within acceptable limits.

Esophageal pressure monitoring

Esophageal pressure monitoring provides an estimate of pleural pressure and allows calculation of transpulmonary pressure, defined as the difference between airway and pleural pressure [54]. Transpulmonary pressure more directly reflects lung stress and enables separation of lung and chest wall mechanics, facilitating individualized ventilator adjustments [54].

In patients with obesity, elevated pleural pressure often results in low or negative end-expiratory transpulmonary pressure, promoting airway closure and atelectasis [55]. Adjusting PEEP to maintain a positive end-expiratory transpulmonary pressure may improve lung recruitment, oxygenation, and respiratory mechanics. Similarly, limiting end-inspiratory transpulmonary pressure may help prevent excessive lung distension [55].

Despite these physiological advantages, clinical outcome benefits remain uncertain. The EPVent-2 trial

did not demonstrate superiority of esophageal pressure-guided PEEP compared with empirical high-PEEP strategies in unselected ARDS populations [56] although secondary analyses suggest that maintaining end-expiratory transpulmonary pressure near 0 cmH_2O may be associated with improved outcomes [57]. Importantly, baseline esophageal pressure is higher in patients with obesity, and elastance-derived transpulmonary pressure estimates may overestimate lung stress in severe obesity [58].

Technical limitations must also be considered. Accurate measurement requires appropriate catheter positioning and balloon inflation, and esophageal pressure reflects regional rather than global pleural pressure. Accordingly, esophageal pressure monitoring should be viewed as a physiological adjunct that enhances interpretation of airway pressures rather than a definitive measure of lung stress. Also, in patients with obesity, the interpretation of transpulmonary pressure remains debated, as elastance-derived estimates may differ from absolute measurements, and robust evidence favoring one approach is currently lacking.

Electrical impedance tomography

EIT is a non-invasive, bedside imaging modality that provides real-time assessment of regional ventilation distribution [59]. By detecting impedance changes during ventilation, EIT allows identification of regional lung collapse and overdistention, supporting individualized ventilatory management. In patients with obesity, EIT may be particularly useful for optimizing PEEP by balancing recruitment against overdistention [60]. EIT-guided PEEP titration has been associated with improved oxygenation and respiratory mechanics compared with conventional approaches [19].

Limitations of EIT include its assessment of a single thoracic cross section and reliance on relative rather than absolute ventilation measurements. Signal acquisition and interpretation may be affected by body habitus and electrode positioning [59]. As EIT does not directly measure lung stress, combining EIT with pressure-based monitoring such as esophageal pressure measurement may provide complementary information and further support individualized ventilatory strategies.

Adjunctive therapies

Adjunctive therapies, such as prone positioning, neuromuscular blockade, and extracorporeal membrane oxygenation (ECMO), play an important role in the management of severe respiratory failure. In patients with obesity, altered respiratory mechanics and increased susceptibility to atelectasis may influence both the physiological response and the technical implementation of these interventions.

Prone positioning

Prone positioning is an established therapy for moderate-to-severe ARDS and improves survival when applied early and for prolonged durations [61]. By redistributing lung stress, reducing dorsal lung compression, and improving ventilation–perfusion matching, prone positioning promotes more homogeneous lung inflation. These physiological effects may be particularly relevant in patients with obesity, who are prone to dependent lung collapse due to elevated pleural pressure and reduced lung volumes; prone positioning may therefore improve dorsal aeration, redistribute lung stress, and help mitigate atelectasis-related hypoxemia [5].

Despite technical challenges related to patient positioning, body habitus, and potential increases in intra-abdominal pressure, available evidence supports its feasibility and effectiveness in patients with obesity [62]. Observational studies and feasibility trials have demonstrated that prone positioning is safe in mechanically ventilated patients with obesity and may result in comparable or greater improvements in oxygenation compared with patients without obesity [62]. Accordingly, prone positioning should be considered in patients with obesity using the same clinical indications as in the general ARDS population.

Neuromuscular blockade

Neuromuscular blockade may improve patient–ventilator synchrony, reduce oxygen consumption, and facilitate lung-protective ventilation in selected patients with severe ARDS. Although randomized trials have not consistently demonstrated a mortality benefit, current guidelines conditionally recommend short-term use in early severe ARDS when lung-protective ventilation cannot otherwise be achieved [61].

In patients with obesity, dosing of neuromuscular blocking agents presents additional challenges due to uncertainty regarding the optimal weight scalar for drug calculation. Inappropriate dosing may result in underdosing, compromising ventilatory control, or overdosing, increasing the risk of prolonged neuromuscular weakness. Careful titration and monitoring are therefore essential when neuromuscular blockade is used in this population.

Extracorporeal membrane oxygenation

ECMO is an effective rescue therapy for patients with severe respiratory failure refractory to conventional management [63]. Obesity has historically been considered a relative contraindication due to technical challenges.

However, contemporary data consistently demonstrate that ECMO can be safely and effectively implemented in patients with obesity [64, 65]. Large registry analyses report comparable, and in some series even favorable, survival outcomes compared with patients without obesity [64]. Taken together, current evidence does not support withholding ECMO solely on the basis of BMI, and eligible patients with obesity should be evaluated using the same physiological criteria applied to the general ARDS population.

Liberation from mechanical ventilation

Patients with obesity are often perceived to be at increased risk of extubation failure. However, observational data suggest obesity may be associated with a lower reintubation risk [66, 67]. This observation aligns with the broader "obesity paradox," whereby patients with obesity in the ICU often experience similar or improved outcomes compared with patients without obesity.

Spontaneous breathing trials (SBTs) performed with a T-piece most closely reproduce post-extubation physiological conditions, whereas SBTs conducted with pressure-support ventilation (PSV) reduce the work of breathing and may be less physiologically demanding [68], particularly in patients with obesity [69]. Consistently, a physiological study showed that inspiratory effort during SBTs performed without positive pressure (T-piece or PSV 0 with PEEP 0) was similar to post-extubation effort, while trials with positive pressure overestimated the true work of breathing [69]. Despite this, robust evidence indicates that SBTs performed with PSV facilitate extubation decision-making without increasing the risk of reintubation, even among patients considered at high risk [70]. Notably, one of these trials included a substantial proportion of patients with obesity, suggesting that these findings are applicable to this population [70]. Whether the addition of PEEP during SBTs further improves outcomes remains uncertain. A recent clinical trial reported that SBTs performed with PSV and PEEP were associated with shorter time to extubation compared with PSV alone [71]. However, these findings should be interpreted cautiously, as no studies to date have specifically focused on populations at highest risk of extubation failure, including patients with severe obesity.

Following extubation, prophylactic NIV has been increasingly studied as a strategy to prevent respiratory failure although specific recommendations for patients with obesity remain limited. Nevertheless, several recent studies suggest a potential benefit of prophylactic NIV in this population, supported by physiological data demonstrating reduced respiratory effort in patients with obesity receiving NIV after extubation [51, 72–74]. It is important to mention that available data suggest that the clinical benefit of post-extubation NIV may depend on the duration and intensity of its application rather than NIV use alone.

In one randomized trial, prophylactic NIV applied for 13 h within the first 24 h after extubation significantly reduced the risk of reintubation and death compared with HFNC in patients with overweight or obesity [74]. In contrast, a second study in which NIV was administered for at least four hours during the first 24 h did not demonstrate a statistically significant reduction in reintubation compared with HFNC or standard oxygen therapy [51] although crossover between groups was allowed and NIV was frequently used as rescue therapy. However, a post hoc crossover analysis showed significantly lower reintubation rates among patients who actually received NIV compared with those managed with oxygen alone. Similarly, a third trial reported a nearly 10% absolute reduction in reintubation with prophylactic NIV applied for 22 h during the first 24 h although this difference did not reach statistical significance [72]. More recently, a meta-analysis of trials evaluating NIV as either a prophylactic or therapeutic intervention in patients with obesity found that NIV was associated with reduced risks of reintubation and mortality compared with HFNC or standard oxygen therapy [75].

GAPS in knowledge and future directions

Best practices for mechanical ventilation in patients with obesity remain incompletely defined, largely due to the limited number of high-quality randomized controlled trials specifically targeting this population. Most available evidence is extrapolated from large observational studies or randomized trials conducted in general ICU populations, which include patients with obesity only in subgroup analyses, studies of ventilatory strategies during surgery under general anesthesia [41], and small physiological investigations [9, 19]. Consequently, many current recommendations lack obesity-specific validation.

Important uncertainties remain regarding optimal PEEP selection. Physiological studies suggest that patients with obesity often require higher PEEP levels to improve lung mechanics and restore lung volumes [10, 19]. However, large randomized trials evaluating higher PEEP strategies have not consistently demonstrated clinical benefit [41]. These findings highlight the need for dedicated trials evaluating individualized PEEP strategies in critically ill patients with obesity. Such approaches may benefit from advanced monitoring tools, including EIT [76], and measurement of transpulmonary pressure [42], to guide patient-specific ventilatory adjustments.

Similarly, optimal strategies for liberation from mechanical ventilation in patients with obesity remain uncertain. Although general principles, such as daily SBTs and minimization of sedation, apply to this population, the optimal SBT modality, the role of added PEEP during SBTs, and the most effective prophylactic respiratory support after extubation, including NIV [72], require further investigation in obesity-specific cohorts.

In patients with severe obesity, the feasibility, safety, and efficacy of prone positioning also warrant further study. Current evidence is largely derived from subgroup analyses of large randomized trials conducted in general ICU populations [77, 78] or small observational studies suggesting potential benefit but limited by sample size and methodological constraints [62]. Dedicated studies are needed to better define the risk–benefit profile of prone positioning in this population.

Finally, emerging therapies such as glucagon-like peptide 1 receptor agonists are increasingly used and may induce rapid and substantial weight loss [79]. Future research should evaluate how these changes influence respiratory mechanics, metabolic reserve, and pharmacokinetics in critically ill patients with obesity, and whether ventilatory strategies should be adapted according to the timing and magnitude of weight loss.

Conclusion

Obesity profoundly alters respiratory mechanics, gas exchange, and cardiopulmonary interactions, increasing susceptibility to respiratory failure and complicating ventilatory management in critically ill patients. Lung-protective ventilation using V_T indexed to PBW, careful PEEP titration, and appropriate adjunctive therapies, such as prone positioning and NIV, are key components of management. Interpretation of airway pressures requires consideration of altered chest wall mechanics, and adjunctive monitoring tools may support individualized care. Despite growing recognition of these physiological differences, high-quality obesity-specific evidence remains limited. Dedicated clinical trials are needed to optimize ventilatory strategies and improve outcomes in this increasingly prevalent ICU population.

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Funding
Open Access funding enabled and organized by CAUL and its Member Institutions.

Data availability
Not applicable.

Declarations

Conflicts of interest
GH is a Section Editor for Intensive Care Medicine. He has not taken part in the review or selection process of this article.

Publisher's Note

Received: 28 February 2026 Accepted: 14 March 2026
Published online: 09 April 2026

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