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Obesity: biomechanical implications for mechanical ventilation

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Abstract

Obesity is a highly prevalent chronic disease that profoundly affects respiratory physiology. Excess adipose tissue alters lung volumes, chest-wall mechanics, and pleural pressures, leading to airway closure, atelectasis, and ventilation–perfusion mismatch. These changes have important implications for mechanical ventilation in both perioperative and critical care settings. This narrative review examines the impact of obesity on respiratory physiology during mechanical ventilation and discusses strategies for optimizing ventilatory management in patients with obesity and acute lung injury. Obesity primarily reduces functional residual capacity and expiratory reserve volume through cephalad displacement of the diaphragm and increased intra-abdominal pressure. These alterations promote airway collapse, impaired ventilation distribution, and mild hypoxemia. During general anesthesia, additional reductions in lung volume and increased pleural pressure further predispose patients with obesity to airway closure and atelectasis. Ventilatory management should include adequate preoxygenation, recruitment maneuvers when appropriate, and individualized positive end-expiratory pressure (PEEP) titration. In patients with acute hypoxemic respiratory failure or acute respiratory distress syndrome, excess adiposity may worsen respiratory mechanics by increasing superimposed pressure and the prevalence of airway closure. These mechanisms complicate interpretation of respiratory mechanics. Advanced bedside monitoring tools, including esophageal pressure manometry and electrical impedance tomography, may provide valuable insights for optimizing ventilatory settings and balancing lung recruitment with the risk of overdistension. In moderate-to-severe acute respiratory distress syndrome, prone positioning is feasible and safe. Furthermore, individualized weaning approaches, and early use of noninvasive ventilation after extubation may further improve respiratory management.

Keywords Obesity, Mechanical ventilation, ARDS, AHRF, Airway closure, Esophageal pressure, Electrical impedance tomography

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Introduction

Obesity, defined as a body mass index (BMI) ≥ 30 kg/m^2, affects more than 40% of the adult population in the United States [1]. After decades of steady increase, prevalence appears to have plateaued, with preliminary signals suggesting a modest decline following the COVID-19 pandemic, possibly related to the rapid uptake of GLP-1 receptor agonists and post-pandemic behavioural changes [2].

Obesity is a chronic disease characterized by excess and/or abnormal fat distribution. Its global prevalence has reached alarming levels, leading to its classification as worldwide pandemic. Obesity is associated with a markedly increased incidence of chronic, health-threatening conditions, including diabetes mellitus, cardiovascular diseases, and cancer, largely driven by chronic inflammation and metabolic dysfunction [3].

From a biomechanical perspective, excess adipose tissue significantly alters respiratory mechanics during both spontaneous breathing and mechanical ventilation. These alterations pose challenges in airway management and ventilatory support, and may contribute to increased disease severity and morbidity in acute conditions such as acute hypoxemic respiratory failure (AHRF) and acute respiratory distress syndrome (ARDS) [4, 5].

This narrative review aims to examine the impact of obesity on respiratory physiology during mechanical ventilation, and to discuss how ventilatory strategies should be adapted accordingly in both the operating room (OR) and the intensive care unit (ICU). This review is based on a non-systematic literature search conducted in PubMed. The search aimed to identify key studies on mechanical ventilation in obese patients, including physiological investigations, clinical studies, and randomized controlled trials. Study selection was guided by the authors' expertise in the field, with particular attention to seminal papers and high-quality evidence, as well as more recent publications to provide an updated overview.

Respiratory mechanics alteration in obesity: pathophysiology

Lung volumes and mechanics

Obesity alters respiratory physiology primarily through the biomechanical effects of excess in body adipose tissue. Importantly, fat distribution may significantly modify the relationship between BMI and lung volumes. Fat accumulation in the thoracic, abdominal, and visceral compartments, referred to as android obesity, directly impairs pulmonary function by increasing intra-abdominal pressure and promoting cephalad displacement of the diaphragm [6]. In contrast, gynoid obesity (i.e., fat predominantly distributed in the subcutaneous tissue of the hips, thighs, arms, and legs) is less likely to exert a significant mechanical effect on lung volumes [7, 8].

In obesity, upward displacement of the diaphragm by abdominal contents results in a restrictive ventilatory pattern, characterized primarily by reduction in the expiratory reserve volume (ERV) and in the functional residual capacity (FRC) [9, 10]. FRC decreases proportionally to the severity of obesity, with the most pronounced reductions occurring with mild increases in BMI (25–35 kg/m^2)[10]. The impact of obesity on the extreme of lung volumes is generally less marked. Total lung capacity (TLC) usually remains above the lower limit of normal, even in severe obesity [10, 11]. Residual volume (RV) is typically preserved [11, 12]. Consequently the RV-to-TLC ratio remains normal or may be slightly increased [10, 13]. Diaphragm displacement and FRC reduction promote airway collapse and atelectasis formation in dependent lung regions [14, 15], leading to a reduction in lung compliance [16].

In mechanically ventilated patients, transpulmonary pressure can be calculated by combining airway pressure with esophageal pressure, which serves as a surrogate for pleural pressure:

Transpulmonary pressure = airway pressure – esophageal pressure [17].

Transpulmonary pressure represents the distending pressure applied across the lung parenchyma (i.e., the pressure responsible for maintaining alveolar patency and preventing alveolar collapse).

Elevated esophageal pressure may result in negative transpulmonary pressure, particularly at end-expiration, promoting lung collapse, reduced lung compliance, and hypoxemia [18]. As a consequence, mechanically ventilated patients with obesity often required higher positive end-expiratory pressure (PEEP) to achieve a positive transpulmonary pressure, thereby preventing alveolar collapse [19].

Chest wall mechanics

Chest wall compliance is often assumed to be decreased in obesity, as reported in studies of spontaneously breathing subjects [20]. Therefore, a frequent clinical misinterpretation is that the elevated airway pressures observed in patients with obesity reflect reduced chest-wall compliance. However, physiological studies, in both spontaneously breathing [21] and anesthetized and paralyzed subjects consistently demonstrate that chest wall compliance is generally preserved in obesity [16, 22]. This can be explained by the mass loading effect of the adipose tissue, which induces a rightward shift of the chest wall pressure-volume curve without altering its slope (i.e., compliance) [23]. In this context, excess in body mass may act as an inspiratory threshold load; once this threshold is overcome, chest wall mechanics behaves normally [24]

(Fig. 1). Although patients with obesity exhibit elevated absolute esophageal pressure values, the esophageal pressure swing between end-inspiration and end-expiration is often relatively small. Therefore, the elevated difference between plateau pressure and PEEP (i.e., driving pressure, DP), should not be misinterpreted as reflecting reduced chest-wall compliance. On the contrary, in most cases, the respiratory system DP, which can be easily measured at the bedside from the ventilator, closely approximates transpulmonary DP.

Airway mechanics

Airway mechanical properties, particularly airway resistance, are highly dependent on lung volume and are therefore influenced by the reduction in FRC observed in obesity. Although total airway resistance increased in individuals with obesity [25], specific airway resistance - adjusted for the lung volume at which the measurements were made - remained within the normal limits [25–27]. These findings suggest that increased airway resistance in obesity is primarily attributable to reduced lung volumes rather than intrinsic mechanical airway obstruction. Regarding spirometry parameters, forced expiratory volume in one second (FEV1) and forced vital capacity (FVC) tend to decrease modestly with increasing BMI, while generally remaining within normal ranges [28, 29]. Accordingly, the FEV1-to-FVC ratio is usually preserved or slightly increased [28, 29].

Gas exchanges

Individuals with obesity are at high risk for ventilation distribution abnormalities and impaired gas exchange.

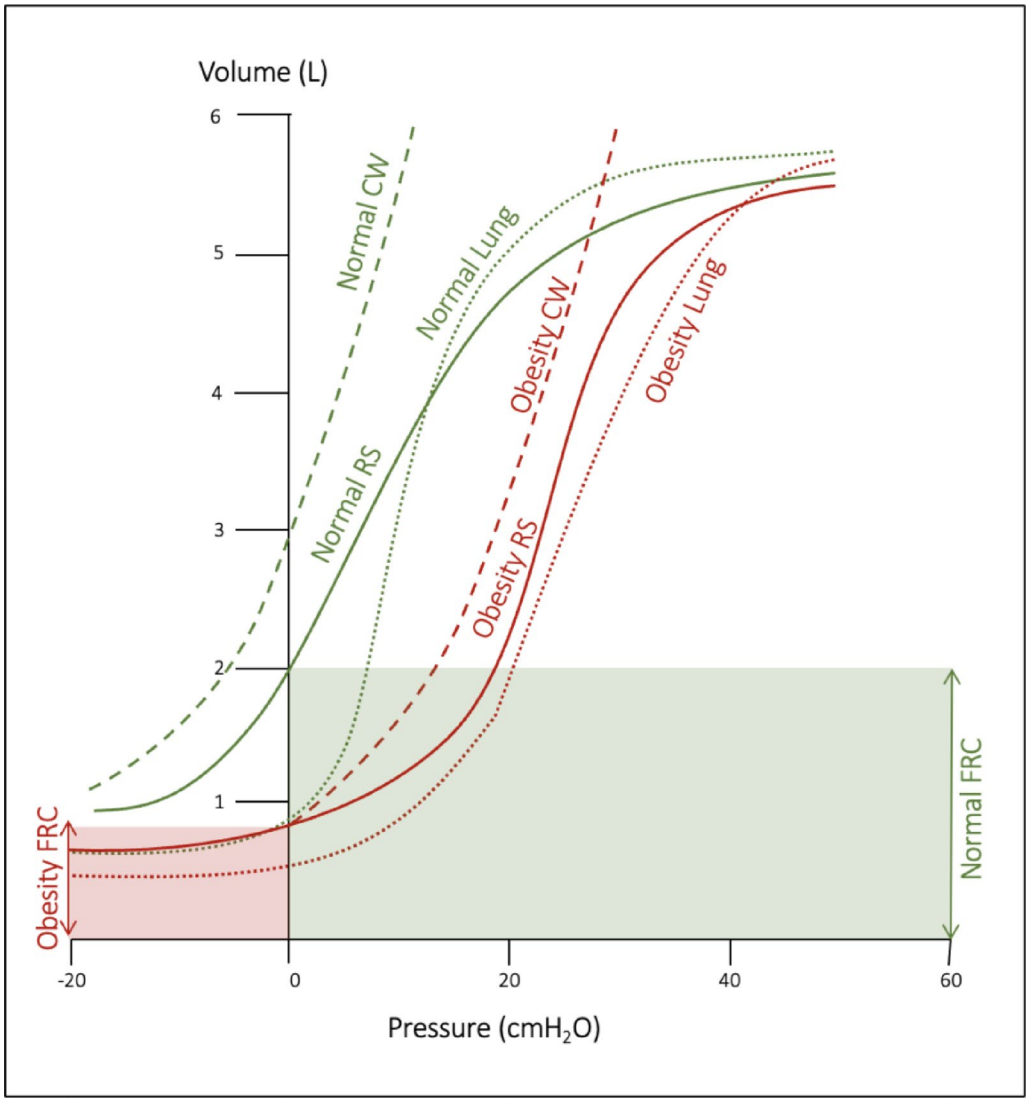

Fig. 1 Pressure – volume curve changes. In the hypothetical pressure-volume curves of the normal respiratory system, FRC is above closing volume, while in obesity, the high pleural pressure lowers FRC to closing volume, thereby reducing RS compliance. CW, chest wall; FRC, functional residual capacity; RS, respiratory system

In upright lean subjects, regional ventilation typically increases from the upper lung zones toward the dependent lower regions. In obesity, this pattern may be reversed, with preferential ventilation of the upper lung zones [30, 31]. Since pulmonary perfusion is predominantly in the lower zones, this altered ventilation distribution promotes ventilation-perfusion mismatch [31], leading to mild hypoxemia even in eucapnic individuals [14, 16, 22].

Work of breathing

During physical exercise, individuals with obesity are more prone to respiratory fatigue than lean subjects. Individuals with morbid obesity are generally able to increase ventilation sufficiently to prevent hypercapnia [32], and the relationships between perceived breathlessness and both oxygen consumption and minute ventilation appears similar to that observed in normal-weight subjects [33]. However, at equivalent workloads, greater oxygen consumption and minute ventilation have been reported in subject with obesity [33], contributing to greater dyspnea during exercise. Several factor may underlie the increased work of breathing and oxygen consumption, including greater tissue displacement to achieve adequate tidal volume, and airway collapse.

Respiratory mechanics in the presence of lung injury

In the presence of acute lung injury, excess adiposity further compromises respiratory mechanics. The increased lung weight characteristic of injured lung may combine with the mass-loading effect of excess adipose tissue [34, 35]. This interaction results in elevated superimposed pressure, which promotes alveolar collapse [10], and further impairs lung mechanics and gas exchange [16, 19, 36] (Fig. 2). Additionally, the increased superimposed pressure, together with alterations in alveolar surface tension caused by lung injury, favours airway collapse. As a result, distal airways close at end-expiration, and part of the applied airway pressure is spent reopening collapsed airways before tidal inflation begins. This phenomenon, characterized by complete interruption of communication between proximal airways and distal alveoli, is known as "airway closure" [37], and the pressure point at which the gas flow begins to expand the alveoli is termed "airway opening pressure" (AOP) [37] (Fig. 3). A higher incidence of complete airway closure has been reported both in mechanically ventilated patients with obesity without lung injury and in those with ARDS, compared with normal-weight patients [36, 38]. In the presence of airway closure, and when the PEEP is set below the AOP, DP calculated from airway pressures may overestimate

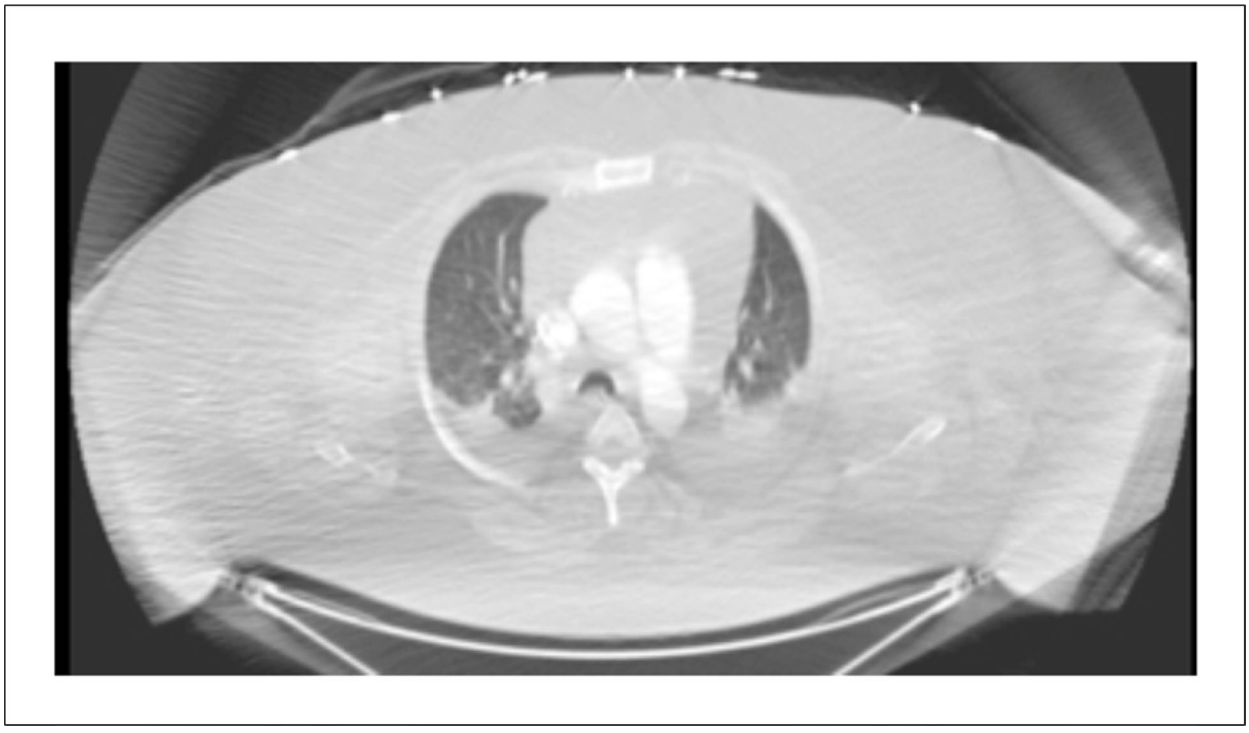

Fig. 2 Axial chest computed tomography image obtained in an intubated and mechanically ventilated patient with severe obesity (BMI 92 kg/m^2). The image was obtained at the level of the tracheobronchial carina. It demonstrates markedly reduced aerated lung volumes with extensive bilateral dependent (posterior) atelectasis. The cardiac silhouette occupies more than half of the maximal transverse thoracic diameter, reflecting markedly reduced intrathoracic space available for lung expansion. These anatomical features likely contribute to increased superimposed pressure on dependent lung regions and reduced functional lung volume

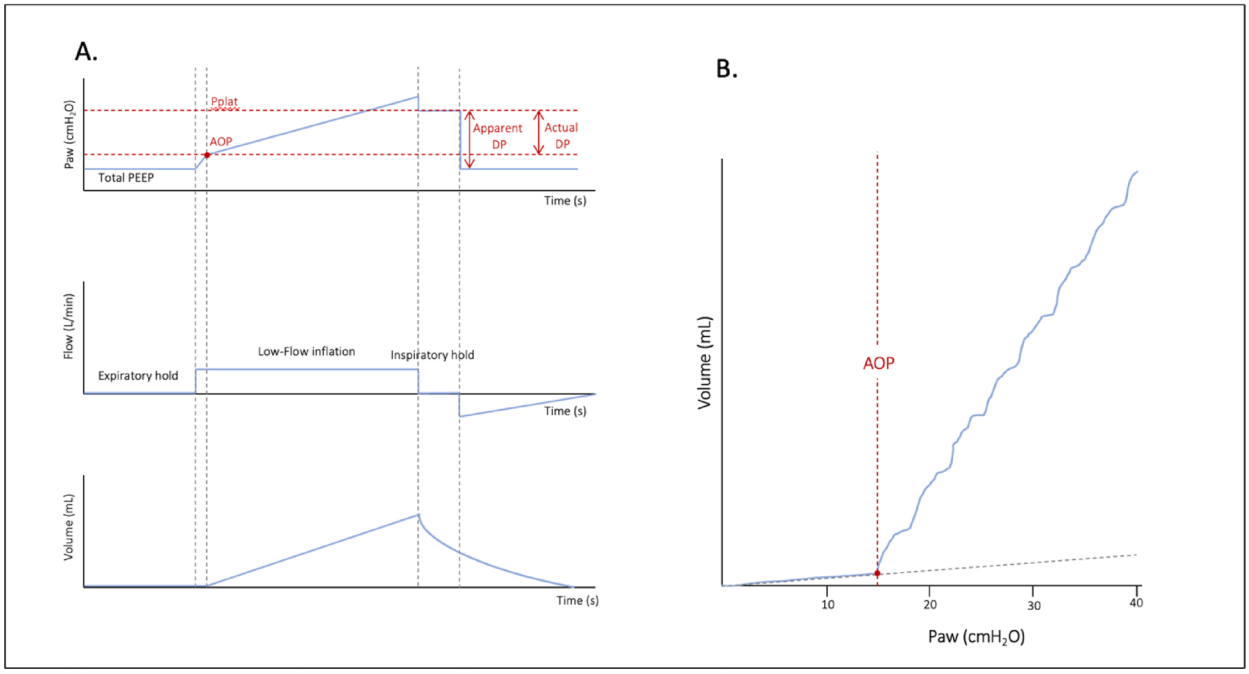

Fig. 3 Airway closure assessment using a low flow (e.g., 5 L/min) and a pressure-time curve during volume-controlled ventilation (**Panel A**), and a pressure-volume loop tool (**Panel B**). In **Panel A**, after ruling out the presence of intrinsic PEEP by an expiratory hold, the initial steep increase of pressure in the absence of an inspiratory flow generating tidal volume suggests the presence of airway closure; the change of the slope of airway pressure identifies the AOP corresponding to the point from which the gas flow starts to expand the alveoli. In the pressure–volume curve (**Panel B**) of the respiratory system the absence of cardiac oscillations, and a value of respiratory system compliance close to that of the occluded breathing circuit (between 1.5 and 2.5 mL/cmH_2O) below the inflection point (indicating AOP) were taken to indicate complete airway closure. AOP, airway opening pressure; DP, driving pressure; Paw, airway pressure

the effective distending pressure applied to the lung parenchyma [39, 40]. In this context, pressure-control ventilation may also result in lack of effective ventilation if the critical AOP required to generate inspiratory flow is not reached.

In conclusion, in patients with lung injury and obesity, elevated values of DP may be incorrectly interpreted as reflecting reduced chest wall compliance. Furthermore, the appropriate tidal volume is frequently overestimated in severe obesity [41, 42]. Consequently, the pressure applied to the respiratory system - largely dissipated in expanding the lung – may expose it to elevated stress and strain, especially when adequate PEEP is not applied.

Mechanical ventilation without lung injury: implications for operating room management

Positioning and anaesthesia induction

The transition from standing or sitting position to the supine position at the anesthesia induction induces specific changes in respiratory mechanics in patients with obesity. First, in the supine position, a smaller-than-expected reduction in FRC or ERV has been observed [25, 43], suggesting that individuals with obesity in the upright position are already breathing near their residual volume. A possible explanation is that sustained activity of the inspiratory muscles at end-expiration may maintain lung volume above residual volume, thereby attenuating the decrease in FRC and ERV in the supine position, limiting airway closure and helping to preserve oxygenation. However, the extent of this effect is likely variable and influenced by individual factors such as fat distribution, chest wall mechanics and muscular tone. Second, in the upright position, reduced lung compliance partially compensates for the decrease in FRC and the increase in airway resistance, thereby preserving expiratory flow until the ERV is obliterated [44]. However, after assuming the supine position, additional factors, such as increased intrathoracic blood volume and upper airways narrowing due to fat loading, promote expiratory flow limitation and air trapping [43, 45]. Consequently, in supine patients with obesity, the respiratory pattern shifts from a purely restrictive to mixed restrictive-obstructive pattern [46].

As a result, patients with obesity may present to the pre-induction phase with pre-existing gas-exchange impairment, airways prone to collapse, and a reduced safe apnea time due to decreased lung volume available for denitrogenation [47, 48] (Fig. 4).

In this context, the priority is to prevent desaturation and ensure oxygen delivery. Adequate preoxygenation in ramped head-up positioning with at least a 30° head-up tilt is recommended, where feasible [49, 50], as it

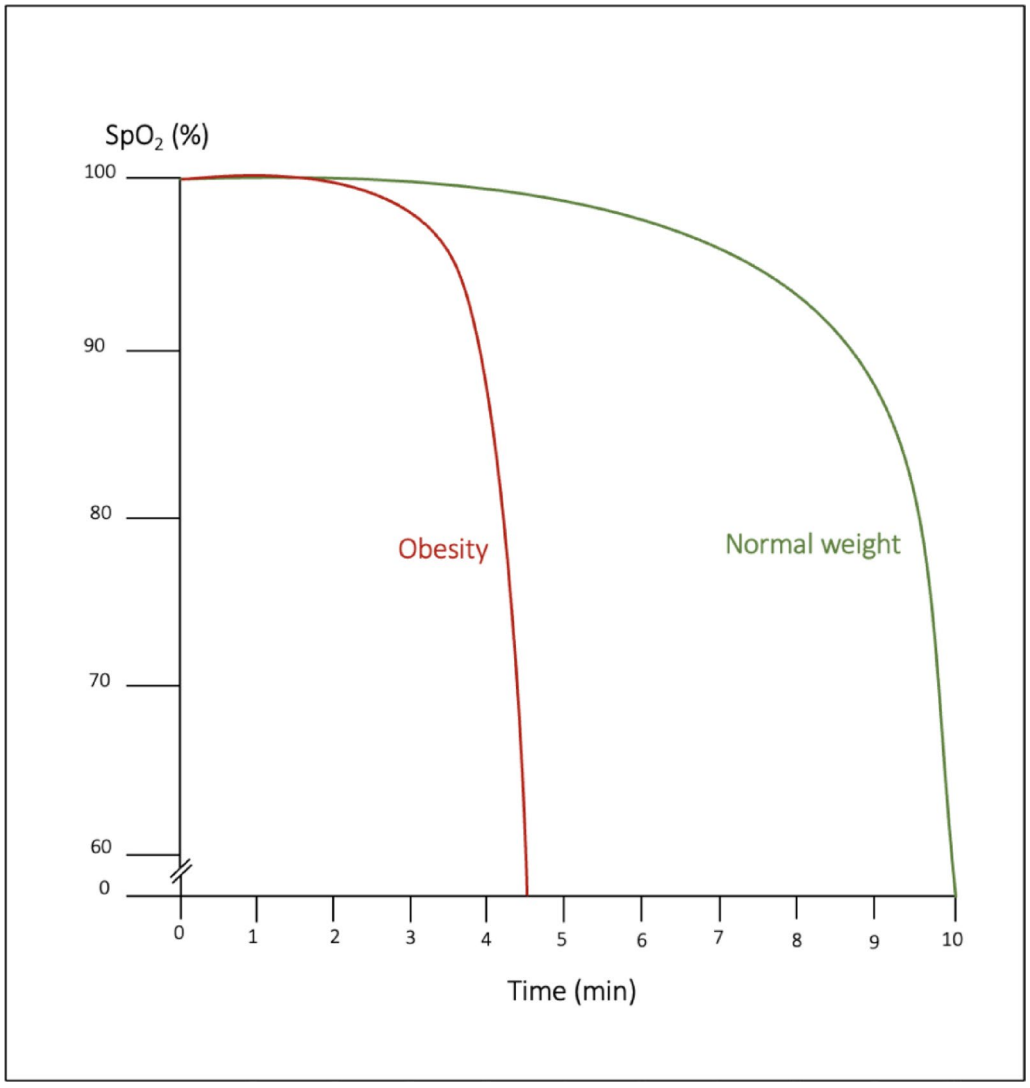

Fig. 4 Desaturation time in obesity. The reduced FRC in obesity decreases oxygen reserve, promotes airway closure and ventilation–perfusion mismatch. Combined with the increased oxygen consumption typically observed in obesity, this results in more rapid oxygen desaturation and a shorter "safe apnea time" during anesthesia induction

prolongs the period of apnoea without desaturation [51]. During elective intubation, an effective oxygenation strategy is the application of positive airway pressure, delivered as continuous positive airway pressure (CPAP) or bi-level positive airway pressure (BiPAP) [52, 53]. During emergent endotracheal intubation, preoxygenation with noninvasive ventilation reduced the risk of hypoxemia by approximately half, without affecting the incidence of aspiration compared with preoxygenation with an oxygen-mask. When the study population was stratified according to increasing BMI, the incidence of hypoxemia increased in the oxygen-mask group, while it remained constant in the noninvasive ventilation group [54].

In both lean patients and patients with obesity, the use of high flow nasal oxygen (HFNO) combined with a facemask for preoxygenation and apnoeic oxygenation has been associated with higher end-tidal oxygen levels after intubation and a lower rate of oxygen desaturation compared with facemask preoxygenation alone [55]. Therefore, HFNO should be considered as a valuable mode for preoxygenation in the operating room [49].

However, obesity-related anatomical and morphological characteristics may contribute to difficult facemask ventilation and airway management, necessitating alternative approaches. In this context, ventilation via a supraglottic device between facemask pre-oxygenation and tracheal intubation attempts is safe and effective [49].

Ventilation setting during anesthesia

The administration of sedation and neuromuscular blockade further reduces FRC, and worsens arterial oxygenation [56], particularly in patients with obesity [57].

In patients with obesity undergoing general anaesthesia, the adequate application of PEEP is crucial in the ventilator setting, as, it maintains EELV, enhances ventilation—perfusion matching, and improves respiratory mechanics and gas exchange [58, 59].

After setting a volume of 6–8 ml/kg of predicted body weight [60], PEEP titration according to the characteristics of the respiratory system – particularly respiratory system compliance and DP [84]- represent a valuable bedside tool, and has been associated with improvements in respiratory mechanics and gas exchange [61]. Even in patients with BMI 50 kg/m^2 or higher undergoing laparoscopic surgery, although compliance-based PEEP titration has been shown to result in slightly negative end-expiratory transpulmonary pressures, this approach improves lung volumes, and oxygenation without causing hemodynamics impairment [58].

Many patients with obesity require high PEEP levels to counteract the high pleural pressures, resulting in plateau pressure exceeding 28 cmH_2O. However if DP remains withing the safety range (i.e., below 15 cmH_2O), the elevated plateau pressure does not necessarily indicate excessive lung stress [62]. An inspiratory hold maneuver after setting mechanical ventilation may serve as a bedside tool whenever clinical conditions change (e.g., induction of pneumoperitoneum or Trendelenburg position), especially when elevated peak pressure is observed. Performing an inspiratory hold is essential to determine whether the increase in peak pressure is due to increased airway resistance or is accompanied by a corresponding increase in plateau pressure. If this function is not available on the anaesthesia ventilator in the OR, ventilation can be set in volume-control mode with an inspiratory pause time to allow visual assessment of the gap between peak and plateau pressures.

Although no specific BMI threshold reliably predicts airway pressure or PEEP requirements, adjusting PEEP based on BMI (e.g., the BMI/3 rule) during general anesthesia has been shown to reduce DP, and decrease postoperative loss of lung aeration. In this context, the BMI/3 rule provides a practical approach to initial PEEP titration, and may serve as a starting point for further individualization [63].

Advanced respiratory monitoring tool may have a role in the operating room setting. PEEP titrated to achieve a positive end-expiratory transpulmonary pressure has been associated with improvements in respiratory mechanics and gas exchange [61]. PEEP titration guided by electrical impedance tomography (EIT) has also been associated with improved oxygenation, EELV, ventilation distribution, and lung mechanics in morbidly obese patients during anesthesia [64]. However, EIT, as well as esophageal manometry in the OR is often limited to research setting, and should be reserved for selected patients in whom concerns arise regarding the interpretation of bedside respiratory mechanic measurements.

The application of a recruitment maneuver (RM), followed by PEEP corresponding to positive end-expiratory transpulmonary pressure after intubation, has been shown to be more effective than PEEP alone in reducing atelectasis, and improving both oxygenation and lung compliance[61]. Although in patients with obesity increased pleural pressures protect the cardiovascular system against higher airway pressures associated with high PEEP [66], the high airway pressures required to overcome increased pleural pressure (e.g., up to 50 cmH_2O), and to achieve lung recruitment in patient with obesity [15, 57], can be clinically relevant [67]. Moreover, since in obesity the increase in airway pressure mainly reflects elevated transpulmonary pressure, a substantial portion of these pressures is transmitted to the lung. A recent large randomized controlled trial comparing a low PEEP strategy (4 cmH_2O) with a strategy including regular RMs and a PEEP of 12 cmH_2O did not demonstrate any benefit on the incidence of postoperative complications in patients with obesity undergoing general anesthesia [67]. It should also be considered that approximately half of the patients enrolled in this trial were studied in the reverse Trendelenburg position, which likely mitigated the mechanical effects of obesity [67]. This may have potentially attenuated the beneficial effects of higher PEEP on respiratory mechanics while amplifying its negative hemodynamic consequences. In conclusion, the routine application of RMs cannot be universally recommended and should instead be individualized after careful evaluation of the risk–benefit balance [68], including the clinical need for alveolar recruitment, the patient's hemodynamic and volemic status, and the presence of conditions associated with an increased risk of barotrauma (e.g., emphysema).

The induction of pneumoperitoneum, particularly when combined with the Trendelenburg position, further aggravates respiratory mechanics in patients with obesity. Particularly, the induction of pneumoperitoneum in the Trendelenburg position has been demonstrated to worsens airway closure by increasing AOP [69]. Pneumoperitoneum elevates intra-abdominal pressure, which is transmitted to the pleural space and increases the pressure required to reopen collapsed airways due to both atelectasis formation or airway collapse [69]. This potentially influences end-expiratory transpulmonary pressure and end-expiratory lung volume (EELV), ultimately affecting lung compliance [70]. Interestingly, in patients with obesity, EELV has been shown to remain relatively unchanged after pneumoperitoneum [69]. One possible explanation is that, in the presence of airway closure and set PEEP lower than AOP, end-expiratory alveolar pressure rises in parallel with airway opening pressure,

partially offsetting the pneumoperitoneum-induced increase in pleural pressure. Conversely, matched lean subjects typically experience a significant reduction in EELV due to pneumoperitoneum [69]. Furthermore, in obesity, changes in chest-wall geometry induced by the increase in intra-abdominal pressure may exert a protective effect on the lung [71], limiting its compressive deformation, thereby reducing the risk of alveolar derecruitment.

In clinical practice, the operating room represents a highly dynamic physiological environment, where changes in intravascular volume, body position, abdominal pressure, and surgical forces may substantially alter respiratory mechanics, requiring frequent reassessment of ventilatory settings. Accordingly, ventilation setting, particularly PEEP, often requires repeated intraoperative titration to accommodate these evolving conditions, as limited adjustments may be insufficient to optimize ventilation [72]. In this context, a personalized approach to PEEP setting should include a comprehensive assessment of the risk for pulmonary atelectasis and other postoperative pulmonary complications, integrating the evaluation and monitoring of respiratory mechanics, gas exchange, and surgical factors such as positioning and pneumoperitoneum, which also influence chest wall compliance. Furthermore, a careful risk–benefit assessment of cardiopulmonary function is essential to identify patients at risk of hemodynamic impairment during RM and PEEP titration, such as those with cardiovascular disease, hypovolemia, or circulatory shock [59].

Postextubation and postanesthesia care

Patients with obesity are particularly prone to developing atelectasis and reductions in lung volumes after extubation [73]. In addition, the prevalence of obstructive sleep apnea and obesity-hypoventilation syndrome is very high is this population, with increasing rates with increasing BMI [65, 74]. Of note, patients with underlying sleep-related breathing disorders are at greater risk of postoperative respiratory failure, particularly hypercapnic respiratory failure, heart failure, and prolonged intubation compared with those with obstructive sleep apnea alone [75]. For these reasons, careful postoperative management is crucial to prevent postsurgical pulmonary complications.

The application of non-invasive ventilation, either CPAP [76] or BiPAP [77], has been demonstrated to improve oxygenation in patient with obesity during the postoperative period.

Accordingly, early initiation of non-invasive ventilation immediately after tracheal extubation is recommended [78], as soon as the patient is awake, responsive, and breathing spontaneously. The application of incentive spirometry may be beneficial in this population, as it has been associated with improved oximetry saturation and faster lung function recovery after surgery [79]. However, the clinical efficacy of postoperative incentive spirometry remains uncertain, as a randomized trial failed to demonstrate improvement in hypoxemia or postoperative pulmonary complications [80].

Figure 5 summarizes key aspects of perioperative ventilatory management in patients with obesity.

Mechanical ventilation with lung injury: implications for intensive care management

In patients with obesity and acute lung injury (e.g., ARDS and AHRF), the interaction between altered respiratory mechanics and lung injury makes individualized ventilatory management essential. It should be noted that patients with severe obesity are often underrepresented or excluded from most clinical trials investigating ventilatory management [81]. Consequently, current ARDS guidelines [82, 83] do not provide specific recommendations for patients with obesity.

For these reasons, a "one-size fits all" approach cannot be applied to patients with obesity, particularly in the presence of lung injury. Mechanical ventilation should be individualized, taking into account the pathophysiological alteration related to obesity and the presence of lung injury, as well as bedside assessment of respiratory mechanics, and, when available, advanced monitoring tools, such as esophageal pressure manometry and EIT.

Bedside respiratory mechanics

After setting tidal volume to 4–8 ml/kg of predicted body weight [68, 83–85], an appropriate PEEP level should be selected. Adequate PEEP stabilizes the alveoli, prevents end-expiratory collapse and atelectrauma, minimize overdistention, and improves gas exchange [86]. Current ARDS guidelines recommend higher PEEP levels in moderate-to-severe ARDS, as these have been associated with an improved survival [82]. Based on the mechanisms discussed above, patients with obesity may require higher PEEP levels than lean individuals with comparable degree of disease severity.

Selecting the PEEP level associated with the lowest DP, following a RM to reopen collapsed alveoli, represents a valuable bedside strategy. This approach has been shown to improve oxygenation and lung mechanics compared with standard PEEP-FiO_2 Table [87]. In the same study, the PEEP level corresponding to the lowest DP was associated with a positive end-expiratory transpulmonary pressure, and an optimal balance between lung overdistension and collapse, as assessed by EIT [87]. Although DP has not been associated with mortality in ARDS patients with obesity [88], it remains a key parameter in ventilatory management, as it represents a reliable bedside estimate of the elastic load applied to the lung

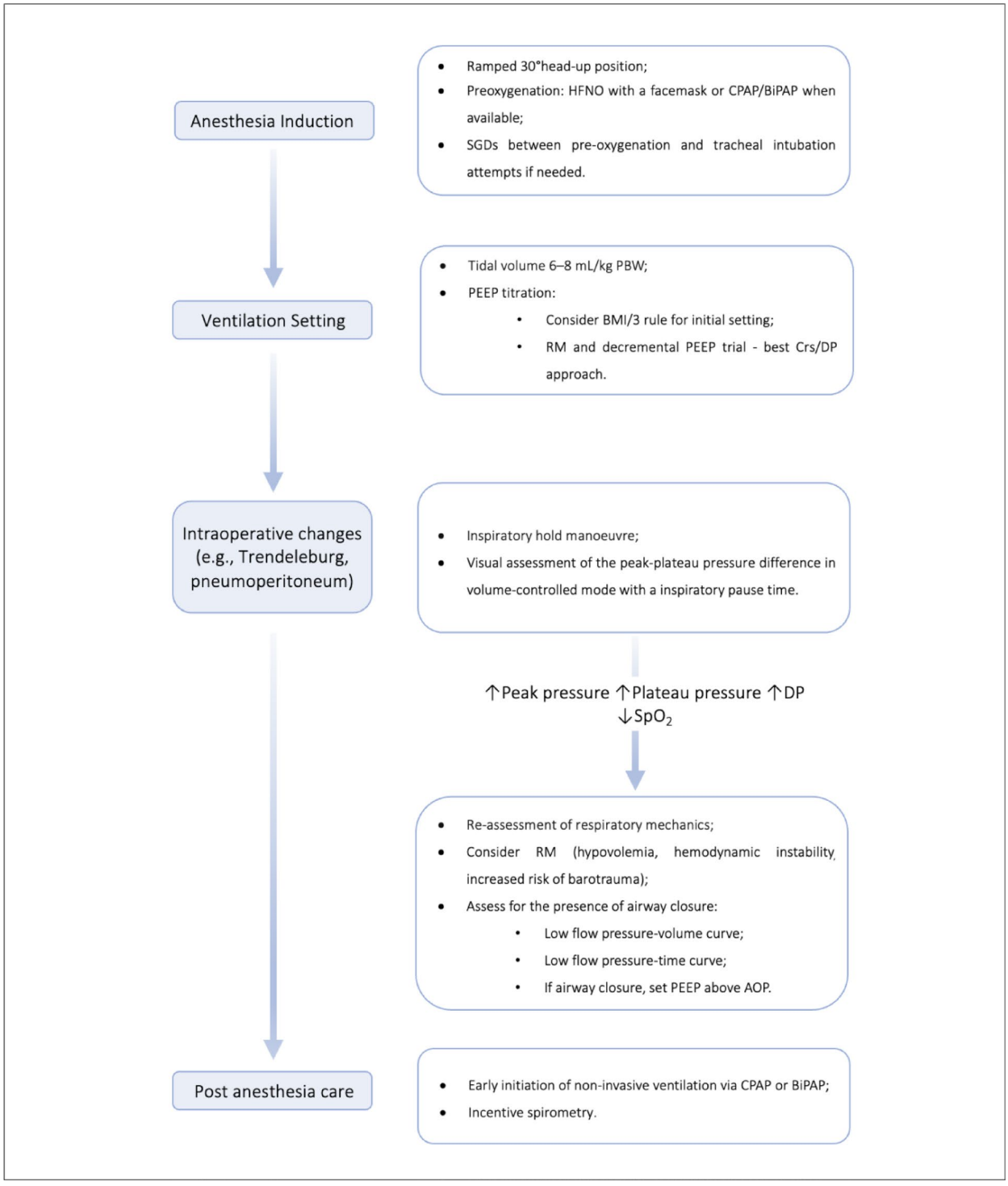

Fig. 5 Perioperative ventilatory management flowchart for patients with obesity. BiPAP, bi-level positive airway pressure; CPAP, continuous positive airway pressure; DP, driving pressure; HFNO, high flow nasal oxygen; PBW, predicted body weight; PEEP, positive end-expiratory pressure; RM, recruitment maneuver; SGD, supraglottic device

parenchyma during tidal inflation (i.e., lung strain) and a major determinant of ventilator-induced lung injury. DP measurements can be easily obtained using inspiratory and expiratory hold maneuvers without requiring additional equipment or expertise. However, it reflects the mean DP (or compliance) measured at the airway opening, thereby representing the combined mechanics of the lung and chest wall, as well as different - likely

heterogeneous - lung regions. The high prevalence of complete airway closure in patients with obesity and ARDS must be considered when assessing respiratory mechanics and titrating PEEP. When PEEP is set below the AOP, distal airways undergo cyclic opening and closing, thereby exacerbating alveolar and bronchiolar injury (e.g., atelectrauma). Additionally, DP of the respiratory system may be overestimated if its calculation is not adjusted for the presence of complete airway closure. In this context, the high prevalence of airway closure in patients with ARDS patients and obesity may partly explain the lack of a clear association between DP and mortality in this population [88], as well as the conflicting evidence regarding the benefit of transpulmonary pressure–guided PEEP strategies [89, 90]. However, a promising association between a positive end-expiratory transpulmonary pressure and higher survival was reported in a prospective observation multicentre registry in ARDS patients with obesity [91].

At the bedside, airway closure can be detected using a low flow inflation (e.g., 5 L/min) pressure–volume diagnostic tool [37]. Alternatively, during volume-controlled ventilation, airway closure can be identified by applying a low inspiratory flow and analysing the pressure—time waveform [86]. A steep initial rise in airway pressure in the absence of inspiratory flow and delivered tidal volume suggests complete airway closure. The subsequent inflection point, at which the slope of the pressure-time curve changes, corresponds to the AOP, beyond which inspiratory flow resumes and tidal inflation of the alveoli occurs (Fig. 3). Furthermore, as recently reported by Haudeborg et al. the assessment of conductive pressure on the pressure-time waveform during tidal ventilation may provide some hints on the presence of airway closure in ARDS [92]. AOP identification can be technically challenging for clinicians who do not routinely perform it, and the presence of auto-PEEP may be misinterpreted as airway closure. Although auto-PEEP can coexist with airway closure, the two phenomena can be distinguished: auto-PEEP can be reduced by prolonging expiratory time, whereas the level of AOP at the subsequent insufflation remains constant [93].

Therefore, at the bedside, the presence of airway closure should always be assessed and PEEP should be adjusted to exceed the AOP and to achieve the lowest DP.

Of note, positioning patients with obesity in at least 30° head-up or sitting position may facilitate unloading the diaphragm from increased abdominal pressure and may thereby improve aeration of dependent lung areas.

Patients with obesity and obstructive sleep apnea requiring endotracheal intubation for acute respiratory failure represent a particularly high-risk population. Reduced FRC, increased oxygen consumption, and frequent coexistence of obesity hypoventilation syndrome predispose these patients to rapid oxygen desaturation during apnea and induction. In addition, severe hypercapnia, pulmonary hypertension, and right ventricular dysfunction may increase the risk of hemodynamic instability and hypotension during intubation. After intubation, lung-protective ventilation strategies and PEEP titration should be applied as previously discussed.

Advanced respiratory monitoring

Although further research is need to determine the impact of advanced respiratory monitoring tools on clinical outcomes, both esophageal pressure measurement and EIT provide valuable physiological insights for ventilatory titration in patients with obesity and acute lung injury.

While the difference between end-inspiratory and end-expiratory transpulmonary pressure accurately reflects the risk of excessive lung stress, the absolute value of transpulmonary pressure at end-expiration reflects the risk of alveolar collapse. In critically ill patients with obesity, elevated pleural pressure may result in negative transpulmonary pressure at end-expiration. Therefore, targeting a positive end-expiratory transpulmonary pressure during PEEP titration has a strong physiological rationale.

In normal-weight patients with ARDS, adjusting PEEP to achieve a positive end-expiratory transpulmonary pressure has shown to improve oxygenation and respiratory system compliance, without a significant impact on clinical outcome [89, 94]. In patients with obesity, transpulmonary pressure-guided lung-protective ventilation - compared with a conventional approach – has been associated with higher PEEP levels, restoration of EELV, improved lung compliance and oxygenation, prevention of lung overdistention, and acceptable hemodynamic tolerance [19, 61, 66]. Moreover, in patients with BMI > 40 Kg/m^2, this strategy, together with an individualized approach based on patient-specific cardiopulmonary physiology and implemented by a dedicated lung rescue team, has been associated with reduced in-hospital mortality [95].

We suggest monitoring esophageal pressure either by considering the absolute end-expiratory esophageal pressure value or by evaluating the esophageal pressure swing during tidal insufflation [17]. This tool is particularly useful when high PEEP levels are required to achieve protective DP, or when elevated plateau pressure - traditionally considered unsafe in ARDS - may actually correspond to acceptable transpulmonary pressure values. However, esophageal balloon placement is a relatively invasive procedure, and the correct positioning and interpretation requires specific expertise. For example, its calibration may be altered in the presence of airway closure, and provide unreliable interpretation of the esophageal and

transpulmonary pressures [96]. Furthermore, esophageal manometry provides an overall estimate of the pleural pressure, primarily reflecting the pleural pressure in the dependent lung [97].

EIT is a noninvasive, radiation-free imaging technique that provides real-time, bedside monitoring of regional lung ventilation. It is based on the measurement of impedance changes within the thorax through surface electrodes placed around the chest. As air content in the lungs varies during the respiratory cycle, corresponding changes in electrical impedance allow reconstruction of cross-sectional images reflecting the distribution of ventilation.

EIT provides dynamic information on the regional distribution of tidal ventilation and allows the evaluation of changes in end-expiratory lung impedance (EELI), a surrogate for EELV, following PEEP adjustments. PEEP titration using EIT is typically based on regional compliance changes during a decremental PEEP trial preceded by a RM: compliance loss towards higher PEEP is interpreted as overdistension, whereas compliance loss towards lower PEEP represents collapsed lung regions [98] (Fig. 6). The "optimal" PEEP, defined as the level minimizing both collapse and overdistention in the explored PEEP range, has been shown to correspond to a positive end-expiratory transpulmonary pressure [87].

PEEP titration using EIT requires DP measurement to estimate regional compliance. Therefore, it should be performed in volume-controlled ventilation (with inspiratory pause > 0.5 s and no intrinsic PEEP) if DP can be automatically collected by the EIT device. Otherwise, pressure-controlled mode, with constant DP should be used [99]. An alternative approach involves evaluating EELI stability at different PEEP levels, aiming to achieve a stable EELI signal during tidal ventilation [100]. EIT-guided PEEP titration may be particularly valuable in patients with inhomogeneous lung injury (e.g., unilateral pneumonia or the presence of emphysematous bullae) [101], where the balance between collapse and hyperdistention should be individualized [102].

In addition, EIT can be useful to predict the effect of patient positioning. Tidal impedance and EELI variations could inform about the effects of both prone and lateral positioning [103, 104].

Prone positioning

Given its demonstrated benefits on clinical outcomes, prone positioning is widely employed in patients with severe ARDS [105].

From a pathophysiological perspective, the prone position improves respiratory mechanics and mitigates ventilator-induced lung injury by promoting a more homogeneous distribution of transpulmonary pressure. This derives from dorsal lung regions recruitment, and overdistension reduction in the ventral ones. Consequently, lung compliance may improve due to increased participation of previously collapsed alveolar units in tidal ventilation [106, 107]. Regarding chest wall mechanics, prone positioning may lead to a reduction in anterior chest wall compliance due to restricted expansion against the supporting surface [108]. However, this effect is often offset by improved diaphragmatic mechanics and a more favorable distribution of pleural pressure, particularly in the dorsal lung regions. As a result,

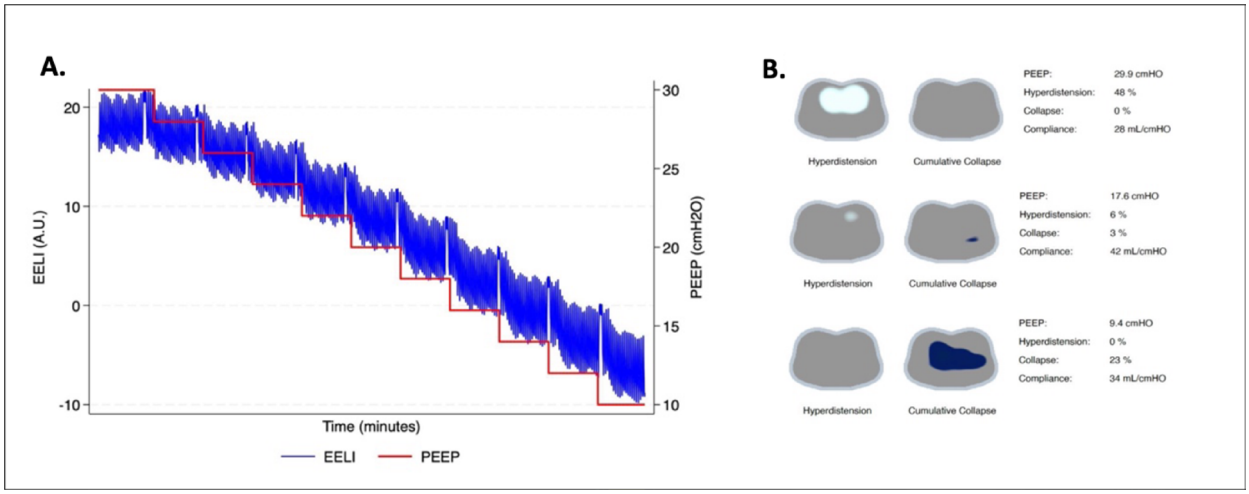

Fig. 6 End-expiratory lung impedance measured by EIT (Panel A) and, representative EIT images at different PEEP levels (Panel B) during a decremental PEEP trial in a patient with BMI 69 kg/m^2. Progressive PEEP reduction (red line) is associated with decrease in EELI (blue), reflecting reduction in end-expiratory lung volume (Panel A). Representative EIT maps at different PEEP levels during the decremental trial, analyzed using the method described by [98] show the distribution of overdistention (left) and collapse (right). At higher PEEP levels overdistention predominates, while progressive PEEP reduction is associated with increasing collapse. Respiratory system compliance at each PEEP level is also shown (Panel B). EIT, electrical impedance tomography; EELI, end-expiratory lung impedance; PEEP, positive end-expiratory pressure.

the overall compliance of the respiratory system may remain unchanged or improve, depending on the balance between lung recruitment and chest wall constraint. Prone position also promotes a more even distribution of ventilation, while pulmonary perfusion remains relatively preserved in dorsal regions due to gravitational and anatomical factors. This leads to improved ventilation–perfusion matching and reduced intrapulmonary shunt [109].

Patients with obesity may be particularly responsive to prone positioning [110], as this intervention may counteract the reduction in EELV.

However, when assessing global respiratory system mechanics in patients with obesity, it should be considered that increased abdominal load may further influence chest wall behavior.

Recent physiological data indicate that regional airway closure, assessed by EIT, occurs more frequently in dorsal than in ventral lung regions. Furthermore, body position appears to influence AOP degree, supporting a role for gravitational gradients in airway closure [111]. As an example, Fig. 7 shows a pressure–volume curve illustrating global and regional airway closure in a swine model of lung injury and obesity. In this context, positional therapy may attenuate airway closure - particularly in patients with predominant thoracic obesity - by reducing compressive forces exerted by adipose tissue on dependent lung regions.

Overall, prone position represents a feasible and safe therapeutic strategy in patients with severe ARDS and obesity [105, 110]. However, a higher incidence of renal failure and hypoxic hepatitis has been observed in patients with abdominal obesity compared with normal-weight patients [112]. Therefore, particular caution is required during pronation, including appropriate abdominal positioning and consideration of a reverse Trendelenburg position to minimize increases in intra-abdominal pressure and prevent abdominal organ compression [68].

Heart-lung interactions during mechanical ventilation

Individuals with obesity are at increased risk of cardiovascular disease because of the high prevalence of metabolic syndrome [113]. The increased adipose tissue mass results in greater circulatory demand, characterised by increased blood volume and cardiac output, ultimately leading to ventricular hypertrophy, diastolic dysfunction, and an increased risk of atrial fibrillation. Consequently, when the increased circulating blood volume related to body mass is combined with diastolic dysfunction, cardiac filling pressures may rise [114].

The application of positive airway pressure during mechanical ventilation can significantly affect heart—lung interactions mainly driven by changes in intrathoracic pressure. In patients with obesity, the detrimental

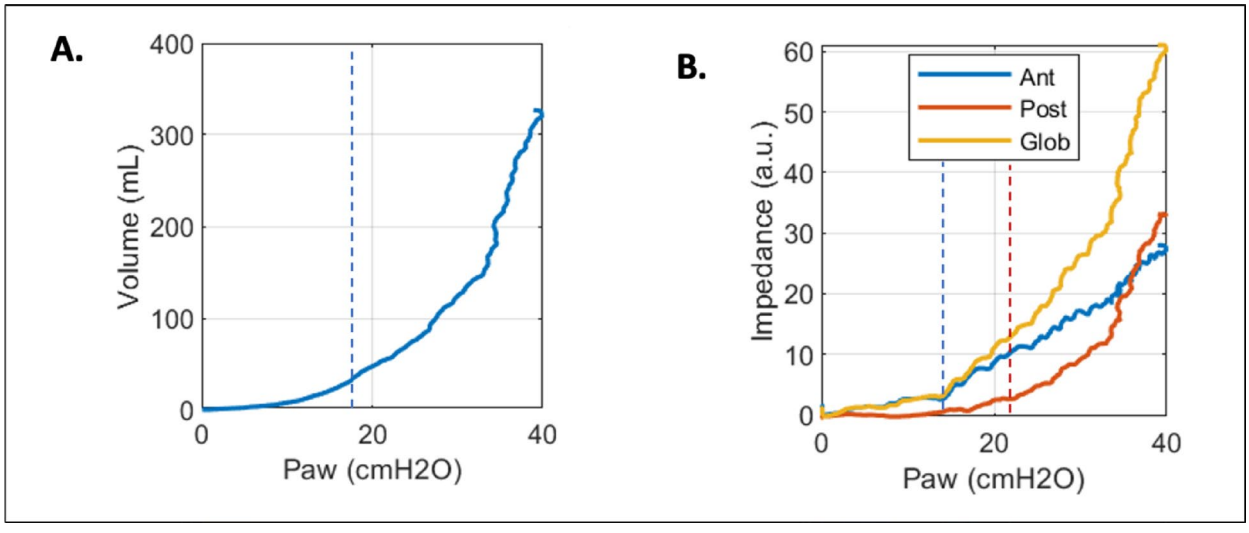

Fig. 7 Representative pressure-volume curve (**Panel A**), and pressure impedance curve (**Panel B**) during a low flow inflation in a swine model of lung injury and obesity (6 Kg abdominal load). The pressure-volume curve reveals the presence of airway closure, and the blue dashed line represents the airway opening pressure (**Panel A**). The global (yellow line), ventral (blue line), and dorsal (red line) pressure–impedance curves are shown in **Panel B**. The presence of airway closure was assessed off-line by analyzing the recorded airway pressure-volume tracings. Complete airway closure was inferred from the pattern of the pressure-volume curve during low-flow inflation, as described previously [37, 38, 69]. Below an inflection point (e.g., airway opening pressure) early in the initial part of the curve, the absence of cardiac oscillations, and a value of respiratory system compliance close to that of the occluded breathing circuit (between 1.5 and 2.5 mL/cm H_2O) were taken to indicate complete airway closure. Once pressure exceeded the AOP, cardiac oscillations became apparent in the airway pressure tracing, and compliance increased abruptly. In this representative model, the airway opening pressure is higher in the dorsal dependent regions (red dashed line) compared with the ventral nondependent regions (blue dashed line). AOP, airway opening pressure; Paw, airway pressure

effects of PEEP on venous return and right ventricular preload may be partially offset by elevated pleural pressures. The resulting increase in upstream venous pressures may help maintain venous return despite high PEEP levels [66].

In the presence of lung injury, as well as significant atelectasis during general anaesthesia, changes in lung volumes together with the impaired gas-exchange may increase pulmonary vascular resistance and impede right ventricular ejection [115]. Appropriate levels of PEEP can maintain alveolar recruitment and restore lung volume toward FRC, thereby reducing pulmonary vascular resistance and right ventricular afterload. In contrast, excessive PEEP may compress low-resistance pulmonary capillaries, increasing right ventricle afterload, according to the U-shaped relationship curve between lung volume and pulmonary vascular resistance [116, 117]. The application of PEEP may also reduce left ventricular afterload through a reduction in left ventricular transmural pressure (e.g., difference between left ventricle intracavitary pressure and pleural pressure), potentially improving left ventricular ejection fraction [118].

Of note, the high prevalence of pulmonary hypertension—— caused by obstructive sleep apnoea/obesity hypoventilation syndrome, elevated left ventricular filling pressures, or chronic gas exchange impairment——may further complicate the cardiopulmonary management of individuals with obesity [119]. In patients with preserved right ventricular function, transient increases in pulmonary vascular resistance are usually well tolerated. However, in patients with obesity, preexisting pulmonary hypertension may lead to varying degree of right ventricular dysfunction. In this context, impaired gas exchange, acidosis, and inadequate PEEP setting resulting in excessive alveolar collapse or overdistention may further increase pulmonary vascular resistance and impair right ventricular function [116, 117]. The resulting right ventricular pressure and/or volume overload may cause interventricular septum shifts toward the left ventricle, thereby limiting left ventricular filling and cardiac output.

For all the reasons, careful PEEP titration aimed at minimizing both alveolar collapse and overdistension, together with prompt correction of factors that increase pulmonary vascular resistance [120], is essential to preserve right ventricular performance and, through ventricular interdependence, maintaining left ventricular function [59, 121, 122].

Weaning from mechanical ventilation

During weaning and the peri-extubation period, patients with obesity may exhibit an increased work of breathing, as a substantial proportion of inspiratory effort is required to overcome the airway closing pressure and generate airflow [123, 124]. This mechanism may prolong the weaning phase and increase the risk of extubation failure.

In this context, careful assessment of inspiratory effort during spontaneous breathing trial is essential. Pressure support should be progressively reduced while monitoring tidal volume and respiratory rate, and indices of inspiratory effort such as the pressure muscle index [125] and the occlusion pressure [126]. Additionally, the reduced mean airway pressure combined with the possible increased work of breathing can result in marked decreases in intrathoracic pressure. This may increase right ventricular afterload and venous return, potentially leading to pulmonary edema and extubation failure [127]. Therefore, cardiac function and volemic status should be carefully assessed during weaning phase.

A spontaneous breathing trial performed with a T-piece - where the patient remains intubated but disconnected from the ventilator and breathes humidified oxygen - may accurately reflect the patient ventilatory workload [128]. Alternatively, if the work of breathing due to airway closure is intolerable, the ventilator may be set to CPAP mode without additional pressure support.

Application of a PEEP level sufficient to counteract expiratory airway collapse may be beneficial, although conventional PEEP levels are often inadequate in mechanically ventilated patients with obesity [19, 61]. The use of relatively high PEEP levels at the time of extubation (e.g., 15–30 cmH_2O), aimed at maintaining positive transpulmonary pressure throughout the respiratory cycle, has been shown to accelerate weaning process [129]. However, this approach did not change the proportion of patients successfully weaned [129].

Additionally, patient positioning plays a key role during the weaning phase in patients with obesity. A head-elevated position, with the upper body elevated by at least 30°, is recommended to reduce intra-abdominal pressure, thereby decreasing pleural pressure and improving respiratory system compliance. As a result, diaphragmatic mechanics is optimized, lung volumes increase, and the work of breathing is reduced. These physiological benefits may facilitate spontaneous breathing and improve tolerance to weaning [46].

Positive airway pressure should be maintained throughout the peri-extubation period. Following discontinuation of invasive mechanical ventilation, positive pressure can be delivered noninvasively to prevent airway collapse, to preserve end-expiratory lung volume, and to offset the increased work of breathing, while maintaining hemodynamic stability without impairing the right heart function [21]. After extubation, patients with obesity should be promptly transitioned to noninvasive support, such as CPAP (PEEP ≥ 10 cmH_2O, equivalent to the PEEP applied during weaning, or matching their prescribed

CPAP setting for obstructive sleep apnea) or BiPAP [130]. The prevention of airway collapse through positive pressure is particularly relevant in patients with obstructive sleep apnea and obesity hypoventilation syndrome.

In a multicenter, randomized controlled trial involving critically ill adults with obesity, the use of noninvasive ventilation after extubation was associated with reduced treatment failure - including reintubation or the need to switch to noninvasive ventilation - compared with HFNO or conventional oxygen therapy [131]. These results were also confirmed in a post hoc analysis including postoperative patients only [132]. Additionally, a post hoc analysis of a multicenter randomized controlled trial suggests that prophylactic BiPAP alternating with HFNO may reduce the risk of reintubation and mortality compared with HFNO alone in overweight patients or patients with obesity at high risk of extubation failure (e.g., older than 65 years or had any underlying chronic cardiac or lung disease). By contrast, prophylactic BiPAP was not effective in normal or underweight patients [133].

A recent systematic review and meta-analysis further support these findings, suggesting that noninvasive ventilation, alone or in combination with HFNO, may be superior to standard oxygen or HFNO alone to prevent reintubation [134]. Accordingly, early initiation of CPAP or BiPAP following extubation appears advisable in patients with obesity [130, 135]. Non-invasive ventilation should be applied for prolonged periods (i.e., at least 12 h) within the first 24–48 h following extubation, and tolerance should be optimized through interface selection, device rotation, active humidification, and appropriate setting [135].

Figure 8 includes key aspects of ventilatory management for patients with obesity and lung injury.

Conclusions

Obesity profoundly modifies respiratory physiology through complex interactions between altered chest wall mechanics, elevated pleural pressures, reduced lung volumes, and a high prevalence of airway closure. These changes have important implications for mechanical ventilation across perioperative and critical care settings, both in the absence and presence of lung injury.

In patients with obesity, standard ventilatory approaches derived from normal-weight populations may lead to misinterpretation of respiratory mechanics and inappropriate ventilatory settings, potentially increasing the risk of atelectasis, ventilator-induced lung injury, and weaning failure. Importantly, elevated airway pressures or DP in obesity should not automatically be attributed to reduced chest wall compliance; in many cases they reflect the presence of airway closure and altered lung volume rather than intrinsic chest wall stiffness.

Individualized ventilation strategies based on predicted body weight, careful assessment of DP, and recognition of airway closure are essential. Advanced physiological monitoring tools, including esophageal pressure manometry and EIT, offer valuable insights to guide PEEP titration and optimize lung protection. Ultimately, trials adopting physiology-driven, patient-specific approach—rather than a “one-size-fits-all” strategy—are required to validate and improve respiratory management and outcomes in patients with obesity undergoing mechanical ventilation.

Future research should focus on improving the evidence base for mechanical ventilation strategies in patients with obesity. Dedicated studies specifically targeting patients with obesity, or ensuring adequate representation of this population in large trials, are needed to better define optimal ventilatory approaches and cardiopulmonary management. In addition, most currently available studies stratify patients exclusively according to BMI, which may inadequately reflect the heterogeneity of obesity phenotypes. Future investigations should therefore explore the impact of fat distribution patterns, including android versus gynoid obesity, using anthropometric or imaging-based measures such as waist-to-hip ratio, as these phenotypes may differently affect respiratory mechanics, heart–lung interactions, and response to mechanical ventilation.

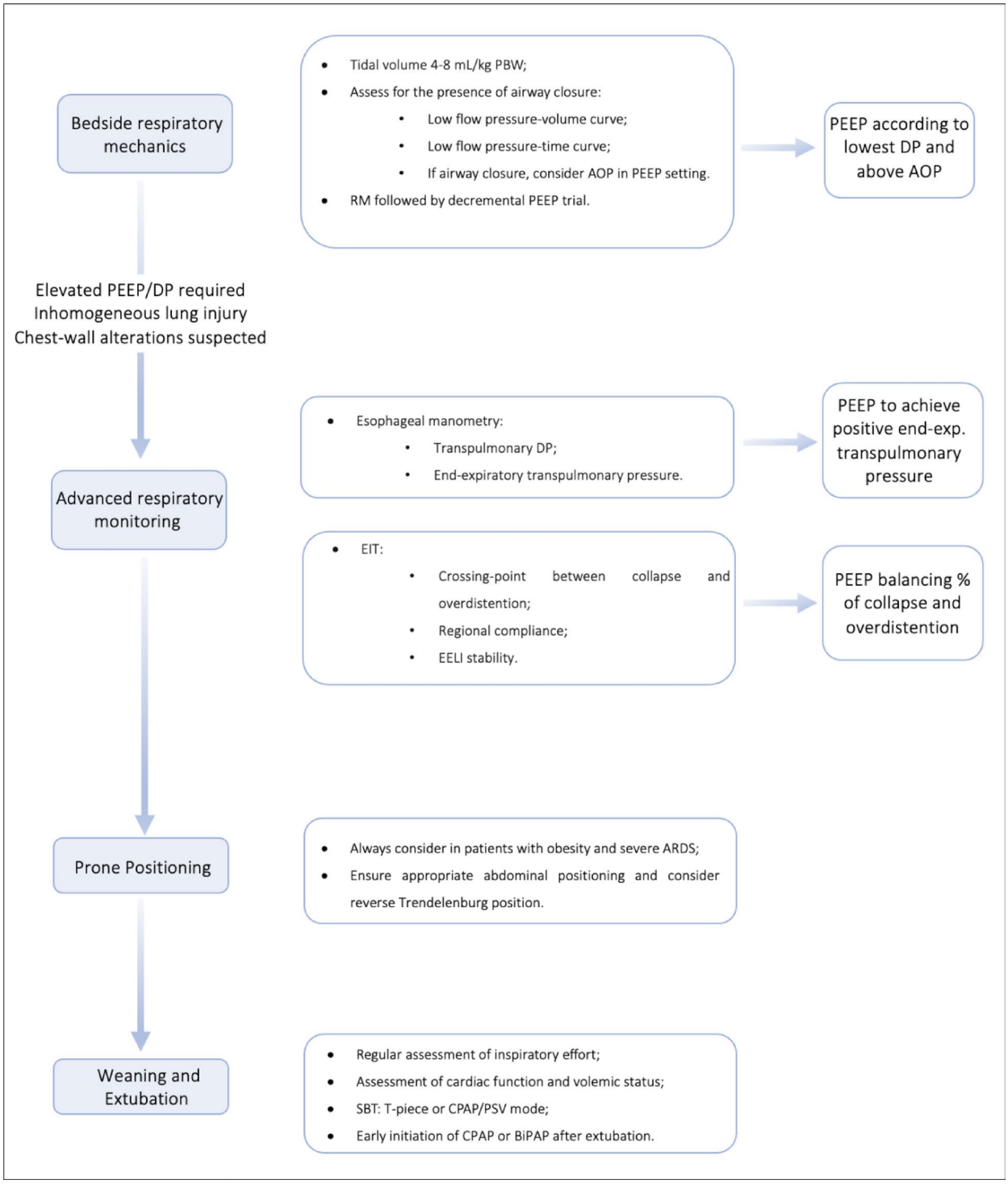

Fig. 8 Ventilatory management flowchart for patients with obesity and lung injury in the intensive care unit. ARDS, acute respiratory distress syndrome; BiPAP, bi-level positive airway pressure; CPAP, continuous positive airway pressure; PSV, pressure support ventilation; DP, driving pressure; EIT, electrical impedance tomography; PBW, predicted body weight; PEEP, positive end-expiratory pressure; RM, recruitment maneuver; SBT, Spontaneous breathing trial

Abbreviations

AHRF	Acute hypoxemic respiratory failure
ARDS	Acute respiratory distress syndrome
AOP	Airway opening pressure
BiPAP	Bilevel positive airway pressure
BMI	Body mass index
CPAP	Continuous positive airway pressure
CW	Chest wall
DP	Driving pressure
EELI	End-expiratory lung impedance
EELV	End-expiratory lung volume
EIT	Electrical impedance tomography

ERV	Expiratory reserve volume
FEV1	Forced expiratory volume in one second
FRC	Functional residual capacity
FVC	Forced vital capacity
HFNO	High flow nasal oxygen
ICU	Intensive care unit
Paw	Airway pressure
PEEP	Positive end-expiratory pressure
PBW	Predicted body weight
OR	Operating room
RM	Recruitment maneuver
RS	Respiratory system
RV	Residual volume
SBT	Spontaneous breathing trial
SGD	Supraglottic device
TLC	Total lung capacity

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Author contributions
A.N. conceived the study, searched literature, drafted images, and wrote the manuscript; E.R. conceived the study, searched literature, drafted images, wrote the manuscript and supervised the project; C.M. helped drafting Figure 6 and revised the manuscript; D.T., revised the manuscript; M.B.P.A., M.C., searched literature and revised the manuscript. L.B. conceived the study, searched literature, revised the manuscript, and supervised the project. All authors gave final approval of the version of the manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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