Respiratory Consequences of Obesity

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Introduction

Prevalence and global burden of obesity

Obesity is a significant global health issue, characterized by excess body fat that impairs health. It is measured using various parameters such as Body Mass Index (BMI), waist circumference, and waist-to-hip ratio (WHR). The World Health Organization (WHO) defines overweight as a BMI of 25 or more and obesity as a BMI of 30 or more. Obesity has reached epidemic proportions, with its prevalence increasing rapidly over the past few decades.

The prevalence of obesity has been increasing globally, affecting both developed and developing countries. According to recent data, approximately 1.6 billion adults were overweight, and at least 400 million adults were obese in 2005. By 2015, these numbers were projected to increase to 2.3 billion overweight adults and over 700 million obese adults. This trend is not limited to adults; childhood obesity has also become a significant concern, with every year more children affected globally.

The rise in obesity rates is attributed to a combination of genetic susceptibility, increased availability of high-energy foods, and decreased physical activity in modern society. This epidemic is no longer confined to high-income countries; it is also prevalent in low- and middle-income countries, particularly in urban areas, due to changes in diet and lifestyle.

Obesity is a leading cause of morbidity and mortality worldwide and contributes to a wide range of health complications. It is associated with an increased risk of cardiovascular diseases, type 2 diabetes, certain cancers, and respiratory disorders. The economic burden of obesity on healthcare systems is substantial, as it leads to increased healthcare utilization, reduced productivity, and a diminished quality of life.

Pathophysiological mechanisms linking obesity and respiratory dysfunction

Obesity significantly affects the respiratory mechanics. Thoracic and abdominal adipose tissues reduce lung and chest wall compliance, leading to reduced lung volumes (particularly functional residual capacity (FRC) and expiratory reserve volume (ERV). The work of breathing increases, and a marked decrease in ERV causes closure of the small airways in the basal parts of the lungs and ventilation-perfusion mismatch. Airway resistance in obese individuals increases due to narrowing of the airways in the presence of reduced lung volume. Although specific resistance is normal, chronic inflammation and adipokines can cause airway remodeling and further worsening obstruction. In addition to mechanical factors, obesity leads to systemic inflammation with elevated cytokine levels of cytokines (e.g. TNF-α,

IL-6, and CRP), which can promote airway inflammation and bronchial hyper-responsiveness. Insulin resistance in metabolic syndrome further impairs lung function via epithelial damage and airway smooth muscle proliferation. Adipokines from adipose tissue also contribute to respiratory dysfunction; adiponectin (a protective adipokine) levels are decreased, and leptin levels are increased in obesity. Leptin influences ventilatory drive, and elevated leptin levels are associated with poorer lung function and a higher risk of asthma.

Impact of Obesity on Respiratory Mechanics

Physiological changes in respiratory mechanics

Obesity significantly alters the respiratory mechanics. Excess adipose tissue around the chest and abdomen places pressure on the chest wall, reducing its compliance and displacing the diaphragm upward. Increased chest mass leads to a greater work of breathing, so obese individuals breathe faster and shallower (higher respiratory rate, lower tidal volume) to reduce effort. Overall respiratory compliance is reduced due to a combination of reduced chest compliance and a slight decrease in lung compliance (due to increased pulmonary blood flow and microatelectasis). The consequence of these changes is a shift of the respiratory curve towards lower volumes and more rapid fatigue of the respiratory muscles during exercise.

Increased airway resistance

Obesity may cause functionally higher airway resistance, although there is no marked narrowing of the airways at rest when adjusted for lung volume. The main reason is reduced functional residual capacity (FRC), obese people breathe at a lower lung volume, which reduces radial bronchial distension and allows airway smooth muscle to shorten excessively. This narrows the lumen of the small airways and increases resistance to airflow. In addition to the mechanical effect, obesity is also associated with mild chronic inflammation that may increase the tendency for bronchoconstriction (e.g., elevated leptin and cytokines may promote airway hyperreactivity). In practice, this means that airflow in obese people is somewhat lower, and resistance is higher, especially at low volumes, which normalizes when the lungs are inflated to a higher volume.

Reduced chest wall compliance

Obese individuals have reduced chest wall compliance owing to the accumulation of adipose tissue in the chest wall and abdominal area. Adipose tissue mechanically restricts chest expansion and diaphragmatic movement, resulting in a stiffer (less compliant) wall. Studies show that reduced chest compliance is the main cause of lung volume disorders in obesity, rather than changes in lung compliance itself.

Consequently, greater negative pleural pressure is required for inspiration, which increases the work of respiratory muscles. The impact of obesity on compliance is particularly pronounced in central (abdominal) obesity; individuals with more abdominal fat have a greater reduction in diaphragmatic mobility and lung volume than individuals with peripheral obesity. Reduced chest compliance contributes to the restrictive breathing pattern in the obese.

Obesity and lung function are very complex, and their mutual influence has not yet been fully investigated. These changes are summarized in Figure 1.

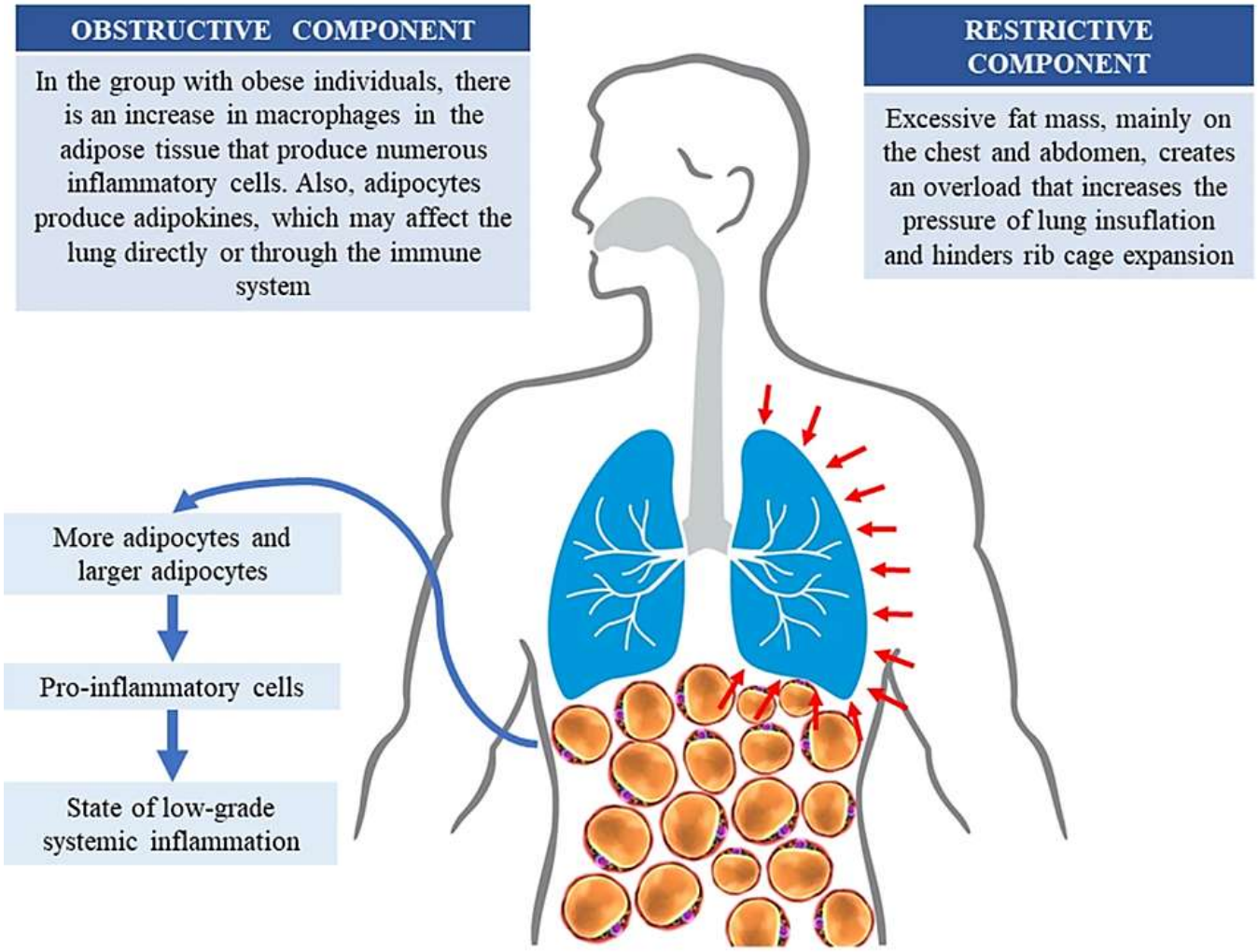

Figure 1: The impact of obesity on the lungs. From reference 10.

Impact of Obesity on Lung Volumes and Capacities

Changes in lung volumes

Obesity causes a reduction in most static lung volumes, consistent with a restrictive disorder. The most prominent changes are a decrease in functional residual capacity (FRC) and expiratory reserve volume (ERV). Due to the lower FRC, the volume at which the lungs normally rest at the end of expiration is also reduced. The residual volume (RV) remains largely unchanged, while vital capacity (VC) and total lung capacity (TLC) are often preserved or only slightly reduced. In many obese individuals, standard spirometric parameters (such as FVC and FEV_1) may remain within normal limits precisely because of the preserved TLC, despite the fact that their lung volumes during rest breathing are reduced. In other words, obesity does not significantly impair maximal lung inflation but significantly reduces the volume of air remaining in the lungs after passive expiration.

Reduced functional residual capacity (FRC)

FRC is the volume of air remaining in the lungs after normal expiration and is significantly reduced in obesity. Abdominal fat pushes the diaphragm upward and shifts the working point of breathing to a lower volume, which reduces FRC. The reduction in FRC is observed even with slight weight gain and is proportionally more pronounced with a higher body mass index. Studies show that overweight (BMI 25–30) reduces FRC by about 10%, moderate obesity (BMI ~30–35) by ~22%, and severe obesity (BMI >40) by up to 33% compared to normal. The decrease in FRC leads to obese people breathing at a volume close to the residual, which reduces the reserve for additional expiration. Consequently, the airways in the basal parts of the lungs can close at the end of expiration because the FRC falls to the level of the bronchiole closing volume. This increases the risk of atelectasis and ventilatory imbalance and represents one of the main mechanisms of respiratory complications in obesity.

Reduced expiratory reserve volume (ERV)

The ERV (the volume that can be exhaled voluntarily after a quiet exhalation) is most affected by obesity. It is often the earliest observed change in lung volumes in obese individuals. As the weight-laden rib cage and diaphragm shift upward, less air is left to exhale after normal expiration. Studies have consistently found a significant decrease in ERV proportional to the degree of obesity. For example, a person with a high BMI may have an ERV that is reduced by more than 50% compared with the normal population. A reduced ERV with a preserved residual volume means that the functional residual capacity is reduced, and the lungs are operating at a lower operating point. Low ERV in obese individuals leads to less flexibility

in adjusting breathing during physical exertion and contributes to the feeling of "running out of air" more quickly. An improvement in ERV is observed after weight loss, which is accompanied by an improvement in FRC.

Impact on total lung capacity (TLC) and vital capacity (VC)

The total lung capacity (TLC) and vital capacity (VC) in obesity are relatively spared compared with FRC and ERV. In mild and moderate degrees of obesity, TLC remains within the normal range or is only slightly reduced. Even in severe obesity, TLC is usually not drastically reduced; a reduction of about 15-20% is expected only in morbid obesity. However, the impact on lung volume is more pronounced in abdominal obesity due to the pressure on the diaphragm. If a significantly reduced TLC is observed in a person with a BMI <40, other causes of restriction (e.g., pulmonary fibrosis) should be considered, as obesity itself rarely causes a large decrease in TLC. Vital capacity (maximum expiration after maximum inspiration) also remained mostly normal or was slightly reduced in obese individuals. FVC and FEV_1 may be slightly lower than predicted because of a lower basal volume, but the FEV_1/FVC ratio remains normal, indicating no obstruction. This suggests that obesity does not significantly limit maximal lung inflation and deflation, but rather primarily reduces the volumes of air present in the lungs during quiet breathing.

Breathing during exercise in obese individuals

Obese individuals have significantly different lung volumes and breathing patterns during exercise than normal-weight individuals. During cyclic exercise, they breathe more rapidly and shallowly, but have a greater inspiratory capacity, allowing them to increase their expiratory volume without limiting their total lung capacity. This breathing pattern may be an adaptation that reduces the elastic work of the lungs. Although dynamic hyperinflation may be observed during exercise, it does not directly affect the sensation of breathlessness but rather represents an adaptation mechanism. These differences are also shown in Figure 2.

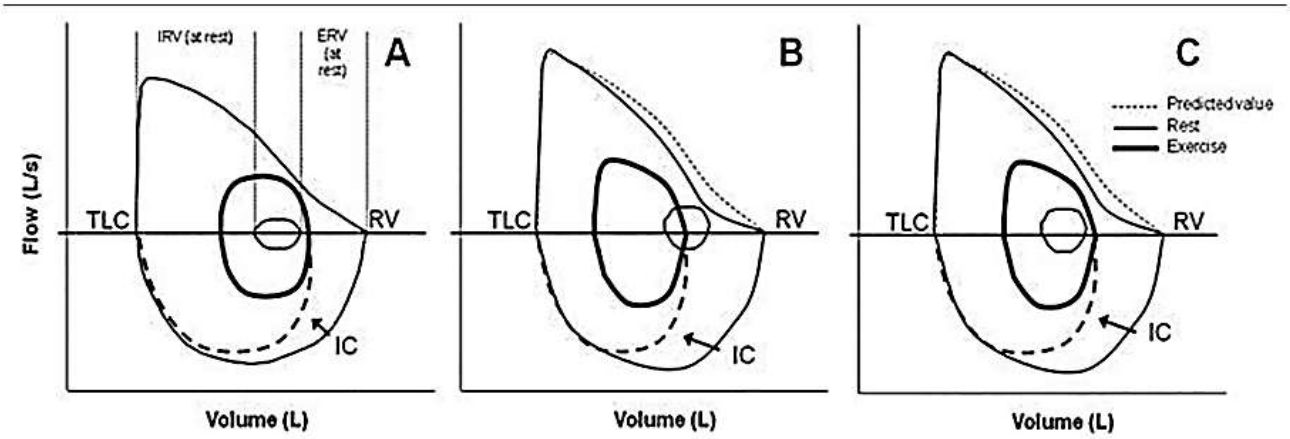

Figure 2: Lung Volume-Flow curves of normal, low and highly obese person. From reference 12.

Effects on Gas Exchange and Ventilation-Perfusion Mismatch

Although pulmonary diffusing capacity (DLCO) remains normal or even elevated in obese individuals (due to increased pulmonary blood flow), obesity can cause mild impairment of blood oxygenation. Reduced FRC and ERV lead to closure of the smaller airways in the basal zones of the lungs during expiration, which creates uneven ventilation of the lungs. The result is a ventilation-perfusion mismatch – the areas of the lung base receive blood (perfusion) but are insufficiently ventilated (ventilated) due to microatelectasis and alveolar closure. As a result, obese patients may have mild hypoxemia at rest, i.e., reduced partial oxygen saturation in arterial blood. Typically, these are small deviations (e.g., a few mmHg lower PaO_2), but in combination with the supine position or lung disease, this may be more pronounced. Also, due to uneven ventilation, there is greater variability in the ventilation-perfusion ratio across lung segments (V/Q mismatch), which is responsible for the aforementioned hypoxemia. In obese individuals without other lung diseases, the diffusion capacity for CO remains preserved or increased, which means that the alveolar-capillary membrane is not damaged; the problem with gas exchange primarily lies in mechanically caused poorer ventilation of parts of the lungs. Weight loss usually improves the uniformity of ventilation and arterial oxygenation.

Comparison of lung function in supine and upright positions in obese individuals

Body position significantly affects lung function, particularly in obese individuals. In the supine position (supination), abdominal contents and adipose tissue push the diaphragm more strongly towards the thoracic cavity, further reducing the FRC. Consequently, FRC and ERV values are lower in the supine position than in sitting or standing, while they are highest in the upright position. Obese patients often experience shortness of breath in the supine position because of this reduction in lung volume and the tendency for the airways to collapse. Interestingly, in the extremely obese (morbidly obese), the effect of position may be reversed, and one study showed that before weight loss, there was no additional drop in FRC when moving from sitting to a supine position (because FRC was already extremely low), whereas after significant weight loss, the expected drop in FRC in the supine position appeared again. This suggests that in extreme obesity, FRC reaches its minimum value already in the upright position, so that position changes have less of an effect, while moderate obesity shows a more pronounced postural effect.

In addition to volume, position also affects the respiratory musculature: in obese individuals, the maximum inspiratory pressure (diaphragmatic strength) in the supine position is significantly lower than in the upright position, which reflects a mechanically less favorable position of the diaphragm and a

greater workload during breathing. All of the above indicate that it is more favorable for obese individuals to breathe in the upright position, while lying down worsens respiratory function; this effect can be partially corrected by losing body weight. Recent scientific publications support these claims and discuss in detail the impact of obesity on lung function, including changes in respiratory mechanics, lung volumes, and gas exchange, as well as the impact of body position.

Role of Weight Loss in Improving Respiratory Function

Effects of weight loss on lung mechanics

Obesity alters respiratory mechanics: excess adipose tissue around the chest and abdomen reduces the overall compliance of the lungs and chest and increases airway resistance, which increases the effort of breathing. Weight loss removes this mechanical burden by improving the elasticity of the respiratory system and reducing the airway resistance. Research confirms that the negative effects of obesity on lung mechanics can be reversed by weight loss. For example, moderate weight loss has been shown to reduce the energy cost of breathing by about 16% in obese individuals, meaning that they need less work from their respiratory muscles to ventilate. Thus, weight loss improves diaphragm and chest function, and breathing becomes more efficient with less effort.

Improved chest compliance

In obese individuals, fat tissue on the trunk physically restricts expansion of the chest and movement of the diaphragm. As a result, their chest compliance is reduced – studies have documented a decrease in chest wall compliance in obesity, leading to a stiffer respiratory system. Weight loss reduces this restriction: with less fat around the lungs, the chest expands more easily during inspiration. Increased chest compliance means that less transpulmonary pressure is required for the same volume, which makes breathing more efficient. This return of compliance to normal improves ventilation – the lungs can fill with air more easily with less resistance. Weight loss normalizes the biomechanics of breathing, subjectively reducing the feeling of “heavy breathing” during exertion.

Increased lung volumes and expiratory flows

Loss of body weight directly improves lung volume and airflow. Many studies have shown a significant increase in static lung volume after weight loss; in particular, total lung capacity (TLC), functional residual capacity (FRC), and expiratory reserve volume (ERV) increase. In parallel, higher expiratory flows are also recorded: forced expiratory volume in 1 second (FEV_1) and peak flow (PEF) increase significantly after weight loss. For example, a recent study found that in men, forced vital capacity (FVC) increases by approximately 1.4% of the predicted value for each 1 kg/m² decrease in BMI. In addition, the FEV_1/FVC ratio improves and airway resistance decreases. The largest increase was observed in FRC, which reflects the release of the restrictive pressure of obesity on the lungs. Overall, weight loss improves ventilation capacity and airflow through the airways, contributing to better respiratory function.

Obesity: Clinical Implications

Obesity-Hypoventilation Syndrome

Obesity Hypoventilation Syndrome (OHS) is defined as a condition in which obesity (BMI > 30kg/m²) is accompanied by chronic hypercapnia ($PaCO_2$ > 45mmHg) and hypoxemia, excluding other causes of respiratory insufficiency. The pathophysiology of OHS is based on mechanical airway restrictions; the increased mass of the chest and abdominal adipose tissue limit the expansion of the lungs, while the reduced mobility of the diaphragm leads to inadequate ventilation. As a result, chronic hypercapnia and hypoxemia occur, which worsens the condition. Leptin resistance, common in obesity, affects the central respiratory drive and further reduces ventilatory response to changes in blood gas concentration. OHS is often associated with obstructive sleep apnea (OSA), where respiratory dysfunction is further exacerbated. The clinical picture includes symptoms such as drowsiness, fatigue, headaches and difficulty breathing, and the risk of developing OHS increases with the severity of obesity. The differential diagnosis must distinguish OHS from chronic obstructive pulmonary disease (COPD) and neuromuscular respiratory disorders, which is crucial for choosing adequate therapy. Long-term consequences of OHS include an increased risk of cardiovascular disease, pulmonary hypertension, and higher mortality rates.

Management of OHS in the clinical setting primarily involves the use of noninvasive ventilation (NIV) strategies, with continuous positive airway pressure (CPAP) and dual positive airway pressure (BiPAP) being the most common modalities. CPAP is often the first-line treatment because of its simplicity and cost-effectiveness, especially in patients with concomitant obstructive sleep apnea (OSA). However, for patients who do not respond adequately to CPAP or do not have OSA, BiPAP may be more effective because it provides additional ventilatory support. Although some studies have shown that both CPAP and NIV improve respiratory function, reduce hypercapnia, and improve quality of life in patients with OHS.

Obesity and Anesthesia

Obese patients present a significant challenge in perioperative care, especially with respect to changes in respiratory function during anesthesia. In the awake state, functional residual capacity (FRC) is reduced owing to the accumulation of adipose tissue around the chest wall and diaphragm. During general anesthesia and muscle relaxation, the FRC decreases further, sometimes to 50% of normal values, leading to alveolar collapse, especially in the basal parts of the lungs. As a result, obese patients quickly become desaturated owing to reduced oxygen stores and increased oxygen consumption. Therefore, preoxygenation is essential; the use of 100% oxygen with prolonged mask ventilation is recommended,

with the upright or "ramped" position significantly increasing the time of safe apnea. This position facilitates laryngoscopy and intubation, allowing good visualization of the vocal cords in morbidly obese patients. During anesthesia, ventilation parameters need to be adjusted: higher PEEP and tidal volumes calculated according to ideal body weight, as well as an increase in respiratory rate to normalize CO_2. Features such as obstructive apnea and hypoventilation syndrome further increase the risks in the postoperative period; therefore, timely identification and therapy, for example, CPAP therapy is crucial for reducing complications. Timely assessment of respiratory function, careful monitoring of parameters, and a multidisciplinary approach enable safer anesthesia flow and a significant reduction in postoperative complications in obese patients.

In summary, anesthetic care for obese patients requires careful preparation: optimization of positioning and oxygenation before induction, application of appropriate ventilation strategies during the procedure (protective ventilation with PEEP), and increased monitoring and respiratory support after awakening. Multidisciplinary collaboration (anesthesiologists, surgeons, and pulmonologists) is important to minimize risks. With these measures, most obese patients can safely undergo anesthesia, although their intrinsic risk of respiratory complications is higher than that of normotensive patients.

Obesity in the ICU: implications for mechanical ventilation

Obese critically ill patients on mechanical ventilation present significant challenges due to altered respiratory biomechanics and the presence of comorbidities. The massive chest wall and heavy abdomen reduce the compliance of the respiratory system, which increases the work of breathing and resistance to diaphragmatic movement. Consequently, higher ventilatory pressures are often required to achieve adequate volume. However, modern approaches focus on protective ventilation to prevent lung damage caused by excessive volume and pressure overload. Adaptations, such as the use of low tidal volumes (approximately 6 ml/kg ideal body weight) and limiting plateau pressure, are used for obese patients, as reduced lung volumes allow for lower tidal volumes. In addition, due to the increased susceptibility to alveolar collapse due to high pleural pressure, higher PEEP (often 10 cmH_2O or more) is typically used in these patients with careful hemodynamic monitoring, as excessive PEEP may reduce venous return. Intermittent recruitment maneuvers may be useful immediately after intubation or during ventilation but are not routinely used because of the potential risk of barotrauma. Thus, the strategy is higher PEEP, low VT for ideal weight, and possibly higher respiratory rate (to ensure pH within limits with low volumes). Optimal positioning (e.g., elevating the head of the bed and avoiding a completely supine position) also aids ventilation and reduces diaphragmatic strain. In very obese patients with ARDS, early use of the

prone position (turning to the stomach) is sometimes resorted to improve oxygenation. Although this is logistically demanding, studies have shown that even very obese patients can benefit from prone ventilation with an experienced team.

Obesity is a significant risk factor for the development of ARDS in critically ill patients, with obese individuals having a 1.5–1.9 times greater risk of developing ARDS/ALI than individuals of normal weight. Abdominal obesity, a proinflammatory state, and the frequent need for larger amounts of fluid contribute to the increased incidence of ARDS in this population. However, the obesity paradox indicates that, despite the higher incidence of ARDS, obese patients with this syndrome show better survival rates, and meta-analyses report lower mortality (OR ~0.74 for patients with BMI 30–39) compared to normal-weight patients. This phenomenon has also been reported in ICU studies, where obese patients on ventilators sometimes have an equal or even better short-term prognosis, despite the challenges in ventilatory management. Possible reasons for this paradox include a specific immune response, in which excess nutritional reserves modulate the catabolic response and more aggressive medical care (earlier intubation and application of higher PEEP). However, it is necessary to individualize ventilation settings to prevent lung damage and ensure adequate oxygenation, with the help of advanced monitoring, such as monitoring of driving pressure, and the application of additional techniques in case of hypercapnia, while avoiding invasive methods such as ECMO unless absolutely necessary.

Conclusion

Obesity is a serious global health problem, with numerous negative effects on the respiratory system. The analyzed text clearly shows that excess adipose tissue, especially in the thoracic and abdominal areas, significantly affects the mechanics of breathing by reducing the flexibility of the lungs and thorax, which results in a decrease in the functional residual capacity (FRC) and expiratory reserve volume (ERV). These mechanical effects, combined with chronic inflammation and changes in adipokine levels, contribute to increased airway resistance and the development of ventilation-perfusion mismatches. The impact of obesity on anesthesia care is also significant, where reduced FRC and increased tendency for alveolar collapse pose a risk of rapid desaturation during the induction of general anesthesia. In this context, the adjustment of ventilation parameters and application of positive end-expiratory pressure (PEEP) are key measures. At the same time, weight loss showed a significant positive effect on improving respiratory function by increasing lung volume and reducing the energy cost of breathing. Therefore, optimal management of respiratory complications in obesity requires multidisciplinary collaboration, including proper anesthesia planning, adaptation of ventilation strategies, and a systematic weight loss program. In addition, research has confirmed that the integration of medical interventions, physical therapy, and nutritional support results in an improved overall quality of life in obese patients. Preventive measures and education regarding a healthy lifestyle are essential to reduce the incidence of respiratory complications and improve lung function in the long term. Therefore, a collaborative approach is required.

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