Submitted: 07 July, 2022 Accepted: 30 August, 2022 Published: 08 September, 2023 DOI:10.22514/sv.2022.076

Open Access
Signa Vitae

REVIEW

Ventilation in critically ill obese patients—Why it should be done differently?

Cristian Cobilinschi[1,2,*], Ana-Maria Cotae[1,2], Raluca Ungureanu[1,2,*], Radu Țincu[1,2], Ioana Marina Grințescu[1,2], Liliana Mirea[1,2]

[1]Carol Davila University of Medicine and Pharmacy, 050474 Bucharest, Romania
[2]Clinical Emergency Hospital Bucharest, 014461 Bucharest, Romania

***Correspondence**
cristian.cobilinschi@umfcd.ro
(Cristian Cobilinschi);
ralucaung@yahoo.com
(Raluca Ungureanu)

Abstract

Considering the increasing prevalence of obesity among the critically ill patient, mechanical ventilation of this type of patients has almost become a daily practice. However, no consensus regarding mechanical ventilation for obese patients has recently been published. Considering the particular pathophysiological features of respiratory system in obese patients, the risk of ventilator induced lung injury (VILI) is highly elevated. This narrative review aims to present the constrains related to mechanical ventilation in obese patients, as well as the features that predispose them to VILI. Moreover, the effects every determinant incriminated in VILI were described with application on the pathophysiological features of obese patients. Increased emphasis was placed on the newly concept of ergotrauma as one of the main determinants of VILI. This is one of the first reviews dedicated to the effects of mechanical power and ergotrauma on mechanically ventilated patients with obesity. Moreover, increased attention was given to the impact of biotrauma related to VILI, considering that the new concept of "pre-conditioning cloud" related to obesity has recently emerged.

Keywords

Obesity; Mechanical ventilation; Ventilator induced lung injury; VILI; Biotrauma; Ergotrauma; Volotrauma; Atelectrauma; Barotrauma

1. Introduction

Since the early 90's, the prevalence of obesity has continuously increased both in Europe and United States. According to the latest World Health Organization (WHO) report obesity affects more than 650 millions of people all over the world becoming a more important cause of morbidity and mortality than starvation [1].

As expected, a higher prevalence of obesity has also been observed among admitted patients in intensive care units (ICU). Although overweight and obese patients admitted to the ICU registered a lower mortality compared to underweight patients, it has been demonstrated that they are more prone to a longer ICU and hospital stay [2–4]. Despite the so-called "obesity paradox of ICU patients" translated by a better prognosis and a higher survival rate of the critically ill obese patients, higher rates of mechanical ventilation on ICU admission were reported along with a greater dependence on mechanical ventilation and an increased risk for infections [2, 4–6]. Moreover, it was reported that obese patients are more likely to develop hypoxemic respiratory disfunction and are more susceptible to undergoing an acute respiratory distress syndrome (ARDS) [6]. Even if there is still insufficient data regarding ICU mortality in obese patients requiring mechanical ventilation, the obesity-related pathophysiological changes make the ventilation very challenging and put these patients at risk for ventilator-induced lung injury (VILI) [5, 7].

Increased lung stress and strain during mechanical ventilation promote the whole set-up for all four mechanisms (barotrauma, volotrauma, atelectrauma and biotrauma) responsible for producing VILI [8].

Despite the increased number of mechanically ventilated obese patients, there is no consensus on recommendations regarding mechanical ventilation in obese patients admitted in the ICU. In this narrative review based on extensive literature search, we summarized the latest data regarding the impact of VILI in critically ill obese patients.

2. Constrains in ventilating obese patients

It has already been demonstrated that obese patients are characterized by a decreased respiratory compliance [9–11]. Specific fat tissue deposition in the mediastinum, abdominal cavities, around the diaphragm and the ribs decrease chest wall compliance [11–13]. According to Lazarus *et al.* [13] chest compliance is mostly affected by central (upper body) distribution of the fat tissue and may be normal in peripheral (lower body) distribution.

Although it may be anticipated that low respiratory compliance is only related to the increased adiposity in the chest wall, numerous studies indicated that this tends to be linked to increased pulmonary blood volume and closure of dependent airways [10, 11]. In other words, low lung compliance results in early closure of the airways at low volumes and atelectasis [14].

Numerous studies indicate that the spirometry and plethysmography measurements in obese patients showed decreased expiratory reserve volume (ERV) and functional residual capacity (FRC) even in initial stages of obesity [11, 12, 15]. Moderate effects or the lack thereof were reported on total lung capacity (TLC) and vital capacity (VC) [14]. Pelosi *et al.* [16] reported since early 90's that these changes make the lungs of obese patients more prone to atelectasis. Decreased ERV and FRC may also be translated into the tendency of obese patients to breathe on a flatter and less efficient part of pressure-volume curve causing an increased work of breathing [17, 18]. Moreover, according to Mafort *et al.* [15] the decreased ERV is further associated with abnormalities in ventilation distribution and alteration of ventilation-perfusion ratio (V/Q). An increased body fat distribution, more than body mass index (BMI), is associated with a slightly decreased forced expiratory volume (FEV_1) and a normal FEV_1/FVC (forced vital capacity) ratio, suggesting an impractical use of these dynamic parameters [11, 12, 19].

Obesity is associated with an increased airway resistance caused by airway narrowing and premature closure, changes that promote bronchial hyperresponsiveness in non-asthmatic patients [15]. Nevertheless, airway narrowing and premature closure are associated with gas trapping which generates intrinsic positive end-expiratory pressure (PEEPi). This in turn contributes to the V/Q mismatch, atelectasis development and impaired oxygenation [12, 14, 20]. PEEPi favours a supplementary load on inspiratory muscles and thus intensifies breathing work [18].

3. Background of ventilator-induced lung injury in obese patients

Apart from the difficulty of managing mechanical ventilation in critically ill patients, often with inhomogeneous lung injuries, the additional obesity-related pathophysiologic changes impede the management of this type of patients. In other words, as already mentioned, the respiratory profile of the obese patients renders the lung susceptible to VILI. Considered one of the most serious complication of mechanical ventilation, VILI includes different pathophysiological mechanisms which in the long run worsen lung injuries [21]:

3.1 Barotrauma and volotrauma

Since publication of the extended research of Dreyfuss *et al.* [22], the main mechanism of VILI was considered to be pressure-related and resulted in pneumothorax, pneumomediastinum or gas embolism [22, 23]. Subsequently, experimental data indicated that excessive lung strain caused by inappropriate tidal volumes (volotrauma) is the hallmark of pathophysiological mechanism of VILI [22–24]. Nowadays it is clear that both mechanisms are in close interaction, considering the fact that stress and strain are related through a proportionality constant represented by the tissue elastance of the lung [21, 23, 25]. In other words, the fundamental pathological mechanism of VILI is considered to be explained through the damage caused by pressure applied directly to the lung tissue (stress) connected to volume change beside the resting volume (strain) [26].

Since the early 2000 experimental evidence was confirmed by large clinical trials, such as the Ventilation with Lower Tidal Volumes as Compared with Traditional Tidal Volumes for Acute Lung Injury and the Acute Respiratory Distress Syndrome—ARMA trial which proved that the use of low tidal volumes and avoidance of excessive lung stress are associated with an increased survival rate [27]. Another more recent trial (A Trial of intraoperative low-tidal-volume ventilation in abdominal surgery—IMPROVE trial) which included 400 patients also demonstrated a higher incidence of pulmonary and extrapulmonary complications in patients ventilated with higher tidal volumes [28].

Guivarch *et al.* [8] demonstrated in his experimental study on obese mechanical ventilated mice with tidal volumes calculated on actual body weight, that inflammation and interstitial oedema may occur after only two hours.

In the face of already proven deleterious effects of volotrauma and despite general recommendations regarding the use of proper tidal volumes according to ideal body weight (IBW), recent data suggest that obese patients are still at high risk for volotrauma occurrence [8]. A secondary analysis of the data resulted from the Intraoperative ventilation settings and their associations with postoperative pulmonary complications in obese patients—LAS VEGAS trial. The latter revealed that obese patients received ventilation with higher tidal volumes than predicted body weight [29]. Jaber *et al.* [30] also reported in his research which included almost 3000 patients that obese patients were ventilated with higher tidal volumes than patients with normal BMI.

3.2 Atelectrauma

Repeated alveolar recruitment and derecruitment is one of the most prominent factor contributing to atelectrauma [31]. The lack of lung homogeneity and the presence of regions with different elasticity amplify stress and strain [21, 32, 33]. As a result, increased shearing forces between aerated and atelectatic areas of the lung may occur, thereby causing atelectrauma [21, 34].

As previously mentioned, respiratory pathophysiological changes like airway narrowing and premature closure, typically identified in obese patients, expand the atelectatic areas promoting the lack of uniformity in the lung [14, 35]. Increased intraabdominal pressure, also present in obese patients, is further associated with lung collapse, atelectasis and increased transpulmonary pressure [36]. It was observed that collapsed lung areas affect neighbouring aerated areas causing deformation and increased stretch [37]. These structural changes eventually promote shear forces exacerbation [37]. So, beside the already existing atelectatic areas, mechanically ventilated patients with obesity are particularly prone to developing at-

electrauma, especially in the absence of adequate ventilation parameters.

In order to prevent lung atelectasis and to minimize the risk of atelectrauma, researchers of the Effect of intraoperative high positive end-expiratory pressure (PEEP) with recruitment maneuvers *vs.* low PEEP on postoperative pulmonary complications in obese patients—PROBESE trial evaluated the effects of protective intraoperative ventilation using different levels of PEEP [38]. Despite expectations, no benefit was obtained in the group ventilated with increased PEEP [38]. Nestler *et al.* [39] demonstrated that an increased PEEP together with recruitment manoeuvres may be beneficial for intraoperative mechanical ventilation of obese patients, but these positive effects were not sustained during the postoperative period. Given these findings, Ball and Pelosi recommend using an increased fraction of inspired oxygen (FiO_2) over PEEP in order to prevent driving pressure increase and hyperinflation [40]. Moreover, these authors suggest that recruitment manoeuvres should only be used as a rescue method in severe hypoxemic dysfunctions [40].

3.3 Mechanical power or "ergotrauma"

Beyond the already incriminated mechanical factors implicated in producing VILI, a recent concept has been advanced through which the mechanical power delivered during mechanical ventilation may be an independent factor for VILI development [41]. If until recently all the physical parameters implicated in mechanical ventilation (pressure, flow, resistance and frequency) were studied as individual data, it is assumed that the combined effects of these parameters should be considered [42]. Gattinoni *et al.* [42] proposed that mechanical power and its determinant factors may be appreciated through a complex equation derived from the equation of motion. Through this mathematical expression the authors could easily determine mechanical power as the result of the product between respiratory frequency and energy, the latter being the sum of the forces which open the lung, push the air into the lung and keep the lung open [25, 42, 43]. The importance of evaluating mechanical power may be useful in the pathogenic process of VILI because repeated tidal energy cycles above the lung tissue tolerance is associated with local structural disruption [43, 44]. Although this concept has recently aroused much interest, there is a lack of practical measurement tools for mechanical power in daily practice [41]. Nevertheless, Marini *et al.* [44] suggest a special consideration when it comes to setting a plateau pressure, or driving pressure and PEEP, underlining therefore that neither is reliable enough to measure the exact amount of energy delivered to the tissue with every cycle.

Considering the novelty of the concept and the lack of studies, both experimental and clinical, specifically targeting the evaluation of the impact of ergotrauma on mechanically ventilated obese patients, its effects are estimated based on the well-known patient-dependent factors.

According to Marini *et al.* [43] one conditional factor that may potentiate the deleterious effects of ergotrauma is represented by chest wall elasticity. Notwithstanding, this means that the energy transferred to the lung may be lower than anticipated due to the spent energy used to overcome the decreased chest wall compliance specific to obese patients [41]. In clinical practice this translates into an elevated threshold for the plateau pressure [41].

As stated above, lung homogeneity in obese patients is usually impaired due to the constitution of numerous atelectatic areas. As stated by the authors of the concept of ergotrauma, mechanical heterogeneity of the lung may predispose to raised regional transpulmonary pressures associated with alteration of dynamic micromechanics and increased shearing forces and stress [43].

Last but not least, it is underlined that a susceptibility of the patient to VILI, which is also the case of the obese patient, may also induce an elevated sensitivity to the harmful effects of mechanical power [43, 45]. Considering that there are no studies regarding the effects of mechanical power on the lungs of obese patients, Ball and Pelosi recommended using the same threshold as in non-obese patients, namely a mechanical power $<$20 J/min [40].

3.4 Biotrauma and the pre-conditioning mechanism

The effects of every injurious ventilator-related stimuli are considered to be biologically translated into an increased inflammatory mediator release and leucocyte recruitment [23, 34]. Beyond the local lung injuries, the systemic release of these inflammatory mediators may by associated with multiple organ failure and even death [37, 46].

The “biotrauma hypothesis” as part of VILI was initially outlined through the results obtained in experimental studies on subjects with ARDS ventilated in a protective manner. A lower level of proinflammatory mediators was also detected in the bronchoalveolar lavage after changing the ventilation strategy and increasing the tidal volume [23, 47]. However, the experimental data of Ricard *et al.* [47] concluded that solely large tidal volumes are not deleterious enough to induce ARDS and a previous lung damage may be mandatory.

The molecular mechanisms underlying biotrauma were mostly highlighted through experimental studies and are summarized in Table 1 [48–50].

The impact of biotrauma in obese patients lacks reliable studies. However, some assumptions may be made considering the data regarding obesity-related immune status.

On the one hand, obesity is associated with a chronic inflammatory state induced by augmented proinflammatory adipocytokines release, decreased anti-inflammatory cytokines and macrophage activation [52]. On the other hand, recent reports indicate that this continuous inflammatory state may offer some protection against further lung injuries, calling this effect as “pre-conditioning cloud” [53]. The concept of preconditioning evolved from the pharmacological process called hormesis and was recently applied in many medical fields including ischemic preconditioning [54]. The “pre-conditioning cloud” in obese patients proposed by Fernandez-Bustamante *et al.* [7] has evolved from the obesity-paradox according to which critically ill obese patients have a decreased rate of mortality [2–4, 53]. Considering that all these hypotheses are based on observations, further research studies are required.

TABLE 1. Molecular mechanisms underlying biotrauma [48–51].

Trigger	Effect
■ proinflammatory mediators release	interleukin (IL)-1, IL-6, IL-8, tumor-necrosis factor (TNF-α), macrophage inflammatory protein (MIP)-2, C-X-C motif ligand 1 (CXCL1) and CXCL10
■ mechanosensation and mechanotransduction	Increased Ca^{2+} and ATP release, voltage-gated Ca^{2+} channels activation, genomic dysregulation
■ complement activation	matrix metalloproteinases activation

ATP: adenosine triphosphate.

4. Some practical aspects

The reported findings suggest that the available recommendation regarding mechanical ventilation for ARDS patients may not fit all groups of patients, such as obese patients [24]. However, there is limited data regarding current practice of mechanical ventilation in critically ill obese patients and most information comes from intraoperative clinical trials [29, 33]. Although ARDS net trial excluded obese patients, this protocol is extensively used for this group of patients [40, 55]. Taking into account that very few studies dedicated to the evaluation of different ventilatory strategies are currently available, an individualized approach should be adopted in obese patients with ARDS [45].

Experts recommend initiation of mechanical ventilation for adopting a protective strategy [40]. Ventilation with low tidal volume (6 mL/kg), titrated according to ideal body weight, may be associated with a more homogenous ventilation of aerated lung [40, 56, 57]. However, if tidal volume is too low in order to avoid overdistension, high PEEP use is recommended in order to avoid atelectasis [32]. Recruitment manoeuvre and PEEP titration are often reported, but their clinical impact is usually lacking [45]. Using electric impedance tomography may offer the possibility for PEEP optimization in obese patients [58].

Jong *et al.* [45] reported that driving pressure may not have an impact on obese patients mortality. Taking into account the elevated chest wall elastance, routinely monitoring oesophageal pressure as a surrogate for transpulmonary pressure should be considered [24, 45]. Since this type of monitoring is not widely available, Ball *et al.* [40] suggest that driving pressure of 17 cmH_2O should be targeted.

5. Conclusions

This review synthetises the latest data available regarding the challenges that we face in managing critically ill obese patients with ARDS. In the absence of clear available guidelines, practical decisions may have to be made based on experts' opinion and on a better understanding of underlying pathophysiology processes.

Although it was observed that obese patients admitted in ICU have a lower mortality rate, a higher risk of prolonged mechanical ventilation should not be neglected together with its associated adverse effects.

This paper review proves that obese patients may have an increased susceptibility to the injuries induced by mechanical ventilation. Considering their high BMI there is a general tendency to use increased tidal volumes predisposing to increased risk of volotrauma and barotrauma. Taking into account the obesity-related pathophysiological changes of the respiratory system, a raised exposure level to the effects of atelectrauma is to be expected. Notwithstanding, considering the difficulties usually met during mechanical ventilation, obese patients may be a perfect target for the effects of the newly described ergotrauma. As obese patients may be the target of all types of ventilator-induced injuries, it should also be expected that they carry an exacerbated inflammatory burden. Since a "preconditioning effect" caused by the chronic inflammatory status is usually seen in obese patients, the impact of biotrauma still remains to be debated.

AVAILABILITY OF DATA AND MATERIALS

Not applicable.

AUTHOR CONTRIBUTIONS

CC, RU—designed the research study. CC—performed the research. AMC, RT and IMG—analyzed the data. CC and LM—wrote the manuscript. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

ACKNOWLEDGMENT

Not applicable.

FUNDING

This research received no external funding.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

[1] WHO. Obesity and overweight. 2021. Available at: https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight (Accessed: 15 September 2020).

[2] Sakr Y, Alhussami I, Nanchal R, Wunderink RG, Pellis T, Wittebole X, *et al*. Being overweight is associated with greater survival in ICU patients. Critical Care Medicine. 2015; 43: 2623–2632.

[3] Hutagalung R, Marques J, Kobylka K, Zeidan M, Kabisch B, Brunkhorst F, *et al*. The obesity paradox in surgical intensive care unit patients. Intensive Care Medicine. 2011; 37: 1793–1799.

[4] Pan J, Shaffer R, Sinno Z, Tyler M, Ghosh J. The obesity paradox in ICU patients. 2017 39th Annual International Conference of the IEEE Engineering in Medicine and Biology Society (EMBC). 2017; 2017: 3360–3364.

[5] Tocalini P, Vicente A, Amoza RL, García Reid C, Cura AJ, Tozzi WA, *et al*. Association between obesity and mortality in adult patients receiving invasive mechanical ventilation: a systematic review and meta-analysis. Medicina Intensiva. 2020; 44: 18–26.

[6] M. N. Gong, E. K. Bajwa, B. T. Thompson, D. C. Christiani. Body mass index is associated with the development of acute respiratory distress syndrome. Thorax. 2010; 65: 44–50.

[7] Fernandez-Bustamante A, Hashimoto S, Serpa Neto A, Moine P, Vidal Melo MF, Repine JE. Perioperative lung protective ventilation in obese patients. BMC Anesthesiology. 2015; 15: 56.

[8] Guivarch E, Voiriot G, Rouzé A, Kerbrat S, Tran Van Nhieu J, Montravers P, *et al*. Pulmonary effects of adjusting tidal volume to actual or ideal body weight in ventilated obese mice. Scientific Reports. 2018; 8: 6439.

[9] Tomescu DR, Popescu M, Dima SO, Bacalbaşa N, Bubenek-Turconi Ş. Obesity is associated with decreased lung compliance and hypercapnia during robotic assisted surgery. Journal of Clinical Monitoring and Computing. 2017; 31: 85–92.

[10] J. Porhomayon, P. Papadakos, A. Singh, N. D. Nader. Alteration in respiratory physiology in obesity for anesthesia-critical care physician. HSR Proceedings in Intensive Care & Cardiovascular Anesthesia. 201; 3: 109–118.

[11] Parameswaran K, Todd DC, Soth M. Altered respiratory physiology in obesity. Canadian Respiratory Journal. 2006; 13: 203–210.

[12] U. Peters, A. E. Dixon. Obesity and Lung Compliance. Expert Review of Respiratory Medicine. 2019; 12: 755–767.

[13] Lazarus R, Sparrow D, Weiss ST. Effects of obesity and fat distribution on ventilatory function. Chest. 1997; 111: 891–898.

[14] A. H. Gifford, J. C. Leiter, and H. L. Manning. Respiratory function in an obese patient with sleep-disordered breathing. Chest. 2010; 138: 704–715.

[15] Mafort TT, Rufino R, Costa CH, Lopes AJ. Obesity: systemic and pulmonary complications, biochemical abnormalities, and impairment of lung function. Multidisciplinary Respiratory Medicine. 2016; 11: 28.

[16] Pelosi P, Croci M, Ravagnan I, Cerisara M, Vicardi P, Lissoni A, *et al.* Respiratory system mechanics in sedated, paralyzed, morbidly obese patients. Journal of Applied Physiology. 1997; 82: 811–818.

[17] A. Banerjee, E. Heiden. Obesity and the effects on the respiratory system. Practical Guide to Obesity Medicine. 2018; 109–121.

[18] C. K. Lin, C. C. Lin. Work of breathing and respiratory drive in obesity. Respirology. 2012; 17: 402–411.

[19] Fischer AJ, Kaese S, Lebiedz P. Management of obese patients with respiratory failure—a practical approach to a health care issue of increasing significance. Respiratory Medicine. 2016; 117: 174–178.

[20] J. L. Pépin, J. F. Timsit, R. Tamisier, J. C. Borel, P. Lévy, S. Jaber. Prevention and care of respiratory failure in obese patients. The Lancet. Respiratory Medicine. 2016; 4: 407–418.

[21] Tonetti T, Vasques F, Rapetti F, Maiolo G, Collino F, Romitti F, *et al.* A driving pressure and mechanical power: new targets for VILI prevention. Annals of Translational Medicine. 2017; 5: 1–10.

[22] D. Dreyfuss, G. Saumon. State of the Art Ventilator-induced Lung Injury. American Journal of Respiratory and Critical Care Medicine. 1998; 157: 294–323.

[23] de Prost N, Ricard J, Saumon G, Dreyfuss D. Ventilator-induced lung injury: historical perspectives and clinical implications. Annals of Intensive Care. 2011; 1: 28.

[24] F. S. L. Vianna, F. J. Do Amaral Pfeilsticker, A. S. Neto. Driving pressure in obese patients with acute respiratory distress syndrome: one size fits all? Journal of Thoracic Disease. 2018; 10: S3957–S3960.

[25] Gattinoni L, Marini JJ, Collino F, Maiolo G, Rapetti F, Tonetti T, *et al.* The future of mechanical ventilation: Lessons from the present and the past. Critical Care. 2017; 21: 1–11.

[26] Marini JJ, Rocco PRM, Gattinoni L. Static, Dynamic contributors to ventilator-induced lung injury in clinical practice. Pressure, Energy, and Power. American Journal of Respiratory and Critical Care Medicine. 2020; 201: 767–774.

[27] R. G. Brower, M. A. Matthay, A. Morris, D. Schoenfeld, B. T. Thompson, A. Wheeler. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The New England Journal of Medicine. 2000; 342: 1301–1308.

[28] Futier E, Constantin J, Paugam-Burtz C, Pascal J, Eurin M, Neuschwander A, *et al*. A Trial of intraoperative low-tidal-volume ventilation in abdominal surgery. New England Journal of Medicine. 2013; 369: 428–437.

[29] Ball L, Hemmes SNT, Serpa Neto A, Bluth T, Canet J, Hiesmayr M, *et al.* Intraoperative ventilation settings and their associations with postoperative pulmonary complications in obese patients. British Journal of Anaesthesia. 2018; 121: 899–908.

[30] Jaber S, Coisel Y, Chanques G, Futier E, Constantin J-, Michelet P, *et al*. A multicentre observational study of intra-operative ventilatory management during general anaesthesia: tidal volumes and relation to body weight. Anaesthesia. 2012; 67: 999–1008.

[31] Z. L. Chen, Y. L. Song, Z. Y. Hu, S. Zhang, Y. Z. Chen. An estimation of mechanical stress on alveolar walls during repetitive alveolar reopening and closure. Journal of applied physiology. 2015; 119: 190–201.

[32] Gattinoni L, Quintel M, Marini JJ. Volutrauma and atelectrauma: which is worse? Critical Care. 2018; 22: 264.

[33] Grieco DL, Russo A, Romanò B, Anzellotti GM, Ciocchetti P, Torrini F, *et al*. Lung volumes, respiratory mechanics and dynamic strain during general anaesthesia. British Journal of Anaesthesia. 2018; 121: 1156–1165.

[34] Leong R, Marks JA, Cereda M. How does mechanical ventilation damage lungs? what can be done to prevent it? Evidence-Based Practice of Critical Care. 2020; 68–73.e1.

[35] Reinius H, Jonsson L, Gustafsson S, Sundbom M, Duvernoy O, Pelosi P, *et al*. Prevention of atelectasis in morbidly obese patients during general anesthesia and paralysis. Anesthesiology. 2009; 111: 979–987.

[36] Kubiak BD, Gatto LA, Jimenez EJ, Silva-Parra H, Snyder KP, Vieau CJ, *et al.* Plateau and transpulmonary pressure with elevated intra-abdominal pressure or atelectasis. The Journal of Surgical Research. 2010; 159: e17–e24.

[37] J. R. Beitler, A. Malhotra, B. T. Thompson. Ventilator-induced lung injury. Clinics in Chest Medicine. 2016; 37: 633–646.

[38] Bluth T, Serpa Neto A, Schultz MJ, Pelosi P, Gama de Abreu M. Effect of intraoperative high positive end-expiratory pressure (PEEP) with recruitment maneuvers *vs.* low PEEP on postoperative pulmonary complications in obese patients. JAMA. 2019; 321: 2292.

[39] Nestler C, Simon P, Petroff D, Hammermüller S, Kamrath D, Wolf S, *et al*. Individualized positive end-expiratory pressure in obese patients during general anaesthesia: a randomized controlled clinical trial using electrical impedance tomography. British Journal of Anaesthesia. 2017; 119: 1194–1205.

[40] L. Ball, P. Pelosi. How I ventilate an obese patient. Critical Care. 2019; 23: 1–3.

[41] Cruz FF, Ball L, Rocco PRM, Pelosi P. Ventilator-induced lung injury during controlled ventilation in patients with acute respiratory distress syndrome: less is probably better. Expert Review of Respiratory Medicine. 2018; 12: 403–414.

[42] Gattinoni L, Tonetti T, Cressoni M, Cadringher P, Herrmann P, Moerer O, *et al*. Ventilator-related causes of lung injury: the mechanical power. Intensive Care Medicine. 2016; 42: 1567–1575.

[43] Marini JJ. Dissipation of energy during the respiratory cycle. Current Opinion in Critical Care. 2018; 24: 16–22.

[44] J. J. Marini, P. R. M. Rocco. Which component of mechanical power is most important in causing VILI? Critical Care. 2020; 24: 1–3.

[45] A. De Jong, D. Verzilli, S. Jaber. ARDS in obese patients: specificities and management. Critical Care. 2019; 23; 1–9.

[46] G. F. Curley, J. G. Laffey, H. Zhang, A. S. Slutsky. Biotrauma and ventilator-induced lung injury: clinical implications. Chest. 2016; 150: 1109–1117.

[47] Ricard J, Dreyfuss D, Saumon G. Production of inflammatory cytokines in ventilator-induced lung injury: a reappraisal. American Journal of Respiratory and Critical Care Medicine. 2001; 163: 1176–1180.

[48] P. M. Spieth, T. Bluth, M. G. De Abreu, A. Bacelis, A. E. Goetz, R. Kiefmann. Mechanotransduction in the lungs. Minerva Anestesiol. 2014; 80: 933–941.

[49] Chen L, Xia H, Shang Y, Yao S. Molecular mechanisms of ventilator-induced lung injury. Chinese Medical Journal. 2018; 131: 1225–1231.

[50] Takahashi K, Saha D, Shattino I, Pavlov VI, Stahl GL, Finnegan P, *et al.* Complement 3 is involved with ventilator-induced lung injury. International Immunopharmacology. 2011; 12: 2138–2143.

[51] Matthay MA, Bhattacharya S, Gaver D, Ware LB, Lim LHK, Syrkina O, *et al*. Ventilator-induced lung injury: *in vivo* and *in vitro* mechanisms. American Journal of Physiology-Lung Cellular and Molecular Physiology. 2002; 283: L678–L682.

[52] Wang C. Obesity, inflammation, and lung injury (OILI): the good. Mediators of Inflammation. 2014; 2014: 1–15.

[53] Bustamante AF. Adipose-lung cell crosstalk in the obesity-ARDS paradox. Journal of Pulmonary & Respiratory Medicine. 2013; 3: 144.

[54] Calabrese EJ. Preconditioning is hormesis part II: how the conditioning dose mediates protection: dose optimization within temporal and mechanistic frameworks. Pharmacological Research. 2016; 110: 265–275.

[55] ARDSnet. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The New England journal of medicine. 2000; 342: 1301–1308.

[56] Samary CS, Santos RS, Santos CL, Felix NS, Bentes M, Barboza T, *et al.* Biological impact of transpulmonary driving pressure in experimental acute respiratory distress syndrome. Anesthesiology. 2015; 123: 423–433.

[57] A. De Jong, G. Chanques, S. Jaber. Mechanical ventilation in obese ICU patients: from intubation to extubation. Critical Care. 2017; 21: 1–8.

[58] K. Erlandsson, H. Odenstedt, S. Lundin, O. Stenqvist. Positive end-expiratory pressure optimization using electric impedance tomography in morbidly obese patients during laparoscopic gastric bypass surgery. Acta Anaesthesiologica Scandinavica. 2006; 50: 833–839.

How to cite this article: Cristian Cobilinschi, Ana-Maria Cotae, Raluca Ungureanu, Radu Țincu, Ioana Marina Grințescu, Liliana Mirea. Ventilation in critically ill obese patients—Why it should be done differently? Signa Vitae. 2023; 19(5): 23-28. doi: 10.22514/sv.2022.076.