Ventilatory Mechanics in the Patient with Obesity

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Obesity is a pathologic increase in the body adipose tissue that is associated with an augmented incidence of chronic health-threatening conditions, such as diabetes, cardiovascular diseases, and cancer.[1] In 2015, 12% of the world adult population and 5% of the world pediatric population were obese, with the highest prevalence among women aged 60 to 64 yr living in high-income countries.[2] Since 1980, the incidence of obesity has globally increased across all age classes and sociodemographic levels.[2] The higher rates of this increase were observed among children. In the United States, one of the countries most affected by the "obesity pandemic," the prevalence is approaching 40% among adults, 20% among adolescents, and 14% among children, and it is higher in women, Hispanic whites, and black Americans.[3]

In the adult, obesity is classically defined as a body mass index greater than 30 kg/m^2 (normal range: 20 to 25 kg/m^2, with subjects between 26 and 30 considered overweight). Body mass index is the weight (expressed in kilograms) divided by the square of height (expressed in meters). The body mass index is easy to use, and high values have been shown to correlate with an increased incidence of comorbidities.[1] However, there is only an indirect correlation between body mass index and total body fat, and this correlation is variable among different ethnicities.[4] Body mass index expresses an excess in body weight, and factors other than the overall body fat, such as the muscular, bone, and connective tissue mass, can influence weight.

Moreover, the distribution of the excessive adipose tissue, as well as its absolute amount, should be considered when evaluating the detrimental effects of obesity. For example, the amount and distribution of body fat show some sex-related differences: men have less adipose tissue than women for the same body mass index,[5] but are more likely to be affected by central obesity, where the adipose tissue accumulates around visceral organs, particularly in the abdominal cavity, whereas women follow more often a gynoid pattern, with fat accumulation around the hips and the proximal extremities.[6] The central subtype has been linked to a higher cardiovascular risk than gynoid subtype.[7] In this context, complementary tools to classify obesity could help to stratify the clinical risk. For example, waist circumference has been pointed out as a marker of central obesity, with higher values predicting a greater cardiovascular risk for the same body mass index.[8] This concept would be particularly useful to the field of anesthesia since what impairs respiratory physiology is the visceral fat pushing against the diaphragmatic muscle, causing a reduction in expiratory reserve and decreased ventilation/perfusion ratio[9,10] (see fig. 1 and following paragraph). However, except for a few retrospective studies underlying the contribution of waist circumference, rather than absolute body mass index, to worsened perioperative outcomes,[11,12] current literature in anesthesia and intensive care still rely on body mass index to identify and categorize obesity.[13] This focused review will discuss how abdominal fat influences airway management and the mechanics of respiration during spontaneous breathing and artificial ventilation. On the other hand, specific topics such as use of neuromuscular blocking agents, extracorporeal lung support, and tracheostomy will not be covered.

Mechanics during Spontaneous Breathing

In humans, the majority of the adipose tissue is distributed in the subcutaneous space, particularly in the abdomen; as the body mass index increases, both the subcutaneous and the visceral adipose components show a tendency to increase.[14] Abdominal fat can be thought of as a liquid mass influencing the pressures in the ventral coelom cavity. The interplay between the increased abdominal pressure and the elastic structures in the thoracic space, such as the lungs, has been extensively studied. In obesity, the cephalic displacement of the diaphragm by the abdominal fat affects the lung volumes, producing a restrictive pattern whose hallmark is the reduction in the functional residual capacity (FRC) and in the expiratory reserve volume[15–17] (fig. 1). The more significant decreases in FRC and expiratory reserve volumes are observed for mild increments of body mass index (between 25 and 35 of body mass index, corresponding to overweight and class I obesity[15]), and are accompanied by a reduction in total lung capacity and vital capacity, but not in inspiratory capacity and residual volume.[17] In most of the cases, the ratio between the forced expiratory volume during the first second and the forced vital capacity (forced expiratory volume during the first second/forced vital capacity) is preserved.[15,17] However, the behavior of the respiratory system changes when a subject with obesity transitions from sitting to the supine position. In the flat situation, there is no further reduction in FRC and

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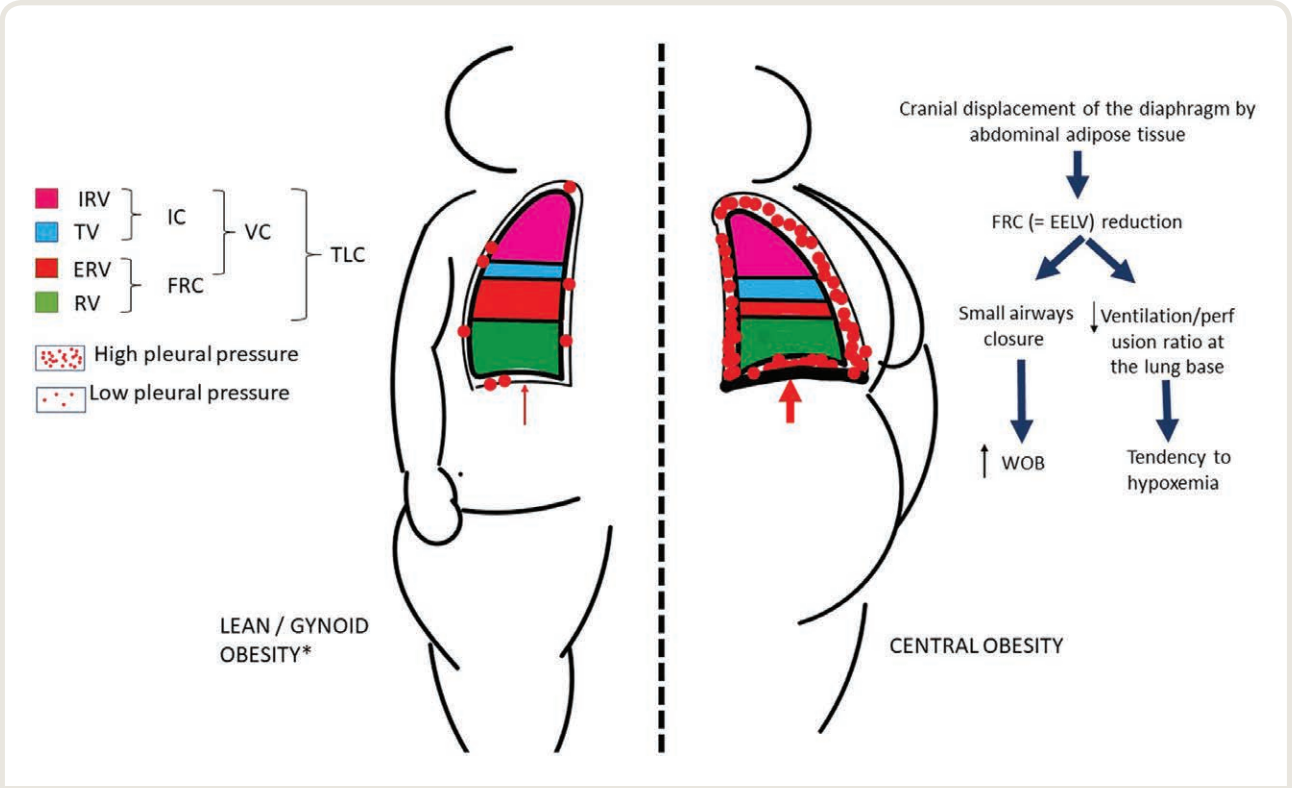

Fig. 1. Changes induced by central obesity during spontaneous ventilation in upright position. Differences in classical lung volumes between the lean and the obese patient. Expiratory reserve volume (ERV) is greatly reduced in the obese, leading to a reduction of functional residual capacity (FRC) and total lung capacity (TLC). Residual volume (RV) is unchanged. Tidal volume (TV) and inspiratory capacity (IC) are only slightly reduced. The main mechanism for this pattern is the cephalic displacement of the diaphragm by the abdominal content *(thick red arrow* on the "obese" side), which leads to an increase in pleural pressure (*high-density red spots*, as compared to *scarce red spots* on the "lean/gynoid obesity" side). *Intended as a pattern where adipose tissue distributes mainly around the hips and the proximal extremities, whereas abdominal fat is relatively lower. EELV, end-expiratory lung volume; IRV, inspiratory reserve volume; VC, vital capacity; WOB, work of breathing.

expiratory reserve volume, contrary to what is observed in the lean person, indicating that, when standing, people with obesity are already breathing near their residual volume. Instead, there is a significant increase in airway resistance, with consequent limitation in the expiratory flow and the development of intrinsic positive end-expiratory pressure (PEEP).[16] Intrinsic PEEP results in an increase in the work of breathing.[18] Hence, in a subject with obesity lying supine, expiratory flow limitation and consequent air trapping at low FRC become the main feature, with the respiratory pattern converting from simple restrictive to mixed restrictive-obstructive. This phenomenon has not been fully elucidated. The person with obesity breathes at very low lung volumes, and low lung volumes are known to be associated with expiratory flow limitation since elastic recoil is a determinant of the airway's caliber.[19] On the other hand, in the upright position, the increased elastance observed in the obese lung is able to compensate for the reduction in FRC and the increased airway resistance, preserving expiratory flows until the expiratory reserve volume is obliterated.[20] It is possible that once supine, this labile compensation is altered by other factors that further contribute to airway closure, such as an increased intrathoracic blood volume or the occlusion of the upper airways by fat loading, with the resulting flow limitation and air trapping.[16] Some of the changes observed in the supine position are illustrated in figure 2.

Mechanics during the Periinduction Period

Breathing at low respiratory volumes impacts gas exchange, in particular oxygenation, which worsens as expiratory reserve volume approaches the residual volume.[21] Indeed, an impaired expiratory capacity seems to be the primary determinant of a decreased ventilation/perfusion ratio leading to shunting and hypoxemia. Redistribution of ventilation toward the apices with the bulk of perfusion delivered to the bases has been observed in spontaneously breathing obese patients at the end of exhalation, especially in those whose expiratory reserve volume was reduced less than 300 ml.[22] The hyperperfusion of poorly ventilated alveolar units in the most dependent parts of the lungs could explain the increased alveolar to the arterial ratio for oxygen and the consequent hypoxemia in otherwise healthy obese

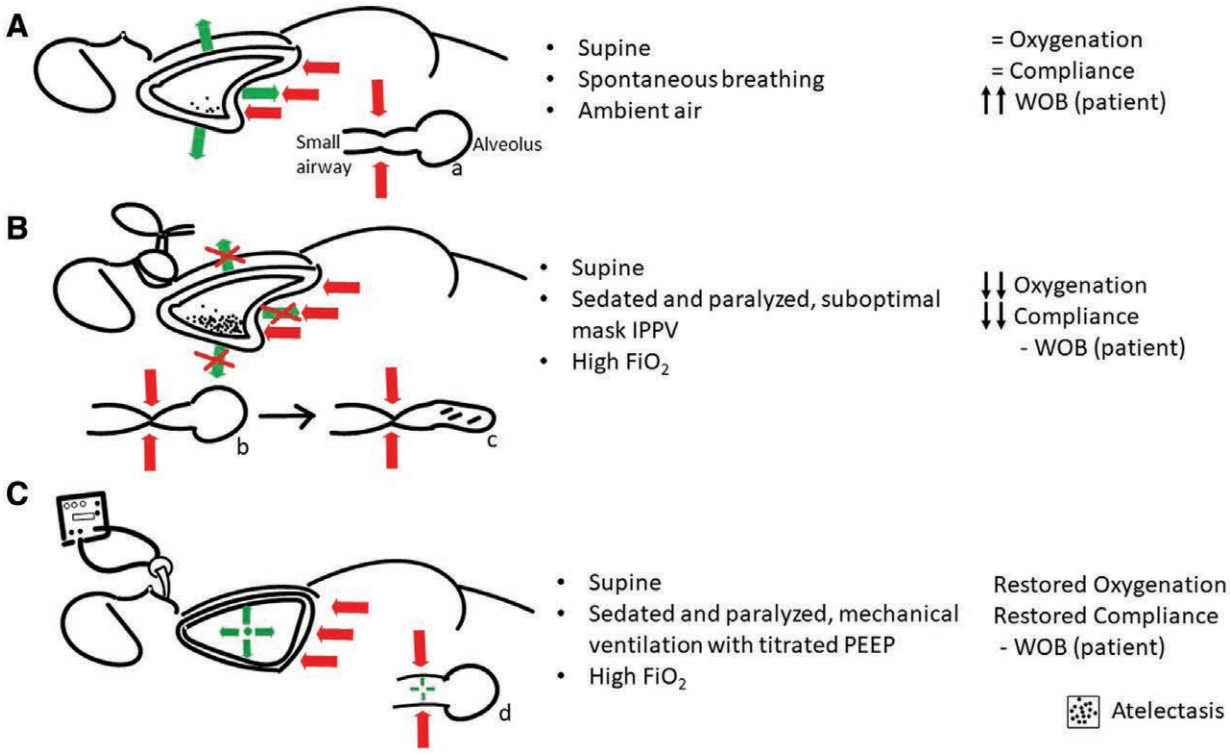

Fig. 2. Changes induced by central obesity in supine position during spontaneous breathing, sedation and paralysis, and mechanical ventilation. (*A*) The patient is actively breathing. Displacement of the diaphragm by a high abdominal load (*horizontal red arrows*) leads to high pleural pressure and lung volumes reduction with consequent narrowing of the small airways (*red arrows* in subpanel *a*, representing a partially collapsed airway-alveolar unit). Airway collapse at low lung volume is partially counteracted by the contraction of the inspiratory muscles (*green arrows*), which lowers pleural pressure, at the cost of a high work of breathing produced by the patient (work of breathing [WOB] patient). (*B*) The patient is sedated and paralyzed, with suboptimal manual intermittent positive pressure ventilation (IPPV). Paralysis prevents inspiratory muscular contraction (*red crosses on the green arrows*) and pronounced total collapse of the small airways predominates (subpanel *b*). Concomitant administration of a high fraction of inspired oxygen (F_{IO_2}) results in reabsorption atelectasis in underventilated alveolar units (subpanel *c*), with further reductions in expiratory lung volumes, deterioration of compliance, and shunting leading to hypoxemia. (*C*) Mechanical ventilation with a titrated level of positive end-expiratory pressure (PEEP) counteracts small airways collapse (subpanel *d*), restoring lung volumes and, consequently, lung mechanics and oxygenation.

individuals. Of note, the decreased ventilation/perfusion ratio improves after weight loss after bariatric surgery, probably reflecting a restoration in end-expiratory volumes.[10]

Moreover, obesity may be associated with hypoventilation, and hence hypercapnia.[23] As a result, the patient with obesity may present to the preinduction period with blood gases values already altered. In addition, the mass loading by cervical fat facilitates the collapse of the upper airways,[24] and a high body mass index is a predictive factor for difficult mask ventilation.[25] Last, but not least important, the decrease in functional respiratory capacity translates into a lower volume available for denitrogenation (time to safe apnea), decreasing the safe apnea time with faster desaturation during laryngoscopy, similar to what happens in children.[26] In this setting, the importance of providing adequate oxygenation before intubation is emphasized. When technically feasible, the best way to deliver oxygen to the patient with obesity during elective intubation is by providing pressure support and PEEP utilizing noninvasive positive pressure ventilation.[27] Noninvasive positive pressure ventilation is particularly helpful since it recruits alveolar units closed at low values of FRC and prevents atelectasis after induction of general anesthesia, an occurrence particularly relevant in the obese.[28] By maintaining alveolar recruitment, positive pressure at end inspiration increases the nonhypoxic apnea time during the induction maneuvers.[29] It has been suggested to keep a 25° head position while delivering preoxygenation.[30] The combination of upright positions and positive airway pressure should be effective in improving the expiratory volume available for denitrogenation.

There is no consensus about the proper fraction of oxygen to deliver. A high percentage of inspired oxygen (greater than 80%) would denitrogenate the functional residual capacity faster and more efficiently but at the cost

of reabsorption atelectasis formation during the intubation time, with consequent rebound hypoxemia.[31]

On the other hand, lower inspired oxygen (as low as 60%) prevents atelectasis but is associated with a shorter safe apnea time.[31] The problem of atelectasis after preoxygenation is accentuated in patients with obesity since the mechanism is reabsorption in alveoli upstream from a closed airway, and closed airways are at the center of obese respiratory pathophysiology, as assessed in the preceding paragraph (see also fig. 2B). As noted above, however, this could yield a very short time for laryngoscopy before desaturation starts. A good practical compromise would be to target inspired oxygen of 80%, to limit atelectasis formation while not shortening safe apnea time too much.

Contrasting data exist about laryngoscopy in the population with obesity. The incidence of difficult airway management appears to be higher in the intensive care unit,[32] most likely because of the urgency of most of the cases, while during elective surgery, the body mass index is a poor indicator of difficult endotracheal intubation.[25] However, as the concept of central obesity is a predictor of comorbidities more accurate than the body mass index itself, so the distribution of cervical fat should be taken into consideration when evaluating a possible difficult airway. In this regard, an index such as a neck circumference greater than 42 cm should be complementary to the body mass index.[33] When attempting a direct laryngoscopy, cushions and blankets should be put under the patient's head and neck, to achieve a "ramped position."[34] Alternatively, the head can be elevated between 25° and 40° by tilting the bariatric table. These approaches facilitate the alignment of oral, pharyngeal, and laryngeal axes, simplifying laryngoscopy. If facing coexisting predictors of both difficult intubation and mask ventilation (body mass index greater than 50 kg/m^2, neck circumference greater than 42 cm, male sex, age greater than 45 y, poor dentition, a thyromental distance of less than 6 cm, the presence of a beard, and so forth), video laryngoscopy or awake fiberoptic intubation should be considered. Table 1 summarizes maneuvers that can help improve the management of the obese patient before induction.

Mechanics during Artificial Ventilation without Lung Injury

Sedation and use of neuromuscular blocking agents make it feasible to study the intrinsic mechanical characteristics of the respiratory system. By coupling the measurements of the airway pressure (P_{aw}) with those given by esophageal pressure (a surrogate for pleural pressure), the transmural pressure distending the lung parenchyma is obtained (transpulmonary pressure = P_{aw} – pleural pressure, approximated as transpulmonary pressure = P_{aw} – esophageal pressure). Thus, one could explore the relative contribution of the lung and the chest wall to the global respiratory system alteration in the population with obesity. Obesity is characterized by an increased respiratory system elastance, and the major contributor to this increase is the lung, while the elastic properties of the chest wall are less affected or substantially unchanged.[35–38] The abdominal load is associated with an elevated esophageal pressure (pleural pressure), which in turn results in negative transpulmonary pressure, especially at the end of exhalation. Indeed, the subject with obesity might spend most of his respiratory cycle (inspiration and exhalation) below the threshold for positive transpulmonary pressure, even when mechanically ventilated with positive pressures, and this results in lung collapse and reduced volumes at FRC.[39] Lung collapse produces increased lung elastance and impacts gas exchange, with the development of hypoxemia. A direct consequence of this mechanism is that an artificially ventilated obese subject requires higher levels of airway pressure to keep the lung open, especially during exhalation. In subjects with high body mass index

Table 1. Management during Preinduction Period

Pathologic Mechanisms	Consequence	Phase	Interventions
(1) Preexisting hypoxemia due to ventilation-perfusion mismatch (2) ↓FRC →↓EELV available for denitrogenation (3) Collapse of the upper airways	Rapid desaturation during even short periods of apnea	Preinduction	• Positioning in reverse Trendelenburg • Use of noninvasive positive pressure ventilation while delivering preoxygenation • Use of high fractions of inspired oxygen (between 85 and 100%; 100% if difficult laryngoscopy predicted) • Monitor for end-tidal oxygen >80% or end-tidal nitrogen <5%
		Ventilation after induction	Consider obese patients as full stomach. Rapid sequence intubation with succinylcholine or high-dose rocuronium allows fast securing of the airway avoiding difficult mask ventilation. Sellick maneuver (cricoid pressure to occlude the esophagus) can be applied.
		Intubation	• Return in supine position but with the patient previously fixed in ramp position or attempt laryngoscopy directly in reverse Trendelenburg • When available and if confident with the technique, use directly videolaryngoscopy • Laryngeal masks as a rescue device should always be ready for use • When coexisting factors of difficult intubation, consider awake intubation with flexible fibroscopy

EELV, end-expiratory lung volume; FRC, functional residual capacity.

undergoing general anesthesia, a higher level of end-expiratory pressure is associated with positive transpulmonary pressure and higher compliance of the respiratory system, although PEEP alone is not able to restore oxygenation.[40] The best approach to restore both lung mechanics and gas exchange seems to be to associate a higher titrated level of PEEP with the performance of recruitment maneuvers.[41] In critically ill patients with obesity, recruitment maneuvers (performed through a stepwise increment of end-expiratory airway pressure) associated with PEEP tailored on the best compliance or a positive transpulmonary pressure at end expiration results in higher end-expiratory volumes and in an improved elastance of the lung, with a consequent positive impact on oxygenation.[37] By restoring a positive transpulmonary pressure, this approach ameliorates the gas distribution in the lung, without significant hemodynamic drawbacks.[38] Despite these strong physiologic premises, a recent large randomized control trial that compared a low level of PEEP (4 cm H_2O) with a strategy providing regular recruitment maneuvers and a PEEP of 12 cm H_2O to obese patients undergoing general anesthesia failed to demonstrate a benefit in terms of clinical outcomes (*i.e.,* incidence of postoperative complications).[42] A possible explanation for such surprising results could be that obese patients require a PEEP titrated based on their specific respiratory characteristics rather than a fixed predetermined value of "high PEEP." Up to now, no level of body mass index predicts PEEP requirements.

The use of transpulmonary pressure to titrate lung mechanics has limitations. First, as stated above, esophageal pressure is a surrogate for the actual pressure in the pleural cavity, which is prohibitive to measure in a human being. As a surrogate, absolute values are different and generally overestimated by at least 3 cm H_2O in the general population.[43] Second, esophageal pressure reflects better pleural pressure at the level of the esophagus or in the more dependent lung, while less can be inferred about nondependent pleural pressure.[44] Furthermore, as of today, there is no available knowledge about the distribution of pleural pressure in morbid obesity, and it is unknown if the gradient between the dependent and nondependent lung is small (as in a lean subject with healthy lungs), or if it is significantly higher (as in adult respiratory distress syndrome [ARDS]) due to atelectasis and changes in lung geometry (fig. 3). In the presence of this gradient, absolute values of a set transpulmonary pressure could be higher than those calculated with esophageal manometry, especially in the nondependent lung, thus resulting in overdistension. Figure 3 illustrates some aspects

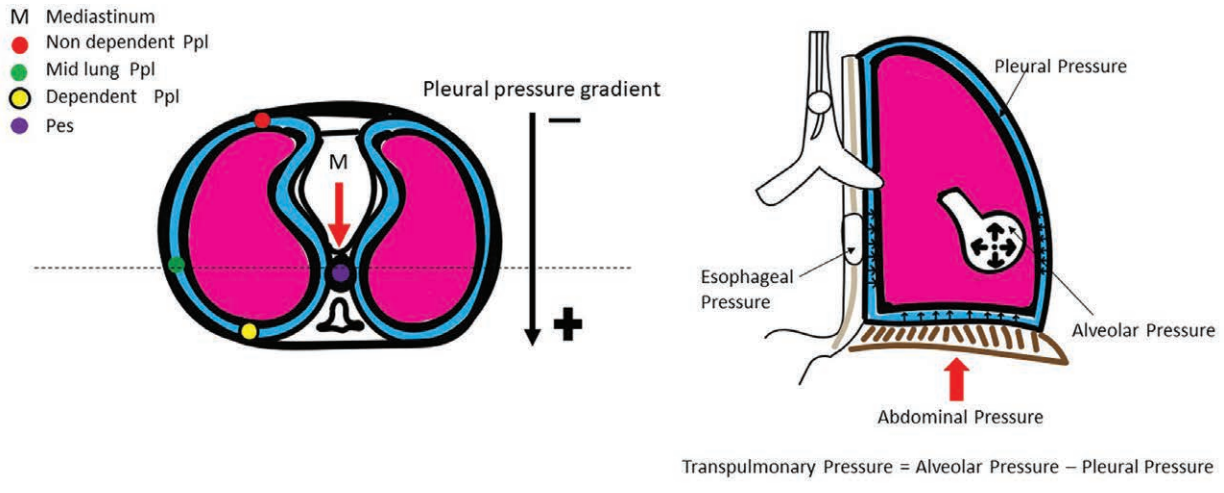

Fig. 3. Role of esophageal manometry in the assessment of transpulmonary pressure. (*A*) A supine model, as it could be seen in a chest computed tomography scan, is shown. The pleural space, in *blue*, is subdivided into three zones (nondependent space, in *red*; the midlung space, in *green*; and the dependent space, in *yellow*). The esophagus, with the esophageal balloon in place, is represented by the *violet mark*. From classic physiology, it is known that the pleural pressure in the dependent pleura is around 2 cm H_2O higher than the one in the nondependent space so that a gradient exists (*vertical black arrow*). Theoretically, esophageal pressure (Pes) is directly exposed to the pleural space and reflects pleural pressure at its same gravitational level (*dotted line*), but the absolute value could be overestimated due to compression by the mediastinum (M) on the esophagus, intrinsic tone of the esophageal musculature, esophageal content, and intrinsic tone of the esophageal balloon. In conditions like adult respiratory distress syndrome, high superimposed pressure caused by inflammatory edema increases this gradient, so that Pes is likely to significantly overestimate nondependent pleural pressure. The entity of this gradient is unknown in morbid obesity. (*B*) An upright model is shown, during endotracheal intubation and positive pressure mechanical ventilation. Transpulmonary pressure is calculated as the difference between alveolar pressure and pleural pressure. A high abdominal load increases pleural pressure, thus resulting in lower transpulmonary pressure. Being exposed to the pleural space, esophageal manometry can be assumed to be a surrogate for pleural pressure, otherwise not measurable in a human being in the clinical setting.

of transpulmonary pressure monitoring through esophageal manometry. Table 2 includes some experienced base recommendations on how to use esophageal manometry to set PEEP in the obese patient.

The effects of pneumoperitoneum on the respiratory system mechanics differ from those of abdominal fat. Pneumoperitoneum induces an increase in the respiratory system elastance sustained mainly by an increased chest wall elastance, while the lung's elastic properties are mostly unaffected.[45] An acute increase in abdominal pressure deforms the chest wall cavity, whose change in geometry is protective for the lung, which is spared from squeezing and compression. Pneumoperitoneum and obesity have a negative additive interaction on the respiratory system during laparoscopic surgery.[46] An approach based on lung recruitment can improve lung mechanics and oxygenation and, when feasible, a beach chair position can be combined to an appropriate level of PEEP to counteract the combined effect of abdominal fat and pneumoperitoneum best.

Finally, the Trendelenburg position negatively impacts lung mechanics. As stated above, obesity is characterized by early airway closure, with a high threshold for airway opening pressure. When pneumoperitoneum is associated with the Trendelenburg position, lung collapse increases, and the airway opening pressure rises even further.[47] This mechanism has some clinical implications: for example, pressure control ventilation can result in hypoventilation and apnea in obese patients in the Trendelenburg position, since the critical opening threshold to generate flow may not be reached. In the same way, if one decides to assess lung mechanics, the level of intrinsic PEEP should be accounted for. For instance, driving pressure is defined as the difference between airway pressure during an inspiratory pause (plateau pressure) and total PEEP, which is the sum of applied and intrinsic PEEP. In this setting, the value of driving pressure would be overestimated if not corrected for the intrinsic PEEP.

Mechanics during Artificial Ventilation with Lung Injury

Obesity has been often a criterion of exclusion in studies focused on lung injury in the intensive care unit. Consequently, to date, hard evidence that can help understanding the complex interaction between the high abdominal load and the injured lung is lacking. For example, the driving pressure of the respiratory system (plateau pressure – total end-expiratory pressure), which is the force needed to overcome the elastic pressures during inflation,[48] is known to directly correlate with mortality in lean patients with ARDS, but the same correlation is not observed for the obese patients.[49] The driving pressure has been associated with the concept of strain (the ratio of the tidal volume to the end-expiratory lung volume), which describes the dynamic force applied to the lung parenchyma during tidal elongation, and is one key mechanism of volutrauma, a type of ventilator-induced lung injury in ARDS.[50] Contrary to what is generally believed, high pleural pressure does not protect against ventilator-induced lung injury if the compliance of the chest wall is normal, as seems to be the case in the obese patient (see discussion in the previous paragraph). In fact, given the partitioned driving pressure of the lung as dPlung = (plateau pressure – PEEP) – (pleural $\text{pressure}_{\text{end-inspiration}}$ – pleural $\text{pressure}_{\text{end-exhalation}}$),[51] it is evident as high absolute values of pleural pressure with a small difference between end inspiration and end expiration expose

Table 2. General Rules and Recommended Settings

Recommended Settings in Mechanically Ventilated Obese Patients in ICU
- Mode: PC or VolC.
- Tidal volume: 6 ml/kg PBW (4–8 ml/kg PBW).
- Inspiratory time: 0.6–1.0 s.
- Plateau pressure: <28 cm H_2O, or end-inspiratory transpulmonary pressure ≤20 cm H_2O as measured with esophageal manometry.
- Driving pressure ≤15 cm H_2O.
- Minute volume 10 l/min.
- PEEP setting in the context of hypoxemia: perform lung recruitment maneuvers and decremental PEEP titration as described by Kacmarek *et al.*[55]
- Monitor transpulmonary pressure. End-expiratory transpulmonary pressure should be slightly positive at the PEEP level coinciding with best compliance.
- Set FIO_2 to maintain SpO_2 ≥88%.
- Keep head elevated 30°.
- Noninvasive ventilation up to 48 h after extubation.

Recommended Settings in Mechanically Ventilated Obese Patients in the Operating Room
- Mode: PC or VC. Note: with Trendelenburg position and pneumoperitoneum PC could result in hypoventilation in airway closure present. In this setting VC preferable.
- Tidal volume: 6 ml/kg PBW (4–8 ml/kg PBW).
- Inspiratory time: 0.6–1.0 s.
- Recruitment maneuvers[55] after any event that could cause atelectasis (example: disconnection from the circuit).
- If refractory hypoxemia: perform recruitment maneuver and decremental PEEP titration as described by Kacmarek *et al.*[55]
- Extubate with the head elevated at 30°.
- NIV after extubation, up to 48 h after, depending on patient condition.

FIO_2, fraction of inspired oxygen; ICU, intensive care unit; NIV, noninvasive ventilation; PBW, predicted body weight (PBW_{MEN} = 50.0 + 0.905 ([height in cm] – 152.4); PBW_{WOMEN} = 45.5 = 0.905 ([height in cm] – 152.4); PC, pressure control; PEEP, positive end-expiratory pressure; SpO_2, oxygen saturation measured by pulse oximetry; VolC, volume control.

the lung to strain and hence volutrauma, if a sufficient PEEP is not provided. It is true, on the other hand, that high pleural pressure can have a protective effect against static stress across the lung parenchyma, especially at the end of inflation (*i.e.*, a decreased transpulmonary pressure during an inspiratory pause, resulting in less alveolar overstretching, particularly in nondependent lung zones). It is difficult to understand what effect is more important in the single patient; it is worth noting that dynamic strain has been associated with greater injury than static stress in some studies.[52] In this context, it would be wise to implement a personalized physiologic approach complementary to the protective ventilatory strategy that has been shown to improve outcomes in ARDS.[53] This approach allows choosing the PEEP corresponding to the best compliance of the respiratory system, after the performance of a recruitment maneuver to reopen collapsed areas of the lung parenchyma, and, in obese patients with a diagnosis of ARDS, has been shown to improve oxygenation and lung mechanics when compared to pressure-oxygenation tables.[54] Decremental PEEP titration based on best compliance may result in a higher PEEP than the one set with predefined tables (with the benefit of a decreased driving pressure), and the use of recruitment maneuvers implies a progressive rise in airway pressures as high as 50 cm H_2O.[55] Such high pressures could raise concerns of important side effects, especially from a hemodynamic point of view,[56] and a recent large randomized trial showed an increase in mortality, barotrauma, and hypotension when decremental PEEP titration was applied to a general population with ARDS.[57] Excessive pressure at end exhalation can decrease cardiac output by decreasing venous return (mainly through an increase in venous resistance[58]) and increased pulmonary vascular resistance, thus impairing the function of the right ventricle. However, pulmonary vascular resistance is augmented at the extremes of lung volumes.[59] That is, the right ventricle is exposed to increased afterload if the lung volumes are too high or too low. Patients with obesity and ARDS theoretically experience very low lung volumes (given that both the conditions are associated with decreased FRC) and may require a higher PEEP to restore a functional residual capacity compatible with a normal resistance in the pulmonary circulation. Hence, the application of high pressures in the obese patient with ARDS would have a strong rationale, once granted an optimal volemic status in order to prevent decreases in venous return. However, future physiologic studies should yield more knowledge about the lung-heart interaction dynamics, and randomized trials are needed to demonstrate any benefit in clinical outcomes of recruitment maneuvers and decremental PEEP titration in this specific population.

Although technically demanding, prone positioning is another therapeutic opportunity to consider: it can be undergone safely in obese patients and has been proven to be beneficial in terms of physiologic outcomes when obesity is associated with severe refractory hypoxemia.[60] In the lean person, pronation works through redistribution of ventilation, releasing the pressure imposed by lung edema on the most dependent lung zones. More studies are needed to prospectively investigate its mechanisms and effects on clinical outcomes in the obese population.

Mechanics during Weaning from Mechanical Ventilation

When extubating a patient with obesity, especially after prolonged mechanical ventilation in a critical care setting, it can be useful to consider the concept of work of breathing. The work of breathing can be defined as the effort made by the respiratory muscles to overcome the elastic and resistive forces that oppose the expansion of the respiratory system.[61] In the periextubation period, muscular weakness and residual activity of sedatives and muscle relaxants can impair the action of respiratory muscles, negatively impacting the weaning process. In patients with obesity, the situation is further complicated by the elevated pleural pressure. High pleural pressure by diaphragmatic displacement results in a negative transpulmonary pressure at the end of exhalation, leading to atelectasis and small airways collapse. As a result, the work of breathing is increased,[18,62] given that a significant part of it is spent to overcome the closing pressure of the airways to generate a flow. This mechanism can prolong the weaning phase.

In this context, a PEEP sufficient to contrast airway collapse during exhalation might be beneficial even if, as discussed above, standard levels of end-expiratory pressure are usually too low to meet the needs of mechanically ventilated obese patients.[37,38] A strategy providing higher levels of end-expiratory pressure at extubation (in the range of 15 to 30 cm H_2O) in order to achieve a positive transpulmonary pressure throughout the respiratory cycle can significantly facilitate the weaning process.[63] Positive pressures in the airways should be maintained along the whole periextubation period. After invasive ventilation has been removed, positive pressure in the small airways can be maintained by means of noninvasive ventilation. The role of simple systems to create a continuous positive airway pressure such as the Boussignac mask has been investigated in the elective postoperative period, with established benefits in terms of oxygenation and improved lung volumes.[64] Noninvasive positive pressure is physiologically attractive because it keeps the small airways inflated and counteracts the collapse of the soft tissue at the level of the upper airways. Obstruction of the upper airways is a common phenomenon in the obese, which is magnified by sedative agents. So far, there is a lack of large studies assessing the benefits of noninvasive positive pressure in the critically ill obese population after prolonged ventilation. Preferably, the subject should be positioned with the upper part of the body upright (sitting position), in order to

improve lung volumes by releasing the pressure caused by abdominal content.[65]

When weaning a patient from mechanical ventilation, it would be ideal to determine the work of breathing, which indicates the level of effort, the patient is required to produce during spontaneous ventilation. Good clinical practice suggests gradually decreasing the pressure support while monitoring variables such as the tidal volume and the respiratory rate. In this context, a spontaneous breathing trial through a T piece (the patient is still intubated but disconnected from the ventilator, and breathes through a system that provides a humidified oxygenated flow of air) can accurately simulate the patient's ventilatory effort.[66]

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Competing Interests

Dr. Kacmarek declares a financial relationship as a consultant with Orange Medical (Hialeah, Florida) and Medtronic Airway (Minneapolis, Minnesota); in the past he has received research grants from Orange Medical and Medtronic Airway. He has presented a webinar for Nihon Kohden (Tokyo, Japan) on high flow nasal cannulas. The other authors declare no competing interests.

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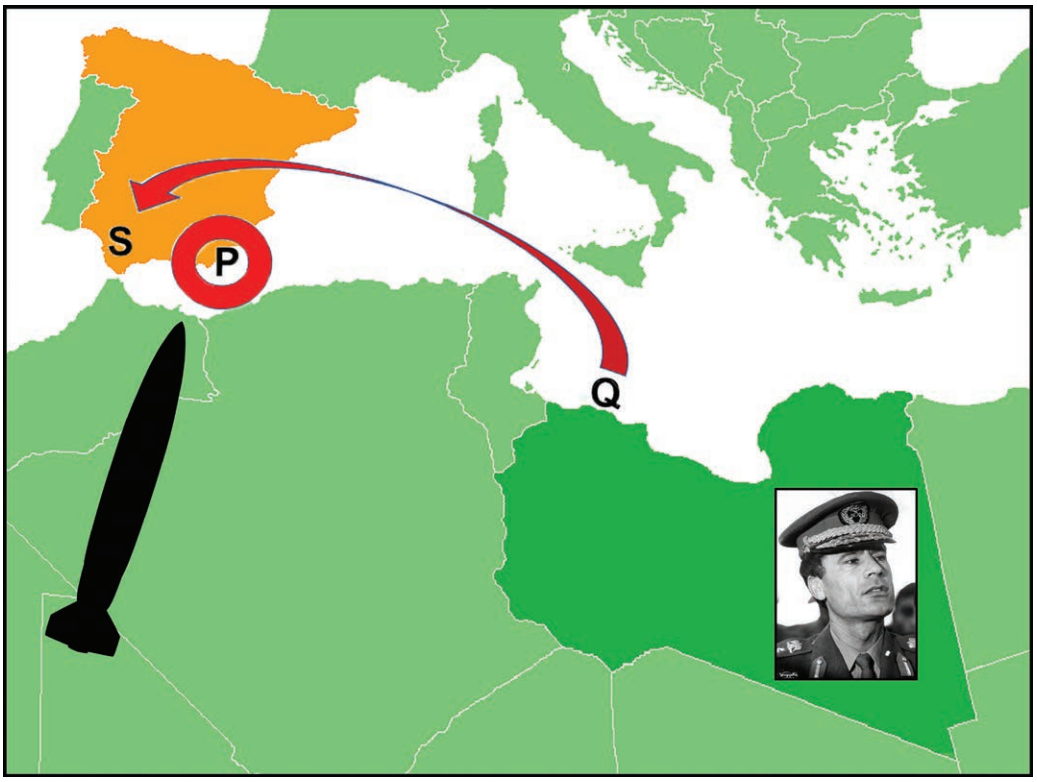
Byline Backstory No. 4: International Ps and Qs for a Future Medical Antiques Courier



In 1965 the Air Force transferred my family from Florida to Seville, Spain (S), so as a pre-teenager, I had to learn my international "Ps and Qs." The "P" was for the 1966 midair refueling collision which scrambled most of Spain's American military, including my father, to find four hydrogen bombs dropped near the fishing village of Palomares (P). The "Q" of the Ps and Qs was for Qaddafi (*lower right*), then Lt. Col. Muammar Qaddafi, one of the Libyan officers forcing the evacuation of hundreds of American military dependents from an air base near Libya's Tripoli (Q) to our base in Spain during the 1967 Six Days' War. Living four years in Europe (three in Spain and, later, one in Scotland) taught me about airport security, passport control, customs, packing valuables—all the international Ps and Qs that would help me as a future medical antiques courier for the Wood Library-Museum. (Copyright © the American Society of Anesthesiologists' Wood Library-Museum of Anesthesiology.)

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