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EDITORIAL

Driving pressure in obese ventilated patients: another brick in the (chest) wall

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The incidence of obesity is increasing worldwide, and obese or severely obese patients often present with several comorbidities. Obesity is associated with significant complications, and an increase in the mortality of obese patients after hospital admission compared to non-obese patients is justified [1]. The proportion of obesity in patients presenting with acute respiratory distress syndrome (ARDS) has not been investigated systematically, but data from the influenza A (H1N1) pandemic in 2009 suggested that obese patients were overrepresented compared to the general population [2]. The management of ARDS in patients with obesity is a special challenge, since obesity and severe obesity are associated with marked derangements in lung and chest wall mechanics [3].

In clinical studies on obese mechanically ventilated patients without lung injury [4] or with ARDS [5], specific changes in respiratory system mechanics were observed (Table 1). These findings may help develop a better understanding of a suitable ventilation strategy in obesity. In all studies, static respiratory compliance ($C_{st,rs}$) was reduced with a rising level of obesity [expressed by body mass index (BMI)], while elastance (the reciprocal of compliance) tended to increase. These changes in $C_{st,rs}$ were due to compliance reduction in both parts of the respiratory system: the lung and the chest wall. In other words, during mechanical ventilation in obese patients, the lungs and predominantly the chest wall become 'stiffer'. This finding—observed in obese lung-healthy patients as well in ARDS patients—deserves attention and interpretation. Physiologically, the chest wall (rib cage and abdomen) is elastic, and the interaction between the lungs and the chest wall is finely balanced under spontaneous breathing [7]. The chest wall tends to expand outward, while the lungs tend to collapse. The 'mediator' between both is the pleural pressure. The reduction of chest wall compliance ($C_{st,w}$) in obesity is mainly due to the increase of intra-abdominal pressure in these patients [3], which results in a lower lung volume for the same pleural pressure.

In a large retrospective analysis of the data of 3562 ARDS patients from nine studies, Amato et al. [8] demonstrated that elevated driving pressure (ΔP), a parameter expressing the relation between tidal volume and functional lung size (compliance of the respiratory system) $= V_T/C_{st,rs}$, was an independent ventilation variable strongly associated with increased mortality, while changes in tidal volume or positive end-expiratory pressure were not independent predictors of the outcome. An actual systematic review [9] reconfirmed the retrospective data of Amato et al. [8] by analysing the data of four prospective randomized studies (3252 patients): a higher ΔP was associated with a significantly higher mortality in ARDS patients.

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Table 1 Characteristics of respiratory mechanics in obese mechanically ventilated patients (findings from clinical studies)

Study/patients	Static respiratory system compliance ($C_{st,rs}$) (mL cmH_2O^{-1})	Lung compliance ($C_{st,L}$) (mL cmH_2O^{-1})	Chest wall compliance ($C_{st,w}$) (mL cmH_2O^{-1})
Pelosi 1996 [4] 20 pts., general anaesthesia	BMI < 25 = 66 ± 14 BMI > 40 = 35 ± 5	107 ± 32 55 ± 15	191 ± 45 112 ± 47
Pelosi 1998 [5]* 24 pts., general anaesthesia	BMI < 25 ≈ 75 BMI 25–40 ≈ 55 BMI > 40 ≈ 46	≈ 150 ≈ 85 ≈ 53	≈ 250 ≈ 180 ≈ 110
Normal findings in spontaneously breathing adult subjects [14]	50–100	200	100–200

Study/patients	Static respiratory system elastance ($cmH_2O\ L^{-1}$)	Lung elastance ($cmH_2O\ L^{-1}$)	Chest wall elastance ($cmH_2O\ L^{-1}$)	Driving pressure (ΔP) (cmH_2O)
Chiumello 2016 [6] 101 ARDS patients	BMI < 25 = 25 (19–31) BMI 25–30 = 27 (21–36) BMI > 30 = 26 (24–32)	18 (14–25) 21 (16–30) 21 (15–24)	5 (3–9) 6 (4–9) 8 (4–10)	11 (10–14) 15 (12–17) 14 (13–17)

BMI body mass index

*Values estimated from figures

Surprisingly, this important finding seems not to be of value for obese ARDS patients. The retrospective analysis presented by de Jong et al. [10] on the impact of ΔP on 90-day mortality in 100 obese (BMI ≥ 30) and 262 non-obese ARDS patients revealed a significant relationship between ΔP and mortality only in non-obese patients. The ΔP on day 1 was significantly lower in non-obese ARDS patients (11.9 ± 4.2 cmH_2O) who had survived 90 days compared to non-obese non-survivors (15.2 ± 5.2 cmH_2O, $p < 0.001$). In obese patients, the ΔP did not differ between survivors and non-survivors. Additionally, in a multivariate Cox analysis, ΔP was identified as an independent predictor of day 90 mortality in non-obese, but not in obese patients. Interestingly the mortality did not differ between non-obese (47%) and obese ARDS (46%) patients. Although the current study has some limitations (the monocentric and retrospective design), an important finding was that the day 1 mean values for ΔP were similar in the survivors (13.7 ± 4.5 cmH_2O) and in the non-survivors (13.2 ± 5.1 cmH_2O, $p = 0.41$), and on a level which has been identified as being within a 'safe' border. Amato et al. [8] described a ΔP of 15 cmH_2O as a threshold discriminating between a good outcome of ARDS patients (relative risk of death ≤ 1 with $\Delta P \leq 15$ cmH_2O) and a higher risk of mortality with $\Delta P > 15$ cmH_2O. The study by de Jong et al. [10] presented 'safe' ΔP levels (≤ 15 cmH_2O) for the majority of obese ARDS patients. Is obesity per se a lung protective precondition generating low(er) ΔP values? If, yes, which are the specific (patho)physiologic mechanisms for such a distinction from non-obesity?

The reduction in $C_{st,w}$ is a characteristic finding in the respiratory mechanics of mechanically ventilated obese patients (Table 1), and this distinctiveness could be responsible for the lower ΔP values, as seen in the 'obesity paradox'. A meta-analysis on the causal relation between obesity and mortality in ARDS [11] evaluating the combined data of nine studies found that obesity was significantly associated with a reduced risk of ARDS mortality (pooled OR 0.63, 95% CI 0.64–0.84). In consequence, further studies should carefully investigate the potential role of chest wall dynamics, pleural pressure and transpulmonary pressure in obesity with the aim of examining the hypothesis that adiposity offers a certain protection against ventilator-induced lung injury. Furthermore, the shape of obesity is various, and in recent years it was demonstrated that the pattern of body fat distribution (general or abdominal adiposity) has a great impact on outcomes. Reis et al. [12] reported that in middle-aged healthy persons, higher values of the waist-to-thigh ratio (in both sexes) or the waist-to-hip ratio (in women) were positively associated with increased mortality, while the BMI exhibited U- or J-shaped associations. In intensive care, no data exists on the pattern of obesity, and in future researchers should investigate not only BMI but also the effects of abdominal obesity on outcomes.

Are the results of the study by de Jong et al. [10] a setback for those who found in the parameter ΔP (easily assessed at the bedside!) a new tailor-made solution for an individualized lung protective ventilation strategy in all ARDS patients? No, quite the opposite! The data from de Jong et al. provides an important step forward to a personalized strategy in intensive care medicine [13]. Again, it becomes clear that a simple 'one variable fits all' will not work in critical care.

Compliance with ethical standards

Conflicts of interest
The author declares no conflict of interest.

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