BJA

British Journal of Anaesthesia, 116 (1): 113–21 (2016)

doi: 10.1093/bja/aev378
Respiration and the Airway

RESPIRATION AND THE AIRWAY

Effect of body mass index in acute respiratory distress syndrome

D. Chiumello[1,2,*], A. Colombo[2], I. Algieri[2], C. Mietto[2], E. Carlesso[2], F. Crimella[2], M. Cressoni[2], M. Quintel[3] and L. Gattinoni[1,2]

[1]Dipartimento di Anestesia, Rianimazione (Intensiva e Subintensiva) e Terapia del Dolore, Fondazione IRCCS Ca' Granda – Ospedale Maggiore Policlinico, Milano, Italy, [2]Dipartimento di Fisiopatologia Medico-Chirurgica e dei Trapianti, Università degli Studi di Milano, Milano, Italy, and [3]Department of Anesthesiology, Emergency and Intensive Care Medicine, Georg-August University of Göttingen, Göttingen, Germany

*Corresponding author: Dipartimento di Anestesia, Rianimazione e Terapia del dolore, Fondazione IRCCS Cà Granda- Ospedale Maggiore Policlinico, Via F. Sforza 35, Milano, Italy. E-mail: chiumello@libero.it

Downloaded from http://bja.oxfordjournals.org/ at Orta Dogu Teknik University Library (ODTU) on December 22, 2015

Abstract

Background: Obesity is associated in healthy subjects with a great reduction in functional residual capacity and with a stiffening of lung and chest wall elastance, which promote alveolar collapse and hypoxaemia. Likewise, obese patients with acute respiratory distress syndrome (ARDS) could present greater derangements of respiratory mechanics than patients of normal weight.

Methods: One hundred and one ARDS patients were enrolled. Partitioned respiratory mechanics and gas exchange were measured at 5 and 15 cm H_2O of PEEP with a tidal volume of 6–8 ml kg^{-1} of predicted body weight. At 5 and 45 cm H_2O of PEEP, two lung computed tomography scans were performed.

Results: Patients were divided as follows according to BMI: normal weight (BMI≤25 kg m^{-2}), overweight (BMI between 25 and 30 kg m^{-2}), and obese (BMI>30 kg m^{-2}). Obese, overweight, and normal-weight groups presented a similar lung elastance (median [interquartile range], respectively: 17.7 [14.2–24.8], 20.9 [16.1–30.2], and 20.5 [15.2–23.6] cm H_2O $litre^{-1}$ at 5 cm H_2O of PEEP and 19.3 [15.5–26.3], 21.1 [17.4–29.2], and 17.1 [13.4–20.4] cm H_2O $litre^{-1}$ at 15 cm H_2O of PEEP) and chest elastance (respectively: 4.9 [3.1–8.8], 5.9 [3.8–8.7], and 7.8 [3.9–9.8] cm H_2O $litre^{-1}$ at 5 cm H_2O of PEEP and 6.5 [4.5–9.6], 6.6 [4.2–9.2], and 4.9 [2.4–7.6] cm H_2O $litre^{-1}$ at 15 cm H_2O of PEEP). Lung recruitability was not affected by the body weight (15.6 [6.3–23.4], 15.7 [9.8–22.2], and 11.3 [6.2–15.6]% for normal-weight, overweight, and obese groups, respectively). Lung gas volume was significantly lower whereas total superimposed pressure was significantly higher in the obese compared with the normal-weight group (1148 [680–1815] *vs* 827 [686–1213] ml and 17.4 [15.8–19.3] *vs* 19.3 [18.6–21.7] cm H_2O, respectively).

Conclusions: Obese ARDS patients do not present higher chest wall elastance and lung recruitability.

Key words: chest wall elastance; intra-abdominal pressure; lung elastance; lung gas volume; obesity; respiratory distress syndrome, adult

Obesity, defined as a BMI between 30 and 39.9 kg m^{-2}, and extreme obesity, with BMI more than 40 kg m^{-2},[1] is a disease with a worldwide increasing prevalence that already affects more than 400 million people.[2] Obesity increases the risk for cancer, heart failure, diabetes, dyslipidaemia, sleep apnoea, and death compared with people of normal body weight.[3] In addition,

Accepted: October 2, 2015

© The Author 2015. Published by Oxford University Press on behalf of the British Journal of Anaesthesia. All rights reserved.
For Permissions, please email: journals.permissions@oup.com

Editor's key points

- Obesity is associated with a reduced functional residual capacity and a stiffening of lung and chest wall elastance, but the changes in respiratory mechanics in obese acute respiratory distress syndrome (ARDS) patients are not known.
- Respiratory mechanics and gas exchange were measured in 101 ARDS patients with various body weights.
- There were no significant differences between obese and non-obese ARDS patients in the lung and chest wall elastance, but lung gas volume was significantly lower and total superimposed pressure significantly higher in obese patients than in non-obese.

owing to the presence of several co-morbid conditions, obesity has been associated with a significant increase in complications and mortality after hospital admission.[4] In contrast, the influence of body weight on acute illness is less clear.[5–7] A retrospective analysis of a randomized trial comparing low *vs* higher tidal volume ventilation in patients with acute lung injury found that obesity did not affect the severity of hypoxaemia.[8] Recent studies have found that critically ill obese patients had no difference or even a decrease in hospital length of stay and mortality.[8–11] The most commonly reported effects of obesity during spontaneous breathing and general anaesthesia are a reduction in functional residual capacity[12–15] and stiffening of lung and chest wall elastance, which promote alveolar collapse and hypoxaemia.[16] To counteract these respiratory changes in mechanically ventilated healthy obese patients, higher tidal volume,[17 18] moderate PEEP levels (5–10 cm H_2O),[16 19] and recruitment manoeuvres have been suggested.[20–22] Kikewise, obese patients with acute respiratory distress syndrome (ARDS) may suffer from greater derangements of respiratory mechanics and gas exchange than patients of normal body weight. Higher PEEP levels could be necessary to optimize lung recruitment, to limit the intratidal opening and closing, and to improve oxygenation. In addition, the possible increase in chest wall elastance could also decrease the transpulmonary pressure.[23 24] These two factors could be relevant in setting the mechanical ventilation to minimize the ventilator-associated lung injury. To date, no major studies have examined the role of chest wall elastance in influencing the effects of PEEP and tidal volume in these patients.

The aim of the present study was to evaluate the effect of the BMI in a group of ARDS patients on chest wall elastance, lung recruitability, and transpulmonary pressure.

Methods

Study population

A total of 101 patients with acute lung injury or ARDS were included; 71 were previously enrolled in two published works,[25 26] whereas the remaining 30 were enrolled in a prospective study (http://clinicaltrials.gov/show/NCT01670747). In the present study, considering a partly new study population, we evaluated completely new data never analysed and shown before. Thus, this is not a duplication of the data or salami publication. The study was approved by the institutional review board of each hospital, and written consent was obtained according to the regulations applicable in each institution (consent was delayed in Italy until the patients had recovered from the sedation and was obtained in Germany from a legal representative). Patients were classified as having mild, moderate, or severe ARDS according to the Berlin definition.[27]

Study design

All patients were deeply sedated, paralysed, and ventilated in the volume-control mode with a tidal volume of 6–8 ml per kilogram of predicted body weight throughout the study protocol.[26] The oxygen fraction, tidal volume, and respiratory rate were maintained unchanged for the entire study. Immediately before each step of the PEEP trial, a recruitment manoeuvre was performed to standardize the lung volume history. The recruitment manoeuvre was performed in pressure-control ventilation at a PEEP of 5 cm H_2O, with a plateau pressure of 45 cm H_2O, ratio of the duration of inspiration to the duration of expiration (I:E) 1:1, respiratory rate of 10 breaths for 2 min.[28] Subsequently, PEEPs of 5 and 15 cm H_2O were randomly applied, with similar ventilator settings used before the recruitment manoeuvre. At each PEEP level, after 20 min, respiratory mechanics were measured and blood gas analyses performed.

Measurements

Respiratory mechanics

The respiratory flow rate was measured with a heated pneumotachograph (Fleisch no. 2; Fleisch, Lausanne, Switzerland). Airway pressure was measured proximally to the tracheal tube with a dedicated pressure transducer (MPX 2010 DP; Motorola, Solna, Sweden). Oesophageal pressure was measured with a radio-opaque balloon (SmartCath Bicore; Bicore Irvine, CA, USA) inflated with 1.0–1.5 ml of air connected to a pressure transducer.[29] All traces were sampled at 100 Hz and processed on a dedicated data acquisition system (Colligo and Computo; www.elekton.it). The oesophageal balloon was positioned in the lower third of the oesophagus at a depth of 35–40 cm.[26] Intra-abdominal pressure was measured by the bladder technique.[30]

During an inspiratory and expiratory pause, the static airway and oesophageal pressure were measured. Transpulmonary pressure was computed as airway pressure minus oesophageal pressure.

The respiratory system, lung, and chest wall elastances were computed according to the following formulae:[25]

(i) Respiratory system elastance (E_{rs}; in cm H_2O $litre^{-1}$)=(airway pressure at end-inspiratory pause−airway pressure at PEEP)/tidal volume
(ii) Lung elastance (E_l; in cm H_2O $litre^{-1}$)=(transpulmonary pressure at end-inspiratory pause−transpulmonary pressure at PEEP/tidal volume
(iii) Chest wall elastance (E_{cw}; in cm H_2O $litre^{-1}$)=(oesophageal pressure at end-inspiratory pause−oesophageal pressure at PEEP)/tidal volume
(iv) The end-inspiratory transpulmonary pressure was calculated as the airway pressure at end-inspiratory pause×lung elastance/respiratory system elastance[31]
(v) The end-expiratory transpulmonary pressure was calculated as the difference between the airway and oesophageal pressure at PEEP and after a release manoeuvre.[31]

Gas exchange

Arterial and central venous blood gases were analysed. The total dead space was computed according to the Enghoff modification of Bohr's equation, with the mixed expired partial pressure of carbon dioxide being measured by a CO2SMO monitor (Novametrix, Wallingford, UK).

Downloaded from http://bja.oxfordjournals.org/ at Orta Dogu Teknik University Library (ODTU) on December 22, 2015

Download English Version:

https://daneshyari.com/en/article/8931252

Download Persian Version:

https://daneshyari.com/article/8931252

Daneshyari.com