TRANSPULMONARY PRESSURE DURING MECHANICAL VENTILATION

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Avhandlingen baseras på följande delarbeten:

- I. Grivans C, Lindgren S, Åneman A, Stenqvist O, Lundin S.
 A Scandinavian survey of drug administration through inhalation, suctioning and recruitment maneuvers in mechanically ventilated patients. Acta Anaesthesiologica Scandinavica 2009;53(6):710-6.
- II. Grivans C, Lundin S, Stenqvist O, Lindgren S.
 Positive end-expiratory pressure-induced changes in end-expiratory lung volume measured by spirometry and electric impedance tomography.
 Acta Anaesthesiologica Scandinavica 2011;55(9):1068-77.
- III. Stenqvist O, Grivans C, Andersson B, Lundin S.
 Lung elastance and transpulmonary pressure can be determined without using oesophageal pressure measurements.
 Acta Anaesthesiologica Scandinavica 2012;56(6):738-47.
- IV. Grivans C, Lundin S, Stenqvist O
 Lung elastance and transpulmonary pressure can be calculated from the change in endexpiratory lung volume following a change in end-expiratory airway pressure.
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ABSTRACT

Background: Mechanical ventilation can aggravate lung injury by repetitive opening and closing of lung units, overdistention and undue pressure on pulmonary structures. Guidelines exist for lung protective ventilation, but individualized ventilator settings, based on partitioning of respiratory mechanics in pulmonary and chest wall complex components, would be beneficial. This thesis examines the current practice of respiratory care in the Nordic countries and evaluates a method to assess lung volume changes during mechanical ventilation. A new concept to measure lung and chest wall elastance and determine transpulmonary pressure during mechanical ventilation is validated.

Methods: Clinical practice concerning adjunct therapies to mechanical ventilation were addressed in a web-based survey performed in Nordic intensive care units. Changes in lung volume (Δ EELV) were determined by spirometry, where the first ten breaths after a PEEP change were studied. The sum of the differences of inspiratory and expiratory tidal volumes was calculated, with correction for offset. The method was validated in a lung model and in 12 patients with simultaneous measurement of lung volume changes by electrical impedance tomography (EIT). PEEP induced changes in Δ EELV, airway and esophageal pressures were studied both in an animal model and in 12 ventilated patients.

Results: One-third of the patients had more than 12 disconnections from the ventilator circuit during 24 hours, with great variations in the individual lowest and highest oxygenation ratios (PaO_2/FiO_2). The spirometric method showed good agreement with known volume changes in the lung model and with estimated lung volume changes by EIT. PEEP increase resulted in only modest increase in esophageal pressure. The increase in transpulmonary pressure was closely related to increase in PEEP. Lung elastance determined from change in PEEP divided by Δ EELV was closely correlated with that obtained from esophageal pressure measurements.

Conclusion: Routine care of ventilated patients leads to repeated derecruitment episodes due to disconnections of the ventilator circuit by frequent use of aerosol therapy and endotracheal suctioning. Spirometric measurements of inspiratory-expiratory tidal volumes as well as impedance changes by EIT can be used to estimate PEEP-induced changes in lung volume. The minimal increase in esophageal pressure after a PEEP increase indicate that the abdomen and chest wall gradually yields and adapts when the diaphragm is pushed in caudal direction. As a consequence, PEEP increase will cause a corresponding increase in transpulmonary pressure. This may explain why lung elastance can be determined from the change in PEEP divided by the change in lung volume without the need for esophageal pressure measurements.

Keywords: Mechanical ventilation, Acute lung injury; Acute respiratory distress syndrome; Lung volume measurements; Electric Impedance/diagnostic use; Lung elastance; Transpulmonary pressure; Esophageal pressure

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