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Laboratory Investigation

LABORATORY INVESTIGATION

Cardiogenic oscillations to detect intratidal derecruitment and overdistension in a porcine model of healthy and atelectatic lungs

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Background: Low PEEP can result in alveolar derecruitment, and high PEEP or high tidal volume (V_T) in lung overdistension. We investigated cardiogenic oscillations (COS) in the airway pressure signal to investigate whether these oscillations can assess unfavourable intratidal events. COS induce short instantaneous compliance increases within the pressure-volume curve, and consequently in the compliance-volume curve. We hypothesised that increases in COS-induced compliance reflect non-linear intratidal respiratory system mechanics.
Methods: In mechanically ventilated anaesthetised pigs with healthy (n=13) or atelectatic (n=12) lungs, pressure-volume relationships and the ECG were acquired at a PEEP of 0, 5, 10, and 15 cm H_2O. During inspiration, the peak compliance of successive COS (C_{COS}) was compared with intratidal respiratory system compliance (C_{RS}) within incremental volume steps up to the full V_T of 12 ml kg^{-1}. We analysed whether C_{COS} variation corresponded with systolic arterial pressure variation.
Results: C_{COS}-volume curves showed characteristic intratidal patterns depending on the PEEP level and on atelectasis. Increasing C_{RS}- or C_{COS}-volume patterns were associated with intratidal derecruitment with low PEEP, and decreasing patterns above 6 ml kg^{-1} and high PEEP showed overdistension. C_{COS} was not associated with systolic arterial pressure variations.
Conclusions: Heartbeat-induced oscillations within the course of the inspiratory pressure-volume curve reflect non-linear intratidal respiratory system mechanics. The analysis of these cardiogenic oscillations can be used to detect intratidal derecruitment and overdistension and, hence, to guide PEEP and V_T settings that are optimal for respiratory system mechanics.

Keywords: blood pressure; lung compliance; mechanical; respiratory mechanics; ventilators

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Editor's key points

- Evaluation of pulmonary mechanical load in relation with ventilator settings to reduce the risk of overdistension or derecruitment is limited because of the absence of validated measurement methods.
- The beating heart is a natural intrathoracic generator for mechanical pulses that appear as cardiogenic oscillations at the airway opening.
- This study in pigs with healthy or atelectatic lungs shows that cardiogenic oscillations during the course of the inspiratory pressure-volume curve reflect respiratory system mechanics and are indicative of intratidal overdistension or derecruitment.
- Cardiogenic oscillations can be used to guide ventilator settings that are optimal for respiratory system mechanics.

During mechanical ventilation, the current properties of the respiratory system in terms of tidal derecruitment and overdistension are mainly determined by the disease and the mechanical load imposed on the lung parenchyma by PEEP and tidal volume (V_T).[1–4] The analysis of intratidal non-linear mechanics of the respiratory system is potentially helpful to capture the actual mechanical load related with the ventilator settings.[2,5–7] However, at present their availability in routine use at the bedside is limited. Here we adopt a new approach by considering the beating heart as a natural intrathoracic generator for mechanical pulses that travel across the lung parenchyma and appear as cardiogenic oscillations (COS) at the airway opening. COS alter the relationship between airway pressure and lung volume, resulting in characteristic peaks in the intratidal pressure-volume relationship.[8] While COS are often regarded as a disturbance signal,[9] we investigated their potential for providing information about the conditions of the respiratory system. In a study in lung healthy volunteers, we found that COS are sensitive to changes in the mechanical properties of the respiratory system.[10] We hypothesised that the patterns of COS-induced compliance-peaks (C_{COS}) are characteristic for specific lung conditions and would change in the presence of derecruited or overdistended lung parenchyma. Therefore, we investigated C_{COS} in pigs with healthy and atelectatic lungs at various levels of PEEP.

Methods

The study protocol was approved by the local Animal Welfare Committee (Uppsala University, Uppsala, Sweden) and was compliant with the ARRIVE guidelines. Data on intratidal respiratory system mechanics of a subgroup of the animals (atelectasis group) were published earlier.[7]

Study protocol

A total of 25 piglets [mean (standard deviation) body weight: 25 (5) kg] were allocated to either the healthy group (n=13) or the atelectasis group (n=12), and their lungs were mechanically ventilated at randomised PEEP sequences (0, 5, 10, and 15 cm H_2O). Atelectasis was generated by induction of anaesthesia with pure oxygen and amplified by negative pressure application. For the latter, the suction line was connected to the tracheal tube and negative pressure was applied until SaO_2 decreased to below 80%, which occurred usually within 2 min. Ventilation was performed in the volume-controlled mode, throughout. Aiming at prolonged inspiration time and generation of lung overdistension, V_T was set to 12 ml kg^{-1}. Inspiration-to-expiration ratio was 1:1. Ventilation frequency was set to 20 min^{-1} and FiO_2 to 1.0 for the atelectasis group and 0.3 for the lung-healthy group.

For anaesthesia, ketamine (20 mg kg^{-1} h^{-1}) and morphine (0.5 mg kg^{-1} h^{-1}) were given; neuromuscular block was achieved by pancuronium bromide (0.25 mg kg^{-1} h^{-1}). Normal saline (10 mg kg^{-1} h^{-1}) was continuously infused. A bolus of dextrane 10 mg kg^{-1} was given to reduce the haemodynamic effects of positive pressure ventilation and those of the anaesthesia-induced vasodilation.

Monitoring

Ventilation was applied via an tracheal tube (ID 9 mm; Mallinckrodt, Athlone, Ireland) using a Servo[i] ventilator (MAQUET Critical Care AB, Solna, Sweden). All measurements were performed after a 10-min stabilisation period. Respiratory flow rate was measured using a Fleisch pneumotachograph, and airway pressure via a transducer connected to a side port. Arterial pressure was measured via a catheter in a branch of the subclavian artery. Systolic pressure variation (i.e. the intratidal change of the systolic pressure peaks), systolic pulse pressure (i.e. systolic minus diastolic pressure[11]), and maximal systolic pulse pressure increase (dp/dt_{max}) were measured and used as surrogate variables for the energy pulse that is transferred from the heart to the lungs causing COS, determined by preload and contractility. The ECG was obtained from a standard ECG-recording system.

Data were recorded with AcqKnowledge software version 3.2.7 (Biopac System Inc., Santa Barbara, CA, USA) at a sample frequency of 500 Hz. For offline analysis, Matlab (R2014a; The Mathworks, Natick, MA, USA) was used.

Analysis of respiratory system mechanics

To eliminate potential influences of non-linear tracheal tube resistance, all analyses were based on tracheal pressure calculated from airway pressure as described elsewhere.[12] In brief, the pressure decrease across the tracheal tube was calculated from the tubes' polynomial resistance coefficients and measured flow rate. Tracheal pressure was then determined as the difference between the measured airway pressure and the calculated pressure decrease across the tracheal tube.

The intratidal compliance-volume curve of the respiratory system (C_{RS}-curve) was calculated using the gliding-SLICE method (Fig. 1a).[13] In brief, from the inspiratory pressure-volume curve (Fig 1b) the range of 5–95% of V_T was analysed at subsequent volume-steps. For each volume step, C_{RS} was calculated by multiple linear regression analysis from data within a specified volume-range (SLICE-volume) around the volume-step. We chose 150 volume-steps to enable calculating a very smooth course of intratidal compliance.

In a first step, choosing a SLICE-volume of one-sixth of V_T for the multiple regression analysis filtered off the COS-induced disturbances and the resulting sequential intratidal C_{RS} values were minimally, if at all, influenced by heartbeats.[5] In a second step, the filtering procedure was modified to single out those COS-induced compliance-peaks (Fig 1c). To this end, the multiple regression analysis was performed with the SLICE-volume reduced to one-thirty-sixth of V_T around each

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