

probably due to expected more efficient secretion removal. We suggest that effective removal of secretions may be inferred by a combination of a decrease in VRI signal coupled with an increase in air flow rate.

Reference

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P174

Continuous elevation of lung sound amplitudes, recorded at fixed flow rate, may indicate an increase in lung water content

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Introduction Vibration response imaging (VRI) is a bedside lung sound monitoring system. We previously reported that vibration intensity can be significantly elevated in patients with congestion, as opposed to pleural effusion, atelectasis, or normal lung [1]. We hypothesized that changes in lung water content (that is, pulmonary edema) may influence breath sound amplitude and explored the possibility of using continuous digitalized lung sound monitoring as a means to track changes in extravascular lung water (EVLW).

Methods EVLW was increased in three pigs: in two animals by installation of saline into the endotracheal tube, and in one animal with sepsis-induced edema. In both models the increase in extravascular lung water index (EVLWi) was evaluated by the PiCCO system, and lung sound amplitude was monitored with the VRI. Animals were ventilated at a fixed flow rate.

Results In both the saline installation and sepsis animal models, significant elevation in lung sound amplitude was measured. In the saline installation animals, sound amplitude increased from $2.21 \times 10^5 \pm 1.58 \times 10^4$ au to $9.49 \times 10^5 \pm 8.02 \times 10^4$ au (average \pm SEM), concomitant with an increase in EVLWi from 10 ml/kg to 14 ml/kg. Similarly, sound amplitudes changed in correspondence with elevation of EVLWi in the septic animal (see Figure 1).

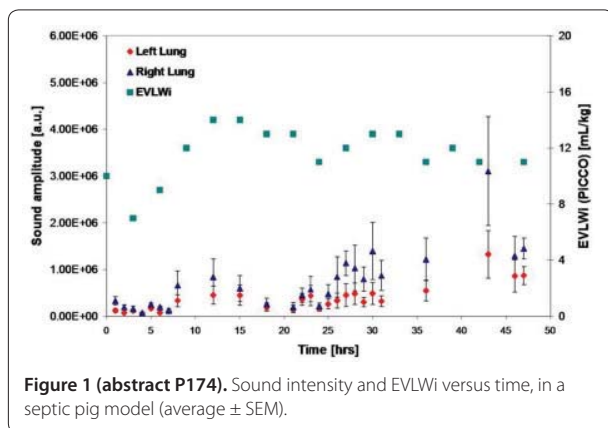


Figure 1 (abstract P174). Sound intensity and EVLWi versus time, in a septic pig model (average \pm SEM).

Conclusions These preliminary results suggest that continuous elevation of lung sound amplitudes, recorded at fixed flow rate, may indicate an increase in lung water content.

Reference

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Impact of normocapnic and permissive hypercapnic one-lung ventilation on arterial oxygenation

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Introduction Physiologically, an approximately 5 to 10 mmHg difference exists between end-tidal carbon dioxide (EtCO₂) and arterial carbon dioxide (PaCO₂) measured during double-lung ventilation (DLV) that may increase during one-lung ventilation (OLV) especially if low tidal volume is applied. There is no evidence that during OLV the EtCO₂ or PaCO₂ should be kept in the normal range. The aim of the present work was to test whether different ventilatory strategies to maintain EtCO₂ or PaCO₂ in the normal range during OLV have any impact on arterial oxygenation (PaO₂).

Methods Data were obtained from 100 patients undergoing thoracic surgery necessitating OLV. Patients were randomized into two groups. In GrEtCO₂ (n = 50) the OLV was guided by capnography, and the respiratory rate (RR) was adjusted to maintain EtCO₂ in the normal range. In GrPaCO₂ (n = 50) the OLV was guided by arterial blood gas analysis (ABG) and RR was adjusted to maintain PaCO₂ in the normal range. ABG was performed in a supine position after induction and in a lateral decubitus position during DLV and every 15 minutes during OLV. During OLV 5 ml/kg tidal volume with 5 cmH₂O PEEP, I:E = 1:2 ratio and FiO₂ 1.0 was used.

Results There were no significant differences in PaO₂ values between groups during DLV and at the 15th minute of OLV. There were significant differences in PaO₂ at the 30th and 45th minutes between groups. In GrPaCO₂ mean airway pressure and RR was higher, and the inspiratory and expiratory time was shorter than in GrEtCO₂.

Conclusions The relatively high RR impairs the emptying of alveoli and results in increased functional residual capacity. So the normocapnic lung-protective OLV results in significantly higher PaO₂ than permissive hypercapnic OLV.

References

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P176

Titration of analgosedation with neurally adjusted ventilatory assist in the ICU

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Introduction The patient-ventilator asynchrony (PVA) is a cause of over sedation that prolongs mechanical ventilation unnecessarily. The current tools for measurement of sedation are inadequate for assessing the PVA. Neurally adjusted ventilatory assist (NAVA) is an innovative ventilatory mode that provides an excellent real-time monitor of the neural signal of diaphragmatic electrical activity (EAdi) and consequently highlights the PVA. Whether EAdi can be of help to titrate the level of sedation has not yet been proved, so we want to verify this conjecture. To titrate the level of analgosedation, we used this signal, which informs us continuously on changes in lung mechanics and synchrony.

Methods A prospective observational study on 50 coma patients, ventilated with Maquet SERVO-I, was performed, following monitoring chart EAdi and recording the numerical values of Edi peak and Edi min during the different ventilatory modes. We recorded the analgosedation via continuous infusion; the dose was titrated to achieve a score of the Richmond Agitation-Sedation Scale from -2 to +1 and the Behavioral Pain Scale \leq 4.

Results The average duration of mechanical ventilation was 5.9 days ($P = 0.004$), the average of analgosedation was 4.8 days while the average length of stay was 6.4 days ($P = 0.02$). The average dose of remifentanyl was varied between 0.075 ± 0.025 μ g/kg/minute, propofol 0.5 ± 0.2 mg/kg/hour and clonidine 0.025 ± 0.02 μ g/kg/minute. Comparing the pressure, volume and EAdi traces we identified all