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Background: Ventilator-induced lung injury (VILI) remains a major challenge associated with mechanical ventilation. A primary mechanism underlying VILI is cyclic alveolar recruitment and derecruitment (R/D) (atelectrauma). To minimise its development, ventilator settings, such as Positive End-Expiratory Pressure (PEEP), must be carefully optimised for each patient. Despite this need for personalisation, current bedside techniques cannot continuously measure local alveolar dynamics. Consequently, clinicians must rely on global parameters, which may not fully capture the heterogeneity of regional lung behaviour. In silico modelling offers a promising solution by linking local alveolar mechanics with global lung function. We sought to investigate whether our in silico modelling framework could simulate the effect of an impaired lung on R/D behaviour. Methods: This work leverages a lumped-parameter model presented in [1], which estimates patient-specific regional elastance and recruitment information derived from dual-volume CT in a mechanically ventilated ARDS patient. The model was used to simulate mechanical ventilation during an incremental PEEP trial consisting of 30-second steps at 5, 10, and 15 cmH₂O. Next, the mean critical pressures were shifted by +5cmH₂O to mimic a severe lung injury, and the trial was repeated. Results: The shifted-critical-pressure trial demanded higher PEEP to maintain alveolar aeration throughout the respiratory cycle. It was observed that tidal recruitment remained high and unstable at 10cmH₂O and was significantly reduced only at the higher PEEP of 15cmH₂O, while it was already reduced at 10cmH₂O in the control simulation. Conclusion: In this study we utilised a methodology that infers local lung properties derived from dual-volume CT. A proof-of-concept simulation shows that the model can reproduce the effect of conventional changes in ventilator settings, such as PEEP, as well as can simulate an impaired lung with worsening biomechanics. As such, this scalable strategy provides a mechanistic foundation for future personalised ventilator optimisation to reduce atelectrauma.

REFERENCES

1. Quicken et al., ESB 2025 conference.

Beyond pulmonary embolism: right ventricular failure and cardiac arrest in decompensated portopulmonary hypertension

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Introduction: Acute right ventricular (RV) failure is a challenging condition in critical care associated with significant morbidity. Portopulmonary hypertension, a severe and under-recognized complication of liver cirrhosis, is an important cause of right-sided heart failure in chronic liver disease. We present the case of a 69-year-old female with cirrhosis who developed cardiac arrest secondary to acute decompensated right ventricular (RV) failure likely due to underlying portopulmonary hypertension. Case report: A 69-year-old female with Child Pugh B7 Hepatitis B cirrhosis and recurrent hepatic hydrothorax presented with syncope and exertional dyspnea. Imaging demonstrated a massive right pleural effusion, which was drained uneventfully. Computed tomography pulmonary angiography excluded pulmonary embolism but revealed right heart strain. The following day, while straining during defecation, she developed sinus bradycardia progressing to pulseless electrical activity arrest. Return of spontaneous circulation was achieved after 14 minutes. Bedside echocardiography demonstrated severe RV dilation with impaired systolic function (TAPSE 11 mm), septal flattening with left ventricular cavity obliteration, severe tricuspid regurgitation, and (estimated) elevated pulmonary vascular resistance. Lactate exceeded 10 mmol/L despite a preserved indirect Fick cardiac index, consistent with acute RV failure and obstructive shock physiology. Acute decompensated RV failure, likely secondary to portopulmonary hypertension, was diagnosed. She required escalating vasopressor support and developed multi-system organ failure. Management targeted RV physiology: preload reduction with continuous renal replacement therapy, inotropic agents, pulmonary vasodilators, preferential vasopressin use, and ventilatory strategies avoiding high PEEP and hypercapnia. Despite transient echocardiographic improvement, she experienced recurrent arrests. Care was subsequently re-directed to palliation. Conclusion: This case underscores the importance of early recognition of RV-predominant shock physiology in cirrhosis and reinforces the need for timely identification of portopulmonary hypertension to mitigate the risk of acute decompensation.

Cultural sensitive interventions and physiological parameters in intensive care unit: a narrative review

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