Mechanical power and Acute Kidney Injury: is there a link?

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Argomento: Insufficienza respiratoria acuta e ventilazione meccanica

Introduction

Mechanical power (MP) is increasingly recognized as a unifying view on Ventilator Induced Lung Injury.

The injurious effects of positive pressure ventilation on the kidney are known, but their understanding is mostly limited to the negative hemodynamic effect of an increased CVP on the renal perfusion. In this work, we explore the lung-kidney interaction in the light of the MP framework.

Methods

78 pigs weighing 23,8 ± 2,2 kg were ventilated with a large spectrum of ventilator settings, with a respiratory system MP ranging from 7,8 J/min to 60,9 J/min. Measurements about gas exchange, acid-base balance, lung mechanics and biochemistry were obtained at baseline, 6, 12, 24, 48 hours. AKI was defined following KDIGO guidelines as 1,5 times increase from the baseline serum creatinine.

Results

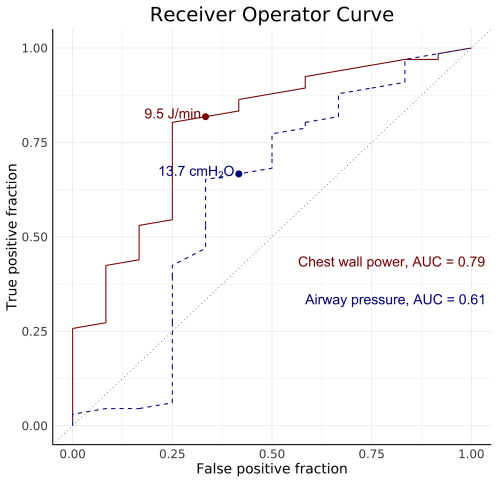
Of all the variables analyzed the mean chest wall mechanical power was the best predictor of AKI development, with an area under the ROC curve of 0,79. We identified a threshold of 9,5 J/min that maximizes sensitivity (80%) and specificity (75%). The population, divided in two groups (above and below threshold), showed different time course in serum creatinine, urine output, uremia, base excess and pH.

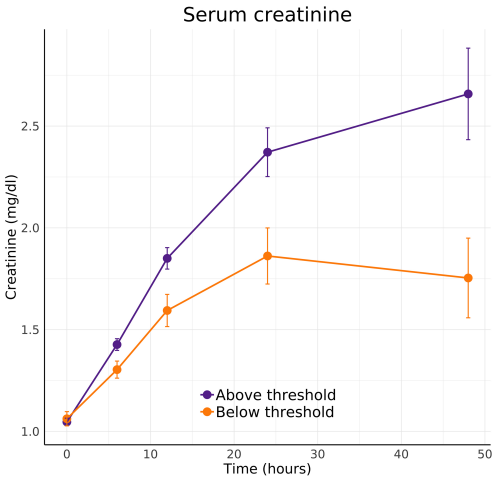
Discussion

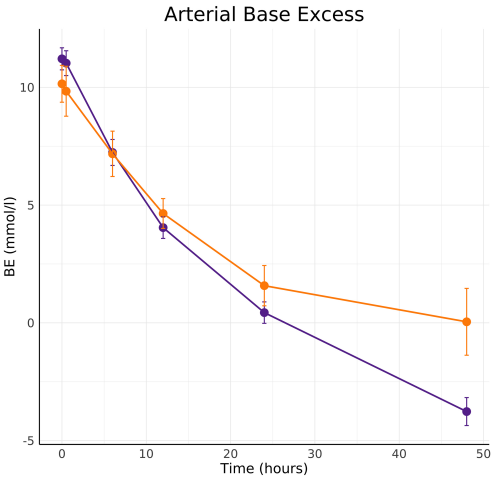
Chest wall mechanical power is the energy delivered to the pleural space during mechanical ventilation. The relevant finding that power was related to AKI even more than airway pressure triggers more doubts than answers. The data provided support the idea that a mechanism other the hemodynamic veno-congestion may concur to AKI development and that the time course of pressure-volume swings within the thorax should not be underestimated.

Conclusions

In an experimental model, chest wall mechanical power was the best predictor of AKI development.


Receiver Operator Curve
True positive fraction
False positive fraction
9.5 J/min
13.7 cmH2O
Chest wall power, AUC = 0.79
Airway pressure, AUC = 0.61


Serum creatinine
Creatinine (mg/dl)
Time (hours)
Above threshold
Below threshold


Arterial Base Excess
BE (mmol/l)
Time (hours)

This might be particularly relevant in the clinical management of patients with high chest wall elastance where AKI could arise even before VILI.