

## Letter

# Myocardial injury associated with hyperinflation of the lung

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A recent study [1], conducted in an animal model of acute lung injury, compared a ventilation strategy in which lungs were inflated by brief application of 50 cmH<sub>2</sub>O pressure followed by increased positive end-expiratory pressure (recruitment strategy) versus conventional ventilation. The study identified an increased inflammatory reaction in liver sinusoids, and increased serum aspartate aminotransferase (AST) and hyaluronate levels with the recruitment strategy. The authors speculated that deficient oxygen delivery and increased sinusoidal pressures may have caused liver injury. The findings of the study suggest an origin of AST outside the liver because there was no elevation in alanine aminotransferase levels, hepatocellular necrosis, or liver dysfunction. Animals in the group receiving the recruitment strategy required volume support and had temporarily reduced cardiac output. This indicated that the origin of AST elevation was a myocardial injury.

Previous studies found that patients with acutely elevated intrapulmonary pressure resulting from bronchospasm [2] and severe respiratory syncytial viral lung disease leading to hyperinflation [3] exhibited evidence of myocardial injury, such as elevated cardiac troponin levels; this was probably due to strain imposed on the right ventricle by pulmonary hypertension. Ventricular strain has previously also been associated with elevated hyaluronate levels [4]. The increase in hepatic neutrophils may be due to a hyperoxia-induced increased inflammatory response found in patients with increased positive end-expiratory pressure and fractional inspired oxygen (FiO<sub>2</sub>) [5]. Future studies need to use specific markers for myocardial injury such as cardiac troponin T in order to discriminate between myocardial and hepatic injury associated with the various ventilation strategies.

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## Authors' response

Markus Kredel and Christian Wunder

We thank Dr Eisenhut for his comments. He raises the issue of whether myocardial injury was the origin of the increased serum AST levels observed in animals treated with the recruitment manoeuvre (RM). Although we did not measure troponin T levels, there was no evidence for myocardial injury during the performed RM. Haemodynamic variables, as shown in Table 1 of our report [1], and mixed venous oxygen saturation (data not shown) were comparable in the two groups at all time points. In addition, the expected and observed increase in central venous pressure and decrease in cardiac output during the RM did not result in differences in pulmonary artery pressure. This casts right ventricular strain into doubt.

In addition to nonspecific transaminases and hyaluronic acid, bilirubin was also elevated in the systemic circulation in the group undergoing the RM. High positive end-expiratory

pressure is known to be an independent risk factor for liver dysfunction, as indicated by hyperbilirubinemia [6].

Regarding the histologic examination, Dr Eisenhut speculates that the increase in liver neutrophils might be due to hyperoxia. The arterial oxygen tension (PaO<sub>2</sub>)/FiO<sub>2</sub> ratio (Table 2 in our report [1]) was improved in the RM group, but PaO<sub>2</sub> levels were even lower after FiO<sub>2</sub> was reduced to 0.4 following the RM. In contrast, in the animals that did not receive the RM the FiO<sub>2</sub> was fixed at 1.0, despite improvement in PaO<sub>2</sub> during the trial. In addition, McClintock and coworkers [5], examining urinary nitric oxide excretion in a subgroup of patients included in the ARDS-Network study, did not address hyperoxia-induced inflammatory responses. Nevertheless, we cannot exclude the possibility that the inflammatory reaction in the liver was a remote effect of ventilation.

## Competing interests

The author declares that they have no competing interests.

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