Letter

Vital organ blood flow during high-frequency ventilation

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See related research by David *et al*., http://ccforum.com/content/10/4/R100

We read with interest the article by David and colleagues [1] comparing the effects of high-frequency oscillatory ventilation with pressure controlled ventilation with respect to vital organ blood flow in an animal model of acute lung injury. However, there are some points we wish to comment on.

Firstly, the authors state that their study is limited due to the lack of more frequent blood flow measurements, as all parameters were measured only once thirty minutes after switching to a new mean airway pressure (P_{mean}). Indeed, changes in hemodynamic variables are most pronounced straight after increasing mean airway pressure [2]. It has been shown that cardiac output initially decreased substantially after an increase in positive end-expiratory pressure (PEEP), but that it adapted to the increased PEEP thereafter due to dynamic hemodynamic changes [3]. Consequently, compensatory mechanisms missed by insufficient data sampling could explain the only slightly decreased cardiac output and unchanged organ blood flow seen in the study by David and colleagues [1]. Therefore, it is of paramount importance to investigate parameters of individual organ perfusion more frequently, and obtain variables of tissue oxygenation and metabolism. Furthermore, brain tissue is extremely susceptible to ischemia, and even a few minutes of compromised cerebral perfusion affect the rate of cerebral oxygen metabolism and tissue integrity. To elucidate the impact of mechanical ventilation on brain tissue, the authors should have analyzed cerebral tissue biochemistry [4] or established biomarkers of cerebral ischemia, such as S-100β or neuron-specific enolase.

Secondly, cardiac filling pressures have repeatedly been shown to only poorly reflect instantaneous cardiac preload. Right ventricular end-diastolic volume and global end-diastolic volume have been demonstrated to be clearly superior for this than cardiac filling pressures, particularly at high intrathoracic pressures in a model of acute lung injury [5], and would have provided more detailed information regarding interaction of recruitment manoeuvre, preload and organ perfusion.

Competing interests

The authors declare that they have no competing interests.

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