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CORRESPONDENCE

Vascular-induced lung injury: another advocate for personalized ARDS management

Discussion on "Inspiratory preload obliteration may injure lungs via cyclical 'on-off' vascular flow"

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Initial correspondence from Dr. Roshdy

Dear Editor,

In a recent issue of *Intensive Care Medicine*, Katira et al. hypothesized pulmonary circulation as the culprit rather than victim of acute respiratory distress syndrome (ARDS) [1]. Based on their recent animal study, cyclical "on-off" flow induces lung injury by means of shear stress and ischemia-reperfusion [1, 2]. The concept is interesting along with its clinical implications.

Intermittent flow can be due to increased right ventricular (RV) pre- or afterload. Simple eyeballing by echocardiography can differentiate both by means of RV dilatation and septum motion. The authors attributed the phenomenon to the first mechanism; however, studies are needed to test whether both mechanisms share the same deleterious effect. Fluid administration can recruit collapsed pulmonary vascular units in ARDS, avoiding reperfusion injury but also reducing shear stress on other units [3]. This fluid-protective role clearly contradicts an increasingly advocated fluid restriction strategy in ARDS [4]. It is true too that fluid can raise the pulmonary hydrostatic pressure. As the balance can shift rapidly from benefit to risk, monitoring is highly recommended (e.g., extravascular lung water, lung ultrasound).

Large swings of pulmonary flow have also been blamed for ventilator-induced lung injury (VILI) [1]. Spontaneously breathing patients, if distressed, can manifest such major swings. As the driving pressure in spontaneous breathing is negative, "off flow" is not expected to occur. Nevertheless, extreme swings can aggravate ARDS by means of shear stress and increased hydrostatic pressure. One solution is the early application of positive pressure ventilation, either invasively or non-invasively, or even minimally by a high flow nasal cannula (HFNC). This can alleviate distress, diminish sympathetic drive and create PEEP, which in turn control pulmonary flow. The drawback can be the generation of an injurious large transpulmonary pressure (sum of the patient's spontaneous negative pressure and the ventilator's positive pressure) [5]. High PEEP is suggested to protect from VILI, but sedation and paralysis with invasive mechanical ventilation may ultimately be warranted to protect the lungs [6].

To sum up, vascular-induced lung injury is shedding light on the complex pathophysiology of ARDS. To meet the challenge, our management should become more personalized, dynamic and based on best evidence as well as sound understanding of a complex pathophysiology. In contrast to oncology where precision medicine is based on molecular and genetic analysis, real-time monitoring and point-of-care imaging can be our precision tools. Vascular-induced lung injury has already been considered by experts, but is expected to gain more attention in the future [7]. Finally, as outcome is correlated with early management, there is a genuine need to transfer knowledge, tools and skills to emergency units.

Reply from Drs. Katira, Kuebler and Kavanagh

We thank Dr. Roshdy for the interest in our article. We agree that the interactions among transpulmonary pressure, ventricular preload, pulmonary vascular flow and lung injury are complex and that, as translational research advances, these concepts may well facilitate better patient care. We advise caution in considering that

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a positive fluid balance will necessarily prevent pulmonary flow interruption—or prevent ventilator-associated lung injury—and prudence is required as every increase in intravascular volume will promote fluid extravasation across a leaky endothelium in the lung. That notwithstanding, we agree that in the future it is likely that care in ARDS will become progressively more individualized.

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Compliance with ethical standards

Conflicts of interest
The corresponding author states that there is no conflict of interest.

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