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Monitoring of oxygen supply and demand during veno-venous extracorporeal membrane oxygenation

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Dear Editor,

We congratulate Dr. Levy and his co-workers for their excellent overview of the treatment possibilities of persistent hypoxemia during veno-venous extracorporeal membrane oxygenation (ECMO) [3]. The authors discuss a multi-step approach in order to optimize oxygen delivery in this critical situation. They propose a clinical algorithm starting with an arterial oxygenation saturation (SaO_2) <88 %. However, one needs to be very careful when using the SaO_2 as a measure of hypoxemia, particularly in critically ill patients. As the authors correctly state, the targeted SaO_2 (>80–88 %) during ECMO therapy remains unclear and needs to be adapted to the hypoxic tolerance of each patient. Most importantly, the SaO_2 as a parameter of hypoxemia does not necessarily reflect tissue oxygenation, as the affinity of oxygen to hemoglobin may be increased and tissue oxygen delivery decreased under certain circumstances (leftward shift of the oxyhemoglobin dissociation curve). In addition, the SaO_2 does not reflect oxygen consumption, which might be critically elevated during a severe ARDS and sepsis.

Most notably, changes in cerebral tissue oxygenation reflect this dilemma during ECMO. A swift removal of carbon dioxide through the extracorporeal circuit during the initial phase of ECMO is associated with a rise of pH, which in turn results in cerebrovascular vasoconstriction in conjunction with a leftward shift of the oxyhemoglobin dissociation curve [2]. Both factors may severely impede cerebral tissue perfusion and oxygenation despite increased SaO_2 values under ECMO support. Although this scenario is due to rapid changes in pH and may not persist during on-going ECMO therapy, it emphasizes the complexity of the pathophysiological changes as well as the fact that tissue hypoxia cannot be fully assessed using a single parameter of hypoxemia such as the SaO_2.

We believe that the SaO_2 should be interpreted in conjunction with several other parameters (e.g., hemoglobin, mixed venous oxygen saturation and cardiac output) in order to correctly assess the oxygen requirement of each patient. Moreover, monitoring of cerebral tissue oxygenation would be desirable during ECMO therapy. In this regard, near-infrared spectroscopy (NIRS) represents a non-invasive technique that allows a continuous monitoring of the regional cerebral oxygen saturation via the frontal cortex [4].

Hence, we propose to include important parameters indicating a mismatch of tissue oxygen supply and demand such as lactate and mixed venous oxygen saturation, as well as cerebral tissue oxygenation into the otherwise excellent algorithm by Dr. Levy and his co-workers. Nevertheless, it is important to keep in mind that hypoxemia as reflected by a drop in SaO_2 is by far the most common cause of tissue hypoxia, and that there is an association between progressively lower arterial oxygen tensions and increasing in-hospital mortality [1].

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