

“Life-threatening” Hypoxemia in One-lung Ventilation

To the Editor:

In January's ANESTHESIOLOGY, Rozé *et al.*¹ describe a thorough and step-wise approach to the management of hypoxemia in one-lung ventilation. Although we agree that “life-threatening” hypoxemia should be treated with resumption of bipulmonary ventilation, we question the definition of life-threatening hypoxemia based only on an arterial oxygen saturation (SpO₂) less than 90%.

Physiologically, end-organ injury caused by inadequate oxygen delivery (DO₂) is dependent on the product of arterial oxygen content (CaO₂) and cardiac output. SpO₂ is only a single component of CaO₂, along with hemoglobin concentration. Thus, in addition to SpO₂, we would like to illustrate the importance of considering hemoglobin concentration and cardiac output before aborting one-lung ventilation.

We agree that an SpO₂ less than 90% may be tolerated poorly in an anemic patient; however, for patients with a normal or high hemoglobin concentration, oxygen content can be maintained at much lower oxygen saturations. It is well known that polycythemia is a compensatory mechanism for hypoxia in people native to high altitudes, with their hemoglobin concentrations being on average 5 g/dl higher than that of their counterparts residing at sea level.² Transfusion of erythrocytes is associated with known complications, but increasing hemoglobin concentration *via* transfusion is associated with decreased work of breathing and minute ventilation in ventilated patients with chronic obstructive pulmonary disease³ and increased successful weaning from mechanical ventilation in anemic patients with chronic obstructive lung disease.⁴

With regard to overall oxygen delivery, cardiac output is another key factor. Although Rozé *et al.* comment briefly on the development of right ventricular dysfunction with hypoxic pulmonary vasoconstriction, vigilance should be kept to maintaining a normal cardiac output in the face of decreased oxygen saturation. Increased cardiac output is a known compensatory mechanism to hypoxia⁵ with additional beneficial effects separate from increased oxygen delivery, such as decreased dead-space ventilation. In addition, although Rozé *et al.* discuss the use of vasodilators to treat hypoxic pulmonary vasoconstriction, dobutamine has been shown to increase oxygen delivery significantly more than does prostacyclin.⁶ Pharmacologic assistance may be needed to maintain a sufficient cardiac output, and patients with low cardiac output states may indeed require oxygen saturations much greater than 90%.

Although in a different population than those with one-lung ventilation but having similar physiologic principles,

* Extracorporeal Life Support Organization (ELSO): ELSO guidelines for extracorporeal life support, Version 1:1. 2009. Ann Arbor, Michigan. <http://www.else.med.umich.edu/Guidelines.html>. Accessed March 11, 2011.

recommendations for management of patients with acute respiratory distress syndrome requiring extracorporeal life support include maintenance of an SpO₂ greater than 80% and a arterial oxygen concentration of 40 mmHg, provided oxygen content is adequate (hematocrit more than 40%) and cardiac function is not threatened.* We have applied similar principles to patients with acute respiratory distress syndrome without extracorporeal life support. In the event of satisfactory hemoglobin concentrations and cardiac function, we have accepted an SpO₂ between 85% and 90% in patients with severe acute respiratory distress syndrome without finding evidence of end-organ malperfusion. In an extreme case, we cared for an 42-yr-old, previously healthy woman with H1N1 infection with superimposed Pseudomonas pneumonia. This patient did not achieve an SpO₂ greater than 90% for more than 10 days despite advanced ventilation maneuvers and pharmacologic therapies (including inhaled nitric oxide); however, oxygen delivery was maintained through cardiac output and oxygen content (hemoglobin goal, more than 12 g/dl). The patient recovered from the acute pathophysiology with no long-term end-organ damage or signs of neurologic impairment.

A complete discussion of mechanisms to increase organ oxygenation is beyond the scope of this letter. Essentially, vigilant consideration must be given to a myriad of parameters, including peak and plateau airway pressures, tidal volumes, rate of cycle delivery or flow, inspired oxygen concentrations, acid–base, markers of end-organ perfusion, cardiac output or ventricular function, and hemoglobin concentration, rather than choosing an SpO₂ of 90% as an arbitrary point of discontinuation of a surgical procedure, sometimes treating “life-threatening” disease.

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