To the Editor,

I read with interest the recent article “Successful Management of Membrane Oxygenator Failure during Cardiopulmonary Bypass: The Importance of Safety Algorithm and Simulation Drills” (JECT 2012;44:78–80). It is commendable and prudent to be prepared for this rare and dangerous risk associated with cardiopulmonary bypass. I am compelled to remark and raise some questions regarding this case report.

I begin by referring to the end of the case report by saying that I agree with the final paragraph that suggests that written protocols and algorithms should be put in place along with simulation so that when a situation such as that described occurs, the perfusionists, surgeons, and anesthesiologists are prepared to diagnose the problem and choose the safest pathway for rectification.

It appears from the information volunteered by the authors that the diagnosis of oxygenator failure was made from the following: 1) progressing respiratory acidosis; 2) “no known history of lung disease and lifelong nonsmoker”; and 3) rule out malignant hyperthermia.

These are not the traditional signs of oxygenator failure and so I would like to address these points in order.

1) The pCO₂ of 8.35 kPa is a respiratory acidosis, which could be expected in the case of true oxygenator failure. Oxygenator failure, like respiratory failure, can be defined as arterial hypoxia that may or may not be accompanied by hypercapnia. Hypoxia can be caused by low FiO₂, ventilation-perfusion mismatch, right to left shunts, a diffusion defect, or hypoventilation. Hypercapnia on the other hand is caused by hypoventilation alone. Any CO₂ increase caused by an oxygenator failure would be expected to be accompanied by a simultaneous fall in the arterial pO₂. However, the authors stated that the patient “was not hypoxic” and the data presented show that the paO₂ increased from 30.7 kPa (230 mmHg) to 60 kPa (450 mmHg) at the same time that the pCO₂ was rising. This evidence seems to contradict the diagnosis of oxygenator failure and points to hypoventilation as the cause of the increase in pCO₂.

2) It is perplexing that the authors found it salient to mention “no known history of lung disease and lifelong nonsmoker” as evidence to eliminate one potential cause of an elevated pCO₂. The condition of the lungs would have no negative impact on the blood gas parameters while the patient was on cardiopulmonary bypass, because the lungs are being bypassed.

3) Malignant hyperthermia (MH) should be suspected in a patient on bypass with an unexplained rise in the pCO₂. Because this, along with an increase in serum lactate and a drop in the SvO₂, has been identified as an early sign of the disease. Ruling out MH based on a patient who was “normothermic,” “not hypoxic” (see #1 above) “and had good muscular tone” could easily lead to a misdiagnosis.

Hyperthermia is a relatively late manifestation of MH and is easily masked in the patient on cardiopulmonary bypass (CPB) as a result of artificial cooling and heating. The patient was not hypoxic, but the increased oxygen consumption that is a hallmark of MH can also be masked by CPB and is better detected by monitoring the SvO₂ and oxygen use, which is not reported in this article. Muscular rigor is also one of the signs of MH that is easily masked in patients undergoing CPB, because the patients are generally paralyzed, which makes the assessment of “good muscular tone” difficult at best. The reported lactate levels did not increase and lends support to the assumption that MH was not present.

The previous three points are not diagnostic of oxygenator failure but strongly suggest hypoventilation of the oxygenator as the cause of the hypercarbia. The progressive rise in the pCO₂ could easily have been caused by a loose connection or leak anywhere in the gas delivery system. It appears that what was needed in this scenario was an algorithm to diagnose oxygenator failure in addition to the reported procedure to change out the oxygenator.

To better understand the circumstances faced by the perfusionists in this case report, additional information would have been useful for the reader and necessary to diagnose oxygenator failure. What were the patient parameters, including height, weight, and hematocrit? What was the blood flow, gas flow, FiO₂, and patient temperatures as the case progressed? Were all gas line connections checked from the source to the oxygenator? Was the patient’s level of anesthesia adequate? Were arterial and venous blood gas analysis performed and what were the calculated oxygen and carbon dioxide transfer rates?

The authors state in the Abstract that this case report presents “real-life aspects to managing” oxygenator failure “without added risk to the patient.” Changing out an oxygenator poses a very significant risk to the patient under the best of conditions even when a safety algorithm and simulation drills have been implemented. The most important aspect of oxygenator algorithms and change-out drills is to train teams to quickly rule out gas supply...
delivery or blood path issues to confirm oxygenator failure before embarking on an unnecessary oxygenator changeout. To engage in changing out an oxygenator while a patient is on bypass when it is unnecessary exposes the patient to substantial risk. Likewise, unnecessary delays may prove equally hazardous.

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