Comments on Point:Counterpoint: Hypoxia is/is not the optimal means of reducing pulmonary blood flow in the preoperative single ventricle heart

COMMENT ON POINT: COUNTERPOINT

To the Editor: Optimal may depend on the circumstances and both points have utility (1, 3). We found carbon dioxide manipulation more useful and less dangerous than alveolar hypoxia in acute transitional settings when mechanical controlled ventilation is required in unrepaired ductal or shunt-dependent pulmonary blood flow (2). The technical aspects of transitioning from spontaneous to controlled mechanical ventilation (anesthetic induction, bag/mask, endotracheal intubation) includes inability to precisely control minute ventilation and requires variable, albeit brief, apnea. Alveolar hypoxemia prior to apnea quickly leads to systemic desaturation and bradycardia and attempts to then quickly deliver oxygen to the alveoli, resulting in hypocapnia, decreasing pulmonary vascular resistance, and transferring of volume to the pulmonary circulation. In the operating room, body temperature and carbon dioxide production decline and hypothermia-induced systemic vasoconstriction combine to shift blood flow to the lung. These observations resulted in using added CO2 along with normoxic inspired gas, which maintained circulatory stability and avoided metabolic acidosis and desaturation-related events. We also observed that the circulatory response to inspired CO2, both increase and decrease, was much more rapid than changing inspired oxygen. This allows more precise control to affect a balanced circulation in acute situations. Understanding that enabling utilization of oxygen and not to suppress energy metabolism— one would expect hypercapnic acidosis only to depress oxidative metabolism and jeopardize cellular energy homeostasis. The purpose of increasing oxygen delivery to tissues is to enable utilization of oxygen and not to suppress energy metabolism.

REFERENCES


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To the Editor: I am grateful for the opportunity to comment concerning methods that limit pulmonary blood flow in patients with congenital heart disease, mixing of venous return, and parallel pulmonary and systemic arterial circulations.

It is concerning that Ebenroth (2) and Liske and Aschner (3) occasionally used the term “hypoxia” inappropriately in their comments. The effects of “alveolar hypoxia” and “systemic arterial hypoxemia” are quite distinct, particularly in the setting of congenital heart disease.

Breathing additional nitrogen or carbon dioxide acutely decreases pulmonary blood flow and improves the volume load of the heart in appropriate patients (1, 4). Both agents may have a beneficial effect on cerebral blood flow (1, 4). Long-term effects are not well known.

Patients are heterogeneous with respect to clinical presentation and the intensity of preoperative care. Individuals may have different short- and long-term needs for balancing the distribution of blood flow between the lung and body.

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To the Editor: Optimal may depend on the circumstances and both points have utility (1, 3). We found carbon dioxide manipulation more useful and less dangerous than alveolar hypoxia in acute transitional settings when mechanical controlled ventilation is required in unrepaired ductal or shunt-dependent pulmonary blood flow (2). The technical aspects of transitioning from spontaneous to controlled mechanical ventilation (anesthetic induction, bag/mask, endotracheal intubation) includes inability to precisely control minute ventilation and requires variable, albeit brief, apnea. Alveolar hypoxemia prior to apnea quickly leads to systemic desaturation and bradycardia and attempts to then quickly deliver oxygen to the alveoli, resulting in hypocapnia, decreasing pulmonary vascular resistance, and transferring of volume to the pulmonary circulation. In the operating room, body temperature and carbon dioxide production decline and hypothermia-induced systemic vasoconstriction combine to shift blood flow to the lung. These observations resulted in using added CO2 along with normoxic inspired gas, which maintained circulatory stability and avoided metabolic acidosis and desaturation-related events. We also observed that the circulatory response to inspired CO2, both increase and decrease, was much more rapid than changing inspired oxygen. This allows more precise control to affect a balanced circulation in acute situations. Understanding that both oxygen and CO2 will independently and synergistically affect pulmonary vascular resistance will optimize management of these patients as they transition through the environments and reconstructive treatments that are required.

REFERENCES


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To the Editor: I am grateful for the opportunity to comment concerning methods that limit pulmonary blood flow in patients with congenital heart disease, mixing of venous return, and parallel pulmonary and systemic arterial circulations.

It is concerning that Ebenroth (1) and Liske and Aschner (2) seem to agree only that hypoxia is the optimal therapeutic strategy for decreasing pulmonary blood flow in patients with unrepaired circulation. In addition, Liske and Aschner (2) contend that in babies who are critically ill and who eventually require mechanical ventilation, the induction of hypercapnic acidosis offers advantages that hypoxic gas ther-
On the basis of the findings of Tabbutt and associates, Liske and Aschner claim that treatment with carbon dioxide may improve oxygen delivery more than treatment with nitrogen (3). They have potentially overstated the findings of a study that included patients with unmeasured amounts of antegrade blood flow across the aortic valve (4). Oxygen delivery was adequate during treatment with nitrogen (4). When necessary, treatment with nitrogen is easy to administer and monitor for short and long periods of time. It is only practical to use carbon dioxide for short periods of time. It is also helpful to consider the oxygen carrying capacity of the blood and maintain an adequate hematocrit whether nitrogen or carbon dioxide is used.

REFERENCES


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