Permissive hypercapnia: Is there any upper limit?

Sir,

We read the article by Garg SK "Permissive hypercapnia: Is there any upper limit?"[1] with great interest. The author has rightly focused on a current protective lung ventilation strategy. Though there are several reports of extreme levels of hypercapnia tolerated by patients no safe upper limit has been described. We would like to add following facts.

Permissive hypercapnia allows for low-tidal volume ventilation and hence minimizes ventilator associated lung injury. It also reduces inflammatory response in lungs by attenuating free radical formation, nuclear factor kappa-β activation and increased tumor necrosis factor-β, interleukin-1, 6, 8 production. On the contrary, it impairs plasma membrane wound healing in injured lungs in a pH dependent fashion[2] and also prevents alveolar fluid resorption by inhibiting Na-K ATPase of alveolar epithelial cells. Sustained hypercapnia in cases of pulmonary infection increases bacterial load and may further aggravate lung injury. Hence in conditions of sepsis associated lung injury permissive hypercarbia would be beneficial. Elevated CO_2 level also induces mitochondrial dysfunction and impairs alveolar epithelial cell proliferation. Hypercapnic acidosis increases ventilatory drive and if patient is not properly sedated and paralyzed, patient-ventilator dyssynchrony may occur and result in increased transpulmonary pressure. Previous animal studies have reported vasogenic cerebral edema in hypercapnic acidosis and in human beings cerebral edema have been reported in severe respiratory acidosis associated with asthma.[3] Hypercapnia has a beneficial effect on brain in an optimal range. Lowering CO_2 causes cerebral vasoconstriction whereas increased CO_2 results in cerebral edema. Hence, like any other drug "permissive hypercapnia" should

be used judiciously and under strict hemodynamic monitoring.

Hypotension in this patient may not be attributed to hypercapnic acidosis. Though hypercapnic acidosis has a negative inotropic effect it increases net cardiac output by sympatho-adrenal stimulation and reduction in systemic vascular resistance.[4]

Usually hypercapnic metabolic acidosis doesn't require buffering as it has less effect on the cardiovascular system. In situations where hemodynamic compromise occurs buffering should ideally be done by tris-hydroxy-amino-methane (THAM/Tromethamine), as it does not release CO_2 and more effective in the correction of pH. Weber *et al*. have also found that THAM attenuates the reversible myocardial depressant action of permissive hypercapnia in patients with acute respiratory distress syndrome.[5]

Last but not the least, it is not clear from the case whether the author tried recruitment maneuvers or how did they achieve optimum positive end-expiratory pressure as a strategy for open lung ventilation and preventing atelectrauma. Moreover, managing elevated $PaCO_2$ by increasing tidal volume is now known to be unacceptable; however, management by increasing the respiratory rate, although common is of uncertain impact. For example, increasing respiratory rate frequency from 17 to 35/min adds over 25,000 additional opening and closing cycles per day to an already injured lung, and laboratory data suggest that this approach is associated with additional lung injury.[6]

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