Letter to Editor

HYPERCARBIA INDUCED OXYGENATION OR OXYGEN INDUCED HYPERCARBIA

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Dear Sir,

Improved arterial oxygenation through permissive hypercarbia (45–50 mmHg) has recently been reported during one-lung ventilation (OLV) in patients undergoing thoracic surgery.[1] In this study after the initiation of OLV, group I patients were first kept at normocarbia (PaCO2: 38-42 mmHg) for 30 min, and thereafter at hypercarbia (45–50 mmHg) for the same duration by reducing the respiratory rate (RR) titrated by targeted end-tidal carbon-dioxide (CO2) levels at a constant tidal volume; vice-versa was followed in group II. They observed that hypercarbia improved oxygenation and pulmonary mechanics during OLV. It induced pulmonary vasoconstriction in the non-dependent non-ventilated lung while decreased pulmonary vascular resistance in the dependent ventilated lung; thus, attributing to improved oxygenation.[1] Even a minor increase in arterial CO2 (PaCO2) is known to induce direct pulmonary vasoconstriction (0.6-1 Torr change in pressure gradient/Torr change in end-tidal CO2 or PaCO2) despite normal oxygenation levels.[2] Increase in PaCO2 from 20-65 mmHg causes a linearly increased bronchodilation in anesthetized, paralyzed subjects.[3] It also induces systemic vasodilation, increased cardiac output, reduced pulmonary inflammation, lower alveolar oxidative stress, increased hematocrit, and increased oxygen delivery leading to overall better oxygenation.[4,5] However, other co-factors that could reduce arterial oxygenation in above study include reduced MV to the dependent lung to achieve hypercarbia and decreased overall surface area for gaseous exchange to half during OLV [compared to two-lung ventilation (TLV)], regardless of same delivered tidal volume. We also noted that the authors did not oxygenate the non-dependent lung during OLV. With chest opened after initiating OLV, there is an initial collapse of non-dependent lung to a point at which smaller airway close. Further atelectasis occurs slowly and in a patchy manner by the ongoing capillary oxygen uptake. As hypoxic pulmonary vasoconstriction (HPV) is a local mechanism diverting blood away from hypoxic regions, pulmonary blood flow is diverted from atelectatic to still aerated areas.[6] Adding apneic oxygen insufflation (AOI) to non-ventilated lung during OLV will better oxygenate these areas. Thus, it will lead to better overall oxygenation and delayed induction of HPV in still aerated lung units, while HPV is not-blunted in atelectatic areas of non-dependent lung.[6,7] The other co-factors that may increase HPV include decrease in cardiac output/pulmonary artery pressure, catecholamine release and high positive end expiratory pressure. Effect of acidosis is not clear as it causes vasoconstriction in both pulmonary beds, but alkalosis opposes HPV. During TLV with closed-chest, an AOI>0.3 L/kg/min is required for adequate CO2 removal.[7] Though an AOI of just 1-3 Liters/min to the non-dependent lung during OLV is expected to improve arterial oxygenation but is per se insufficient to effectively remove CO2 and causes hypercarbia. A higher ventilation/perfusion mismatch (higher dead space with increased perfusion) in non-ventilated lung reduces the CO2 excretion and results in higher PaCO2 values. Due to CO2 retention within the pulmonary vasculature, the arteriovenous CO2 gradient gets reversed, with arterial PaCO2 exceeding the venous values. This effect is further compounded by the Haldane effect wherein arterial oxygenation resulting from apneic oxygenation displaces the hemoglobin-CO2 dissociation curve to the right, releasing CO2 from

hemoglobin and results a further rise in arterial PaCO2.[8] Thus, AOI to the non-dependent lung during OLV may not only improve the overall oxygenation but may also aid in causing oxygen-induced hypercarbia. It may minimize the need to reduce the respiratory rate/minute ventilation to achieve permissive hypercapnia while ventilating the dependent lung during OLV. It may allow for a reduction in tidal volume while switching from TLV to OLV to avoid volutrauma of the dependent lung. The other factors like hyperthermia, metabolic acidosis, dead space ventilation, sepsis, and thyroid disease that may contribute to hypercarbia during OLV, and should also be taken into account during surgery.

We suggest initiating AOI to the non-dependent lung, while reducing minute ventilation in the dependent lung to achieve permissive hypercarbia with better overall oxygenation during OLV. This viewpoint's problem and physiology need to be considered in more detail with greater acceptance of permissive hypercapnia with apneic oxygenation.

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