Impact of respiratory quotient during extracorporeal gas exchange: a theoretical model

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Argomento: Altro

Interactions between natural (NL) and artificial (membrane - ML) lung during extracorporeal gas exchange are complex. Gas exchange through the artificial lung substitutes the alveolar function, possibly dissociating oxygenation from carbon dioxide removal (VCO₂) in the natural lung. During ultra-protective ventilation strategies a significant amount of CO_2 removal is achieved by the ML (ECCO₂R – extracorporeal CO₂removal) to allow a great reduction of NL ventilation, while oxygenation is still provided by the NL. In this setting hypoxemia is a frequent collateral effect, especially in patients with severe respiratory failure.

The alveolar gas equation states that alveolar partial pressure of oxygen (P_AO_2) depends on inspired partial pressure of oxygen (P_1O_2 , function of F_1O_2), oxygen consumption (VO_2) and alveolar ventilation, being the last two integrated in the respiratory quotient ($RQ = VCO_2/VO_2$). If RQ decreases (as a consequence of decreased ventilation and VCO_2 of the natural lung), F_1O_2 needs to be increased to maintain the same P_AO_2 (figure 1). When RQ decreases below 0.2-0.3 (high levels of ECCO₂R) any increase in F_1O_2 becomes less efficient thus enhancing the formation of reabsorption atelectasis already facilitated by hypoventilation, further worsening hypoxemia.

However, RQ is a function of both VCO₂ and VO₂. Keeping constant the total VO₂ and CO₂ production, the relationship between natural lung RQ (RQ_{NL}) and artificial gas exchange can be computed for different amount of extracorporeal oxygenation (ECMO) and CO₂ removal (ECCO₂R), as shown in figure 2: for a given amount of ECCO₂R (VCO_{2ML}/VCO_{2TOT}), increasing the extracorporeal oxygen supply (VO_{2ML}/VO_{2TOT}) will increase RQ_{NL}.

Currently available low-flow (300-500 mL/min) $ECCO_2R$ systems are not able to provide significant oxygen supply. If hypoventilation-induced hypoxemia during ultra-protective ventilation strategies is a concern, an "intermediate-flow" system (able to provide at least 20% of patient total oxygen consumption) could be useful.



Figure 1. The alveolar gas equation solved for $P_ACO_2 35 \text{ mmH}_2O$ and $P_AO_2 100 \text{ mmHg}$, F_1O_2 as dependent variable for different RQ of the natural lung. As you can see, when RQ decreases below 0.2-0.3 F_1O_2 needs to be increased more consistently to maintain alveolar oxygenation (modified from Gattinoni L et al. Control of intermittent positive pressure breathing (IPPB) by extracorporeal removal of carbon dioxide. Br J Anaesth. 1978;50(8):753–8).



Figure 2. QR_{NL}/QR_{TOT} as a funtion of VCO_{2ML}/VCO_{2TOT} (ECCO₂R) for different VO_{2ML}/VO_{2TOT} (ECMO). The relationship is explained by the following equation:

$$\frac{RQ_{NL}}{RQ_{TOT}} = \left(1 - \frac{VCO2_{ML}}{VCO2_{TOT}}\right) \div \left(1 - \frac{VO2_{ML}}{VO2_{TOT}}\right)$$

where: $VCO_{2TOT} = VCO_{2ML} + VCO_{2NL}$, and $VO_{2TOT} = VO_{2ML} + VO_{2TOT}$. As you can see, in case of 70% of extracorporeal CO₂ removal adding about 20% of total VO₂ by the extracorporeal route increases natural lung RQ from about 0.3 to 0.4.