Lungs and Breathing

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CO_2 gas exchange in lungs

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The classical concept of CO_2 gas exchange in the lungs is well established [1]. Thus, the first step in CO_2 gas exchange involves a combination of the physiological processes required for CO_2 gas diffusion from cells in tissues, the catalysis of carbonic acid dehydration, and transmembrane HCO_3^- -Cl^- exchange in red blood cells. The second step involves the oxygenation of hemoglobin and the simultaneous liberation of protons, which are then able to retract with HCO_3^- within red blood cells, resulting in the excretion of CO_2 gas from the red blood cells in the pulmonary capillaries.

Currently, with cellular, molecular, and in vivo animal experiments, the new concept of pulmonary blood flow-mediated CO_2 gas exchange in the lungs is demonstrated [2,3]. Thus, we showed that 10s shear stress stimulation induced stress strength-dependent H^+ release followed by CO_2 gas excretion from pulmonary arteriolar endothelial cells, which was significantly reduced by the inhibition of cell surface F_1/F_0 ATP synthase or carbonic anhydrase type IV. Based on these findings, we proposed the new concept of CO_2 gas excretion in the lungs, pulmonary arteriolar flow-mediated cell surface F_1/F_0 ATP synthase-dependent H^+ secretion, which results in the facilitation of a dehydration reaction involving HCO^- in the plasma followed by CO_2 gas excretion from the arteriolar endothelial cells. To further examine the validity of the proposed new concept, we investigated the effects of intramediastinal balloon catheterization-, pulmonary artery catheterization-, or isoprenaline-induced changes in the pulmonary blood flow on the end-expiratory CO_2 gas pressure ($PECO_2$), the maximal velocity of pulmonary artery (Max Vp), systemic arterial pressure of anesthetized rabbits. We also evaluated the changes in the $PECO_2$ in clinical model of anemia. An almost linear relationship was detected between the $PECO_2$ and Max Vp. When small pulmonary arteries were subjected to stenosis, the $PECO_2$ fell rapidly, and the speed of the reduction was dependent on the degree of stenosis. Isoprenaline produced significant increases in the $PECO_2$. Conversely, the treatment with a cell surface F_1/F_0 ATP synthase antibody caused significant reductions in the $PECO_2$ itself and isoprenaline-induced increase in the $PECO_2$. Neither the $PECO_2$ nor systemic arterial pressure was significantly influenced by marked anemia (%hematocrit, ~70%). On the other hand, in the presence of severe anemia (%hematocrit, ~100%) both the $PECO_2$ and the arterial pressure fell significantly when the rabbit's blood viscosity was decreased. With these findings of in vivo animal experiments, we reaffirm the validity of the proposed new concept of CO_2 gas exchange in the lungs.

The new concept of pulmonary blood flow-mediated CO_2 gas exchange in the lungs may offer reasonable answers for several serious questions which remain still unanswered under the classical concept of CO_2 gas exchange in the lungs. For example, the physiological CO_2 gas excretion was observed in isolated rat lungs that were perfused with a red blood cell-free Krebs-Ringer bicarbonate solution [4]. Moreover, in patients with severe anemia, little or no symptoms of reduced CO_2 transport-mediated acidosis have been confirmed [5].

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