

Pulmonary Gas Exchange during Exercise following Altitude Acclimatization

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Abstract. In man the resting alveolar-arterial O_2 partial pressure difference ($A-aPO_2$) at sea level is very small (5 to 10 Torr). However, during sea-level exercise at O_2 uptakes ($\dot{V}O_2$) of 2.0 to 5.0 l/min, the $A-aPO_2$ has been shown to progressively increase, reaching values as high as 40 Torr during extreme exertion. Furthermore, when exercise is performed during acute hypoxia, the deterioration in pulmonary gas exchange is even more evident in that the $A-aPO_2$ at any given $\dot{V}O_2$ is greater than at sea level. This implies an even greater interference to gas exchange because when PaO_2 is reduced, the effective slope of the O_2 -hemoglobin dissociation curve is increased, and, for any given drop in PO_2 , the associated decrease in O_2 concentration is much larger. By use of the multiple inert-gas elimination technique, it has been shown that the primary mechanism for the widening $A-aPO_2$ and deterioration in pulmonary gas exchange during both normoxic and hypoxic exercise was alveolar-endcapillary diffusion disequilibrium with ventilation-perfusion (\dot{V}_A/\dot{Q}) inequality playing a significant but secondary role.

Even though unacclimatized subjects exercising during acute hypoxia develop a larger A-aPO₂ at any given $\dot{V}O_2$ than at sea level, this does not seem to be the case for acclimatized individuals. In a previous study, Operation Everest II, where subjects were decompressed to a barometric pressure (P_b) of 240 Torr ($F_I O_2=0.22$, $P_I O_2=43$ Torr) over a period of six weeks, the A-aPO₂ at any given $\dot{V}O_2$ was the same as at sea level regardless of the $P_I O_2$. However, in these subjects no comparison of A-aPO₂ during exercise was made between acute and chronic hypoxia. The reasons for this apparent improvement in the efficiency of pulmonary gas exchange following acclimatization are unclear. However, we have hypothesized that the well-known reduction in cardiac output following altitude acclimatization produces a longer pulmonary capillary red blood cell transit time resulting in less alveolar-endcapillary diffusion disequilibrium.

The aim of this study was first to confirm the reduction in A-aPO₂ with acclimatization and second to determine its mechanisms. By use of the multiple inert-gas elimination technique, it was possible to estimate the relative roles of diffusion disequilibrium, \dot{V}_A/\dot{Q} inequality, and pulmonary shunt in determining the A-aPO₂. Subsequent calculations of pulmonary O₂ diffusing capacity permitted differentiation between a change in diffusing capacity and pulmonary capillary red blood cell transit time as determinants of O₂ diffusion equilibration.

Pulmonary gas exchange was studied in eight normal subjects both before and after two weeks of altitude acclimatization at the White Mountain Research Station's Barcroft Facility (3,800 m, 12,470 ft, $P_b=484$ Torr). Respiratory and multiple inert-gas tensions, ventilation, cardiac output, and hemoglobin concentration were measured at rest and during three levels of constant-load-cycle exercise during both normoxia ($P_I O_2=148$ Torr) and normobaric hypoxia ($P_I O_2=91$ Torr). Following acclimatization, the measured alveolar-arterial PO₂ difference (A-aPO₂) for any given work rate decreased ($p<0.02$). The largest reductions were observed during the highest work rates and were 24.8 ± 1.4 to 19.7 ± 0.8 Torr (normoxia) and 22.0 ± 1.1 to 19.4 ± 0.7 Torr (hypoxia). This could not be explained by changes in ventilation-perfusion inequality, pulmonary shunt, or estimated O₂ diffusing capacity, which were unaffected by acclimatization. However, cardiac output for any given work rate was significantly decreased ($p<0.001$) following acclimatization.

We conclude that the reduction in the measured A-aPO₂ following altitude acclimatization is due to less diffusion disequilibrium for O₂ and is primarily the result of the lower cardiac output, which produces a longer pulmonary capillary red blood cell transit time.

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