

Pulmonary O₂ Uptake during Exercise: Conflating Muscular and Cardiovascular Responses

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ABSTRACT

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For moderate-intensity exercise (below lactate threshold, θ_L), muscle O₂ consumption ($\dot{Q}O_2$) kinetics are expressed in a first-order phase 2 (or fundamental) pulmonary O₂ uptake ($\dot{V}O_2$) response: $d\dot{V}O_2/dt + \dot{V}O_2/\tau + \Delta\dot{V}O_2(t) = \dot{V}O_2(ss)$; where $\dot{V}O_2(ss)$ is the steady-state $\dot{V}O_2$ increment, and τ the $\dot{V}O_2$ time constant (which is within approximately 10% of $\dot{Q}O_2$). A likely source of $\dot{Q}O_2$ control in this intensity domain is ADP-mediated, for which intramuscular phosphocreatine (PCr) may serve as a proxy variable. Whether, in reality, this behavior reflects the operation of a single homogeneous compartment is unclear, however; a multicompartment structure comprised of units having a similar $\Delta\dot{V}O_2(ss)$ but with widely varying τ can also yield a “well-fit” exponential response with an apparent single τ . In support of this is the inverse (although poorly predictive) correlation between τ and both θ_L and $\dot{V}O_{2max}$. Above θ_L , the fundamental $\dot{V}O_2$ kinetics are supplemented with a delayed, slowly developing component that can set $\dot{V}O_2$ on a trajectory towards $\dot{V}O_{2max}$, and that has complex temporal- and intensity-related kinetics. This $\dot{V}O_2$ slow component is also demonstrable in [PCr], suggesting that the decreased efficiency above θ_L predominantly reflects a high phosphate cost of force production rather than a high O₂ cost of phosphate production. In addition, the oxygen deficit for the slow component is more likely to reflect a progressive shifting of $\Delta\dot{V}O_2(ss)$ rather than a single $\Delta\dot{V}O_2(ss)$ having a single τ .

Key Words: MUSCLE OXYGEN CONSUMPTION, FEEDBACK CONTROL, 31P-MR SPECTROSCOPY, KINETICS

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