

Hemodynamic responses to graded exercise during acute simulated mild altitude exposure.

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Introduction: Research regarding the physiological effects of acute and chronic altitude exposure on exercise performance was stimulated by the 1968 Summer Olympic Games in Mexico City (~ 2,300 m) (1). This research has generally focused on the effects of high altitude (> 3,000 m) on the physiology of exercise (2-3). However, many recreational areas and athletic contests are located in U.S. cities with mild altitudes (< 2,000 m). The purpose of this study was to examine the effects of acute mild altitude exposure on the hemodynamic responses to graded exercise. **Methods:** Young apparently healthy trained cyclists (n=9) served as subjects for this study. Each subject performed 2 graded exercise tests (GXT) on a mechanically braked bicycle ergometer inside a hypobaric chamber. The GXTs were performed at simulated altitudes, low = 305 m (LA) and mild = 1829 m (MA). The altitude order was randomized, and the GXTs were separated by 5-10 days. The GXT protocol consisted of rest and 3 minute exercise stages until volitional exhaustion or failure to maintain 70 rpm pedal cadence. Oxygen saturation (O₂SAT), heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), rate pressure product (RPP), and oxygen pulse (O₂pulse) were measured at rest and during the last minute of each exercise stage. **Statistics:** The physiological data were analyzed using a 2 altitude by 5 workload (WL) repeated measures ANOVA to determine main and interaction effects with Bonferroni post hoc analysis. Maximal data were compared using paired t-tests. **Results:** O₂SAT was significantly lower during each MA exercise WL, and at maximal WL compared to LA. DBP was not significantly affected by altitude or WL. O₂pulse was significantly lower at MA during max WL, but no altitude effect was observed at other WLs. However, RPP was significantly higher at MA and 140 W, but lower during maximal WL. Maximal WL and time to exhaustion were significantly lower at MA.

WL	O ₂ SAT (%)		HR (bt/min)		SBP (mm Hg)		MAP (mm Hg)	
(W)	LA	MA	LA	MA	LA	MA	LA	MA
0	94±2	90±5	72±12	68±12	123±8	131±20	89±6	96±12
70	93±6	*88±8	99±9	104±12	141±10	140±13	106±7	106±8
140	95±2	*86±9	120±14	*127±14	154±12	*161±7	113±9	118±4
210	96±3	*89±6	145±17	151±16	174±12	175±12	124±8	124±6
280	94±2	*86±3	166±17	167±14	186±14	186±13	129 ±7	130±7
max	93±4	*86±4	177±14	172±12	196±14	189±13	137 ±7	132±9

* Significantly different than LA (p<0.05).

Discussion: Despite the decreased O₂SAT at MA, there were minimal effects on the hemodynamic responses to GXT. This may be attributed to the relatively flat slope of the O₂-dissociation curve at these altitudes. However, maximal exercise performance was lower at MA, and may be the result of decreased arterial PO₂ resulting in lower O₂ diffusion into working muscle. **References:** 1. Craig, A. *Med Sci Sports Exerc.* 1:177-180, 1969. 2. Saltin, B. *Res Quart.* 67:S1-S10, 1996. 3. Squires, R. et al. *Med Sci Sports Exerc.* 14:36-40, 1982.