CARDIOPULMONARY ADJUSTMENTS WITH EXERCISE IN COLD WATER AND HYPERBARIC ENVIRONMENTS

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INTRODUCTION:

Physiological adjustments to exercise include regulating cardiopulmonary responses to changes in workload. Further adjustments are required when exercise is conducted in cold water (1) or in hyperbaric environments (2) to compensate for the added effects of thermal or high pressure stress.

Ventilatory equivalent (VEQ) is the ratio of minute ventilation (V_E) to oxygen consumption (VO_2), and provides a useful index of how well V_E remains coupled to VO_2 for a given workload. Oxygen pulse (OW) is the ratio of VO_2 to heart rate (HR), and serves as an indirect estimate of the product of cardiac stroke volume and arteriovenous O_2 differece; derived from solution of the classical Fick equation.

The purpose of this paper is to describe changes in VEQ and O W during steady-state leg exercise in water at temperatures of 18-31° C, and during hyperbaric exposures to 31 ATA.

METHOD

The data were derived from three human exercise studies conducted between 1987-1990. In each study V_E, VO_2, and HR were obtained while males performed steady-state leg exercise during head-out immersion. VEQ and OXP were calculated from measured variables and analyzed by a two-way ANOVA for repeated measures. Data are presented in this paper for a workload of 1.5 W/kg, which was common to all studies.

Study I (n = 10) involved performing 60 min of exercise at 1.5 W/kg during immersion in 28 and 18° C water at the surface. Steady-state variables were obtained by averaging the last 40 min of exercise. Study II (n = 11) entailed 4 consecutive periods of 5 min rest and 25 min exercise at 1.5 W/kg during immersion in 25° C water, once breathing air at 1 ATA and once breathing HeO_2 at 5.5 ATA (PO, = 0.42 ATA). VEQ and OXP did not change among exercise periods and were therefore averaged across all 4 periods. Study III (n = 12) used step increases in workload (10 min each at 0.5, 1.0, and 1.5 W/kg) during immersion in 31 and 20° C water. Tests at each water temperature were conducted at 1 ATA breathing air and at 31 ATA breathing HeO_2 (PO_2 = 0.42 ATA).

RESULTS

V_E increases exponentially, as VO_2 increases. This relationship is the same in *dry* and immersed conditions up to a VO_2 of about 2.5 L/min. Thereafter the immersed curve increases at a faster rate, but is not influenced by water temperature.

The table below presents VEQ and OXP for the common workload of 1.5 W/kg (mean ± SEM).

DEPTH	STUDY	WATER T°	VEO	OXP
1 ATA	I	18	28.1 ± 1.1	17.8 ± 6.0
	III	20	30.8 ± 1.6	17.9 ± 3 2
	II	25	27.1 ± 0.6	16.9 ± 2.8
	I	28	28.6 ± 1.6	15.9 ± 4.0
	III	31	29.8 ± 1.2	15.5 ± 3.4
5.5 ATA	II	25	24.3 ± 0.7	17.3 ± 2.9
31 ATA	III	20	27.1 ± 2.2	18.2 ± 2.8
		31	275 ± 1.6	15.7 ± 3.1

At 1 ATA, VEQ did not vary significantly with water temperature because of concurrent increases in V_E and VO_2. OXP increased as temperature declined, due to increases in VO_2 with little change in HR. Reductions in V_E at depth, with no change in VO_2, significantly lowered VEQ. No significant change in HR occurred at depth, thus OXP was not altered relative to corresponding 1ATA values.

CONCLUSIONS:

These findings demonstrate that colder water temperatures do not affect the coupling of exercise V_E to VO_2. Cold induced increases in VO_2 were matched by increases in V_E such that VEQ did not change. Lower VEQ at depth was due solely to a reduced V„ which suggests a downward (and more efficient) regulation of ventilation to O_2 demand.

Increases in OXP in colder water were largely a result of higher VO_2; indicating an increase in the product of stroke volume and a-v O_2 difference. Since OXP was not significantly altered at depth (no significant changes in VO_2 and HR) it can be concluded that this index of cardiopulmonary adjustment to exercise was not affected by hyperbaric exposure *per se*.

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