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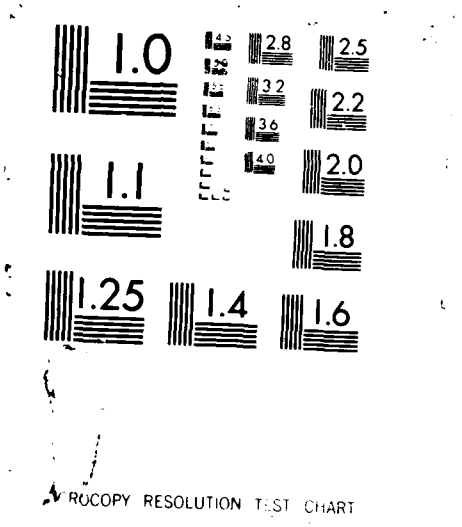
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Maximal cardiorespiratory responses to one- and two-legged cycling during acute and long-term exposure to 4300m

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Maximal oxygen consumption (VO2max) and maximal cardiac output (CO) are reduced at high altitude. The purpose of this investigation was to determine if the reduction in CO was responsible for the sustained reduction in VO2max. Our approach was to have seven male subjects perform one-and two-legged VO2max tests on a cycle ergometer at sea level (SL), after 1 hour at 4300m simulated altitude (SA) and after two weeks of residence at the summit of Pikes Peak, CO. (PP; 4300m). Cardiac output limits maximal performance during two-legged but not during one-legged cycling. Arterial oxygen content decreased from 18.8% to 16.0 vol% (SL to SA, $p < .01$) and increased to 19.9% (SL vs PP, $p < .05$; SA vs PP, $p < .01$). Two-legged VO2max decreased from 3644 ml/min at SL to 2700 ml/min at SA ($p < .01$) to 2858 ml/min at PP (SL vs PP, $p < .01$; SA vs PP, ns). One-legged VO2max decreased from 2952 to 2252 ml/min (SL vs SA, $p < .01$) but improved to 2657 ml/min at PP (SA vs PP; $p < .05$). Since only one-legged VO2max increased as more oxygen was made available to the working muscles then the altitude-induced reduction in cardiac output can be implicated as being responsible for the reduction in VO2max during two-legged cycling

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SUMMARY

During exposure to high altitude, maximal oxygen consumption (V02max) is diminished immediately in proportion to the reduction in oxygen content of the inspired air. As the exposure lasts beyond a couple of days, there is an increase in arterial oxygen content due to hemoconcentration and an increase in arterial oxygen saturation. However, V02max does not increase possibly due to a reduction in cardiac output. The purpose of this investigation was to study the contribution of the increase in oxygen content to the working muscles without the potentially confounded problem of a reduced cardiac output. Seven male subjects (aged 17 to 24 years) performed one- and two-legged V02max tests on a cycle ergometer at sea level (SL), after 1 hour at 4300m simulated altitude (SA) and after two weeks of residence at the summit of Pikes Peak, CO. (PP;4300m). Cardiac output limits maximal performance during two-legged but not one-legged cycling. Oxygen content changed from 18.8 $\pm$ 0.5 to 18.0 $\pm$ 0.6 vol% (SL vs SA, $p<.01$) and to 19.9 $\pm$ 0.4 vol% (SL vs PP, $p<.05$; SA vs PP, $p<.01$). Two-legged V02max decreased from 3644 $\pm$ 257 ml/min SL to 2700 $\pm$ 142 ml/min SA ($p<.01$) to 2858 $\pm$ 161 ml/min PP ($p<.01$). One-legged V02max decreased from 2952 $\pm$ 217 to 2252 $\pm$ 169 ml/min (SL vs SA, $p<.01$) but improved to 2657 $\pm$ 175 ml/min at PP (SA vs PP;$p<.05$). Since only one-legged V02max increased as more oxygen was made available to the working muscles then the altitude-induced reduction in cardiac output can be implicated as being responsible for the reduction in V02max during two-legged cycling.

Key words: One-legged cycling - Altitude acclimatization - Cardiac output - Maximal oxygen consumption

INTRODUCTION

It is generally agreed that maximal oxygen uptake (VO2max) is a valid index of the work capacity of an individual because it reflects both the ability of the cardiovascular system to deliver oxygen to the working muscles and the ability of the tissues to utilize oxygen (Astrand and Rodahl 1977; Frisancho 1975). During exposure to high altitude, VO2max is diminished immediately in an inverse relationship to the elevation beginning at approximately 2200 m (Buskirk 1969; Sutton and Jones 1983). Since maximum cardiac output does not decrease significantly for the first couple of days of exposure to high altitude (Hansen et al. 1967; and Stenberg 1966), the reduction in VO2max is closely related to the reduction in the oxygen content of the inspired air (Gleser 1973). During exposures lasting longer then a couple of days, the cause of the sustained reduction in VO2max is not as firmly established (Alexander et al. 1967; Reeves et al. 1987; Vogel et al. 1967; Wagner et al. 1987).

As the altitude exposure continues and altitude acclimatization occurs, there is an increase in oxygen content due to hemoconcentration (Grover et al. 1976; Grover 1979) and an increase in arterial oxygen saturation (Lenfant and SBullivan 1971; Frisancho 1975). Maximal oxygen uptake, however, does not rise above the levels measured during the acute exposure in proportion to the increase in oxygen content suggesting improvement in VO2max is limited by a reduced cardiac output. Although cardiac output is reduced at altitude, direct evidence that the reduction actually limits exercise performance is lacking (Vogel et al. 1974; Saltin et al. 1968).

Augmentation of cardiac output using methods such as preventing the altitude-induced hemoconcentration by breathing high concentrations of CO2 to maintain stroke volume (Grover et al. 1976) or by increasing the maximal heart rate with atropine (Hartley et al. 1974) were not successful in increasing VO2max. It may be that the enhanced variable (e.g. stroke volume or heart rate) was offset by a compensatory change in another variable (e.g. arterial oxygenation) making it difficult to determine the relative contribution of cardiac output and oxygen content to the VO2max value.

To be able to study the contribution of an enhanced oxygen content to the working muscles without the potentially confounding problem of a reduced cardiac output, we determined VO2max during one- and two-legged cycling at sea level and during two weeks of altitude exposure. Cardiac output limits two-legged VO2max at sea level and possibly at altitude (Gleser 1973; Saltin et al. 1968; Vogel et al. 1967), but does not limit one-legged cycling (Gleser 1973; Stamford et al 1978a). We reasoned that if both one- and two-legged VO2max do not increase as more oxygen is made available to the working muscles by the increase in oxygen content during altitude acclimatization, then other factors such as an impaired ability of the working muscles to fully utilize the available oxygen may be responsible for the reduction in two-legged VO2max. If, on the other hand, only one-legged VO2max increases with altitude acclimatization, then the altitude-induced reduction in cardiac output can be implicated as being responsible for the reduction in VO2max during two-legged cycling.

METHODS

Seven healthy male volunteers served as test subjects. Each gave informed consent and all were highly motivated. Ages ranged from 17 to 24 years (mean: 20.6), heights from 170.2 to 182.9 (mean: 173.8) and weights from 63.5 to 80.0 kg (mean: 72.6). The experiment was conducted in the hypobaric environmental chamber at the U.S. Army Research Institute of Environmental Medicine at Natick, Massachusetts (50m), and at the U.S. Army Pikes Peak Laboratory at the summit of Pikes Peak, Colorado (4300m). During the first week of the study, the subjects were required to practice pedalling frequently on a cycle ergometer against heavy resistance with one and/or two legs to become familiarized and habituated with the tasks and procedures.

One- and two-legged VO2max tests were performed during two-day periods on five separate occasions: twice at sea level during weeks two and three of the study, once at 4300 m simulated altitude (1-hour exposure) during week four, and twice at Pikes Peak at the end of weeks seven and eight (the first and second week of the altitude sojourn). On four occasions during the first day of each two-day period, four subjects completed the two-legged VO2max test and the remaining three subjects completed a one-legged VO2max test. On the following day, the type of VO2max test taken by each of the subjects was reversed. On one occasion at sea level, the order of days on which the subjects took either the one- or two-legged VO2max test was reversed to determine if there was a test-to-test order effect.

One- and two-legged VO2max were determined using a continuous, incremental cycling protocol on an electrically-braked ergometer (Collins,

Inc). All subjects elected to perform the one-legged VO2max test with their right leg. The left leg rested on the middle crossmember of the bike. For all of the one- and two-legged tests, the subjects started pedalling at 50 watts for two minutes followed by an incremental increase of 25 watts every two minutes until the subject could not continue pedalling. For each of the tests, the subjects were required to pedal at a frequency of 60 rpm. However, during the heaviest workloads of the one-legged VO2max tests, some of the subjects found it necessary to increase the pedal revolutions to 65-70 rpm to maintain the cycling motion. Because the resistance was automatically and immediately reduced to offset the increase in pedalling frequency, the work intensity was exactly the same as with 60 rpm. The subjects' feet were secured to the pedals during all VO2max testing.

A Sensormedics Metabolic Measurement Cart Horizon System (MMC; Sensormedics Corp.) was used to collect respiratory metabolic data. The MMC was calibrated prior to each test with medical grade calibration gases. Expired air was channeled from a low resistance valve and tubing into a mixing chamber within the MMC. For each minute, mixed expired gas was sampled from the mixing chamber for 45 seconds alternated with 15 seconds of sampling end-tidal values for oxygen and carbon dioxide directly from the mouthpiece. Analog heart rate signals from a heart rate monitor (IBS,Inc.) were continuously fed to the MMC. Values for heart rate, minute ventilation, oxygen consumption, carbon dioxide production, tidal volume, breathing frequency, respiratory quotient and ventilatory equivalents for oxygen and carbon dioxide were calculated and printed every 15 seconds. A minute-to-minute summary report which averaged the four 15-second periods of each minute was printed at the conclusion of each

test and was subsequently used to provide the information for analysis of the respiratory data.

Hemoglobin and hematocrit were determined on samples obtained from an indwelling catheter in an antecubital vein prior to, during the last thirty seconds of each workload and five minutes after the completion of each VO2max test. Plasma volume reduction was calculated using the equation of Dill and Costill (1974). Oxygen saturation was determined at rest periodically throughout the study using ear oximetry (Hewlett-Packard, Inc.). Oxygen content (vol%) was calculated as the product of saturation (%) X Hb (mg%) X 1.36 ml02/mgHb (Grover, 1979).

The data were analyzed using a two-way, repeated-measures analysis of variance (subject x trial). When a statistically significant F-ratio was calculated, differences between the means were tested for significance using Neuman-Keuls post-hoc test. The level of significance was chosen as $p<.05$.

RESULTS

There were no significant differences in any of the respiratory or hematological parameters measured during rest or either exercise protocol during the two testing sessions at sea level. It was also determined that the test-to-test order of VO2max testing had no influence on any of the results obtained. Therefore, the values collected on the last test session at sea level were used as the sea-level baseline values.

The maximal physiological responses to one- and two-legged exercise are presented in Table 1. Two-legged VO2max was reduced 26% during simulated

altitude and 25% during the first week and 22% during the second week at Pikes Peak. Regardless of the length of exposure, two-legged VO2max was significantly reduced from sea-level values. Conversely, one-legged VO2max was decreased 24% in the altitude chamber but gradually increased 18% by the second week at Pikes Peak to a VO2max value that was not significantly different from sea level. At sea level and in the altitude chamber, one-legged VO2max was 81% and 83% of the two-legged VO2max, respectively. At Pikes Peak, the one- to two-legged VO2max ratio was 91% during week 1 and 93% during week 2.

[TABLE 1 HERE]

Maximal heart rate during two-legged cycling was reduced from sea level in the altitude chamber (-11 beats) and at Pikes Peak (-12 beats). Maximal heart rate during one-legged cycling at altitude was not statistically different from sea level. At sea level and at altitude, maximal heart rate for one-legged cycling was always less then the maximal heart rates for two-legged cycling.

Maximal minute ventilation (VE) increased for both one- and two-legged cycling during the sojourn at Pikes Peak. VE was greater for two-legged cycling then for one-legged cycling only at sea level. In the altitude chamber and during the first week at Pikes Peak, VE for one- and two-legged cycling were not statistically different. However, during the second week, VE during one-legged exercise was 8% higher than VE during two-legged cycling.

[TABLE 2 HERE]

Table 2 presents the values for hemoglobin, hematocrit, plasma volume, oxygen saturation and oxygen content. From sea level to the one hour exposure in the altitude chamber, hemoglobin, hematocrit and plasma volume were not altered while oxygen saturation and oxygen content were reduced 12.3% and 14.8%, respectively. During the Pikes Peak sojourn, however, hemoglobin and hematocrit were increased significantly above sea level baseline values. From these values, it was estimated that plasma volume was reduced by 22% the second week. Also by the second week of the sojourn, oxygen saturation increased 5.1% (84.1% to 88.4%) above the values obtained during the one-hour exposure. Because of the increases in oxygen saturation and hemoglobin at Pikes Peak, oxygen content increased from 16.1 vol% during the one-hour exposure to 18.6 vol% (+15.5%) during week 1 and to 20.0 vol% (+24.2%) during week 2.

DISCUSSION

Attempts were made in the design of this study to eliminate as many extraneous factors as possible so that a true comparison could be made between the maximal responses of one- and two-legged cycling at sea level and at altitude. To that end, the test subjects, equipment, diet, times of testing, testing conditions and cycling protocols were identical for the one- and two-legged VO2max tests. Also, a minimum of one week separated consecutive testing periods so that an exercise "training effect" would not occur.

In this study, one-legged VO2max was approximately 82% of the two-legged value at sea level and during one hour of altitude exposure. This relationship

is similar to values previously reported at sea level (Gleser 1973; Neary and Wenger 1986; Saltin, et al 1968; Stamford et al 1978b) and during acute hypoxia (Gleser 1973). The 25% reduction in VO2max from sea level to one hour of exposure to similar altitudes or levels of hypoxia are also similar to previous investigations during one- (Gleser 1973) and two-legged cycling (Buskirk 1969; Moore et al 1986; Hansen et al 1967; Maher et al 1974). In contrast to some studies (Gleser 1973; Vogel et al 1974; Vogel et al 1972) but not all (Ekblom et al 1975), the reductions in one- and two-legged VO2max from sea level to the one-hour exposure in the present study was not entirely due to the 14.8% reduction in arterial oxygen content. The reductions in the maximal heart rate values obtained during one-legged (-8 b/min;$p>.05$) and two-legged (-11 b/min; $p<.05$) cycling were also contributory.

As the altitude exposure continued, two-legged VO2max did not increase significantly above the value obtained during one hour of exposure and thus remained below sea-level values despite a 24.2% increase in oxygen content. These observations have been reported on numerous occasions (Dua and SenGupta 1980; Hansen et al 1967; Maher et al 1974; Moore et al 1986; Vogel 1967). What has not been not been reported previously is that VO2max during one-legged cycling improved 11.0% and 17.9% during the first and second weeks of altitude acclimatization, respectively. Furthermore, the improvement in one-legged VO2max was so pronounced that the value obtained during week two did not differ from the sea level value.

It is well established from previous studies that cardiac output is reduced after the first few days at altitudes greater than 3000 m (Alexander et al 1967; Klausen 1966; Vogel et al 1967). The 22% reduction in plasma volume

calculated in the present study is consistant with a reduction in cardiac output. It has also been shown that maximal cardiac output during one-legged cycling is 75% to 87% of two-legged cycling and is clearly not a limiting factor to one-legged maximal performance at sea level (Neary and Wenger 1986; Saltin et al 1968; Stamford et al 1978a) or during acute hypoxia (Gleser 1973). Throughout the present study, maximal heart rate during one-legged cycling was not as high as the maximal heart rate during two-legged cycling strongly suggesting that cardiac output does not limit one-legged maximal exercise performance even after two weeks of altitude exposure. One-legged VO2max appears to be limited by the ability of the muscle vasculature to accept the high blood flow (Gleser 1973; Stamford et al 1978a).

During normoxia, maximal two-legged cycling causes a greater arterial desaturation then one-legged cycling because of the limits imposed by cardiac output (Davies and Sargeant 1974; Stamford et al 1978a). Consequently, when air enriched with oxygen is breathed, two-legged but not one-legged VO2max is increased (Davies and Sargeant 1974). In the present study, in a similar manner, there was an increase in availability of oxygen to the working muscles by an increase in oxygen content. During two-legged maximal exercise, the increase in oxygen availability and utilization offset the altitude-induced reduction in cardiac output to maintain the VO2max value at the same level as measured during the chamber exposure (Grover 1979).

During one-legged cycling, VO2max increased as the altitude exposure lengthened. Why would an increase in oxygen availability cause a rise in one-legged VO2max at altitude when it does not affect maximal performance at sea level? It is not likely that this increase was related to arterial desaturation

as cardiac output was not as high as with two-legged cycling. It is also not likely that there was a greater redistribution of flow to the working muscles due to an exercise "training effect" since one-legged $\dot{V}O_2max$ had already started to improve during the first week of exposure even though the test subjects had not performed any cycling in the previous two weeks. One possible explanation may be that structural and/or metabolic adaptations in the working muscles or an increase in diffusion capacity from the capillary to the tissue mitochondria occurred favoring a more complete extraction and utilization of oxygen (Sutton et al 1987). Obviously, actual proof of these changes occurring was beyond the scope of this study.

The maximal values for ventilation during two-legged cycling at sea level and at altitude agree well with previous investigations (Ekblom et al 1975; Hansen et al 1967; Moore et al 1986). Also, the ratio of one- versus two-legged maximal ventilations (0.83) at sea level agrees well with previous work (Neary and Wenger 1986; Stamford et al 1978b). What are truly unique findings are the rate of increase of one-legged maximal ventilation with time at altitude and the observation that one-legged ventilation equalled and exceeded the two-legged value during the first and second weeks of exposure, respectively. Because the same subjects performed the one- and two-legged $\dot{V}O_2max$ tests under the same testing and environmental conditions, any respiratory and metabolic adaptations that may have occurred were obviously present during each of the two types of cycling. Why then was the rate and the magnitude of the increases in maximal ventilation at altitude between the two types of cycling different? The answer is related to the increase in the one-legged value for $\dot{V}O_2max$. From the one-hour exposure in the altitude chamber to

the second week at Pikes Peak, the subjects performed at a higher power output during one-legged cycling (150 watts to 171 watts, Table 1) which resulted in a higher rate of increase in ventilation then expected. Furthermore, if the one-legged submaximal ventilatory value obtained at 150 watts (164.2 l/min, unpublished data, present study) is compared to the two-legged maximal ventilatory value (191.7 l/min) the ratio after two weeks at Pikes Peak becomes .86, not significantly different from the .83 and .88 obtained at sea level and in the altitude chamber, respectively.

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Subjects participated in this study after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 in the use of volunteers in research. The views, opinions and findings in this report are those of the authors and should not be construed as an official Department of the Army position, policy or decision, unless so designated by other official documentation.

TABLE 1

MAXIMAL PHYSIOLOGICAL RESPONSES DURING ONE- AND TWO-LEGGED EXERCISE AT SEA LEVEL AND AFTER 1 HOUR, 1 WK. AND 2 WEEKS EXPOSURE TO 4300-M

PARAMETER	SEA LEVEL		1 HOUR		1 WEEK		2 WEEKS	
	1 LEG	2 LEGS	1 LEG	2 LEGS	1 LEG	2 LEGS	1 LEG	2 LEGS
Workload (watts)	179 *	268	150# *	207#	157# *	218#	171+ *	229#
$\dot{V}O_2$ (ml/min)	2952 *	3644	2253# *	2700#	2501# *	2747#	2657+ *	2858#
$\dot{V}CO_2$ (ml/min)	3417 *	4278	3021#	3417#	3033	3232#	3209	3263
R ($\dot{V}CO_2/\dot{V}O_2$)	1.16	1.18	1.34# *	1.28#	1.22+ *	1.18+	1.21+	1.19+
Heart Rate (b/min)	173 *	188	165 *	177#	172	176#	172 *	176#
Minute Ventilation (L/min)	124.0 *	148.6	132.3	149.9	178.6#+	177.9#+	207.1#+ *	191.7#+
Tidal Volume (ml/b)	2009 *	2639	2055 *	2626	2540	2734#+	2685#+	2812
Frequency (breathes/min)	61	56	64	58	71	65#+	75# *	68#+
$V_E/\dot{V}O_2$	41.9	40.5	58.3#	56.0#	71.3# *	65.1#+	77.2# *	67.5#+
$V_E/\dot{V}CO_2$	36.0	34.4	43.2#	43.5#	58.6# *	55.0#+	63.1#+ *	56.4#+
$P_{ET}O_2$	120	119	63#	61#	71#+	69#+	72#+	69#+
$P_{ET}CO_2$	27	31	25	25	17#+	19#+	15#+	19#+

* the value for 1 leg is significantly different than 2 legs ($p<0.01$).

significantly different from sea level ($p<.05$).

+ significantly different from simulated altitude ($p<.05$).

TABLE I[illegible]

HEMOGLOBIN, HEMATOCRIT, PLASMA VOLUME REDUCTION, AND ARTERIAL BLOOD SATURATION AT SEA LEVEL AND AFTER 1 HOUR, 1 WEEK, AND 2 WEEKS OF EXPOSURE TO 4300 M

PARAMETER	SEA LEVEL		1 HOUR		1 WEEK		2 WEEKS	
	1 LEG	2 LEGS	1 LEG	2 LEGS	1 LEG	2 LEGS	1 LEG	2 LEGS
HEMOGLOBIN (mg%)								
Pre-Exercise	14.5	14.6	14.1	14.0	15.7*	15.8*	16.6*	16.6*
At $\dot{V}O_2$ Max	15.3	15.9	15.3	15.4	16.9*	17.0*	17.8*	17.9*
HEMATOCRIT (%)								
Pre-Exercise	41.6	42.6	41.6	41.5	45.8*	46.3*	47.9*	48.3*
At $\dot{V}O_2$ Max	44.9	46.4	45.3	45.6	48.6*	49.1*	50.6*	50.9*
PLASMA VOLUME								
% Change f/Sea Level:								
Pre-Exercise	---	---	2.8	6.2	-14.0*	-14.1*	-22.1*	-20.8*
At $\dot{V}O_2$ Max	—	—	0.0	4.5	-14.7*	-11.0*	-22.6*	-18.6*
% Change f/Pre-Exercise to $\dot{V}O_2$ Max	-10.6	-14.3	-13.7	-15.5	-12.2	-11.3	-11.6	-11.9
OXYGEN SATURATION (%)	95.9		84.1		86.8		88.4	
OXYGEN CONTENT (VOL%)	18.9	18.8	16.1*	16.0*	18.6	18.6	20.0*	19.9*

*Significantly different from sea level

END

DATE

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