THE EFFECTS OF ACUTE SIMULATED MILD ALTITUDE EXPOSURE ON THE CARDIO-VASCULAR, RESPIRATORY, AND METABOLIC RESPONSES TO GRADED EXERCISE

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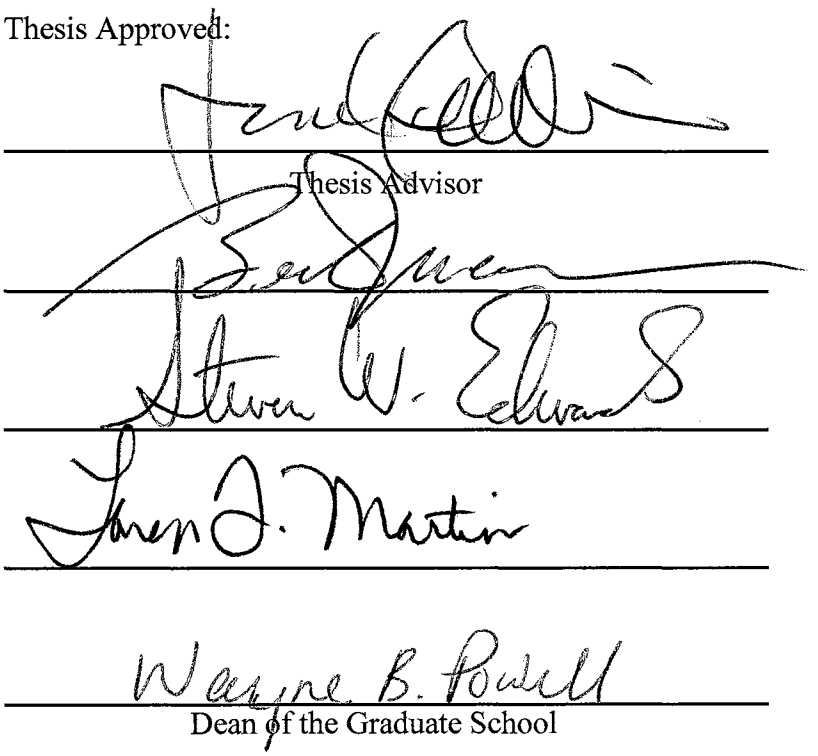

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CHAPTER I

INTRODUCTION

Interest into the physiological effects of altitude on exercise performance was stimulated by the 1968 Olympic Games in Mexico City (altitude ~ 2300 m above sea level) (Craig, 1969; Dill, 1967). In the United States many people travel to the Denver, Colorado area (altitude ~ 1600 m above sea level) to participate in sports or physical activity, and the U.S. Olympic Training Center is located in Colorado Springs, Colorado (alt. approx. 1839 meters above sea level). It is generally believed that performance of aerobic exercise is impaired when the human body is exposed to altitude. The logic of this is based on physics.

At sea level the ambient air (air in the surrounding environment) has a barometric pressure (P_B) of 760 mm Hg, an oxygen concentration of 20.93%, and a partial pressure of oxygen (PO_2) of 159 mm Hg (Guyton & Hall, 1996). When corrected for water vapor pressure in the lungs this yields a partial pressure of oxygen in alveolar air (P_AO_2) of 104 mm Hg (Guyton et al. 1996). Oxygen (O_2) diffuses down a pressure gradient across the alveoli in the lungs into the circulatory system and binds to hemoglobin (Hb) (Berne & Levy, 1993). At sea level the partial pressure of oxygen in arterial blood (P_aO_2) of 100 mm Hg, and the oxygen saturation of hemoglobin (SaO_2) is 96% (Haymes & Wells, 1986). At the tissue level in the human body, O_2 diffuses into cells down a pressure

gradient and is utilized for aerobic metabolism (Grover, 1979). At elevations above sea level O_2 concentration in ambient air remains constant, but the P_B, P_AO_2 and P_aO_2 decrease. With a smaller pressure gradient at the alveoli the SaO_2 and P_aO_2 also decreases resulting in lower O_2 diffusion into cells (Grover, 1979). However, the amount of O_2 bound to Hb is relative to the sigmoidal shape of the standard oxygen dissociation curve. Initial decreases in P_AO_2 during mild altitude result in small decreases in SaO_2, but subsequent decreases in P_AO_2 during moderate to high altitude exposure result in large decreases in SaO_2 (Ganong, 1997). The decrease in O_2 availability due to decreased PO_2 is known as hypoxic hypoxia (Ganong, 1997).

When exposed to hypoxia the decreases in P_aO_2 stimulate the chemoreceptors in the carotid bodies (Berne et al., 1993; Ganong, 1997; Guyton et al., 1996). The increased neural stimulation from the carotid bodies signals an increase in ventilatory drive at the respiratory center in the brain stem (Ganong, 1997). The respiratory center signals a sympathetic increase in ventilation, and also stimulates the cardioaccelerator center in the brain (Berne et al., 1993). Consequently, the sympathetic response of the heart is both chronotropic (increased rate) and inotropic (increased force) (Guyton et al., 1996). The result includes increasing heart rate and cardiac output.

Normal transport of O_2 is essential for optimal human performance. During muscular activity there is an increased demand for O_2 at the cellular level. Dilation of the peripheral vasculature and increased cardiac output, via increased heart rate (HR) and stroke volume (SV), result in increasing the flow of O_2 to the working muscle tissue (Wasserman, Hansen, Sue and Whipp, 1987). Minute ventilation (V_E) also increases, via increased tidal volume (V_T) and breathing frequency (f), to ensure adequate O_2 is

available at the alveoli for diffusion into circulation (Durstine & Pate, 1988). Therefore, to maintain adequate O_2 delivery to match the demand there is a coupling of the muscular, cardiovascular and respiratory systems (Wasserman et al., 1987).

Graded exercise tests (GXT) are commonly used to assess the function of these coupling mechanisms (Wasserman et al., 1987). During a GXT there is a progressive systematic increase in exercise intensity designed to stress these systems. Therefore, the maximal amount of work an individual can perform is a result of the function of O_2 delivery and extraction by the working muscle (Wasserman, et al. 1987). During a normal GXT cellular O_2 uptake increases with increasing workloads, and is demonstrated by an increase in arteriovenous oxygen difference (a-v O_2 diff) and an increase in oxygen consumption (VO_2) (Wasserman et al., 1987). Normal cardiovascular responses include 1) progressive linear increase in HR at each workload, 2) progressive linear increase in SV up to a workload of 40 to 50% of maximum then plateaus, and 3) progressive and linear increase in cardiac output at each workload (Durstine et al., 1988; Nieman, 1990). Total peripheral resistance (TPR) steadily decreases as workload increases, and is a function of vasodilation. Mean arterial blood pressure generally increases at each workload due primarily to increasing systolic blood pressure (SBP), while diastolic blood pressure (DBP) remains relatively constant (Durstine et al., 1988). Normal respiratory responses include 1) a curvilinear increase in f with workload, 2) a linear increase in V_T at low and plateaus at high workloads respectively, 3) curvilinear increase in V_E with workload (Durstine et al., 1988, Nieman, 1990).

To reach maximal aerobic power (VO_2max) each system is required to increase its' function to deliver the maximal amount of oxygen to working muscles (Wasserman et

al., 1987). Therefore, hypoxia due to altitude exposure can reduce aerobic power. However, the level of altitude necessary to reduce VO_2max has been the focus of much debate. Squires and Buskirk (1982) didn't observe reduced VO_2max in recreational runners at 914 m of elevation. Meanwhile, Terrdos, Mizuno, & Anderson (1985) reported that endurance trained athletes experienced a significant reduction in VO_2max at 600 to 900 m above sea level, but that sedentary subjects were unaffected until the elevation reached 1200 m. Young (1990) has suggested that at elevations equal to 1000 to 2000 m, VO_2max responses of individuals are highly variable, but that a significant reduction is observed at altitudes greater than 2000 m. Squires et al. (1982) did observe a 4.9% and 6.9% reduction in aerobic power from GXT's performed at 1219 and 1524 m respectively. Grover, Weil, & Reeves (1986) have observed that beginning at 700 m above sea level there is an 8% reduction in VO_2max for every 1000 m of elevation.

When GXT's are performed at altitudes greater than 2000 m there is general agreement that aerobic power is reduced (Faulkner, Kollias, Favour, Buskirk, & Balke, 1968; Pugh, Gill, Lahiri, Milledge, Ward, & West, 1964; Squires et al. 1982).

Statement of the Problem

The purpose of this study was to determine the effects of mild acute simulated altitude exposure on the cardiovascular, respiratory, and metabolic responses to graded exercise. The specific responses measured include oxygen saturation, heart rate, blood pressure, minute ventilation, oxygen consumption, and blood lactate concentration.

Hypothesis

This study tested the following hypotheses, and significance was accepted at the 0.05 level:

1. There will be no significant differences in oxygen saturation between low and mild altitude exposure during rest, submaximal and maximal exercise.

2. There will be no significant differences in heart rate between low and mild altitude exposure during rest, submaximal and maximal exercise.

3. There will be no significant differences in blood pressure between low and mild altitude exposure during rest, submaximal and maximal exercise.

4. There will be no significant differences in minute ventilation between low and mild altitude exposure during rest, submaximal and maximal exercise.

5. There will be no significant differences in oxygen consumption between low and mild altitude exposure during rest, submaximal and maximal exercise.

6. There will be no significant differences in blood lactate concentration between low and mild altitude exposure during rest, submaximal and maximal exercise.

Limitations

This study was subject to the following limitations:

1. The researchers exerted no control over the participants exercise or training patterns prior to or during the study except for the 24 hours prior to exercise testing.

2. The researchers exerted no control over the participants food or beverage intake prior to or during the study except for the 24 hours prior to exercise testing.

3. No attempt was made to control the participants exposure to other stressors (ie. work, sleep) prior to or during the study.

Delimitations

The delimitations of this study were:

1. The participants were delimited to 8 male and 2 female adult trained cyclists who volunteered to serve as subjects in this study.

2. The physiological measures were delimited to oxygen saturation, heart rate, blood pressure, minute ventilation, oxygen consumption, and blood lactate concentration.

Assumptions

For this study the following assumptions were made:

1. It was assumed that the participants followed recommendations concerning physical activity/exercise prior to each exercise test.

2. It was assumed that the participants dietary pattern were similar prior to both exercise tests, and the participants were instructed in writing regarding appropriate exercise and dietary patterns for the 24 hour period prior to each exercise test.

3. It was assumed that the participants exerted maximal effort during both exercise tests.

Definition of Terms

The following definitions were used in this study:

Altitudes.

HighAltitude. Elevations greater than 3,500 m above sea level or simulated environments producing conditions, barometric pressure or partial pressures of oxygen similar to altitudes greater than 3,500 m.

Low Altitude. Elevations less than 500 m above sea level or simulated environments producing conditions, barometric pressure or partial pressure of oxygen similar to altitudes less than 500 m.

Mild Altitude. Elevations between 500 m and 2,000 m above sea level or simulated environments, barometric pressure or partial pressure of oxygen similar to altitudes between 500 and 2,000 m.

Moderate Altitude. Elevations between 2,001 m and 3,500 m above sea level or simulated environments, barometric pressure or partial pressure of oxygen similar to altitudes between 2,001 and 3,500 m.

Sea Level. The elevation at sea level where the environmental conditions include a barometric pressure of 760 mm Hg or a partial pressure of inspired oxygen equals 159 mm Hg.

Blood Lactate. The concentration of lactate in whole blood obtained from venous or capillary samples.

Cardiac Output. The quantity of blood pumped from the left ventricle per minute of time.

Diastolic Blood Pressure. The pressure exerted by blood on the arterial walls during the diastolic phase of the cardiac cycle.

Heart Rate. The number of systolic contractions of the heart during one minute of time.

Hypoxia. A condition where there is decreased oxygen content in arterial blood.

Mean Arterial Pressure. The average pressure exerted by the blood on the artery walls during the cardiac cycle.

Minute Ventilation. The volume of air inhaled or exhaled by the lungs in one minute of time.

Oxygen Consumption. The amount of oxygen utilized by the body for the purpose of metabolizing fuels to produce energy in one minute of time.

Oxygen Saturation. The percent of hemoglobin in the arterial blood supply that is bound or coupled to oxygen.

Stroke Volume. The quantity of blood pumped by the left ventricle during one cardiac cycle.

Systolic Blood Pressure. The pressure exerted by the blood on the artery walls during the systolic phase of the cardiac cycle.

CHAPTER II

REVIEW OF LITERATURE

The study of the effects of simulated altitude on the acute physiological responses to graded exercise involves examination of the organ systems primarily responsible for performing aerobic exercise. To evaluate these systems with regard to this study, the review of literature will include 1) altitude/hypoxia classifications and comparisons, 2) the physiological responses at rest, during submaximal, and maximal workloads during acute altitude exposure, and 3) maximal exercise performance during acute altitude exposure.

Altitude/Hypoxia Classification

Previous investigators have produced levels of hypoxia similar to altitude environments using different methods. Some investigators have performed research in the field or in laboratories actually located at altitude. Others have simulated altitude by decreasing barometric pressure in a specialized hypobaric chamber. Meanwhile, others have decreased the concentration of oxygen in inspired air to produce a partial pressure of inspired oxygen similar to altitude. Regardless of the technique employed the result is the same, lowered partial pressure of inspired oxygen. For the purposes of this review of literature the altitude equivalent will be used unless otherwise specified.

At present there is no standardized nomenclature for reporting altitude or hypoxia classifications. The classifications used for this review include four different levels of altitude: 1) Low Altitude ($\geq$ sea level & $<$ 500 m), 2) Mild Altitude ($\geq$ 500 m & $\leq$ 2,000 m), 3) Moderate Altitude ($>$ 2,000 m & $\leq$ 3,500 m) and 4) High Altitude ($>$ 3,500 m). The selection of the nomenclature for these categories is based on the authors interpretation of levels of hypoxia observed by previous investigators (Welch 1987) (ie. mild hypoxia is associated with the term mild altitude). This classification system allows one to examine the effects of similar altitude levels on various physiological measurements.

Acute Physiological Responses to Altitude

Cardiovascular Responses

Cardiac Output In reviewing the literature no studies were found where cardiac output was measured during rest, submaximal or maximal exercise during acute exposure to mild altitude. However, there is some evidence to suggest that resting cardiac output does increase significantly during moderate altitude exposure (McManus, Horvath, Bolduan & Miller, 1974; Wagner, Gale, Moon, Torre-Bueno, Stolp, & Saltzman, 1986). Meanwhile, numerous studies have demonstrated a consistent increase in resting cardiac output during acute exposure to high altitude (Asmussen & Chiodi, 1941; Asmussen & Nielsen, 1955; Hansen, Vogel, Stetler, & Consolazio, 1967b; Klausen, 1966; Saltz, Beller, Giamber & Alpert, 1976; Stenberg, Ekblom, & Messin, 1966; Vogel, Hansen, & Harris, 1967; Wagner et al. 1986).

Limited studies suggest that cardiac output is significantly higher at moderate altitude when compared to low altitude at submaximal exercise workloads during a GXT (Hughes, Clode, Edwards, Goodwin, & Jones, 1973; Wagner, et al., 1986). During graded exercise testing subjects inspired oxygen at a concentration of 16% (~ 2,100 m altitude), Hughes et al. (1973) observed a significant increase in cardiac output at all submaximal bicycle ergometer workloads. Wagner et al. (1986) also observed an increase in cardiac output during graded exercise testing at a simulated altitude of approximately 3,000 m. When exposed to high altitude there is a significant increase in cardiac output during submaximal exercise (Assmussen et al. 1941; Asmussen et al. 1955; Hansen et al. 1967b; Hughes et al, 1973, Klausen, 1966; Stenberg et al. 1966; Vogel et al. 1967; Wagner, et al. 1986).

During maximal exercise cardiac output responses are highly variable. Wagner et al. (1986) and Hughes et al. (1973) did not observe a significant difference in maximal cardiac output during acute exposure to moderate altitude conditions. Furthermore, Hughes et al. (1973) and Stenberg et al. (1966) did not observe significant differences in maximal cardiac output during exercise at high altitude. However, there is some evidence to suggest that maximal cardiac output is increased at high altitude (Hansen et al. 1967b; Vogel et al. 1967), while others have observed a decrease (Pugh, 1964; Vogel, Hartley, Cruz & Hogan, 1974).

Heart Rate Under resting conditions at simulated mild altitudes of 1,000 and 2,000 m, moderate altitude of 3,000 m, and high altitude of 4,000 m, Bubb, Howley & Cox (1983) reported no difference in resting heart rates compared to low altitude.

However, the majority of studies have reported significant increases in resting heart rate at moderate (Wagner et al. 1986), and high altitudes (Assmussen et al. 1941; Asmussen et al. 1955; Hoon, Balasubramanian, Matthew, Tivari, Sharma & Chadha, 1977; Klausen 1966; Manchada, Maher & Cymerman, 1975; Reeves, Halpin, Cohen & Dauod, 1969; Saltz et al. 1976; Vogel et al. 1967; Wagner et al. 1986).

A limited number of studies have been performed to study the acute effects of mild altitude on heart rate during submaximal exercise. Bubb et al. (1983) observed no significant difference in heart rate during exercise at a workload equal to 40% of VO_2 max at sea level vs. 1,000 m. Similarly, Squires et al. (1982) reported no difference in steady state heart rate during level treadmill running at 214 m/min at 362, 914, 1219, and 1524 m of altitude.

During exposure to moderate altitudes heart rate responses during submaximal exercise were variable. Bubb et al. (1983) and Squires et al. (1982) reported no significant difference in steady state heart rate between sea level and 2,000 and 2,286 m respectively. However, Hughes et al. (1968) and Saltin (1996) reported higher exercise heart rates at 2,100 m compared to low altitude. At moderate altitudes of 3,000 m and above there is a consensus of agreement that heart rate is increased compared to low altitude during submaximal exercise (Assmussen et al. 1941; Asmussen et al. 1955; Bubb et al. 1983; Hughes et al. 1968; Klausen 1966; Knuttgen & Saltin, 1973b; Manchada et al. 1975; McManus et al. 1974; Pugh 1964; Stenberg et al. 1966; Vogel et a.l 1967; Wagner et al. 1986).

Maximal heart rates are not different at mild (Squires et al. 1982) or moderate altitudes (Fagraeus, Karlsson, Linnarsson & Saltin, 1973; Hughes et al. 1968; Reeves,

Grover & Cohen, 1967; Saltin 1996; Squires et al. 1982; Wagner, Miles, Horvath & Reyburn, 1979) and, it has been reported that acute high altitude exposure does not effect maximal heart rate (Hughes et al. 1968; Pugh 1964; Stenberg et al. 1966). However, some investigators have observed decreases in maximal heart rate during high altitude exposure (Cymerman, Reeves, Sutton, Rock, Groves, Malconian, Young, Wagner & Houston, 1989; Hartley, Vogel & Cruz, 1974; Klausen, Robinson, Michael & Myhre 1966; Knuttgen et al. 1973b; Vogel et al. 1967; Vogel et al. 1974).

Stroke Volume In reviewing the literature very little information can be found regarding the acute effects of mild and moderate altitude exposure on stroke volume at rest or during exercise. McManus et al. (1974) did observe a reduction is stroke volume when subjects exercised for 2 hours at 30% of VO_2max at 3,060 m above sea level. Alexander, Hartley, Modelski & Grover (1967) also reported a reduced exercise stroke volume at 3,100 m, and Mac Dougall , Reddan, Dempsey & Forster (1976) attributed this to myocardial hypoxia resulting in decreased myocardial contractility.

At high altitudes stroke volume responses are variable. Asmussen et al. (1955) observed an 11% reduction in resting stroke volume at 4,300 m altitude. Hoon et al. (1977) also observed decreased resting stroke volume at 3,658 m, but other investigators have reported an increase (Assmussen et al. 1941) or no change in resting stroke volume (Saltz et al. 1976, Vogel et al. 1967). During exercise Asmussen et al. (1955) reported that stroke volume remained 2 to 10% lower than sea level values during submaximal exercise workloads of 60 to 180 W. However, Vogel et al. (1967) did not observe reduced stroke volumes during rest or submaximal exercise at 4,300 m altitude.

Interestingly, Stenberg et al. (1966) reported a decreased stroke volume when subjects exercised at 100 W, but did not observe any significant difference at 150 W when subjects exercised at 4,000 m. When subjects performed graded exercise at 5,800 m Pugh (1964) reported significant reductions in stroke volume for each exercise stage.

During maximal exercise at high altitudes stroke volume responses are highly variable with some reports of reduced (Vogel et al. 1974), elevated (Vogel et al. 1967) or no change (Stenberg et al. 1966).

Arterial Blood Pressure Resting arterial blood pressure does not appear to be altered by acute exposure to mild hypoxia similar to mild altitude. Ekblom, Huot, Stein & Thorstensson (1975) exposed subjects to 15% CO mixture to induce mild hypoxia (P_aO_2 = 96 mm Hg). No significant differences between normal and hypoxic treatments were observed in mean arterial pressure at rest. Alexander et al. (1967) reported no difference in resting systolic, diastolic or mean blood pressure upon acute exposure to moderate altitude. However, variable differences have been observed in resting blood pressures at high altitudes. Resting systolic pressures have been reported to be lower (Reeves et al. 1969) or unchanged (Stenberg et al. 1966; Vogel et al. 1967) at altitudes greater than of 4,000 m. Saltz et al. (1976) reported an increase in pulmonary arterial pressure, but mean arterial, and left atrial pressures were unaffected by 4,300 m.

Lower resting diastolic pressures have been observed during acute exposure to high altitude (Reeves et al. 1969; Stenberg et al. 1966). Meanwhile, resting mean arterial pressure has been observed to be decreased (Stenberg et al. 1966), increased (Weil, Bryne-Quinn, Battock, Grover & Chidsey, 1971), and unaffected (Vogel et al. 1967) by acute high altitude exposure.

During submaximal work at 30% and 70% of VO_2max Ekblom et al. (1975) found no difference in mean arterial pressure during mild CO-hypoxic treatment. At moderate altitude (3,100 m) Alexander et al. (1967) observed an increase in mean arterial pressure in subjects during low cycle ergometry workloads of 25, 50, 75, and 100 W. During submaximal exercise systolic pressures have been lower at high altitude (Reeves et al. 1969) and unaffected (Stenberg et al. 1966; Vogel et al. 1967), diastolic pressures have been lower (Reeves et al. 1969; Stenberg et al. 1966), resulting in no effect of mean arterial pressure (Vogel et al. 1967).

No significant altitude effect has been observed on systolic, diastolic (Squires et al. 1982) or mean arterial pressure (Ekblom et al. 1975) during maximal exercise under mild hypoxic conditions. Squires et al. (1982) reported a lower systolic pressure, but unaffected diastolic pressure during maximal exercise at moderate altitude, and Vogel et al. (1967) observed lower systolic and mean arterial pressures during maximal exercise at high altitude.

TABLE I

SUMMARY OF THE CARDIOVASCULAR RESPONSES DURING REST AND EXERCISE TO ALTITUDE

Physiological Variable	Mild Altitude	Moderate Altitude	High Altitude
Cardiac Output			
Resting	?	+	++
Submaximal Ex.	?	+	++
Maximal Ex.	?	=	= + -
Heart Rate			
Resting	=	= +	++
Submaximal Ex.	=	= +	++
Maximal Ex.	=	=	= -
Stroke Volume			
Resting	?	?	+ = -
Submaximal Ex.	?	-	+ = -
Maximal Ex.	?	?	+ = -

= no effect, + some evidence of increase, ++ strong evidence of an increase, - some evidence of a decrease, ? no literature reported

Respiratory Responses

Minute Ventilation Under resting conditions at mild altitude it is reported that there is no significant change is minute ventilation at BTPS (Adams & Welch, 1980; Bubb et al. 1983; Ekblom et al. 1975; Tucker, Stager & Cordain, 1984). There is also no effect on minute ventilation under resting conditions when subjects are exposed to moderate altitude (Bubb et al. 1983; Reeves et al. 1967; Tucker et al. 1984; Wagner et al. 1986). However, at higher elevations some investigators have observed increases in resting minute ventilation (Assmussen et al. 1941; Bender, McCullough, Huang, Wagner, Cymerman, Hamilton & Reeves, 1989; Reeves et al. 1969, Wagner et al. 1986). Others have not seen changes in resting minute ventilation at high altitude (Bubb et al. 1983;

Hansen et al. 1967b).

During submaximal exercise at mild altitude minute ventilation is reported to be higher than at low altitude (Adams et al. 1980; Astrand, 1954; Hogan et al. 1983), or unchanged (Bubb et al. 1983). However, Squires et al. (1982) observed no difference in minute ventilation at 914 or 1219 m, but did report significantly higher values at 1524 m. As the environment increases to moderate altitude minute ventilation is increased significantly for a given submaximal workload (Astrand 1954; Grover, Reeves, Grover & Leathers, 1967; Hughes et al. 1968; McManus et al. 1974; Reeves et al. 1967; Squires et al. 1982; Wagner et al. 1986). However, Bubb et al. (1983) did not observe a significant difference in minute ventilation at 3,000 m when exercising at 40% of VO_2max. At high altitude there is an increase in minute ventilation during submaximal work (Assmussen et al. 1941; Astrand 1954; Bubb et al. 1983; Hansen et al. 1967b; Hughes et al. 1968; Knuttgen et al. 1973b; Maher, Jones & Hartley, 1974; Stenberg et al. 1966; Wagner et al. 1986).

A significant increase in maximal minute ventilation during acute mild altitude exposure has been reported (Hogan et al. 1983; Tucker et al. 1984). However, Squires et al. (1982) did not observe significant changes in maximal minute ventilation during a treadmill GXT when subjects ran at 914, 1219, and 1524 m of elevation. When the subjects performed the GXT at 2286 m, maximal minute ventilation was significantly higher. Other investigators have reported similar increases in maximal minute ventilation during moderate altitude exposure (Fagraeus et al. 1973; Tucker et al. 1984; Wagner et al. 1979). Interestingly, not all researchers have observed increased maximal minute ventilation (Hughes et al. 1983; Reeves et al. 1967). The conflicting results in minute

ventilation have also been reported during maximal exercise at high altitude with some investigators reporting increases (Buskirk, Kollias, Akers, Prokop & Reategui, 1967; Cymerman et al. 1989; Hansen et al. 1967b; Knuttgen et al. 1973a) while others observed no differences (Hughes et al. 1968; Klaussen et al. 1966, Stenberg et al. 1966).

Oxygen Saturation At rest oxygen saturation is significantly lower at mild (Tucker et al. 1984), moderate (Reeves et al. 1969; Wagner et al. 1986), and high altitude (Assmussen et al. 1941; Bender et al. 1989; Hansen et al. 1967b; Vogel et al. 1974; West, Lahiri, Gill, Milledge, Pugh & Ward, 1962). When the demands for oxygen increase during submaximal and maximal exercise oxygen saturation declines further at mild (Squires et al. 1982; Tucker et al. 1984), moderate (Reeves et al. 1967; Squires et al. 1982; Tucker et al. 1984), and high altitude (Assmussen et al. 1941; Cymerman et al. 1989; Hansen et al. 1967b; Stenberg et al. 1966; Vogel et al. 1974; West et al. 1962). These decreases in oxygen saturation occur at increasing altitude, decreasing barometric pressure, P_IO_2 and P_AO_2, and could not be offset by coupling the increases observed in cardiac output and minute ventilation at rest and exercise.

Metabolic Responses

Oxygen Consumption Resting oxygen consumption does not appear to be affected by acute mild (Adams et al. 1980; Hogan, Cox & Welch, 1983; Tucker et al. 1984), moderate (Tucker et al. 1984; Wagner et al. 1986) or high altitude exposure (Assmussen et al. 1941; Hansen et al. 1967b; King, Dodd & Cain, 1987; Reeves et al. 1969; Wagner et al. 1986). During submaximal exercise, oxygen consumption also does

not appear to be affected by acute mild (Adams et al.1983 ; Ekblom et al. 1975; Hogan et al. 1983; Squires et al. 1982), moderate (Hughes et al. 1968; McManus et al. 1974) or high altitude exposure (Assmussen et al. 1941; Hansen et al. 1967b; Hartley et al. 1971; Hughes et al. 1968; Katz et al. 1987; King et al. 1987; Knuttgen et al. 1973b; Pugh et al. 1964; Stenberg et al. 1966). However, Squires et al. (1982) observed a reduced oxygen consumption when subjects exercised at 2,286 m. Others reported reduced oxygen consumption during submaximal exercise between 3,000 and 4,600 m (Hansen et al. 1967b; Wagner et al. 1986).

Maximal oxygen consumption has generally been reported to be reduced at altitudes from 600 (Terrados et al. 1985) to 7,400 m (Pugh et al. 1964). However, at mild altitudes of 914 and 1,600 m Squires et al. (1982) and Hogan et al. (1986) respectively, did not observe a significant reduction in maximal oxygen consumption. Others have reported reduced oxygen consumption during maximal exercise at mild altitudes between 1,200 and 1,524 m of elevation (Squires et al. 1982; Terrados et al. 1985; Tucker et al. 1984). There is agreement among investigators that maximal oxygen consumption is reduced at moderate (Bouissou, Peronnet, Brisson, Helie & Ledous, 1986; Fragraeus et al. 1973; Faulkner et al. 1968; Grover et al. 1967; Reeves et al.1967 ; Saltin 1996; Squires et al. 1982; Tucker et al. 1984; Wagner et al. 1979) and high altitude (Buskirk et al. 1967; Cymerman et al. 1989; Faulkner et al. 1968; Hansen et al. 1967b; Hartley et al. 1971; Klaussen et al. 1966; Knuttgen et al. 1973b; Linnarsson et al. 1974; Maher et al. 1974; Pugh et al. 1964; Stenberg et al. 1966).

Upon review of the research Grover et al. (1986) has suggested that maximal oxygen consumption is reduced by 8% for every 1,000 m of altitude above 700 m.

However, Young (1990) suggests that altitude has no effect on maximal oxygen consumption up to 1,000 m, at 1,000 to 2,000 m there is a small and variable decrease in maximal oxygen consumption, and above 2,000 m there is a decrease of 10% for every 1,000 m increase in altitude. Meanwhile, Haymes et al. (1986) concluded that a threshold of 1,500 m exists, before maximal oxygen consumption is reduced by altitude.

Blood Lactate Resting blood lactate responses to acute altitude exposure have been variable. McManus et al. (1974) reported no change in blood lactate upon elevation to above 3,000 m, but Wagner et al. (1986) and Edwards (1936) observed increased resting blood lactate at altitudes above 2,800 to 6,100 m. At high altitude others did not observe a change in resting blood (Hansen et al. 1967a; Harboe, 1957) or muscle lactate (Katz et al. 1987). Harboe (1957) did report a rise in blood lactate at 6,100 m.

During submaximal exercise at 2,000 m, Adams et al. (1980) did not observe a difference in blood lactate at 55% or 90% of VO_2max. Hogan et al. (1983) reported no difference in blood lactate at workloads up to 120 W, but lactate was significantly higher when workloads were greater than 195 W in subjects exercising at 1,600 m. Meanwhile, Lundin & Strom (1947) did observe a higher blood lactate when subjects exercised at altitudes between 1,400 to 5,500 m. There does appear to be an increase in blood lactate during submaximal exercise at moderate altitude (Edwards 1936; Lundin et al. 1947; McManus et al. 1974; Saltin 1996; Wagner et al. 1986). However, Hughes et al. (1968) and Lorentzen (1962) have reported conflicting (unchanged to higher) blood lactate values when subjects exercised between 2,100 and 5,100 m. At high altitude, lactate is generally higher in blood (Edwards et al. 1936; Linnarsson, Karlsson, Fagraeus & Saltin,

1974; Lundin et al. 1947; Stenberg et al. 1966; Wagner et al. 1986) and muscle (Katz et al. 1987; Linnarsson et al. 1974) at similar exercise workloads. Hansen et al. (1967a) did not report changes in blood lactates during submaximal exercise at 4,300 m, but Hughes et al. (1968) and Lorentzen (1962) reported variable differences in blood lactates during exercise at high altitude.

At mild altitude, maximal blood lactates have been observed to be similar at 1,600 m (Hogan et al. 1983) and elevated at 2,000 m (Adams et al. 1980) compared to sea level. There does not appear to be any difference in maximal blood lactate at moderate altitude (Bouissou et al 1986; Fagraeus et al. 1973; Saltin 1996; Wagner et al. 1973). Klaussen et al. (1966) reported decreased maximal blood lactate at 3,800 m. This may be due to lower maximal work at performed 3,800 m. Others have not observed a change in maximal blood (Hansen et al. 1967a; Linnarsson et al. 1974; Stenberg et al. 1966) or muscle lactate at high altitude (Linnarsson et al. 1974).

Maximal Performance

At mild altitude beginning at 1,600 m of elevation there is a debatable effect on human performance aerobic in nature. Adams et al. (1980) observed a decrease in time to exhaustion from 12.5 min to 9.7 min when trained subjects exercised at 90% of VO_2max while breathing 16.82% oxygen (similar to 2,000 m). Hogan et al. (1983) reported a decrease in maximal workload when subjects performed GXT's while breathing 17.2% oxygen (similar to 1,600 m). At moderate altitudes some investigators have reported decreases in middle distance and distance track events (Craig 1969; Faulkner et al. 1968; Grover et al. 1967). Others observed decreases in performance measured as time to

exhaustion during laboratory exercise (Fagraeus et al. 1973) and maximal exercise workload during a GXT (Hughes et al. 1968). At high altitude previous investigators have reported decreases in maximal workload (Hughes et al. 1968; Klaussen et al. 1966; Linnarsson et al. 1974; Maher et al. 1974) and time to exhaustion (Young, et al. 1984).

TABLE II

SUMMARY OF THE RESPIRATORY AND METABOLIC RESPONSES DURING REST AND EXERCISE TO ALTITUDE

Physiological Variable	Mild Altitude	Moderate Altitude	High Altitude
Minute Ventilation			
Resting	=	=	=+
Submaximal Ex.	=+	+	=+
Maximal Ex.	=+	=+	=+
Oxygen Saturation			
Resting	-	--	--
Submaximal Ex.	-	-	--
Maximal Ex.	--	--	--
Oxygen Consumption			
Resting	=	=	=
Submaximal Ex.	=	=	=
Maximal Ex.	=-	--	--
Blood Lactate			
Resting	?	=+	=+
Submaximal Ex.	=+	=+	=+
Maximal Ex.	=+	=	=-

= no effect, + some evidence of increase, ++ strong evidence of an increase,
- some evidence of a decrease, - - strong evidence of a decrease, ? no literature reported

CHAPTER III

METHODS AND PROCEDURES

The purpose of this study was to determine the effects of mild acute simulated altitude exposure on the cardiovascular, respiratory, and metabolic responses to graded exercise. The specific responses measured include oxygen saturation, heart rate, blood pressure, minute ventilation, oxygen consumption, and blood lactate concentration.

Prior to initiating this study, approval from Oklahoma State University's Institutional Review Board was obtained, and subsequent modifications to the experimental protocol were also approved (Appendix A). All of the procedures were conducted in accordance with the guidelines for ethical treatment of human subjects, and exercise testing followed established guidelines (American College of Sports Medicine, 1995).

Participants

The participants were ten (8 male, 2 female) adult cyclists (age = 31 $\pm$ 2 yrs.; mean $\pm$ sem). Eight of the participants were actively trained as members of the Tulsa Wheelmen Bicycle Club in Tulsa, Oklahoma, and two trained independently in Stillwater, Oklahoma. These participants were classified as trained, using the criteria of a minimum bicycle training routine of 3 days per week for 30 minutes per session for the

last 6 months. Prospective participants were solicited via flyers and personal contact at organizational weekend training rides. Prior to testing the participants completed a medical history questionnaire (Appendix B). All participants were classified as apparently healthy using ACSM guidelines (American College of Sports Medicine, 1995): asymptomatic with no more than one major coronary risk factor. Furthermore, the participants were evaluated by a physician for medical clearance (Appendix C) and safety prior to exercise testing. The participants then gave informed consent to serve as participants (Appendix D).

Upon acceptance each participant scheduled 2 graded exercise tests (GXT) to be performed within 5 to 10 days of each other inside the Oklahoma State University College of Osteopathic Medicine Hypobaric Chamber (United States Air Force 20 man Guardite Stratosphere Chamber) at the Tulsa Technology Center. Prior to reporting for testing the subjects were given the following instructions:

1. Refrain from consuming food, alcohol, caffeine, tobacco or drugs within three hours of the scheduled test. Water consumption is recommended to maintain hydration.
2. Avoid any vigorous exercise within twelve hours of the scheduled test.
3. Dress for maximal exercise should include: shorts, short sleeve t-shirt, athletic bra (for females), and athletic shoes.

Upon arrival for their first exercise test the participants descriptive information was collected. The age of each participant was self reported in years. Each participant was then measured in their exercise clothes without shoes for height and weight using a

Detecto Standard Physicians Scale and Stadiometer. Skinfold thickness was measured on the right side of the body using Lange calipers at the following seven sites: 1) chest, 2) subscapula, 3) tricep, 4) mid-axilla, 5) suprailiac, 6) abdominal, and 7) thigh. The skinfold site locations followed those outlined by Jackson and Pollock (1978). Each site was measured twice, and if a difference was observed to be greater than 2 mm a third measurement was obtained. The skinfold trials were then averaged for each site and summed together. Body density was estimated from the sum of the seven skinfold sites and age using gender specific formulae (Jackson et al. 1978; Jackson, Pollock & Ward, 1980). Percent body fat was estimated from body density using the Siri equation (Siri, 1961).

Preparation for Exercise Testing

Following collection of descriptive data the participant entered the hypobaric chamber with the researcher. The chamber operator closed and sealed the door to the chamber, and decreased the internal barometric pressure to either 733 mm Hg (altitude equivalent of 305 m above sea level) or 609 mm Hg (altitude equivalent of 1829 m above sea level). The rate of assent and later descent was 914 m/minute. The simulated altitude was blind to the participant, and the order was randomly assigned.

Once the desired simulated altitude was attained a Monark 818E mechanically braked bicycle ergometer was calibrated using a 2.25 kg weight according to standard procedures (Howley, 1988). The seat height of the bike was then adjusted by the participant to allow for near complete knee extension of the participant during the down stroke of pedaling. The participant also adjusted the handle bar position for comfort. The

seat height and handle bar position were measured and recorded for use in the second GXT. The toe straps of the bike were adjusted to secure the participants feet to the pedals.

After the bike was set, the participant was prepped for collection of the physiological data. The preparation included placement of a blood pressure cuff on the participants right arm. The blood pressure cuff was attached to a floor standing mercury sphygmomanometer. A portable pulse oximeter (Burdick Model 100) was secured to the participants index finger on their dominant hand to measure oxygen saturation. A Polar telemetry heart rate transmitter was strapped to the participants chest at the level of the xiphoid process. The Polar Beat telemetry heart rate receiver was strapped to the handle bars of the bicycle ergometer. A Hans-Rudolph mask was then placed on the participants face and secured by straps around the head. The mask consisted of two small one way valves for inspiration and one large one way valve for expiration. The mask was attached to a three meter tube which was later secured to a metabolic cart (Quinton Q-Plex I Cardio-Pulmonary Exercise System). The participants descriptive data was then entered into the metabolic cart. The metabolic cart was then calibrated for volume and gas concentrations according to manufacturers specifications (Quinton Instrument Company, 1989), and programmed to calculate minute ventilation (BTPS) and oxygen consumption (STPD) in 30 second intervals. Following calibration the expiration tube was attached to the internal mixing chamber of the metabolic cart.

Exercise Testing and Physiologic Data Collection

Following preparation and equipment calibration the participant remained in the

seated position on the bike for 5 minutes prior to data collection. During this period proper functioning of the laboratory instruments was verified. Afterwards, 3 minutes of resting data was collected while the participant remained seated. Heart rate and oxygen saturation were observed and recorded during the last 5 seconds of each minute. Blood pressure was measured after the first minute. Systolic blood pressure was determined as the first Korotkoff sound, and diastolic pressure as the change from the 4th to 5th Korotkoff sound. Rate of perceived exertion (RPE) was self-reported following blood pressure using the Borg Scale (Borg, 1982). Minute ventilation and oxygen consumption were calculated by the metabolic cart in 30 second intervals.

Blood lactate concentrations were measured between minutes 2 and 3 of each workload using an Accusport portable lactate analyzer via finger tip capillary blood samples from the non-dominant hand of the participants. Immediately prior to obtaining the blood sample an Accusport dry chemical strip was placed in the analyzer. During each sampling period the finger tips were cleaned with an alcohol swab and dried with a cotton ball. A thin layer of petroleum jelly was applied to the area with a cotton swab. A lancet secured to an Autolet was used to puncture the skin. A large drop of blood (~ 20 microliters) was collected in a heparinized capillary tube attached to a rubber bulb and then expelled on the dry chemical strip for analysis. The blood lactate concentration (mM) was determined by the analyzer in 60 seconds and recorded. The used lancet and capillary tube was disposed of in a sharps container. A cotton ball was placed over the puncture site to stop blood flow. The used alcohol swab, cotton swab, cotton ball, and dry chemical strip were disposed of in a biohazard container. Each subsequent blood sample was taken from a different finger and/or a different location on the same finger.

The GXT began at a workload of 70 W (pedal cadence of 70 rpm with 1 kg of friction) and increased by 70 W (1 kg of friction) every 3 minutes until volitional exhaustion. Volitional exhaustion was determined to be either the point where the participant quit or when the participant was unable to maintain the appropriate pedal cadence.

During the GXT, heart rate and oxygen saturation were monitored and recorded from the appropriate receiver at the end of each minute of exercise. Blood pressure was monitored during the second minute of each exercise stage. Rate of perceived exertion was reported by the participant at the end of the second minute of each stage. Minute ventilation and oxygen consumption were measured and recorded in 30 second intervals throughout the rest and exercise periods using a Quinton Q-Plex I met cart. Blood samples were obtained for lactate analysis during the last minute of each stage.

Analysis of Data

Prior to data analysis, each participants resting oxygen consumption, minute ventilation, heart rate, and oxygen saturation at each altitude were averaged. Subsequently, for the last minute of each stage oxygen consumption and minute ventilation data were also averaged and were considered a representative value for that workload. Maximal or peak oxygen consumption and minute ventilation was determined to be the highest measured over 30 sec. Maximal workload is defined as the highest workload the participant exercised at for at least 1 min. Maximal heart rate was the highest recorded heart rate during the GXT.

All data was entered into a SAS Institute, Inc. data file for analysis. The maximal

data were analyzed using paired t-tests to reveal significant differences between the two altitude treatments. A randomized block on subjects with split-plot on altitude and workload ANOVA was used to determine the main effects and interaction effects of the altitude treatment on the physiological responses at each workload (Steel & Torrie, 1980). Any significant main effects due to altitude or interaction effects were further analyzed with a Bonferroni multiple comparison technique using the appropriate error term (Milliken & Johnson, 1992). All measures are reported as mean $\pm$ standard error of the mean (sem). And subsequently the significance level for this experiment is $p < 0.05$.

CHAPTER IV

RESULTS AND DISCUSSION

The purpose of this study was to determine the effects of mild acute simulated altitude exposure on the cardiovascular, respiratory, and metabolic responses to graded exercise. Eight male and two female trained cyclists served as the participants. The descriptive characteristics of these participants are reported in Table III.

TABLE III

DESCRIPTIVE CHARACTERISTICS OF PARTICIPANTS (mean $\pm$ sem)

Variable	Male (n=8)	Female (n=2)
Age (yrs)	32 ± 3	29 ± 1
Body Fat (%)	9.5 ± 1.0	17.0 ± 1.7
Height (m)	1.78 ± 0.02	1.66 ± 0.01
Weight (kg)	75.5 ± 2.8	64.3 ± 2.9
VO_2max (ml/kg/min)	63.8 ± 2.2	51.8 ± 2.4

Results

When the participants of this study were seated on the bike and exposed to 1,829 m of altitude there were no significant changes in heart rate, blood pressure, minute ventilation, oxygen saturation, or blood lactate. However, when oxygen demand

increased with exercise there was a significant reduction in oxygen saturation ($p < 0.05$) at 1829 m for 70, 140, 210, 280 W, and maximal workloads respectively (Table IV & Figure I). This decrease was not affected further by increasing workload. At the highest workload (350 W) only 2 participants were able to exercise for greater than 1 minute at both altitudes. Therefore, no meaningful statistical comparisons were made for any of the physiological variables at 350 W. Oxygen saturation during the highest workload for each participant was significantly reduced from 94% at 305 m to 86% at 1829 m.

TABLE IV

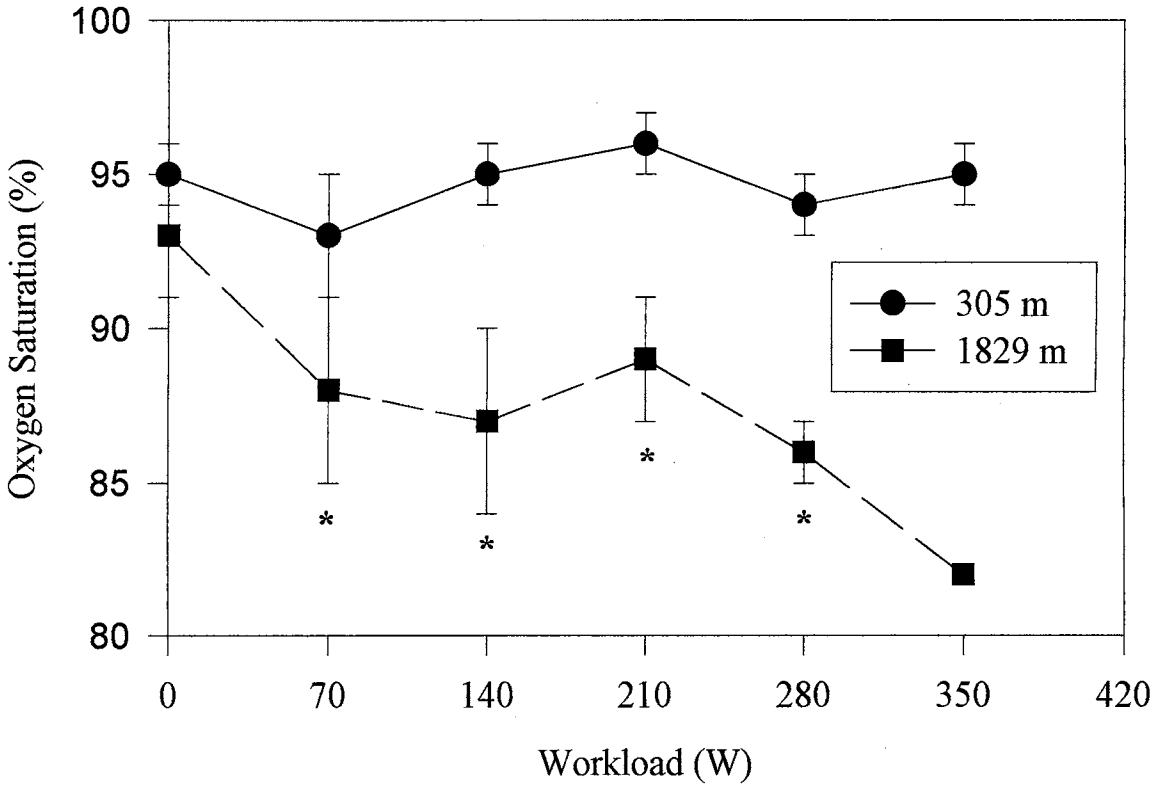
OXYGEN SATURATION (%) RESPONSES TO GRADED EXERCISE AT 305 m AND 1829 m OF ALTITUDE (mean ± sem).

Workload (W)	305 m.	1829 m.
Rest	95 ± 1	93 ± 2
70	93 ± 2	88 ± 3*#
140	95 ± 1	87 ± 3*
210	96 ± 1	89 ± 2*
280	94 ± 1	86 ± 1*#
350	95 ± 1	82 ± 0
Maximal	94 ± 1	86 ± 1*

* significantly different than 305 m. ($p < 0.05$).
# significantly different than previous workload ($p < 0.05$).

FIGURE I

OXYGEN SATURATION RESPONSES TO GRADED EXERCISE AT 305 m AND 1829 m OF ELEVATION



* Significantly different than 305 m ($p<0.05$)

Resting heart rate was not affected by altitude. There was also no significant altitude effect on exercise heart rate at any workload during the GXT, and maximal heart rate was not effected by 1829 m of altitude (Table V & Figure II). However, there was a significant increase in heart rate due to exercise workload regardless of altitude.

TABLE V

HEART RATE RESPONSES TO GRADED EXERCISE AT 305 m AND 1829 m OF ELEVATION (mean ± sem).

Workload (W)	305 m	1829 m
Rest	71 ± 3	69 ± 4
70	99 ± 3#	104 ± 4#
140	120 ± 4#	128 ± 5#
210	145 ± 5#	152 ± 5#
280	166 ± 6#	167 ± 5#
350	179 ± 7	171 ± 9
Maximal	176 ± 5	172 ± 4

significantly different than previous workload ($p < 0.05$).

Systolic blood pressure response data are shown in table VI and figure III. Resting systolic blood pressure wasn't affected by altitude (305 m: 122 ± 3; 1829 m 129 ± 6 mm Hg). Systolic blood pressure increased significantly during each workload, but was not effected by altitude. During maximal exercise workload attained by each participant, systolic blood pressure was also not altered due to altitude. Meanwhile diastolic blood pressure remained unchanged regardless of exercise workload or altitude (Table VII & Figure III).

FIGURE II

HEART RATE RESPONSES TO GRADED EXERCISE AT
305 m AND 1829 m OF ELEVATION

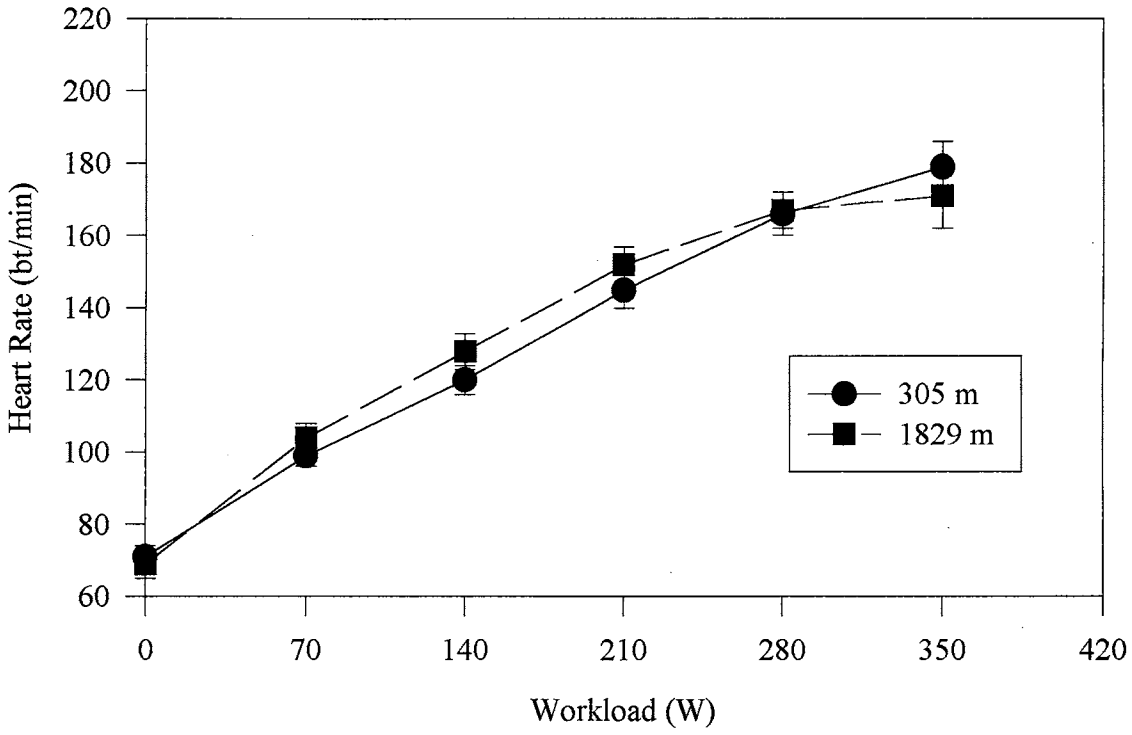


TABLE VI

SYSTOLIC BLOOD PRESSURE (mm Hg) RESPONSES TO GRADED EXERCISE AT 305 m AND 1829 m OF ELEVATION (mean ± sem).

Workload (W)	305 m	1829 m
Rest	122 ± 3	129 ± 6
70	140 ± 3#	139 ± 4#
140	153 ± 4#	159 ± 3#
210	172 ± 4#	174 ± 4#
280	186 ± 5#	186 ± 4#
350	206 ± 6	203 ± 7
Maximal	189 ± 7	187 ± 5

significantly different than previous workload ($p < 0.05$).

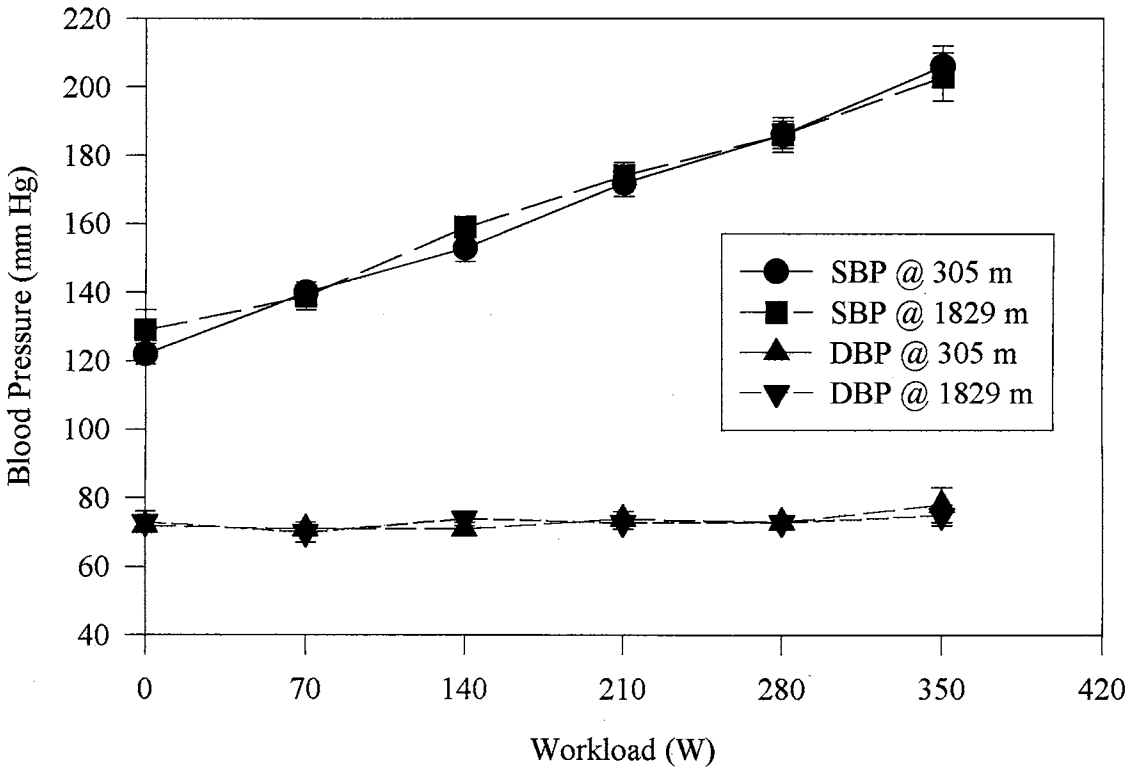
TABLE VII

DIASTOLIC BLOOD PRESSURE (mm Hg) RESPONSES TO GRADED EXERCISE AT 305 m AND 1829 m OF ELEVATION (mean ± sem).

Workload (W)	305 m	1829 m
Rest	72 ± 2	73 ± 3
70	71 ± 2	70 ± 3
140	71 ± 2	74 ± 2
210	74 ± 2	73 ± 2
280	73 ± 1	73 ± 1
350	78 ± 5	75 ± 3
Maximal	74 ± 2	73 ± 1

FIGURE III

BLOOD PRESSURE RESPONSES TO GRADED EXERCISE AT
305 m AND 1829 M OF ELEVATION



Minute ventilation measured at BTPS was not affected by altitude at rest (305 m: 15.9 ± 3.3; 1829 m: 16.4 ± 3.8 l/min) or at exercise workloads of 70 or 140 W (Table VIII & Figure IV). However, at higher workloads, 210 and 280 W, minute ventilation was significantly higher when the participants exercised at 1829 m. Workload also exerted a significant increase in minute ventilation. Despite the altitude effect on the higher workloads there was no effect on maximal minute ventilation.

TABLE VIII

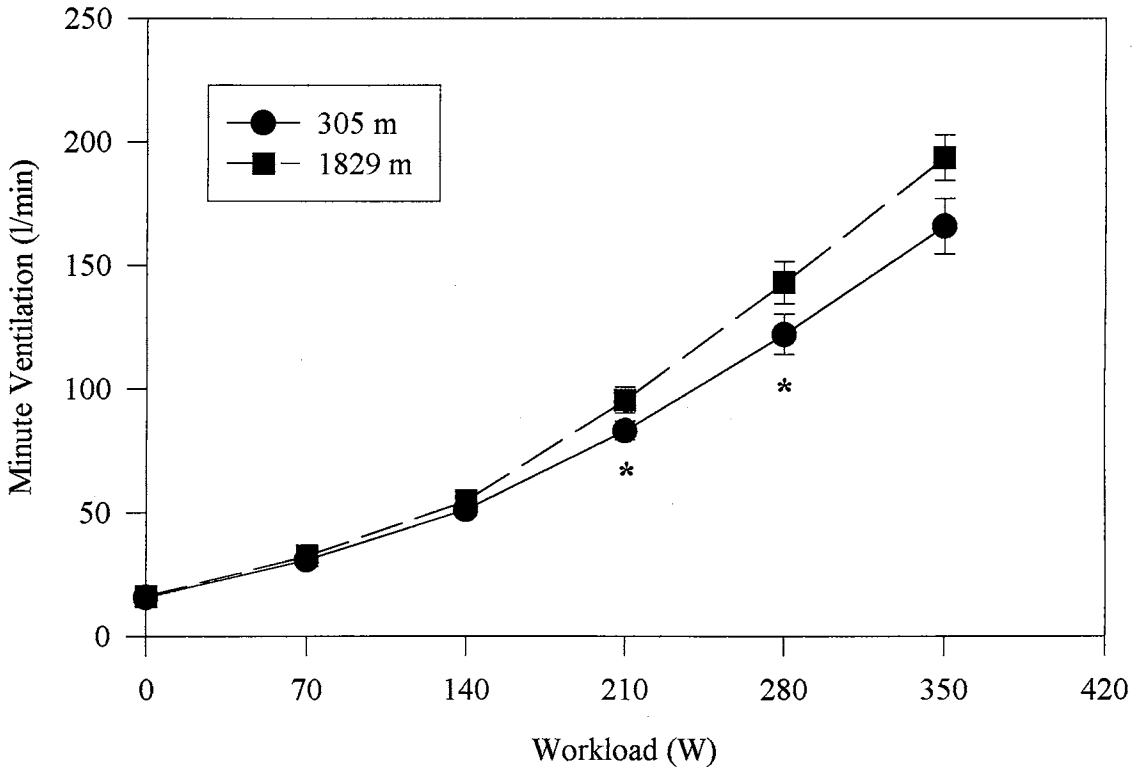
MINUTE VENTILATION (l/min) RESPONSES TO GRADED EXERCISE AT 305 m AND 1829 m OF ELEVATION (mean ± sem).

Workload (W)	305 m	1829 m
Rest	15.9 ± 3.3	16.4 ± 3.8
70	30.9 ± 1.0#	32.7 ± 1.0#
140	51.3 ± 1.6#	54.8 ± 1.7#
210	83.2 ± 3.8#	95.6 ± 5.3*#
280	121.9 ± 8.2#	143.0 ± 8.7*#
350	166.0 ± 11.2	193.6 ± 9.2
Maximal	166.4 ± 8.3	168.8 ± 10.0

* significantly different ($p < 0.05$).
# significantly different than previous workload ($p < 0.05$).

FIGURE IV

MINUTE VENTILATION (BTPS) RESPONSES TO GRADED EXERCISE AT 305 m AND 1829 m OF ELEVATION



* significant difference ($p<0.05$).

The oxygen consumption data is presented in Table IX and Figure V. Resting oxygen consumption was not altered during exposure to 1829 m of altitude. Exercise oxygen consumption was not effected by altitude at any submaximal exercise workload, but maximal oxygen consumption was significantly lower when the participants exercised at altitude (305 m: 4.45 $\pm$ 0.18; 1829 m: 4.04* $\pm$ 0.11 l/min). Exercise workload also exerted a significant increase in oxygen consumption regardless of altitude.

TABLE IX

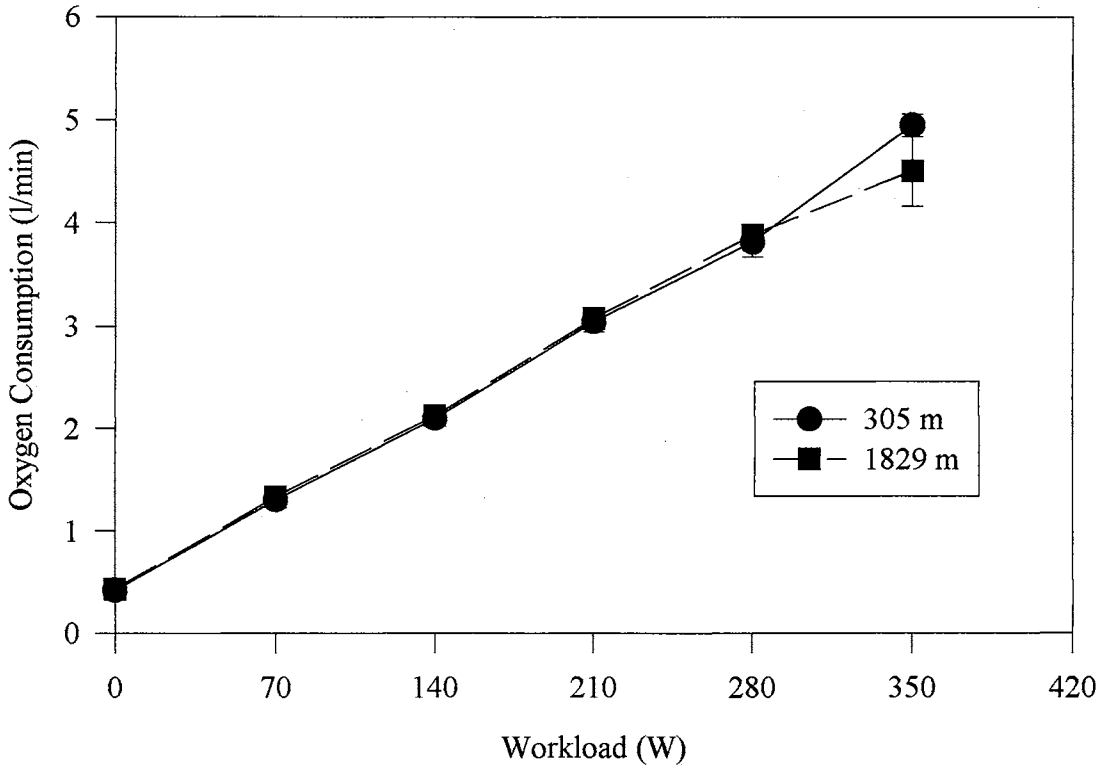
OXYGEN CONSUMPTION (l/min) RESPONSES TO GRADED EXERCISE AT 305 m AND 1829 m OF ELEVATION (mean $\pm$ sem).

Workload (W)	305 m	1829 m
Rest	0.42 $\pm$ 0.04	0.43 $\pm$ 0.03
70	1.30 $\pm$ 0.04#	1.33 $\pm$ 0.04#
140	2.10 $\pm$ 0.07#	2.13 $\pm$ 0.07#
210	3.05 $\pm$ 0.10#	3.08 $\pm$ 0.05#
280	3.81 $\pm$ 0.14#	3.88 $\pm$ 0.10#
350	4.95 $\pm$ 0.11	4.50 $\pm$ 0.34
Maximal	4.45 $\pm$ 0.18	4.04 $\pm$ 0.11*

* significantly different than 305 m ($p < 0.05$).
# significantly different than previous workload ($p < 0.05$).

FIGURE IV

OXYGEN CONSUMPTION RESPONSES TO GRADED EXERCISE AT 305 m AND 1829 m OF ELEVATION



Resting and exercise blood lactate concentrations were not effected by altitude (Table X & Figure V). There was a significant workload effect on blood lactates, but samples obtained during the highest workload attained were not altered at altitude (305 m: 7.49 ± 0.84; 1829 m: 8.34 ± 0.95 mM).

TABLE X

BLOOD LACTATE (mM) RESPONSES TO GRADED EXERCISE AT 305 m AND 1829 m OF ELEVATION (mean ± sem).

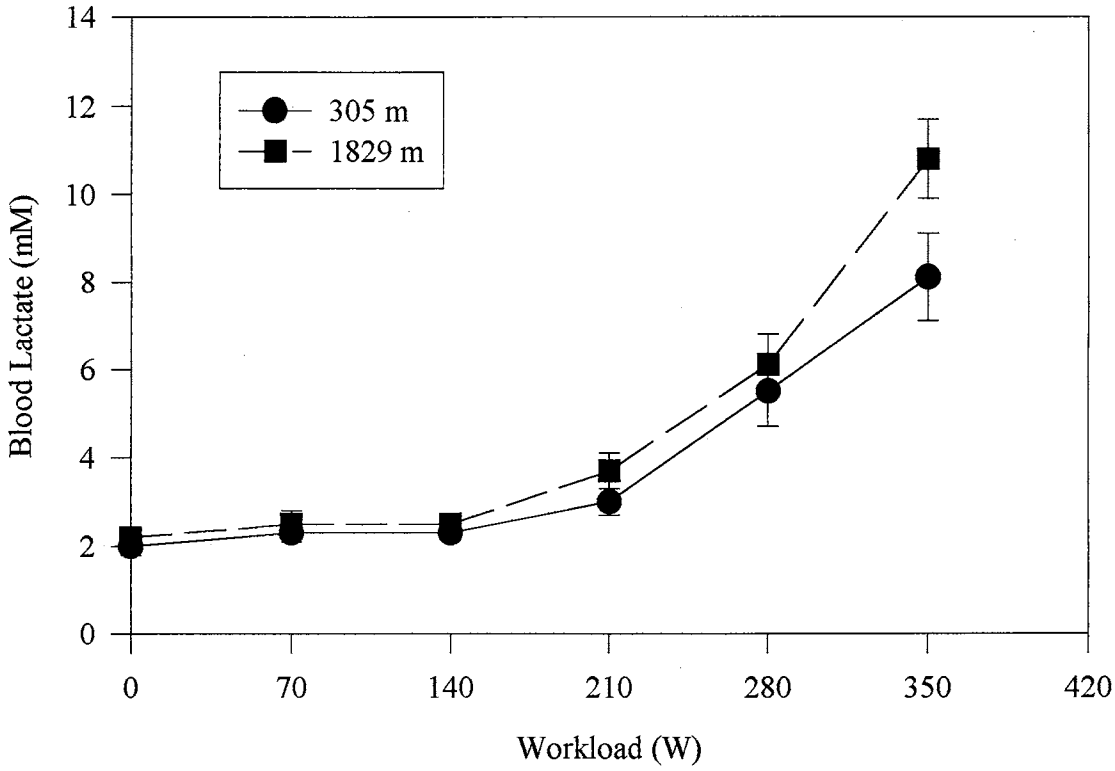
Workload (W)	305 m	1829 m
Rest	2.0 ± 0.2	2.2 ± 0.2
70	2.3 ± 0.2	2.5 ± 0.3
140	2.3 ± 0.1	2.5 ± 0.2
210	3.0 ± 0.3#	3.7 ± 0.4#
280	5.5 ± 0.8#	6.1 ± 0.7#
350	8.1 ± 1.0	10.8 ± 0.9
Maximal	7.5 ± 0.8	8.3 ± 1.0

#significantly different than previous workload ($p < 0.05$).

The total exercise time was reduced from 14.02 ± 0.63 to 12.96* ± 0.61 min when the participants exercised at 1829 m. As a result the maximal workload was also significantly reduced from 335 ± 14 W at low altitude to 308* ± 19 W at 1829 m.

FIGURE VI

BLOOD LACTATE RESPONSES TO GRADED EXERCISE AT
305 m AND 1829 m OF ELEVATION



CHAPTER V

DISCUSSION

The findings of this study indicate that at 1829 m there is a trend towards decreased resting oxygen saturation (2%) compared to 305 m of elevation. However, in a previous study (Tucker et al.1984) a significant decrease of 3.6% in resting oxygen saturation, measured by ear oximetry, was reported in trained runners when exposed 1,520 m altitude versus sea level. Under resting conditions no significant hypoxia was observed in the present study. This may be due to the relatively flat slope for the relationship between oxygen saturation and the partial pressure of oxygen associated with this altitude and individual variability.

During the submaximal and maximal stages of the graded exercise test, mild hypoxia is evident by the significant decrease in oxygen saturation in cyclists at 1829 m. This is consistent with previous investigations at mild altitude (Squires et al. 1982; Tucker et al. 1984). As exercise began at 1829 m there was an immediate 5% reduction in oxygen saturation. This reduction remained fairly constant throughout the exercise test. Tucker et al. (1984) previously reported a significant reduction in oxygen saturation when comparing maximal work to resting regardless of altitude. The increased demand for and extraction of oxygen, as measured by increased arterial-venous oxygen difference (Grover et al. 1986), during exercise was not offset by the cardiovascular and respiratory

systems.

Despite the presence of mild hypoxia there was no altitude effect on heart rate at rest or during submaximal and maximal work. This is consistent with previous studies at similar altitudes (Bubb et al. 1983; Squires et al. 1982). Since heart rate has been reported to be the main factor contributing to increased cardiac output at high altitude (Grover et al. 1986), it is assumed that cardiac output was not altered by altitude during exercise. This would suggest that the levels of hypoxia observed during submaximal and maximal exercise at mild altitude is not sufficient enough to cause increased sympathetic response to the heart when compared to low altitude.

Blood pressure measures at rest, during submaximal, and maximal exercise were not effected by mild altitude. This is consistent with previous reports (Ekblom et al. 1975; Squires et al. 1982). Systolic blood pressure increased and diastolic blood pressure remained relatively constant during the graded exercise test as expected (Durstine et al. 1988) regardless of altitude.

As expected resting minute ventilation was not altered at mild altitude (Adams et al. 1980; Bubb et al. 1983; Tucker et al. 1984). This may be the result of maintained oxygen saturation at rest. During exercise at low intensity workloads of 70 and 140 W (29 and 47% of low altitude maximal oxygen consumption) minute ventilation was not effected by mild altitude exposure. However, at workloads of 210 and 280 W (68 and 86% of low altitude maximal oxygen consumption) minute ventilation was significantly higher when the participants were at 1829 m. These findings do not clarify the issue of minute ventilation response to submaximal work at mild altitude. Previous investigators have observed increased (Adams et al. 1980; Astrand 1954; Hogan et al. 1983; Squires et

al. 1982) or unchanged minute ventilation (Bubb et al. 1983; Squires et al. 1982) at mild altitude during submaximal exercise.

In the present study, a decreased arterial partial pressure of oxygen, due to the observed decrease in oxygen saturation and possibly increased arterial-venous oxygen difference, at the higher workloads most likely stimulated the carotid chemoreceptors. However, it is possible that increased catecholamines produced during hypoxia and strenuous exercise (Welch 1987) may also stimulate peripheral chemoreceptors resulting in increased minute ventilation. It should be noted that partial pressure of carbon dioxide and hydrogen ion concentration in blood are decreased during hyperventilation when exercising at altitude (Welch 1987), and therefore do not provide excitatory chemical stimuli to the chemoreceptors. In fact Ganong (1997) stated that "the chemical stimulatory effects of hypoxia on ventilation are not clearly manifest until they become strong enough to override the counterbalancing inhibitory effects of a decline in arterial H^+ concentration and PCO_2". This may explain the observed minute ventilation response in the present study.

Maximal minute ventilation at BTPS was not observed to significantly different in this study. In a review of the literature Welch (1987) reached a similar conclusion concerning hypoxia and maximal minute ventilation. However, some investigators have reported significantly higher maximal minute ventilation at mild altitude (Hogan et al. 1983; Tucker et al. 1984).

Oxygen consumption was not significantly different at rest or during any submaximal exercise workload at 1829 m. Previous investigators observed the same results at mild altitude (Adams et al. 1980; Hogan et al. 1983; Tucker et al. 1984). It is

apparent that the amount of energy required to perform an absolute workload on a stationary bicycle ergometer is not affected by mild altitude.

In the present study there was a significant reduction of 9.2% in maximal oxygen consumption. At slightly lower altitudes of between 1,200 and 1,600 m Squires et al. (1982) reported 5 and 7% reductions, and at 1,520 m Tucker observed a 6.5% reduction in maximal values compared to sea level. However, at 900 m Squires et al. (1982) reported an insignificant reduction of 2%, and at 1,600 m Hogan et al. (1983) reported an insignificant 4% reduction in maximal oxygen consumption. The physiological mechanisms responsible for the observed decrease in maximal oxygen consumption are debatable. However, it is apparent that the ability of the respiratory and cardiovascular system to deliver oxygen to the muscles during strenuous exercise is impaired at mild altitude. As a result of hypoxia, muscles may be required to rely to a greater extent on anaerobic metabolism to produce the energy required to perform work.

However, blood lactate values were not significantly different between the two altitudes during rest, submaximal or maximal exercise conditions. Previous studies at mild altitude have reported mixed findings regarding blood lactates during submaximal and maximal exercise (Adams et al. 1980; Hogan et al. 1983; Lundin et al. 1947). The lack of altered blood lactate differences during submaximal exercise may be due to the relatively mild hypoxia present at the altitude in question. At moderate and high altitudes there is generally a significant higher blood lactate concentration at the same submaximal workload (Young 1990). It should be noted that the stage lengths were 3 minutes and the work increments were 70 W. Previous investigations have been performed with sampling done at 3, 5, and 10 minutes with various exercise protocols (Adams et al. 1980; Hogan et

al. 1983; Lundin et al. 1947). The study by Hogan et al. (1983) employed a similar graded exercise test with small increments (15 W/stage) and 3 minute stages at sea level and at 1,600 m. The researchers observed no significant altitude effect on submaximal blood lactate until the workload exceeded 195 W. At higher workloads there was a significantly higher blood lactate at altitude, but the peak blood lactate was not significantly effected.

As a result of the decrease in maximal oxygen consumption observed in the present study there was a reduction in maximal exercise performance as indicated by a lower maximal workload, and time to exhaustion at mild altitude. Previous investigators have also observed decreases maximal workload (Hogan et al. 1983) during graded exercise testing at 1,600 m. However, when exercising at a workload equal to 90% of maximal oxygen consumption, Adams et al. (1980) did not observe a significant reduction in time to exhaustion.

CHAPTER VI

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

In summary, in the present study acute altitude exposure at 1829 m did not alter resting measures of oxygen saturation, heart rate, blood pressure, minute ventilation, oxygen consumption or blood lactate. Acute exposure to mild altitude may create mild hypoxia at rest and during submaximal and maximal exercise (Squires et al. 1982; Tucker et al. 1984). Under resting conditions this level of hypoxia is not sufficient enough to alter normal functioning of the cardiovascular (Bubb et al. 1983; Ekblom et al. 1975) and respiratory systems (Adams et al. 1980; Bubb et al. 1983; Ekblom et al. 1975; Tucker et al. 1989) or alter energy metabolism (Adams et al 1980; Hogan et al. 1983; Tucker et al. 1984).

In the present study normal heart rate, blood pressure, oxygen consumption and blood lactate responses to graded exercise were observed at 1829 m during submaximal exercise workloads (Durstine et al. 1988; Nieman 1990; Wasserman et al. 1987). Therefore, there appears to be no effect on cardiovascular and metabolic responses to submaximal exercise during acute mild altitude exposure (Adams et al. 1980; Bubb et al. 1983; Hogan et al. 1983; Squires et al. 1982).

Also, at low intensity and maximal workloads during graded exercise the respiratory response is normal, but during moderate intensity workloads minute

ventilation is significantly higher when at mild altitude. The levels of hypoxia produced at mild altitude generally appears to increase the minute ventilation response to submaximal exercise (Adams et al. 1980; Astrand 1954; Hogan et al. 1983) and may increase maximal minute ventilation (Hogan et al. 1983; Tucker et al. 1982). When the magnitude of the hypoxia created by the environment and exercise is great enough, it appears to increase respiratory stimulation.

In the present study the level of hypoxia caused by acute mild altitude exposure resulted in a 9.2% decrease in maximal oxygen consumption, 8.1% decrease in maximal workload, and a 7.8% decrease in time to exhaustion measurements during graded exercise testing. Therefore, decreased performance of high intensity exercise is expected of individuals upon arrival to mild altitude environments (Adams et al. 1980; Hogan et al. 1983).

The purpose of the present study was to examine the effects of acute mild altitude exposure on the cardiovascular, respiratory and metabolic responses to graded exercise. Therefore, the physiological findings from this study are limited to application towards graded exercise testing. The three minutes stages of the test protocol are similar to the Bruce treadmill test (Wasserman et al. 1987), but may not be of sufficient length to yield steady state physiological data in all subjects during mild altitude exposure. Therefore, the submaximal data should be interpreted in the context of a graded exercise test, and application to longer duration steady state submaximal exercise should be made with caution.

Care should also be taken when attempting to predict lactate threshold from the present data. The continuous test protocol, short three minute stages and large workload

increments at each stage may not yield accurate predictions of lactate threshold for the subjects.

It should also be noted that the results of this study are limited to the two altitudes under investigation. As altitude increases and the partial pressures decrease there appears to be a continuous impact on the cardiovascular, respiratory and metabolic processes of the body at rest and during exercise. Therefore, it is possible that a similar experiment performed at sea level and 2,000 m may yield slightly different results.

The subjects in the present study were young, apparently healthy, highly trained cyclists. Previous investigators (Saltin 1996; Terrados et al. 1985) have suggested that training status impacts the physiologic effects of altitude on exercise performance. Therefore, untrained persons or individuals with cardiovascular, pulmonary or metabolic disease may demonstrate different results when performing exercise at mild altitude.

Future investigations into the effects of acute mild altitude exposure on exercise performance should include various participant populations and altitudes. Exercise test protocols should also include long duration steady state exercise, workloads and stage durations appropriate for lactate threshold prediction. To increase the level of knowledge regarding the cardiovascular, respiratory and metabolic response to exercise at mild altitude, additional physiological variables be included (ie. arterial blood-gases and pH, stroke volume, cardiac output, respiratory rate, tidal volume, muscle metabolites and pH, and blood hormones and catecholamines).

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APPENDIX A

INSTITUTIONAL REVIEW BOARD FOR HUMAN SUBJECTS APPROVAL

OKLAHOMA STATE UNIVERSITY
INSTITUTIONAL REVIEW BOARD
HUMAN SUBJECTS REVIEW

Date: 03-06-98 **IRB #: ED-98-080**

Proposal Title: THE EFFECTS OF SIMULATED ACUTE MILD ALTITUDE EXPOSURE ON THE CARDIOVASCULAR, METABOLIC AND RESPIRATORY RESPONSES TO GRADED EXERCISE

Principal Investigator(s): Frank A. Kulling, Curtis L. Hart

Reviewed and Processed as: Modification

Approval Status Recommended by Reviewer(s): Approved

ALL APPROVALS MAY BE SUBJECT TO REVIEW BY FULL INSTITUTIONAL REVIEW BOARD AT NEXT MEETING, AS WELL AS ARE SUBJECT TO MONITORING AT ANY TIME DURING THE APPROVAL PERIOD.
APPROVAL STATUS PERIOD VALID FOR DATA COLLECTION FOR A ONE CALENDAR YEAR PERIOD AFTER WHICH A CONTINUATION OR RENEWAL REQUEST IS REQUIRED TO BE SUBMITTED FOR BOARD APPROVAL.
ANY MODIFICATIONS TO APPROVED PROJECT MUST ALSO BE SUBMITTED FOR APPROVAL.

Comments, Modifications/Conditions for Approval or Disapproval are as follows:

Signature

Chair of Institutional Review Board

Date: May 19, 1998

cc: Curtis L. Hart

OKLAHOMA STATE UNIVERSITY
INSTITUTIONAL REVIEW BOARD
HUMAN SUBJECTS REVIEW

Date: March 06, 1998 **IRB #: ED-98-080**

Proposal Title: THE EFFECTS OF SIMULATED ACUTE MILD ALTITUDE EXPOSURE ON THE CARDIOVASCULAR, METABOLIC AND RESPIRATORY RESPONSES TO GRADED EXERCISE

Principal Investigator(s): Frank A. Kulling, Curtis L. Hart

Reviewed and Processed as: Full Board

Approval Status Recommended by Reviewer(s): Approved

ALL APPROVALS MAY BE SUBJECT TO REVIEW BY FULL INSTITUTIONAL REVIEW BOARD AT NEXT MEETING, AS WELL AS ARE SUBJECT TO MONITORING AT ANY TIME DURING THE APPROVAL PERIOD.
APPROVAL STATUS PERIOD VALID FOR DATA COLLECTION FOR A ONE CALENDAR YEAR PERIOD AFTER WHICH A CONTINUATION OR RENEWAL REQUEST IS REQUIRED TO BE SUBMITTED FOR BOARD APPROVAL.
ANY MODIFICATIONS TO APPROVED PROJECT MUST ALSO BE SUBMITTED FOR APPROVAL.

Comments, Modifications/Conditions for Approval or Disapproval are as follows:

Signature

Chair of Institutional Review Board

Date: March 16, 1998

cc: Curtis L. Hart

APPENDIX B

MEDICAL HISTORY QUESTIONNAIRE

Medical History Questionnaire

Name: _____ Age: _____ yrs. Date: _____

Major Coronary Risk Factors

Please check if you have any of the following risk factors:

___ 1. Diagnosed high blood pressure on at least two separate occasions (SBP≥ 140 or DBP≥90), or currently taking medication for high blood pressure

___ 2. Blood cholesterol values ≥ 200 mg/dl or HDL < 35 mg/dl

___ 3. Currently smoke cigarettes

___ 4. Diabetes Mellitus

___ 5. Family history (parents or siblings prior to age 55) of coronary heart disease of other atherosclerotic disease

___ 6. Sedentary lifestyle without regular physical activity

Symptoms of Cardiopulmonary or Metabolic Disease

Please check if you've recently experienced any of the following symptoms:

___ 1. Pain or discomfort in the chest or surrounding areas

___ 2. Unaccustomed shortness of breath or shortness of breath with mild exertion

___ 3. Dizziness of syncope (fainting)

___ 4. Orthopnea (difficulty breathing except when in upright position) paroxysmal nocturnal dyspnea (recurring labored breathing at night)

___ 5. Ankle edema (swelling)

___ 6. Palpitations or tachycardia (rapid heart rate)

___ 7. Claudication (pain in extremities upon exertion)

___ 8. Known heart murmur

APPENDIX C

MEDICAL CLEARANCE FORM

REVIEW BY MEDICAL STAFF

PHYSICAL EXAMINATION

BLOOD PRESSURE: _____

TEMPERATURE: _____

JUMP TEST: _____

_____ TM's

_____ INFECTION

_____ FLUID

_____ MOBILITY

_____ THROAT

_____ HEART

_____ RHYTHM

_____ MURMUR

_____ LUNGS

_____ CLEAR

_____ PATELLAR REFLEX

RECOMMENDATIONS:

CLEARED FOR CHAMBER FLIGHT: YES NO

_____ _____

SIGNATURE DATE

APPENDIX D

INFORMED CONSENT FORM

INFORMED CONSENT FORM

School of Applied Health and Educational Psychology

Oklahoma State University

Principle Investigators: Frank Kulling, Ed.D., Curtis L. Hart, M.S.

Project Title

The effects of simulated acute mild altitude exposure on the cardiovascular, metabolic and ventilatory responses to graded exercise.

Purpose

The purpose of this experiment is to evaluate the human body's response to exercise intensity when exposed to simulated mild altitude.

General Procedures

Prior to this experiment you will evaluated for medical clearance by a physician that is a FAA Certified Flight Examiner. If you qualify, you will perform two exercise tests on a bicycle ergometer in the Oklahoma State University - College of Osteopathic Medicine altitude chamber located at Tulsa Airpark. The exercise tests will be separated by approximately one week. One bicycle test will be performed at a simulated altitude (1000 ft) slightly higher than Tulsa, and the other will be at a simulated altitude (6000 ft) similar to Colorado Springs, CO. Each experimental trial/exercise test will last approximately one hour. The rate of assent and descent will be 3000 ft/min. The order of

the test conditions will be randomly assigned, and only the research team will have knowledge of the altitude during each session. During the bicycle test you will be pedal at a set cadence equal to 70 revolutions /minute. In the beginning the tension you pedal against will be light and will increase every three minutes until exhaustion. While in the chamber you will be attached to a heart rate monitor, blood pressure cuff, and finger tip pulse oximeter to monitor your cardiovascular conditions. You will also wear a face mask attached to a metabolic analyzer to monitor your respiratory and metabolic response. Every three minutes during the exercise test and five and ten minutes after maximal effort, a member of the research team will obtain a blood sample from a finger stick for determination of blood lactate concentration.

The exercise test and simulated altitude may be stopped at any time by the investigators based on signs of fatigue, equipment failure or abnormal physiological responses. You also have the right to stop the test whenever you wish due to feelings of fatigue or any other discomfort.

Risks and Discomforts

There exists the possibility of abnormal changes during the experiment due to exercise. They include abnormal blood pressure and/or heart rate, fainting, and in rare instances, heart attack, stroke, or death. Every effort will be made to minimize these risks by evaluation of preliminary information regarding your health and fitness and by observations during testing. The possibility also exists that you may experience slight headaches, nausea, sinus or ear blocks, and mild disorientation during the tests due to the simulated altitude. These symptoms should rapidly dissipate. Emergency equipment and

trained personnel will be available to deal with unusual situations that may arise.

Potential Benefits

The results of this study will expand the body of knowledge related to physiological responses of exercise at mild altitude in apparently healthy trained participants. If you desire a copy of the results of this study will be provided upon request.

Confidentiality

The individual information obtained from the procedures listed will be treated as privileged and confidential. It will not be release or revealed without your written consent. The information will be used for statistical analysis for scientific purposes with your right to privacy maintained.

Freedom of Consent

Your participation in this investigation is voluntary. You are free to deny consent if you desire. You are free to withdraw your consent and discontinue the investigation at any time. If you have any questions about the procedures you may contact Frank Kulling or Curtis Hart at 744-4837. You may also contact Ms. Gay Clarkson, Institutional Review Board, 305 Whitehurst Hall, Oklahoma State University, Stillwater, OK 74078, telephone number (405) 744-5700.

I acknowledge that I have read and fully understand the consent form. I sign it freely and voluntarily and consent to participation in this investigation. A copy has been

given to me.

_____ _____ _____

Signature Date Time

_____

Witness

I certify that I have personally explained all elements of this form to the subject before requesting the subject to sign it.

_____

Principal Investigator

APPENDIX E

ANOVA TABLES OF PHYSIOLOGICAL RESPONSES TO GRADED EXERCISE AT 305 m AND 1829 m ELEVATION

TABLE XI

ANALYSIS OF VARIANCE: OXYGEN SATURATION RESPONSE

Source	df	Sums of Squares	Mean Square	F
Subject	8	918.17	114.77	
Altitude, factor A	1	764.76	764.76	22.22*
Subject x Altitude, Error (a)	8	275.29	34.41	
Workload, factor B	4	136.24	34.06	2.37
Altitude x Workload, AB	4	93.64	23.41	1.63
Error (b)	61	877.63	14.39	
Total	94	3065.72		

* significant difference ($p < 0.05$).

TABLE XII

ANALYSIS OF VARIANCE: HEART RATE RESPONSE

Source	df	Sums of Squares	Mean Square	F
Subject	9	12134.45	1348.27	
Altitude, factor A	1	240.29	240.29	2.45
Subject x Altitude, Error (a)	9	884.23	98.25	
Workload, factor B	4	103176.63	25794.16	444.01*
Altitude x Workload, AB	4	335.47	83.87	1.44
Error (b)	67	3892.25	58.09	
Total	94	120663.32		

* significant difference ($p < 0.05$).

TABLE XIII

ANALYSIS OF VARIANCE: SYSTOLIC BLOOD PRESSURE RESPONSE

Source	df	Sums of Squares	Mean Square	F
Subject	9	11465.200	1273.911	
Altitude, factor A	1	208.663	208.663	2.07
Subject x Altitude, Error (a)	9	909.037	101.004	
Workload, factor B	4	44440.874	11110.218	277.47*
Altitude x Workload, AB	4	213.807	53.452	1.33
Error (b)	70	2802.919	40.142	
Total	97	60040.500		

* significant difference ($p < 0.05$).

TABLE XIV

ANALYSIS OF VARIANCE: DIASTOLIC BLOOD PRESSURE RESPONSE

Source	df	Sums of Squares	Mean Square	F
Subject	9	2938.059	326.451	
Altitude, factor A	1	8.582	8.582	0.15
Subject x Altitude, Error (a)	9	500.018	55.558	
Workload, factor B	4	117.406	29.352	1.95
Altitude x Workload, AB	4	58.162	14.541	0.96
Error (b)	70	1056.231	15.089	
Total	97	4678.459		

TABLE XV

ANALYSIS OF VARIANCE: MINUTE VENTILATION RESPONSE

Source	df	Sums of Squares	Mean Square	F
Subject	9	8964.98	996.11	
Altitude, factor A	1	871.23	871.23	4.81
Subject x Altitude, Error (a)	9	1628.79	180.98	
Workload, factor B	4	147457.05	36864.26	291.08*
Altitude x Workload, AB	4	1466.79	366.70	2.90*
Error (b)	65	8232.07	126.65	
Total	92	160388.84		

* significant difference ($p < 0.05$).

TABLE XVI

ANALYSIS OF VARIANCE: OXYGEN CONSUMPTION RESPONSE

Source	df	Sums of Squares	Mean Square	F
Subject	9	6.65076	0.73897	
Altitude, factor A	1	0.00245	0.00245	0.04
Subject x Altitude, Error (a)	9	0.52826	0.05870	
Workload, factor B	4	122.56023	30.64006	1084.08*
Altitude x Workload, AB	4	0.01839	0.00460	0.16
Error (b)	65	1.83714	0.02826	
Total	94	131.59723		

* significant difference ($p < 0.05$).

TABLE XVII

ANALYSIS OF VARIANCE: BLOOD LACTATE RESPONSE

Source	df	Sums of Squares	Mean Square	F
Subject	9	27.55222	3.03136	
Altitude, factor A	1	2.91960	2.91960	1.44
Subject x Altitude, Error (a)	9	18.24468	2.02719	
Workload, factor B	4	162.89678	40.72419	35.19*
Altitude x Workload, AB	4	1.14673	0.28267	0.25
Error (b)	68	78.68499	1.15713	
Total	95	291.44500		

* significant difference ($p < 0.05$).

VITA

CURTIS L. HART

Candidate for the Degree of

Doctor of Education

Thesis: THE EFFECTS OF ACUTE SIMULATED MILD ALTITUDE EXPOSURE ON THE CARDIOVASCULAR, RESPIRATORY, AND METABOLIC RESPONSES TO GRADED EXERCISE

Major Field: Applied Educational Studies - Health, Physical Education and Leisure

Biographical:

Education: Graduated from Abraham Lincoln High School, Des Moines, Iowa, in May 1977; studied Biology at Marshalltown Junior College, Marshalltown, Iowa in 1977-78; received Bachelor of Arts degree in Physical Education and Recreation from Central University of Iowa, Pella, Iowa, in May 1981; received Master of Science degree in Physical Education and Leisure Studies from Iowa State University, Ames, Iowa, in August 1989; studied Chemistry at Tarrant County Community College, Ft. Worth, Texas in 1990-92; completed requirements for Doctor of Education degree at Oklahoma State University, Stillwater, Oklahoma, in December, 1998.

Experience: Graduate Assistant, Department of Physical Education and Leisure Studies, Iowa State University, Ames, Iowa, 1982 to 1983; Laboratory Technician, Department of Physiology and Pharmacology, University of Osteopathic Medicine and Health Sciences, Des Moines, Iowa, 1983 to 1984; Physical Fitness Instructor, Supreme Court Racquetball and Health Clubs, Austin, Texas, 1984 to 1985; Specialist, Department of Exercise, Sport and Health Studies, University of Texas at Arlington, Arlington, Texas, 1985 to 1995; Research Assistant, Department of Physiology and Pharmacology, Oklahoma State University - College Osteopathic Medicine, Tulsa, Oklahoma, 1995; Graduate Assistant, School of Health, Physical Education and Leisure, Oklahoma State University, Stillwater, Oklahoma, 1995 to 1998.