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ENERGY SOURCES DURING MUSCULAR WORK UNDER NORMOXIC AND HYPOXIC CONDITIONS

DISSERTATION

Presented in Partial Fulfillment of the Requirements for
the Degree Doctor of Philosophy in the Graduate
School of The Ohio State University

By

Robert Bason, B.S., M.S.

* * * * *

The Ohio State University
1970

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To my wife, Paula Ann
and to my daughter, Cheryl Lynn

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Mathews, D. K., E. L. Fox, C. E. Billings and R. Bason: Changes in Work Tolerance During Prolonged Exposure at 3800 Meters. Presented at Aerospace Medical Association annual meeting, Washington, D. C., April, 1967.

Turner, H. S., G. W. Hoffler, C. E. Billings and R. Bason: An Attempt to Produce Acclimatization to Hypoxia by Intermittent Altitude Exposure with Vigorous Exercise. Presented at Aerospace Medical Association annual meeting, Miami, Florida, May, 1968.

Chisholm, D. M., C. E. Billings, and R. Bason: Studies on the Behavior of Naive Subjects During Decompression: An Evaluation of Passenger Oxygen Equipment. Presented at Aerospace Medical Association annual meeting, San Francisco, California, May, 1969.

Billings, C. E., R. E. Brashear, R. Bason and D. K. Mathews: Medical Observations During 20 Days at 3800 Meters. Arch. Environ. Health 18:987-995, June, 1969.

Bason, R. and C. E. Billings: Effect of High Altitude on Lipid Components of Human Serum. Arch. Environ. Health 19:183-185, August, 1969.

Turner, H. S., G. W. Hoffler, C. E. Billings and R. Bason: An Attempt to Produce Acclimatization to Hypoxia by Intermittent Altitude Exposure with Vigorous Exercise. Aerospace Med. 40:971-976, September, 1969.

Billings, C. E., R. Bason, and R. J. Gerke: The Physiological Cost of Piloting Rotary Wing Aircraft. Aerospace Med. 41:256-258, 1970.

RESEARCH REPORTS

Billings, C. E., D. K. Mathews, E. L. Fox and R. Bason: Effects of Physical Conditioning and Partial Acclimatization to Hypoxia on Work Tolerance at High Altitudes. The Ohio State University Research Foundation Report No. RF 2002-1, July, 1966.

Fox, E. L., C. E. Billings, R. Bason and D. K. Mathews: Improvement of Physical Fitness by Interval Training: II. Required Training Frequency. The Ohio State University Research Foundation Report No. RF 2002-3, April, 1967.

Billings, C. E., R. L. Wick, Jr., G. W. Hoffler, H. S. Turner, and R. Bason: Use of Passenger Oxygen Equipment Following Rapid Decompressions to 30,000 Feet. The Ohio State University Research Foundation Report No. 2505-2, March, 1968.

Billings, C. E., D. K. Mathews, R. L. Bartels, E. L. Fox, R. Bason and D. Tanzi: The Effects of Physical Conditioning and Partial Acclimatization to Hypoxia on Work Tolerance at High Altitudes. The Ohio State University Research Foundation Report No. RF 2002-4 (final), June, 1968.

FIELDS OF STUDY

Major Field: Work Physiology

Studies in Exercise Physiology. Professors Charles E. Billings, Jr. and Donald K. Mathews

Studies in High Altitude Physiology. Professor Charles E. Billings, Jr.

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CHAPTER I

Introduction

"The diminution of barometric pressure acts upon living beings only by lowering the oxygen tension in the air they breathe, and in the blood which supplies their tissues and by exposing them thus to the dangers of asphyxia" (6).

Ever since Paul Bert made this famous statement in 1878, the adjustment of man to low ambient oxygen pressure has been an object of intensive study. One area of particular interest has been the energy cost of a given amount of muscular activity at altitude.

Numerous studies have been concerned with the amount of oxygen consumed during muscular activity at various altitudes ranging from sea level to 24,000 feet. The results of these studies unanimously indicate that one's maximal aerobic work capacity is reduced at altitude (2, 4, 12, 14, 21, 22, 46, 55, 56, 59). The average decrement is approximately 2-3% per thousand feet of altitude (12).

With regard to the oxygen consumption for submaximal work, however, this unanimity is not present. It is generally agreed among most investigators that the oxygen consumed for a given amount of submaximal work will not vary as a function of altitude (4, 14, 15, 24, 28, 34, 59). On the other hand, there is some evidence to suggest that the oxygen consumption for submaximal work is not independent of altitude. Billings et al. (7, 8), for instance, have shown slight but significant increases in oxygen uptake at two submaximal workloads. More recently, Billings

and Bason (9) found that during work of higher intensity, the oxygen uptake at altitude was slightly lower than at sea level. Similar findings were reported by Cronin and MacIntosh (16) and Naimark et al. (54).

The amount of oxygen consumed during a given quantity of work has long been used as a measure of the energy cost of that work. However, this oxygen uptake reflects only the energy derived through the aerobic metabolic pathway. It has been well established (46, 49, 50, 51, 63) that there are, in addition to the aerobic pathway, two other pathways from which the body can derive its energy, namely, the lactic acid and alactic acid energy sources collectively referred to as the anaerobic metabolic pathway. It is necessary, therefore, that when the energy cost of a given amount of work is to be determined all three energy sources should be considered.

The apparent disagreement among investigators concerning the aerobic energy cost of work at altitude could perhaps be due to the role each of these energy sources plays in contributing to the total energy cost of work. For instance, a decrease in the energy derived from the aerobic metabolic pathway at altitude might be associated with an increase in the energy derived from the anaerobic pathway. The total energy cost of work, however, might remain the same or even be higher.

It has been shown in a number of studies that there is a greater lactic acidemia at altitude than during sea level for the same amount of muscular activity (28, 30, 33, 39, 47, 48, 53, 54, 55, 59, 60). This would tend to suggest that perhaps one derives more energy from the lactic acid energy sources at altitude than at sea level for identical

workloads. There is little information, however, regarding the energy derived from the alactacid energy source for the same set of conditions.

Statement of the Problem

The purpose of this study, therefore, was to partition man's energy sources into three component parts - aerobic, lactacid and alactacid - and to assess whether the power derived from each of these components is utilized differently at altitude than at sea level.

The following null hypothesis were tested:

- A. The total power input for a given power output is independent of altitude.
- B. The proportional contribution of the aerobic, lactacid and alactacid components to the total power input is independent of altitude.

Related Literature

The effect of hypoxic hypoxia on lactic acid formation both at rest and following muscular activity has been a subject of considerable interest for many years.

Edwards (26), on a high altitude expedition to Chile in 1933, was probably the first to observe initial rise in the resting lactic acid content of the blood on going to altitude (17,520 feet).

Some years later increased amounts of lactic acid in the blood were found by Friedman et al. (31). They conducted a number of experiments on one subject at simulated altitudes up to 24,000 feet. The threshold altitude for the increased lactic acid formation was found at an altitude of 15,000 feet. That this rise was due to hypoxia was further evidenced by the fact that the lactic acid increase was prevented by the

administration of oxygen or by a return to a simulated altitude of 10,000 feet.

Harboe (35) in 1957 probably conducted the first systematic investigation to determine the degree of hypoxia required to produce increased concentrations of lactic acid in the blood at rest. Twenty-two experiments were carried out on 8 healthy subjects, 20 to 28 years of age. The subjects were exposed to hypoxia by breathing atmospheric air at 10,000, 15,000, 20,000 and 26,000 feet, respectively in simulated flights in an altitude chamber, for periods up to 30 minutes. During the ascent, each subject breathed air plus oxygen from an oxygen mask and regulator.

The conclusions drawn from these experiments were that the concentrations of lactic acid in the blood increased during hypoxia, the increase depending on the degree and duration of the hypoxia. The threshold altitude for increased lactic acid was found to be 15,000 feet; a latent period of approximately 15 minutes was required before the gradual increase in the blood lactic acid concentration appeared. In one experiment at 26,000 feet, the subject became unconscious within 4.5 minutes. Blood samples taken during this hypoxic period showed no change in lactic acid concentration as compared to a sample taken prior to hypoxia. Later evidence by Naimark et al. (54) also supports a threshold altitude of 15,000 feet.

The threshold altitude for increased resting blood lactic acid concentrations has not gone unchallenged. Dejours (18) has observed increased concentrations at 12,500 feet while Favour (29) and Hansen et al. (33) have shown increased lactate concentrations at 14,000 feet. In a much earlier study, Terzioglu et al. (61) showed that resting

lactates increased from a mean of 15.2 mg% at sea level to 23.6 mg% on the third day at 6,000 feet and 20.4 mg% on the ninth and tenth day at altitude. However, Bay and coworkers (5) observed no difference in resting blood lactate between sea level and 10,000 or 15,000 feet.

The presence of lactic acid in the blood following muscular activity at altitude has also received much attention. Although most investigators find that lactacidemia accompanies work at altitude, there is a disagreement on the amount of change.

Dill et al. (20) conducted experiments on subjects remaining at an altitude of 10,000 feet for 6 to 8 weeks. The subjects worked on a bicycle ergometer and the oxygen consumption and the blood lactic acid level were determined at the end of work. At that altitude the subjects had diminished work capacity and they showed a higher rise of blood lactic acid than at sea level for a given intensity of work. In one of the subjects, however, a continuous change occurred during the 8-week sojourn. The extra rise of lactic acid following a given amount of work became smaller and eventually disappeared so that at the end of the stay the subject showed the same blood lactic acid rise for a given intensity of work as at sea level.

In 1933, during an international high altitude expedition to Chile, Edwards (26) investigated the effect of acclimatization on blood lactic acid levels. Observations were made on subjects at an altitude of 9,180 feet for 16 days and later at 12,008 feet for 8 days, 15,420 feet for 12 days, and 17,520 feet for 3 weeks. Work was performed on a bicycle ergometer for ten minutes. At the end of work, blood samples were taken for lactic acid determinations. At the beginning of the stay at 9,180

feet, 3 of 7 subjects had increased lactic acid concentrations for a given amount of work as compared with sea level values. However, after this initial rise at 9,180 feet and subsequently at all the higher altitudes, all the subjects showed the same lactic acid rise as at sea level for a given amount of work. Similar findings were reported by Schilling et al. (58).

The failure to show higher levels of lactic acid at the higher altitudes suggested that once acclimatization has begun at lower altitudes, acclimatization to higher altitudes takes place more readily. The diminished accumulation of lactic acid reported by Edwards (26) was assumed to be a protective mechanism preventing an already low arterial oxygen saturation from becoming markedly lower by a shift in the oxy-hemoglobin curve due to the Bohr Effect. Dill (19) suggested that this reduced ability to accumulate large amounts of lactic acid might be due to a reduced alkaline reserve.

In 1948, Asmussen et al. (2) studied the effect of acute hypoxia on the capacity for work and on the ability to accumulate lactic acid. In this experiment there was no disturbance in the acid-base balance of the blood previous to work in contradistinction to experiments made on subjects acclimatized to altitude. In later experiments the alkaline reserve was lowered prior to work by ingesting of large amounts of NH_4Cl . The alkaline reserve was decreased to values found in subjects who had lived at altitudes of 12,139 feet and 19,685 feet. The results of the experiment showed that an acute lowering of the oxygen tension of the inspired air reduced the capacity for maximal work; however, the ability to contract an oxygen debt and to accumulate lactic acid was practically

unchanged even when the alkaline reserve was lowered.

On the basis of their experimental evidence, they suggested that the diminished ability for the formation of lactic acid at high altitude was not the effect of a useful acclimatization, but rather was due to a general weakening of the subjects living under the stresses of high altitude. This weakening affect was probably brought about by an impairment of the central nervous system which is especially sensitive to low oxygen tension. Such a weakening lowered the ability to perform anaerobic work and therefore, to produce lactic acid. Local changes in the muscle could also have caused a decreased ability to perform work if, for instance, the sojourn at altitude caused a decrease in the glycogen content in the muscle or an inhibition of the transformation of muscle glycogen to lactic acid.

In 1947, Friedman and Ivy (30) performed an experiment to determine the ceiling at which lactic acid concentrations increased above those at ground level during acute altitude exposure. Six male subjects performed exhaustive work at a rate of 30.8 kg.m/sec on a bicycle ergometer at a simulated altitude of 15,000 feet. In addition, this work rate was also performed at simulated altitudes of 5,000, 7,000, 10,000, 12,000 and 15,000 feet for five minutes to determine the ceiling at which lactic acid concentrations increased above those at sea level. The results of the experiment showed that during rapid exhausting work rates lactic acid levels were lower at 15,000 feet than at ground level. The level of lactic acid produced was related to the duration of the work, i.e., the shorter the duration of work, the less lactic acid produced. In addition, the data also suggested that between 7,000 and 12,000 feet, man can

exercise at a relatively light rate of work without an increase in lactic acid in the blood above that attained during similar work at ground level. At 15,000 feet, marked changes in lactic acid were noted in all subjects. A rise in lactic acid above that after similar work at sea level has been noted by Lorentzen (47) at 10,000 feet.

Tepperman and Tepperman (60) also showed that the lactic acid concentration in the blood after exercise (750 kg.m/min. - 1200 kg.m/min.) was higher at low barometric pressure (10,000 feet and 15,000 feet) than at sea level. Upward displacement of the lactate was found at a simulated altitude as low as 5,000 feet in three well-trained subjects. Two untrained subjects did not show the same progressive elevation of blood lactic acid with increased hypoxia. They suggested that the muscle of the untrained subject was so hypoxic at sea level that lowering the arterial oxygen saturation produced no evident additional embarrassment.

In 1963, Dejours (18) showed evidence that light grade work (25% max. $\dot{V}O_2$) at 12,467 feet had relatively small and inconsistent effects on blood lactate; however, heavier exercise (50% max. $\dot{V}O_2$) regularly raised it.

Stenberg et al. (59) showed submaximal work (600 kpm and 900 kpm) at 13,123 feet produced greater blood lactic acid concentrations than the same work performed at sea level. Oxygen consumption for the submaximal work, however, remained independent of the altitude. In the 600 kpm exercise, the lactates increased from 25.02 mg% at sea level to 37.80 mg% at 13,213 feet. In the 900 kpm exercise, the blood lactates increased from 47.52 mg% to 99.72 mg% respectively. The data suggested

the limiting factor of an individual's aerobic capacity under acute hypoxic was a decreased amount of oxygen transported from the left heart because of a reduced oxygen content in the arterial blood.

In 1967, Hansen et al. (33) reported similar findings. Exercise at a rate of 367 kg.m/min. and 428-734 kg.m/min. at 14,108 feet induced very significant acidemia and lactacidemia. Oxygen consumption on the other hand, remained independent of altitude.

Studies by Hermansen and Saltin (39) showed similar increases in blood lactic acid during submaximal exercise at acute exposure to altitudes of 7,546 feet and 13,123 feet. They suggest that the main reason for the increased lactic acid formation was the reduction in the arterial oxygen tension and most likely the tissue oxygen tension.

In general, it is agreed that acute hypoxic hypoxia leads to an increased lactate concentration and therefore, does not appear to impair the lactacid mechanism. On the other hand, subjects long exposed to altitude have a reduced lactic acid production due to reduced alkaline reserve. Cerretelli (13) offered an interesting suggestion concerning this reduced lactic acid. He hypothesized that this reduction could be brought about by some blockage of the enzymatic reaction in the glycolytic chain. This could be triggered by the blood pH which then would be the limiting factor of the lactic acid production. Thus he suggests a feed-back mechanism between lactic acid production and blood pH avoiding an intolerable acidosis.

In all of the studies mentioned so far, no one has related the caloric value of the increased lactate to the total energy cost of muscular exercise. As a matter of fact, very little, if anything, has

been done in comparing the total energy cost of a given amount of work at sea level and at altitude.

Naimark (54), in 1965, reported an investigation designed to determine if hypoxia during exercise does in fact result in an increased anaerobic metabolism and related the findings to the total energy cost of muscular exercise. In this study, 8 male subjects exercised at the same workload (about 1.5 l/min. O_2 consumption) for 5 to 7 minutes, breathing air and a hypoxic gas mixture (11-12% O_2 in N_2). The results clearly showed that the concentration of blood lactate in any one subject was higher during hypoxia than during normoxic exercise. Lactates increased from 8.10 mg% to 26.28 mg%. In all subjects, oxygen consumption during hypoxic exercise was less than during normoxic exercise at the same workload. Oxygen consumption decreased from 1.512 ml/min. under normoxia to 1.356 ml/min. under hypoxia; the average difference being 165 ml/min. From the results of his study, Naimark suggested that the total energy expenditure is the same or greater during hypoxic than normoxic exercise.

In 1970, Moncloa et al. (53) measured blood lactates following two levels of work at sea level and at 14,000 feet. Lactates were measured following 6 minutes of work at 2.5 mph up a 5% grade and also after 4 minutes of work at 2.5 mph up a 11% grade. After the first 6 minutes of exercise at 5% grade, one minute was allowed for blood drawing. Exercise was started immediately at the same speed, but at a 11% grade. The results of the experiment showed that at high altitude, acidosis was more severe. Lactate increased from 35.68 mg% to 49.07 mg% at the end of 10 minutes of exercise at sea level and from 40.07 mg% to 62.46 mg%

at the end of 10 minutes of exercise at 14,000 feet. They suggested that since the magnitude of the exercise was the same, the more marked acidosis indicated that proportionally more energy comes from the glycolytic mechanism as one goes to altitude.

While it is still generally accepted that hypoxia leads to an increased anaerobic metabolism and therefore, to an increased lactate formation, other evidence has been presented that would perhaps make it quite inadvisable to draw any conclusions about tissue oxygen supply from the determinations of lactate alone. Huckabee (41), for example, has reported evidence that showed factors such as the infusion of bicarbonates, of pyruvate or glucose and hyperventilation to influence lactate concentration in the absence of oxygen lack. Rises in blood lactate due to glucose injection have been reported by other investigators (10, 40). The results of these experiments would suggest that lactate accumulation ensues not because of tissue hypoxia, but perhaps as a result of its tendency to maintain an equilibrium with the pyruvate present.

CHAPTER II

METHODS AND PROCEDURES

Selection of Subjects

Nine adult males varying in degrees of physical fitness were selected from The Ohio State University for use in this study. Each subject was selected on the basis of his availability during the morning hours rather than on the basis of physical fitness. None of the subjects had actively engaged in competitive sports on a regular basis for at least three years, with the exception of Subject G. This subject was a member of The Ohio State University Track Team. Part-way through the study, Subject D withdrew, reducing the number of subjects to eight. No effort was made to replace him and his data were not considered in the final analysis.

Each subject was given a thorough medical examination, including chest x-ray, electrocardiogram, hematocrit and urinalysis. Particular emphasis was given to noting any cardiovascular disorders. In addition, certain pulmonary function tests were also performed. These included measurement of forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1). Vital data of the subjects are presented in Table 1.

Facilities and Apparatus

The hypobaric chamber and related facilities of The Ohio State

University Aviation Medicine Research Laboratory were used for this experiment. All exercise was performed in a sitting position on a Jacquet Universal bicycle ergometer. Each subject rode at sixty revolutions per minute. All physiological recordings before, during and after exercise were accomplished using a Sanborn 350 multi-channel recorder with appropriate preamplifiers.

TABLE 1

ANTHROPOMETRIC DATA AND PULMONARY FUNCTION TESTS

Subject	Age yr.	Height cm	Weight kg	BSA (m ²)	Hematocrit %	BTPS		
						FEV liters	FEV ₁ liters	FEV ₁ FEV
A	23	162.6	66.82	1.71	45.0	3.9	2.7	70%
B	27	186.7	85.91	2.12	43.5	5.7	4.4	77%
C	20	172.7	58.64	1.64	43.5	4.7	4.2	89%
E	21	185.4	94.54	2.20	45.0	6.7	5.1	76%
F	19	176.4	85.91	2.50	46.0	4.9	4.4	90%
G	22	175.3	62.50	1.82	47.0	5.3	4.5	85%
H	24	181.0	85.45	2.06	44.5	6.4	5.7	89%
I	20	185.4	86.90	2.12	42.0	6.0	5.4	90%
Mean	22	178.4	78.33	2.02	44.6	5.5	4.6	83%

Preliminary Testing

Maximal Oxygen Consumption Test. Preliminary testing was employed to determine the subject's maximal oxygen consumption (aerobic capacity). This was determined in the following manner: after a five minute rest period, each subject performed a warm-up ride of continuous riding for two minutes at a 50 watt power output, followed by three minutes at a 100 watt power output. Following the warm-up ride, each subject rested for ten minutes, then began exercising again at a power output of 150

watts. Every two minutes the power output was increased by 20 watts until the subject was exhausted. The subjects were encouraged to ride as long as possible; each subject returned on two alternate days and the same procedure was carried out with one slight alteration. The workload following the ten minute rest period was arbitrarily increased in an effort to elicit a maximal oxygen consumption. Maximal consumption was taken as the highest value obtained for a given workload.

Heart Rate Determination. Chest and back electrodes fastened by a rubber strap were used to connect each subject to a Sanborn Model 350 multi-channel recorder with preamplifier. Heart rates were obtained by recording sample fifteen second strips and counting the QRS complexes.

Collection of Expired Gas and Gas Analysis. Pulmonary ventilation, oxygen consumption and carbon dioxide output were calculated for all expired air samples. The method employed in the collection of the expired gas and gas analysis will be described. Room air concentration of oxygen and carbon dioxide was determined during each ride, as was barometric pressure. Maximal values for oxygen consumption ($\dot{V}_{O_2}$), pulmonary ventilation ($\dot{V}_E$ STPD and BTPS), carbon dioxide production ($\dot{V}_{CO_2}$), heart rate (H.R.) and respiratory frequency (f) are shown in Appendix A.

Steady State Exercise

Following the determination of maximal oxygen consumption each subject exercised once a week in a hypobaric chamber at sea level (223 meters) and at simulated altitudes of 2286 meters (7500 feet) and 3810 meters (12,500 feet). Initially, workloads were chosen to approximate 25%, 50% and 67% of each subject's aerobic capacity. Determination of

these workloads and corresponding absolute values are shown in Appendix B. In actuality, these workloads more nearly approximated 30%, 60% and 80% of the subjects' maximal aerobic capacities. (From this point, these actual values are used). Each level of work was performed once at each altitude. The work level and corresponding altitude were assigned in a random manner using a table of random numbers. This was done to avoid any conditioning and/or sequence effect. At no time was the subject informed as to the altitude or workload. All sea level runs were conducted with the chamber door closed and the vacuum pump running, in an effort to simulate as accurately as possible, conditions present at altitude. Each subject exercised until he could no longer continue, or up to a maximum of forty-five minutes.

The variables measured during exercise and the time of measurement are shown in Table 2. Deviation from these monitoring periods was necessarily modified when it became apparent that the subject could not continue to ride for the full forty-five minute period. This was especially apparent during the 80% workloads. Monitoring then was at the discretion of the investigator. Heart rate, respiratory rate, rectal temperature, chamber temperature and relative humidity were recorded during the last fifteen seconds of each period. Expired gas was collected for the full collection period.

Collection of Expired Air. During exercise the subject wore a nose clip and breathed through an open circuit system by means of a high velocity, low resistance Collins Triple-J valve. The valve was suspended in front of the subject by means of a flexible conduit. The flexible conduit was attached to a wooden platform which, in turn, was secured to

TABLE 2

VARIABLES AND TIME OF MEASUREMENT DURING SUBMAXIMAL WORK AND RECOVERY

	Collection Period (Min.)	Heart Rate	Respiratory Frequency	Rectal Temperature	Pulmonary Ventilation	Lactic Acid	Chamber Temperature	Relative Humidity	Room Air
Rest	0-5	x	x	x	x	x	x	x	x
Exercise	0-1	x	x	x	x				
	1-2	x	x	x					
	1-3				x				
	2-3	x	x	x					
	3-6				x				
	5-6	x	x	x					
	6-9				x				
	8-9	x	x	x					
	14-15						x	x	
	19-20	x	x	x	x		x	x	
	29-30	x	x	x	x		x	x	
	34-35	x	x	x					
	39-40	x	x	x	x		x	x	x
	44-45	x	x	x	x				
Recovery	0-2				x				
	1-2	x	x	x		x			
	2-6				x				
	4-5	x	x	x		x	x	x	
	5-6	x	x	x					

TABLE 2--Continued

	Collection Period (Min.)	Heart Rate	Respiratory Frequency	Rectal Temperature	Pulmonary Ventilation	Lactic Acid	Chamber Temperature	Relative Humidity	Room Air
Recovery	6-12				x				
	7-8					x			
	9-10	x	x	x					
	11-12	x	x	x		x			
	14-15	x	x	x			x	x	
	15-20				x				
	19-20	x	x	x					
	24-25	x	x	x			x	x	
	25-30				x				
	29-30	x	x	x		x			
	34-35	x	x	x			x	x	
	35-40				x				
	39-40	x	x	x					
	44-45	x	x	x			x	x	
	45-50				x				x
	54-55	x	x	x			x	x	
	55-60				x				
	59-60	x	x	x		x			

the bicycle ergometer. The Collins Triple-J valve was also connected by means of transparent non-kinking plastic tubing (I.D. 1-1/2") to a low resistance parallel plate flow meter which in turn was connected to a Collins Low Turbulence Three-Way "Y" valve, connected to Douglas bags. The Douglas bags were hung in series on a wooden rack and inter-connected with smooth pieces of rubber tubing (I.D. 1-1/4") and Collins T-valves. Prior to each ride, the Douglas bags were evacuated to remove any residual gas.

Analysis of Expired Air. Expired gas was analyzed by first securing an aliquot from the sample arm of the Douglas bag into a 2,028 ml rubber sampling bag. The concentration of oxygen and carbon dioxide in the sample aliquot were then determined by means of the Beckman E-2 and LB-1 electronic analyzers. The two analyzers were calibrated with known concentrations of gas - 100% N₂, 20.86% O₂, 5.80% CO₂ - analyzed by means of the Micro-Scholander method. The remaining gas in the Douglas bag was measured in a 120 liter Collins Chain Compensated Spirometer. Pulmonary ventilation volume, oxygen uptake, carbon dioxide output and respiratory exchange ratio were calculated from all expired air samples.

Room air samples were taken before, during and after exercise as indicated in Table 2. A mean value for oxygen and carbon dioxide was obtained and used in the metabolic calculation. Sea level barometric pressure was obtained just prior to gas analysis.

Heart Rate Determination. Heart rate was obtained using a Model 350-1600 ECG preamplifier and modified NASA electrodes as described by Day and Lipett (17). The electrode was modified with microdot wire (#250-3804). This wire has a low microphonic characteristic (less noise

from movement). The ECG leads were attached at the manubrium, xiphoid process, at approximately the V₅ position in the anterior axillary line and posteriorly at the cervical prominence, after the areas of attachment were thoroughly scrubbed with acetone. The electrodes were held in place by electrode adhesive discs. Heart rate was obtained by recording a fifteen second strip and counting the deflections of the QRS complex and multiplying by four. In addition, a cardiometer provided for continuous monitoring of heart rate.

Respiratory Frequency. Respiratory frequency was sensed from a low resistance parallel plate flow meter placed in the expiratory line. The flow meter was operated in the following manner: Air was passed through a large number of ducts to avoid turbulence. The outermost ducts were used to measure a pressure differential which is created according to Bernoulli's principle of velocity and pressure - the greater the velocity, the lower the pressure. The pressure was then transmitted through tubes to a differential pressure transducer which converted the pressure into an electronic signal. In this experiment, a differential pressure transducer was connected to a preamplifier, Model 350-1100AS in the Sanborn recorder. The pressure transducer used four impedance elements connected in a Wheatstone bridge circuit. When the bridge is loaded, one or more of the bridge arms changes its resistance and unbalances the bridge. The carrier preamplifier measured the load by measuring the bridge unbalanced voltage.

Rectal Temperature. Rectal temperature was continuously monitored using a Yellow Springs #405 thermometer inserted approximately 10-15 cm into the rectum. The probe was connected at the other end to a Tele-

thermometer. In addition, permanent recordings of rectal temperature were obtained by the use of a low level preamplifier, Model 350-1500, driven by the output from the Tele-thermometer. The preamplifier was balanced before each run so that full scale deflection covered a range of 36°C to 41°C with each millimeter deflected representing 0.1°C .

Recovery From Exercise

Recovery from exercise was evaluated for one hour for all workloads. The physiological variables measured during recovery and the times of measurement are shown in Table 2. The techniques employed were the same as previously described under steady state exercise. Deviation from the monitoring procedure occurred only when the subject complained of nausea or vomited.

Lactates. Blood lactates were obtained by inserting a 21 gauge scalp vein needle into a suitable dorsal hand vein or forearm vein. A heparinized saline solution was used to keep the scalp vein needle patent. Approximately one cubic centimeter of blood was drawn into a plastic syringe and then discarded. This blood served to wash out the heparinized saline in the scalp vein needle. A second blood sample was then drawn into a sterile plastic three cubic centimeter syringe. Approximately one cubic centimeter of this blood was expelled into a cool weighed tube containing 10% trichloroacetic acid. The tube was immediately agitated to insure complete mixing. All tubes were kept refrigerated until the time of analysis. Lactate determinations were done by Labs, Incorporated in Columbus, Ohio, using the Barker and Summerson method as modified by Huckabee (38).

Procedure

Each subject reported to the laboratory in a post absorptive state, dressed in shorts and tennis shoes. Prior to entering the altitude chamber, body weight was recorded, electrocardiograph leads were attached and the rectal probe inserted. The subject then entered the altitude chamber and adjusted the bicycle seat and mouthpiece to the most comfortable position. The electrocardiograph leads and rectal probe were connected to the monitoring devices and all systems were checked for proper functioning.

When all physiological monitoring devices were operating satisfactorily, the chamber door was sealed and the chamber decompressed to the specified altitude in approximately ten minutes. On the ascent to altitude, the subject was briefed on the procedures to be followed. He was instructed to hold up one finger if at any time during the ride he felt that only one more minute of exercise could be tolerated. In this way, a timely collection of expired gas could be made. The subject also understood, however, that he was free to stop exercising without any prior warning, if necessary. Also, during the ascent a resting blood lactate was obtained by means of a venipuncture of the anti-cubital vein.

Upon arrival at altitude the subject was instructed to put on a nose clip and breathe through the mouthpiece for several seconds to be certain that the system was air tight and that the dead space in the system was properly flushed with the subject's own expired gas. A five minute resting expiratory gas sample was then obtained.

A five second countdown was used to start the exercise and on the

command, "Begin", timing devices were activated and the subject commenced pedaling at sixty revolutions per minute. The subject was frequently encouraged to ride as long as possible, especially at the higher altitudes and workloads.

At the completion of the 9th, 20th and 30th minutes of exercise, the subject was instructed to continue riding, but was allowed to remove the noseclip and mouthpiece. The relief periods between gas collections allowed the subject to be more comfortable and relaxed. The subject was permitted this rest for eight minutes, then instructed to replace the noseclip and breathe through the mouthpiece.

Upon completion of the exercise, the subject was told to stop, keep the mouthpiece in place and remain on the bicycle. A 21 gauge scalp vein needle was inserted into the dorsal hand vein or forearm vein, and blood samples were taken for lactate determinations. The subject remained on the bicycle for twenty minutes then moved to a comfortable chair for the remainder of the recovery period. At the end of the 20th, 30th, 40th and 50th minutes of recovery, the subject was allowed to remain off the noseclip and mouthpiece for approximately four minutes.

Following completion of the recovery period, the chamber was recompressed. Descent to ground level took approximately ten minutes. The subject was re-weighed upon arrival at ground level.

CHAPTER III

DATA REDUCTION

Computer Program

A program for calculating values related to ventilation and the metabolic cost of work was written. The format and equations used appear in Appendix C. The following values were printed out:

1. Ventilation ($\dot{V}_E$ cc/min.) - STPD, BTPS
2. Tidal volume (V_T cc) - BTPS
3. Oxygen uptake ($\dot{V}_{O_2}$ cc/min.) - STPD
4. Carbon dioxide output ($\dot{V}_{CO_2}$ cc/min.) - STPD
5. Respiratory exchange ratio - R
6. Oxygen extraction - percentage
7. Respiratory frequency - breaths/minute
8. Heart rate - beats/minute
9. Oxygen pulse - ml O_2 /heart beat
10. Lactic acid - mg%
11. Power output - watts
12. Rectal temperature - $^{\circ}C$
13. Gas collection time - minutes

In addition, all these values were punched on cards for later data analysis.

Determination of the Energy Cost of Work

Aerobic Component. The determined values of oxygen consumption per minute ($\dot{V}O_2$) were plotted at the mid-point of each collection period and the points connected with a straight line. Values of oxygen consumed between collection periods were estimated from this curve. For example, $\dot{V}O_2$ determinations were made continuously through the first nine minutes of exercise, but not again until the nineteenth minute. To estimate the oxygen consumed between the ninth and the nineteenth minutes, the oxygen consumption at the fourteenth minute (mid-point between the ninth and nineteenth minutes) was estimated from the curve and multiplied by ten. The gross oxygen consumed was calculated by a summation of these determined and estimated values. The net aerobic cost was then obtained by subtracting the resting oxygen consumption for the same period from the gross oxygen cost. The caloric equivalent of the net oxygen cost was calculated assuming one liter of oxygen to be equal to 5 kcal.

The accuracy of this method for determining the net oxygen cost was compared to integration (planimetry) of the area under the oxygen consumption curve. Several curves were chosen at random for this comparison. The results are shown in Table 3. A "t" test indicated that the two means were not significantly different from one another.

Lactacid Component. The energy in kcal derived from the lactacid mechanism was calculated according to Margaria et al. (52). They found that the caloric equivalent of lactic acid production from glycogen in man was 222 cal/g or 222 kcal/kg. Total body lactate produced during work and removed during recovery was calculated using the following equation (52):

$$LA/kg = (0.61/0.80)LA_b$$

where LA/kg = total body lactate in grams per kilogram of body weight
 0.61 = fraction of total body water (25)
 0.80 = fraction of blood water
 LA_b = blood lactate in grams per liter of blood

The lactic acid formed during exercise was taken as the highest value obtained during the first minutes of recovery.

TABLE 3

PLANIMETRY VERSUS GRAPHIC METHOD
FOR ESTIMATING NET OXYGEN COST

Planimetry	Graph
30.80 liters	31.14 liters
34.47	34.63
28.46	28.83
62.62	63.43
52.06	52.82
$\bar{X}$ 41.68	$\bar{X}$ 42.17

Alactacid Component. The energy derived from the alactacid mechanism was not as easy to calculate and involved a partitioning of the lactacid and alactacid components from the total measured respiratory oxygen debt. From the calculated alactacid component an estimate of the alactacid energy in kilocalories was determined assuming the efficiency of repayment to be 60% (50), and assuming the caloric equivalent of one liter of oxygen to be 5 kcal. The determination of the alactacid component was accomplished in the following manner:

The determined values for oxygen consumption were plotted at the mid-point of each collection period and the points connected with a

straight line. Next, a baseline or steady state value was determined. Since there was no "ideal" way to establish a baseline oxygen value, it was arbitrarily chosen at a point during recovery in which any consecutive oxygen value did not differ by more than 25 ml. These points were averaged and the value obtained was used in determining the net oxygen debt.

Net oxygen consumption during recovery was then plotted at the mid-point of each collection period on four-cycle semi-logarithmic paper. A curve of "best fit" was then described among the plotted points.

The oxygen debt curve was then partitioned into its two components - lactacid and alactacid. The oxygen debt curve can be represented by an equation of the form:

$$O_2 = a_1 e^{-k_1 t} + a_2 e^{-k_2 t} \quad (37)$$

The first term represents the slow or lactacid component while the second term represents the fast or alactacid portion of the total curve. The method for calculating the constants is described in Appendix D. Once the constants were defined, the area under the oxygen consumption curve was then calculated. This was done by integration. The method for integrating the area under the curve is also described in Appendix D. When no excess lactic acid was present in the blood following exercise, only one set of constants (alactacid) were defined for the total oxygen debt curve.

Expression of Each Energy Component in Kcal/kg.hr

The total energy cost of a given amount of work was first calculated by summing the energy derived from the three energy components

(aerobic + lactacid + alactacid). This sum represented the total energy cost of work, expressed in kilocalories, uncorrected for body weight and independent of time. Correction for body weight and time was then done according to the following equation:

$$\frac{\text{Total Energy Cost (kcal)}}{\text{Body Weight (kg)}} \left/ \frac{60 \text{ min.}}{t \text{ min.}} \right. = \frac{\text{Total Energy Cost (kcal/kg.hr)}}{\text{}} \quad \text{where } t = \text{duration of exercise}$$

Each energy component was then expressed in the same units (kcal/kg.hr) in the following manner:

$$\frac{\text{Total Energy Cost (kcal)}}{\text{kcal/kg.hr}} \times \% = \text{Fraction (kcal/kg.hr)}$$

where % = per cent contribution (kcal) of each energy source to the total energy cost of work (kcal)

fraction = amount of the energy from each component expressed in kcal/kg.hr

Data Analysis

Simple averages and measures of dispersion were computed for all variables by means of a BMD01D computer program (23). Output from this program included:

1. Means
2. Standard deviations
3. Standard error of means
4. Maximum values
5. Minimum values
6. Ranges
7. Sample sizes

An analysis of variance was computed for each of the variables by means of a BMD02V computer program (23). This program allows computation of a factorial design breaking down the sums of squares into the main

effects and the first order interactions as indicated in the following analysis of variance table.

TABLE 4
ANALYSIS OF VARIANCE MODEL

Source of Variation	Degrees of Freedom	F Ratio
Altitude (A)	2	$\frac{A}{A \times S}$
Workload (W)	2	
Subject (S)	7	
A x W	4	
A x S	14	
W x S	14	
Residual (A x W x S)	28	
Total	71	

The Duncan multiple range test (29) was used to evaluate the significance of differences between adjacent means.

In a few instances data points for a particular variable were missing. These missing data were filled in by interpolation of measured values whenever possible or by the use of column means.

CHAPTER IV

RESULTS

Mean values and measures of dispersion are presented in Tables 6, 7 and 8, and Figures 1 and 2. All individual data are presented in Appendixes E, F and G. Summaries of analyses of variance and Duncan multiple range tests are shown in Appendixes H, I and J.

Chamber Temperature and Relative Humidity

Mean chamber temperature for all ground level runs was 25.2°C with a range of 22.5°C to 27.4°C. Relative humidity averaged 58.5% with a range of 30% to 91%. At 2286 meters mean temperature was 24.8°C with a range of 21.6°C to 27.4°C. Relative humidity for the same period averaged 57.1% with a range of 29% to 83%. At 3810 meters the chamber temperature ranged from 21.5°C to 27.6°C with a mean of 24.8°C. Relative humidity averaged 51.7% with a range of 31% to 80%.

Body Weight Changes

No significant changes in mean body weight before and after exercise were observed, although individual subjects did vary 1-2 kg.

Duration of Exercise

Table 5 shows the range and mean duration of exercise for each workload at each of the altitudes studied. It can be seen that work duration time was reduced at 2286 meters and 3810 meters for workloads approximating 60% and 80% of sea level aerobic capacity.

TABLE 5

MEAN DURATION OF EXERCISE IN MINUTES
HIGH AND LOW VALUES GIVEN IN PARENTHESES

Per Cent Aerobic Capacity	Altitude		
	223m	2286m	3810m
30	45	45	45
60	40 (9-45)	41 (9-45)	33 (7.5-45)
80	19 (4-29)	11 (5-15)	8 (3-11)

Power Input for a Fixed Quantity of Work (kcal/kg.hr)

Aerobic Power. The power derived from the aerobic energy source (oxygen consumption) is shown in Table 6 and Figure 1. At power outputs of approximately 30% and 60% of sea level aerobic capacity, the power input was independent of altitude. At 80% of sea level aerobic capacity, the power input was significantly less at simulated altitudes of 2286 meters and 3810 meters, as compared to sea level ($p < .05$). No significant difference was observed between the mean values at 2286 meters and 3810 meters.

Lactacid Power. The power derived from the breakdown of glucose or glycogen to lactic acid (glycolysis) is shown in Table 6 and Figure 1. At a power output of 30% of sea level aerobic capacity, the power input from the lactacid energy system was not significantly different from zero at any of the altitudes studied. At 60% of sea level aerobic capacity, the power input from this energy source increased with increasing

altitude. Only the mean value recorded at 3810 meters was significantly higher ($p < .05$) than the mean value at ground level, however. During work at 80% of sea level aerobic capacity the power derived from the lactacid energy source showed a significant increase with increased hypoxia ($p < .05$).

Alactacid Energy Cost. As can be seen in Table 6 and Figure 1, the power input from this source was independent of altitude at workloads approximating 30% and 60% of sea level aerobic capacity. At a power output representing 80% of maximal capacity, the power derived from the alactacid energy source increased with increasing altitude. The mean values were significantly higher at 2286 meters and 3810 meters as compared to sea level ($p < .05$). No significant difference was obtained for the two high altitude values.

Total Power Input. The total power input for a given intensity of work, represented by the summation of the aerobic, lactacid and alactacid power sources, was independent of altitude for all workloads (Table 6, Figure 1).

Relative Contribution of the Three Energy Sources to the Total Power Input

The relative contribution of the three energy components to the total power input for a given quantity of work is shown in Table 7 and Figure 2. Examination of the data in this manner shows slightly different results from those previously reported.

Aerobic. At 30% of aerobic capacity, aerobic power contributed approximately 97% of the power input, independent of altitude. At 60% of aerobic capacity, the per cent contribution of aerobic power was still

TABLE 6

POWER INPUT OF THE AEROBIC, LACTACID, AND ALACTACID ENERGY COMPONENTS
FOR DIFFERENT WORKLOADS AT 223 METERS AND DURING ACUTE EXPOSURES AT 2286 AND 3810 METERS

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
Aerobic Power - kcal/kg.hr									
Mean	2.919	2.935	2.924	6.163	6.329	5.946	8.308	7.671	7.208
S.D.	.571	.578	.639	1.002	.912	1.073	1.512	1.177	1.327
S.E.	.202	.204	.226	.354	.323	.379	.535	.416	.469
N	8	8	8	8	8	8	8	8	8
Lactacid Power - kcal/kg.hr									
Mean	.005	.006	.016	.080	.114	.244	.457	.744	.958
S.D.	.014	.007	.016	.125	.197	.217	.377	.353	.117
S.E.	.005	.003	.005	.044	.070	.076	.133	.125	.041
N	8	8	8	8	8	8	8	8	8
Alactacid Power - kcal/kg.hr									
Mean	.064	.064	.069	.111	.110	.195	.378	.639	.715
S.D.	.022	.019	.019	.040	.033	.156	.233	.436	.258
S.E.	.008	.007	.007	.014	.012	.055	.028	.154	.091
N	8	8	8	8	8	8	8	8	8

TABLE 6--Continued

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
Total Power - kcal/kg.hr									
Mean	2.988	3.001	2.918	6.354	6.553	6.385	9.144	9.054	8.882
S.D.	.582	.573	.724	.954	.924	.910	1.098	1.369	1.141
S.E.	.206	.202	.256	.337	.327	.322	.388	.484	.403
N	8	8	8	8	8	8	8	8	8

FIGURE 1.--POWER INPUT OF THE AEROBIC, LACTACID, AND ALACTACID ENERGY COMPONENTS FOR DIFFERENT WORKLOADS AT 223 METERS AND DURING ACUTE EXPOSURES AT 2286 AND 3810 METERS

independent of altitude. The mean values were 97%, 97% and 93% at 223 meters, 2286 meters and 3810 meters. At 80% of aerobic capacity, a significant reduction ($p<.05$) in the contribution of the aerobic power input was observed only for work performed at 3810 meters. The mean values were 90%, 85% and 81% for ground level, 2286 meters and 3810 meters respectively.

Lactacid. At the low workload, the per cent contribution of the lactacid component to the total power input was significantly increased only at 3810 meters. The mean values were 0.18%, 0.18% and 0.55% at sea level, 2286 meters and 3810 meters respectively. However, since the lactic acid production did not differ significantly from zero, this finding of significant difference is not real. At 60% maximal capacity, the contribution of this power source increased with increasing altitude. At 3810 meters, the mean value of 4.0% was significantly different than the mean values of 1.36% at sea level and 1.70% at 2286 meters ($p<.05$). No significant difference was observed between the mean values for the two lower altitudes. During work at 80% aerobic capacity, there was a difference in the same direction, but it was of greater magnitude. The mean values at this level of work were 5.31%, 8.13% and 11.01% at sea level, 2286 meters and 3810 meters respectively. All three values were significantly different from one another ($p<.05$).

Alactacid. The per cent contribution of alactacid power to the total power input for work at 30% of maximal capacity was found to be independent of altitude. The observed values were 2.20%, 2.39% and 2.33% for sea level, 2286 meters and 3810 meters respectively. At 60% aerobic capacity the observed values were 1.70%, 1.79% and 3.17%. The

TABLE 7

RELATIVE CONTRIBUTION OF AEROBIC, LACTACID, AND ALACTACID POWER
TO THE TOTAL POWER INPUT FOR DIFFERENT SUBMAXIMAL WORKLOADS

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
Aerobic Power - Per Cent of Total Power									
Mean	97.66	97.46	97.12	96.86	96.61	92.83	90.37	85.00	80.68
S.D.	.52	1.04	.66	2.96	3.04	6.66	7.92	7.77	5.76
S.E.	.18	.37	.23	1.05	1.07	2.36	2.80	2.75	2.04
N	8	8	8	8	8	8	8	8	8
Lactacid Power - Per Cent of Total Power									
Mean	.180	.183	.549	1.345	1.698	4.000	5.313	8.129	11.006
S.D.	.509	.224	.550	2.227	2.889	3.922	4.883	3.488	2.224
S.E.	.180	.079	.195	.787	1.021	1.387	1.727	1.233	.786
N	8	8	8	8	8	8	8	8	8
Alactacid Power - Per Cent of Total Power									
Mean	2.200	2.393	2.326	1.791	1.695	3.165	4.320	6.874	8.311
S.D.	.685	1.016	.608	.771	.545	2.793	3.081	4.394	3.708
S.E.	.242	.359	.215	.272	.193	.987	1.089	1.553	1.311
N	8	8	8	8	8	8	8	8	8

FIGURE 2.--RELATIVE CONTRIBUTION OF AEROBIC, LACTACID, AND ALACTACID POWER TO THE TOTAL POWER INPUT FOR DIFFERENT SUBMAXIMAL WORKLOADS

value obtained at 3810 meters (3.17%) was significantly higher ($p < .05$). At 80% aerobic capacity contribution of the alactacid power to the total power input increased with increasing altitude. The mean values at 2286 meters and 3810 meters were significantly higher than the mean sea level values ($p < .05$). The observed values were 4.32%, 6.87% and 8.13% for sea level, 2286 meters and 3810 meters respectively. No significant difference was observed between the two high altitude mean values.

Ventilation

The average ventilation calculated in terms of standard temperature and pressure dry (STPD) is shown in Table 8. This method of expressing gas volumes allows a comparison of the actual number of gas molecules reaching the alveoli. At a work rate approximating 30% of maximal capacity, STPD ventilation was significantly reduced ($p < .05$), 30.6% at 3810 meters and 18.7% at 2286 meters when compared with sea level values. The mean values for the two high altitudes were also significantly different from one another. At 60% of maximal capacity STPD ventilation showed an average decrease of 17.10% at 2286 meters and 16.55% at 3810 meters. While these reductions were not significantly different from one another, they were significantly different from the mean sea level value ($p < .05$). At 80% aerobic capacity, STPD ventilation was reduced 8.36% and 4.41% at 2286 meters and 3810 meters respectively. Only the reduction at 2286 meters was significantly different when compared to the mean sea level value ($p < .05$). It is worth mentioning at this point, that the mean values representing 80% of maximal capacity were taken from the 9th minute of exercise. A few subjects were unable to complete even 9 minutes at this intensity at the various altitudes. Mean values were

TABLE 8

MEASUREMENT OF PHYSIOLOGICAL VARIABLES DURING SUBMAXIMAL WORKLOADS AT 223 METERS, 2286 METERS AND 3810 METERS

ALL VALUES SHOWN FOR 30% AND 60% OF SEA LEVEL AEROBIC CAPACITY ARE THE MEAN VALUES
OBTAINED FROM THE 20TH THROUGH THE 45TH MINUTES OF EXERCISE

ALL 80% VALUES REPRESENT THE MEAN OBTAINED FROM THE 9TH MINUTE OF EXERCISE

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
$\dot{V}_E$ STPD liters/min.									
Mean	28.49	23.13	19.77	50.54	41.90	42.18	70.62	64.77	67.51
S.D.	5.08	5.35	3.97	7.22	5.27	4.46	7.06	7.06	10.68
S.E.	1.80	1.89	1.40	2.55	1.86	1.58	3.51	2.50	3.78
N	8	8	8	8	8	8	8	8	8
$\dot{V}_E$ BTPS liters/min.									
Mean	35.50	37.78	39.96	62.93	68.45	85.28	87.60	105.80	136.44
S.D.	6.31	8.74	8.01	8.64	8.62	9.01	12.04	11.54	21.58
S.E.	2.23	3.09	2.83	3.06	3.05	3.19	4.26	4.08	7.63
N	8	8	8	8	8	8	8	8	8
Tidal Volume BTPS cc									
Mean	1575	1532	1520	2285	2221	2536	2886	2961	3064
S.D.	346	305	296	577	322	320	287	614	347
S.E.	122	108	105	204	114	113	101	217	123
N	8	8	8	8	8	8	8	8	8

TABLE 8--Continued

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
Frequency, breaths/min.									
Mean	23.03	24.95	27.09	30.28	31.59	32.91	30.50	37.38	46.25
S.D.	3.32	3.03	3.87	6.05	4.26	4.24	4.38	6.97	1.98
S.E.	1.17	1.07	1.37	2.14	1.51	1.50	1.55	2.46	0.70
N	8	8	8	8	8	8	8	8	8
$\dot{V}CO_2$ cc/min.									
Mean	838.37	851.12	834.37	1651.63	1658.75	1819.00	2441.00	2589.00	2818.00
S.D.	139.33	183.25	152.98	259.77	199.04	142.88	423.33	367.84	431.00
S.E.	49.26	64.79	54.09	91.84	70.37	50.51	149.67	130.05	152.40
N	8	8	8	8	8	8	8	8	8
$\dot{V}O_2$ cc/min.									
Mean	1079.50	1091.13	1076.00	2071.38	2015.38	2009.63	2661.13	2685.25	2726.38
S.D.	133.49	170.81	149.31	279.22	252.23	253.98	434.57	334.72	411.30
S.E.	47.20	60.39	52.78	98.72	89.18	89.80	153.64	118.34	145.42
N	8	8	8	8	8	8	8	8	8

TABLE 8--Continued

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
R									
Mean	.767	.776	.773	.795	.824	.879	.917	.961	1.033
S.D.	.046	.046	.064	.040	.027	.027	.051	.027	.012
S.E.	.016	.016	.023	.014	.010	.010	.018	.010	.004
N	8	8	8	8	8	8	8	8	8
Oxygen Pulse cc/beat									
Mean	10.02	9.46	8.50	13.02	12.37	11.58	15.20	15.13	15.89
S.D.	1.56	1.49	1.48	1.95	1.89	1.38	2.64	2.07	1.36
S.E.	.55	.53	.52	.69	.67	.49	.93	.73	.48
N	8	8	8	8	8	8	8	8	8
Heart Rate - beats/min.									
Mean	110.27	115.75	127.87	160.62	164.05	174.52	175.25	177.38	184.25
S.D.	14.30	11.36	10.95	13.78	10.78	4.28	7.11	3.46	6.27
S.E.	5.06	4.02	3.87	4.87	3.81	1.51	2.51	1.22	2.21
N	8	8	8	8	8	8	8	8	8

TABLE 8--Continued

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
Oxygen Extract - %									
Mean	3.67	4.57	5.28	3.95	4.66	4.82	3.70	4.11	4.08
S.D.	.23	.46	.62	.41	.41	.25	.36	.31	.32
S.E.	.08	.16	.22	.14	.15	.09	.13	.11	.11
N	8	8	8	8	8	8	8	8	8
Rectal Temperature - °C									
Mean	37.63	37.57	37.74	38.35	38.25	38.28	37.49	37.48	37.32
S.D.	.22	.30	.19	.22	.19	.23	.29	.26	.32
S.E.	.08	.11	.07	.08	.41	.08	.10	.09	.11
N	8	8	8	8	8	8	8	8	8
Excess Lactic Acid - mg%									
Mean	2.16	1.91	6.70	18.31	24.10	50.54	59.10	70.34	73.07
S.D.	6.12	2.20	6.10	14.90	17.20	13.39	12.40	11.16	23.08
S.E.	2.16	.78	2.16	5.26	6.08	4.73	4.39	3.94	8.16
N	8	8	8	8	8	8	8	8	8

TABLE 8--Continued

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
$\dot{V}_E/\dot{V}O_2$ STPD									
Mean	26.30	21.10	18.37	24.48	20.90	21.22	26.77	24.23	24.84
S.D.	2.04	2.27	2.45	2.37	2.08	2.89	2.63	1.81	1.97
S.E.	.72	.80	.87	.84	.74	1.02	.93	.64	.70
N	8	8	8	8	8	8	8	8	8

used to fill in the missing data. For this reason comparisons were difficult and may be misleading in some cases.

The average ventilation in terms of body temperature and pressure saturated, (BTPS) is also shown in Table 8. This method of expressing gas volumes allows for a volumetric comparison of ventilatory values. Ventilation was higher under hypoxic conditions than under normoxic conditions for all workloads.

The amount of increase in BTPS ventilation was higher the greater the degree of hypoxia. When compared to mean sea level values, BTPS ventilation at work rates representing 30% maximal aerobic capacity was increased by 6.04% at 2286 meters and significantly increased by 11.17% at 3810 meters ($p < .05$). A similar trend was noted for work at 60% maximal capacity. At this level of work, ventilation was increased 8.07% and 19.74% at 2286 meters and 3810 meters respectively. Only the increase at 3810 meters, however, was significant ($p < .05$). At 80% maximal capacity, BTPS ventilation was significantly higher at the two altitudes when compared with the mean sea level value ($p < .05$). The observed increases were 17.21% and 22.46% at 2286 meters and 3810 meters respectively.

Heart Rate. Heart rate tended to rise with increased altitude; however, significant elevations in the exercising heart rate were only observed at 3810 meters ($p < .05$). It is worth noting that the magnitude of the differences in heart rate was less the greater the workload. For example, at a workload representing 30% maximal aerobic capacity, the difference in heart rate between sea level and 3810 meters was 17.6 beats; at 60% maximal capacity the difference in heart rate between these two

altitudes was 13.9 beats, and at 80% maximal work the difference was 9.0 beats.

Rectal Temperature. Rectal temperature showed no consistent change with increased altitude for any of the given workloads. The obvious reduction in rectal temperature observed at 80% of aerobic capacity reflects only shorter work times and has no real physiological significance.

Respiratory Exchange Ratio. At work rates above 30% maximal capacity, the respiratory quotient was significantly increased for a given amount of work with increasing altitude, the increase being more marked the greater the degree of hypoxia ($p < .05$). The increased R may be related in part to a release of CO_2 from bicarbonate by lactic acid since it is associated with increased lactic acid production and in part to a decrease in CO_2 stores due to relative hyperventilation.

Blood Lactate. The values given in Table 8 represent the net peak lactic acid concentrations following the termination of exercise. Blood lactate concentration did not increase significantly until a work rate of 60% maximal aerobic capacity. At this workload, the mean concentration of lactate was higher during submaximal work at 2286 meters and 3810 meters than at sea level. However, significant differences were observed only at 3810 meters ($p < .05$). During work at 80% aerobic capacity, no significant changes in blood lactic acid concentration were observed with increased altitude.

Oxygen Extraction. Oxygen extraction increased for a given amount of work at increasing altitude. It is interesting to note that the magnitude of the changes was smaller when work was performed at a

greater percentage of maximal aerobic capacity. At 30% of aerobic capacity, all three values of oxygen extraction were significantly different from one another. At 60% and 80% of aerobic capacity, the values at 2286 meters and 3810 meters were significantly higher than ground level, but not significantly different from one another.

Oxygen Pulse. The oxygen pulse has been used to give a relative measure of the oxygen being consumed or extracted by the tissues per heart beat. At 30% of aerobic capacity, a significantly lower oxygen pulse was observed with increased altitude ($p < .05$). At 60% aerobic capacity, a significant decrease in the oxygen pulse was observed only at 3810 meters. No significant alterations in the oxygen pulse with increased altitude were observed for work at 80% aerobic capacity.

CHAPTER V

DISCUSSION

This experiment was designed to partition man's energy sources into their three component parts - aerobic, lactacid and alactacid - and to assess whether the power derived from each of these components during muscular work was utilized differently at altitude than at sea level. The null hypothesis to be tested was that the power derived from the aerobic, lactacid, and alactacid energy systems was independent of altitude and, therefore, that the total power input for a given power output was independent of altitude.

In evaluating the participation of these energy systems in muscular activity, it is necessary to keep in mind two important concepts: (1) capacity, which is the ability to reach a maximal value, (2) power, which is work per unit of time. In this study it was necessary to express the energy component on a power basis because of decreased performance times at altitude (Table 5).

The results of this study showed that during acute hypoxia, the aerobic power input for work at 60% or less of sea level aerobic capacity was independent of the altitude at which the work was performed. At workloads of 80% aerobic capacity, aerobic power was significantly lower during hypoxic exercise than during exercise at near sea level elevations. A decreased oxygen consumption has been previously reported by some

investigators (9, 16, 54). Cronin and MacIntosh (16), for example, attributed a decreased oxygen consumption during hypoxic exercise (11% oxygen) to increased muscular efficiency. They based their conclusion on the facts that the observed decrease in oxygen consumption was not due to a slower attainment of steady state and that the observed increase in the oxygen debt following exercise (especially at the high exercise levels) at a reduced oxygen tension was not sufficient to account for the oxygen deficit. Naimark et al. (54) showed that decreased oxygen consumptions during moderate work (1.5 l/min. O₂) breathing air and hypoxic gas mixtures (11-12% O₂), were associated with increased blood lactate concentrations. Their findings are more in accord with the results of this study. This reciprocal relationship between the aerobic and anaerobic components observed in this study and in the study of Naimark et al. (54) would tend to suggest an increased reliance on anaerobic power sources at least at higher exercise levels during acute hypoxic exposure. Furthermore, the results of this study do not lend support to the suggestion of increased muscular efficiency during hypoxia, as contended by Cronin and MacIntosh (16).

The accumulation of lactate in the blood is generally accepted as a sign of anaerobic glycolysis. Lactacidemia for a given grade of work during acute hypoxic exposure has been reported in a number of studies (18, 28, 30, 33, 39, 47, 48, 53, 54, 55, 59, 60). However, in none of these studies was the power derived from the lactacid energy system quantitated and related to the total power input for a given amount of work.

In this study, muscular activity approximating 30% of sea level

aerobic capacity resulted in no significant encroachment on the lactacid energy system either at sea level or up to altitudes of 3810 meters. At 60% of aerobic capacity, increased blood lactic acid levels were observed at each altitude. The power derived from this energy source at this workload was minimal at sea level and 2286 meters, but significantly higher at 3810 meters. At 80% of aerobic capacity, there were no significant differences in peak blood lactic acid values among the three altitudes; however, the power input from this energy source was significantly higher with increased hypoxia.

Two important facts are substantiated from the data presented in this study: (1) acute exposure to hypoxia had no effect on the capacity of the lactacid mechanism to reach peak lactic acid values, and (2) the greater power derived from the lactacid mechanism with increased altitude was not due to an increase in the capacity of this mechanism, but was rather due to a similar energy input over a shorter period of work.

It would appear that the degree to which tissue oxygenation is impaired for a given degree of hypoxia may be related to the intensity of the workload. During light workloads approximating 30% of sea level aerobic capacity even under "severe hypoxic" conditions (3810 meters) the oxygen provision to the working muscle is maintained, probably through compensating mechanisms involving a redistribution of blood out of the inactive areas to the working muscles (3), increased cardiac output (32, 44, 57, 62) and stroke volume (32, 62), and an increased oxygen extraction manifested by means of an increased arterial-venous oxygen difference (1, 34, 36, 42). Workloads of 60% or more of sea level aerobic capacity, however, appear to have exceeded the ability of the

subjects to maintain proper tissue oxygenation resulting in a greater reliance on anaerobic glycolysis.

This greater utilization of the lactacid energy system probably occurs for a number of reasons, despite efforts of compensating mechanism to maintain proper tissue oxygenation. It has been shown in this study (Table 8) and in other studies (7, 9), that mass ventilation is reduced at altitude for a given amount of work. Therefore, the number of oxygen molecules reaching the alveoli is less, which results in a decreased oxygen saturation of the blood leaving the lungs. Desaturation of the arterial blood during exercise in low ambient partial pressures has been reported (34, 64). This fall in arterial oxygen saturation occurred despite an increased alveolar - arterial pressure gradient which supports a diffusion limitation to oxygen transfer (34, 64). Stenberg (59), in some recent hemodynamic studies, presented evidence of a decreased amount of oxygen transport from the left heart because of a reduced oxygen content in the arterial blood. This reduction in oxygen transport concomitant with a decreased arterial P_{O_2} and in all probability a decreased tissue P_{O_2} is probably the main reason for the increased utilization of the anaerobic glycolytic pathway. This encroachment on the anaerobic metabolic pathway becomes particularly evident at the higher workloads at altitude since one is working at a much higher relative percentage of his aerobic capacity. At these workloads, the same or even an increased arterial-venous oxygen difference may not be sufficient to meet aerobically the energy demands of the working muscle.

The original concept of the alactacid oxygen debt as proposed by

Margaria (49) was the oxygen required for the resynthesis of phosphagens broken down during muscular activity. Later this concept was broadened to include the restoration of depleted oxygen stores in the myoglobin and in the arterial-venous blood (46, 50, 63).

There is very little information available concerning the effects of hypoxia on the alactacid energy systems. Cerretelli (13) suggested that the alactacid debt for exhaustive work is constant and independent of altitude. He admits, however, that the venous oxygen stores could account for 10% of the over-all alactacid oxygen debt. Recently Karlsson and Saltin (43) showed that phosphagens are depleted to the same extent after 2-3 minutes of work whether the load is exhaustive or not.

The data presented in this study showed that the capacity of this mechanism to deliver energy was constant and independent of altitude. For example, values for the alactacid oxygen debt were 2342 cc, 2393 cc and 2149 cc during work at 80% of aerobic capacity at 223 meters, 2286 meters and 3810 meters respectively. However, the power derived from this energy source was constant and independent of altitude only for workloads of 60% or less of sea level aerobic capacity. At 80% of sea level aerobic capacity, the power produced by the alactacid energy system was significantly increased over sea level values, by 40% at 2286 meters and 47% at 3810 meters. Again the increased power production reflects only a maximal energy release by this system over a shorter period of work performance with increased hypoxia.

It becomes obvious from this study that as the aerobic power was reduced with increased hypoxia, anaerobic power was utilized to counter-balance or offset the decrease in the aerobic power so that the total

power input for a given amount of work remained unaffected by hypoxia.

CHAPTER VI

SUMMARY AND CONCLUSIONS

The purpose of this experiment was to partition man's energy sources into their three component parts - aerobic, lactic acid and alactic acid - and to assess whether the power derived from each of these components is utilized differently at altitude than at sea level.

Eight adult males varying in degrees of physical fitness were selected from The Ohio State University for use in this study. Each subject exercised on a bicycle ergometer once a week in a hypobaric chamber at sea level and during acute exposure to 2286 meters and 3810 meters. Each subject performed at 30%, 60% and 80% of his sea level aerobic capacity. Each level of work was performed once at each altitude. The work level and corresponding altitude were assigned in a random manner.

Within the limits of the experiment, the following conclusions appear to be justified:

1. The total power input for a given power output was independent of altitude.
2. The aerobic power for work at 60% or less of sea level aerobic capacity was independent of altitude. At 80% of sea level aerobic capacity, aerobic power was significantly decreased with hypoxia.

3. At light workloads (30% of sea level aerobic capacity) there was no significant utilization of lactic acid power either at sea level or at altitudes up to 3810 meters. At 60% aerobic capacity, minimal utilization of lactic acid power occurred at sea level and 2286 meters with a significantly greater rate of power expenditure occurring at 3810 meters. At 80% of aerobic capacity, significant increases in power expenditure occurred with increasing altitude.
4. At workloads of 60% or less of sea level aerobic capacity, the power derived from the alactic acid energy source was independent of altitude. At 80% of aerobic capacity, however, a significantly greater power input from this energy system occurred with increasing altitude.

APPENDIXES

APPENDIX A

PHYSIOLOGICAL RESPONSES TO MAXIMAL WORK AT SEA LEVEL
ON A BICYCLE ERGOMETER

Subject	$\dot{V}_{O_2}$ l/min.	$\dot{V}_{O_2}$ ml/kg/min.	$\dot{V}_E$ BTPS l/min.	$\dot{V}_E$ STPD l/min.	Heart Rate beats/min.	f breaths/min.
A	2.47	36.95	110.81	89.54	188	52
B	3.42	40.45	118.84	95.96	192	42
C	2.18	36.61	118.86	96.32	192	49
E	3.67	38.82	166.95	134.75	196	60
F	3.83	45.24	148.41	119.21	194	54
G	3.46	55.36	131.49	104.44	190	48
H	3.27	38.27	149.26	119.31	192	40
I	3.66	42.49	109.581	87.88	184	40

APPENDIX B

DETERMINATION OF SUBMAXIMAL WORK

It is a well established fact that there is a direct linear relationship between oxygen uptake and work rate. Consequently, a correlation and regression analysis can be used to derive a linear approximation between the two variables, keeping in mind that the equation for a straight line is represented by the formula:

$$y = a + bx, \text{ where } \begin{array}{l} a = \text{intercept} \\ b = \text{slope of the line.} \end{array}$$

This principle was used to estimate submaximal work corresponding to 25%, 50% and 67% of one's maximal aerobic capacity. The oxygen uptake data and corresponding workloads were obtained from the third progressive maximal workup (see maximal oxygen consumption test under Methods and Procedures). The actual computation for this line of best fit from a regression analysis has been omitted, since such procedures can be found in any statistical text book. The straight line was then plotted on regular graph paper.

It now remained to determine the power output in watts corresponding to 25%, 50% and 67% of one's maximal aerobic capacity. This was done quite easily and can be best illustrated by the following example.

The resting oxygen uptake was first subtracted from the maximal oxygen consumption.

$$\begin{array}{l} \dot{V}O_2 \text{ Max.} = 3.656 \text{ l/min.} \\ \dot{V}O_2 \text{ Rest} = \underline{.348} \text{ l/min.} \\ \hline 3.308 \end{array}$$

APPENDIX B--Continued

Next, 25%, 50% and 67% of the above value were obtained.

$$\begin{array}{r} 3.308 \\ \underline{.827} \text{ 25\%} \\ 1.654 \text{ l/min.} \end{array} \quad \begin{array}{r} 3.308 \\ \underline{1.654} \text{ 50\%} \\ 2.216 \text{ l/min.} \end{array} \quad \begin{array}{r} 3.308 \\ \underline{2.216} \text{ 67\%} \\ 2.216 \text{ l/min.} \end{array}$$

The resting oxygen uptake was now added to these new values so that,

$$\begin{array}{r} .827 \\ \underline{.348} \\ 1.175 \text{ l/min.} \end{array} \quad \begin{array}{r} 1.654 \\ \underline{.348} \\ 2.002 \text{ l/min.} \end{array} \quad \begin{array}{r} 2.216 \\ \underline{.348} \\ 2.564 \text{ l/min.} \end{array}$$

From a graph containing the line of best fit, relating power output and oxygen consumption, power outputs corresponding to 1.175 l/min., 2.002 l/min. and 2.564 l/min. respectively were obtained.

The following table shows the output value in watts for the three different intensities studied.

Subject	Intensities Studied (Watts)		
	25% Max. $\dot{V}O_2$	50% Max. $\dot{V}O_2$	67% Max. $\dot{V}O_2$
A	45	95	135
B	60	130	175
C	45	90	120
E	40	140	210
F	70	140	200
G	60	125	170
H	60	125	175
I	50	140	205

As previously noted, the above intensities more nearly approximated 30%, 60% and 80%, the subject's aerobic capacities, as estimated from steady state oxygen consumption obtained during much longer periods of work. This discrepancy between predicted and observed values is not

APPENDIX B--Continued

surprising. It will be recalled that each increment in workload during the progressive workup lasted only two minutes. This time period is not sufficiently long for the oxygen consumption to reach a steady state value. Consequently, a lower oxygen consumption is obtained for a given workload. This results in lower relative workloads (oxygen uptake as a fraction of maximal oxygen uptake) when compared to the relative workloads obtained using steady state oxygen consumptions.

APPENDIX C

SYMBOLS AND EQUATIONS USED IN CALCULATING VALUES
RELATED TO VENTILATION AND THE METABOLIC COST OF WORK

Inputs

1. P_A - (Ambient Pressure) Barometric pressure at spirometer when gas is measured volumetrically in mm Hg.
2. P_B - (Body Pressure) Barometric pressure where experiment was done.
3. P_{H_2O} - Water Vapor Pressure - Vapor pressure at T_s , spirometer temperature, from a table to be furnished in mm Hg.
4. T_s - Temperature of gas in spirometer when gas is measured volumetrically, in $^{\circ}C$.
5. D_f - Spirometer displacement when full of sample, in mm.
6. D_e - Spirometer displacement when empty, in mm.
7. K_s - Spirometer bell factor in cc/mm.
8. t - Duration of gas collection in minutes.
9. V_s - Volume of sample aliquot drawn for analysis in cc (ATPS).
10. $F_{I_{O_2}}$ - Fraction concentration of oxygen in inspired air.
11. $F_{E_{O_2}}$ - Fraction concentration of oxygen in expired air.
12. $F_{I_{CO_2}}$ - Fraction concentration of CO_2 in inspired air.
13. $F_{E_{CO_2}}$ - Fraction concentration of CO_2 in expired air.
14. f - Respiratory frequency in, breaths/min.

Outputs - Ventilation

1. $\dot{V}_E$ ATPS - Expired volume at ambient temperature and pressure saturated, in cc/min.
2. $\dot{V}_E$ STPD - Expired volume of air at standard temperature ($0^{\circ}C$) and pressure (760 mm Hg) in cc/min.
3. $\dot{V}_E$ BTPS - Expired volume of air expressed at body temperature ($37^{\circ}C$) and pressure (variable), saturated with water vapor, in cc/min.

APPENDIX C--Continued

4. V_T BTPS - (Tidal volume), volume of gas expired per breath.

Equations - Ventilation

1. $\dot{V}_E \text{ ATPS} = \frac{(\dot{D}_f - \dot{D}_e) K_s + V_s}{t}$
2. $\dot{V}_E \text{ STPD} = \dot{V}_E \text{ ATPS} \frac{P_A - P_{H_2O}}{760} \frac{273}{273 + T_s}$
3. $\dot{V}_E \text{ BTPS} = \dot{V}_E \text{ STPD} \frac{760}{P_B - 47} \frac{310}{273}$
 $= \dot{V}_E \text{ STPD} \frac{863}{P_B - 47}$
4. $V_T \text{ BTPS} = \dot{V}_E \text{ BTPS} / f$

Outputs - Metabolic

1. $\dot{V}_I = \dot{V}_E \text{ STPD} \frac{1 - F_{E_{O_2}} - F_{E_{CO_2}}}{1 - F_{I_{O_2}} - F_{I_{CO_2}}}$
2. $\dot{V}_{O_2} \text{ STPD} = \dot{V}_I \text{ STPD } F_{I_{O_2}} - \dot{V}_E \text{ STPD } F_{E_{O_2}}$
3. $\dot{V}_{CO_2} \text{ STPD} = \dot{V}_E \text{ STPD } F_{E_{CO_2}} - \dot{V}_I \text{ STPD } F_{I_{CO_2}}$
4. $O_2 \text{ Extraction} = (F_{I_{O_2}} - F_{E_{O_2}}) \times 100$
5. $R = \dot{V}_{CO_2} / \dot{V}_{O_2}$
6. $O_2 \text{ Pulse} = \text{ml } O_2 / \text{H.R.}$

APPENDIX C--Continued

Input - Output Data

<u>Identification</u>	<u>Digits</u>	<u>Identification</u>	<u>Example</u>
1. Altitude	1	1 2	E25 N
2. Workload	1	3 6	R40 N
3. Collection Time	3		
4. Subject	1		
<u>Altitude</u>	<u>Workload</u>	<u>Collection Time</u>	
a. 223 meters - 1	30% - 2	E0 - E45	
b. 2286 meters - 2	60% - 5	R0 - R60	
c. 3810 meters - 3	80% - 6		
<u>Inputs</u>	<u>Digits</u>	<u>Range</u>	
1. P _A , mm Hg	xxx.x	470 - 760	
2. P _B , mm Hg	xxx.x	470 - 760	
3. P _{H₂O}	xx.x	17.5 - 47	
4. T _s , °C	xx.x	18 - 30	
5. D _f mm	xxxx.x	60 - 1000	
6. D _e mm	xx.x	5 - 80	
7. K _s cc/mm	xxx.x	133.2	
8. V _s cc	xxxx	2000 - 2500	
9. t min	x.x	0 - 6	
10. F _{E_{O₂}}	.xxxx	.1300 - .1900	
11. F _{E_{CO₂}}	.xxxx	.0200 - .0800	

APPENDIX C--Continued

	<u>Inputs</u>	<u>Digits</u>	<u>Range</u>
12.	$\dot{V}_{I_{O_2}}$	.xxxx	.1900 - .2400
13.	$\dot{V}_{I_{CO_2}}$	.xxxx	.0000 - .0040
14.	f	xx.	5 - 70
15.	Heart Rate	xxx.	40 - 200
16.	Lactic Acid	xxx.x	109 - 200
17.	Power	xxx.	50 - 325
18.	Rectal Temperature °C	xx.xx	36 - 41

Note: Numbers 14, 15, 16, 17 and 18 are to be transferred from input card to output card without manipulation.

	<u>Outputs</u>	<u>Digits</u>	
1.	Identification	6	12E25N
2.	$\dot{V}_E$ STPD cc/min.	6	107536
3.	$\dot{V}_E$ BTPS cc/min.	6	121724
4.	V_T BTPS cc	4	350.5
5.	$\dot{V}_{O_2}$ STPD cc/min.	4	2176
6.	$\dot{V}_{CO_2}$ STPD cc/min.	4	2053
7.	R	3	.955
8.	O ₂ Extraction	3	5.60
9.	Respiratory Frequency	2	15
10.	Heart Rate	3	200
11.	Oxygen Pulse	3	24.2
12.	Lactic Acid	3	200
13.	Power	3	300

APPENDIX C--Continued

<u>Outputs</u>	<u>Digits</u>	
14. Rectal Temperature °C	3	37
15. Gas Collection Time	2	6.0

APPENDIX D

FRACTIONATION OF OXYGEN DEBT CURVE
INTO LACTIC AND ALACTIC COMPONENTS

The total respiratory oxygen debt curve may be described as the sum of two exponential terms, $a_1 e^{-k_1 t}$ and $a_2 e^{-k_2 t}$. The total oxygen curve could therefore be represented by an equation of the form:

$$O_2 = a_1 e^{-k_1 t} + a_2 e^{-k_2 t} \quad (37)$$

The first term represents slow or lactacid component, while the second represents the fast or alactacid component of the total oxygen debt curve. These two components each plot as a straight line on semilog paper and add together vertically to form a theoretical curve which should fit the experimental curve closely (Figure 3). In order to evaluate the constants of the above equation, a time must be chosen at which one of these components does not contribute to the total oxygen debt curve. This enables one to evaluate the constants of each component separately.

To evaluate the two components of the lactacid portion, times are chosen in the region where the lactacid component is the only contributor to the total oxygen debt curve. Therefore,

$$O_2 = a_1 e^{-k_1 t}$$

In the example given in Figure 3, these times are 9 minutes and 47.5 minutes. We can now write a system of two simultaneous equations in a_1 and k_1 . From Figure 3 the following values are selected:

APPENDIX D--Continued

$$t = 47.5 \text{ min.} = .7916 \text{ hours} \quad O_2 = 13.5 \text{ cc}$$

$$t = 9.0 \text{ min.} = .1500 \text{ hours} \quad O_2 = 220 \text{ cc}$$

Then,

$$13.5 = a_1 e^{-.7916k_1}$$

$$220 = a_1 e^{-.1500k_1}$$

(Note: the time t is converted into hours so that the exponents can be manipulated more easily).

By taking natural or Napierian logarithms of both sides of the above system of equations, the following new system is obtained:

$$\ln 220 = \ln a_1 - .1500k_1$$

$$\ln 13.5 = \ln a_1 - .7916k_1$$

or,

$$5.3936 = \ln a_1 - .1500k_1$$

$$2.6027 = \ln a_1 - .7916k_1$$

and, subtracting one equation from another,

$$5.3936 = \ln a_1 - .1500k_1$$

$$2.6027 = \ln a_1 - .7916k_1$$

$$\hline 2.7909 = .6416k_1$$

$$k_1 = 4.35$$

Now, substituting k_1 back into one of the above equations, and solving for a_1

$$5.3936 = \ln a_1 - (.1500)(4.35)$$

$$5.3936 = \ln a_1 - 0.6525$$

APPENDIX D--Continued

$$\ln a_1 = 6.0461$$

$$a_1 = 423.46.$$

Therefore, the first term of the equation can be written as

$$O_2 = 423.46e^{-4.35t}.$$

It now remains to solve for the alactacid portion, or

$$O_2 = 423.46e^{-4.35t} + a_2e^{-k_2t}.$$

The constants a_2 and k_2 are evaluated by choosing $t = 0$ (end of exercise) and $t = 3$ minutes. It is important to choose the second term appropriately; this point is chosen where the slope starts a rather severe change, thus forcing the curve to turn:

$$t = 0 \qquad O_2 = 2538 \text{ cc}$$

$$t = 3 \text{ min.} = .0500 \text{ hours} \qquad O_2 = 450 \text{ cc}$$

$$2538 = 423.46e^{-4.35t} + a_2e^{-k_2t}$$

$$2538 = 423.46e^{-(4.35)(0)} + a_2e^{-0k_2}$$

$$2538 = 423.46e^{-0} + a_2e^{-0}$$

Since at zero all exponential values become one, that is to say, $e^0 = 1$, then

$$2538 = (423.46)(1) + (a_2)(1)$$

$$2538 = 423.46 + a_2$$

$$a_2 = 2538 - 423.46$$

APPENDIX D--Continued

$$a_2 = 2114.54.$$

Then,

$$450 = 423.46e^{-(4.35)(.0500)} + 2114.54e^{-.0500k_2}$$

$$450 = 423.46e^{-.2175} + 2114.54e^{-.0500k_2}$$

Substituting $e^{-.2175} = .8025$, thus,

$$450 = (423.46)(.8025) + 2114.54e^{-.0500k_2}$$

$$450 = 339.83 + 2114.54e^{-.0500k_2}$$

$$110.17 = 2114.54e^{-.0500k_2}$$

$$.0521 = e^{-.0500k_2}$$

By taking the natural logs of both sides of the above equation, and keeping in mind that the log of a number smaller than 1 becomes negative by the definition of a Napierian logarithm, it is determined that:

$$-2.9565 = -.0500k_2$$

Then, dividing the above equation, $k_2 = 59.13$.

The final equation now becomes:

$$O_2 = 423.46e^{-4.35t} + 2114.54e^{-59.13t}$$

The following shows the calculation of the lactic acid, alactic acid and total oxygen debt at any given time "t":

APPENDIX D--Continued

at t = .0166 hours (1 min.)

$$423.46e^{-(4.35)(.0166)} + 2114.54e^{-(59.13)(.0166)}$$

$$423.46e^{-(.07)} + 2114.54e^{-(.98)}$$

$$(423.46)(.9324) + (2114.54)(.3753)$$

$$394.91 + 793.59;$$

at t = .0500 hours (3 min.)

$$423.46e^{-(4.35)(.0500)} + 2114.54e^{(59.13)(.0500)}$$

$$423.46e^{-(.22)} + 2114.54e^{-(2.96)}$$

$$(423.46)(.8025) + (2114.43)(.0518)$$

$$339.83 + 109.53,$$

at t = .0833 hours (5 min.)

$$423.46e^{-(4.35)(.0833)} + 2114.54e^{-(59.13)(.0833)}$$

$$423.46e^{(-.36)} + 2114.54e^{-(4.93)}$$

$$(423.46)(.6977) + (2114.54)(.0072)$$

$$295.45 + 15.22;$$

at t = .1166 hours (7 min.)

$$423.46e^{-(4.35)(.1166)} + 2114.54e^{-(59.13)(.1166)}$$

$$423.46e^{-(.51)} + 2114.54e^{-(6.89)}$$

APPENDIX D--Continued

$$(423.46) (.6005) + (2114.54) (.0010)$$

$$254.29 + 2.11.$$

Summary of above calculations:

<u>Time in hours</u>	<u>Lactacid Portion of O₂ debt cc</u>	<u>Alactacid Portion of O₂ debt cc</u>	<u>Total for time t cc</u>
.0166	394.91	793.59	1188.50
.0500	339.83	109.53	449.36
.0833	295.45	15.22	310.67
.1166	254.29	2.11	256.40
.333	99.34	-	99.34
.500	47.85	-	47.85

A plot of these values is shown in Figure 3.

It now remains to determine the area under both components of respiratory oxygen debt curve. This is done by integrating the equation over the corresponding periods of time, noting that:

$$\int_0^t a e^{-kt} = a \int_0^t e^{-kt} = -\frac{a}{k} e^{-kt} \Big|_0^t$$

The first step is to establish the duration of integration for each component. For the lactacid component this simply corresponds to the time it took for the recovery oxygen consumption to return to baseline. It will be recalled that the baseline was arbitrarily defined at a point during recovery in which any consecutive oxygen value did not differ by more than 25 ml. The duration of integration for the alactacid oxygen debt, on the other hand, corresponds to the time that this component intersects the baseline. As can be seen from Figure 3, then, the alactacid and lactacid oxygen debt components are integrated over 5

APPENDIX D--Continued

minutes and 47.5 minutes respectively.

The integration of each component is as follows:

Lactacid Portion

$$\begin{aligned}
 \int_0^{.7916} 423.46 e^{-(4.35) (.7916)} dt &= 423.46 \int_0^{.7916} e^{-(4.35) (.7916)} dt \\
 &= \frac{-423.46}{4.35} e^{-(4.35) (.7916)} \Big|_0^{.7916} \\
 &= \frac{-423.46}{4.35} \left[e^{-(4.35) (.7916)} - e^0 \right] \\
 &= -97.35 \left[e^{-3.444} - e^0 \right] \\
 &= -97.35 \left[.0321 - 1 \right] \\
 &= -97.35 \left[-.9679 \right] = 94.23
 \end{aligned}$$

The total lactacid debt then is 94.23 cc in .7916 hours, or

$$94.20 \times 60 \text{ min.} = 5683.80 \text{ cc in } 47.5 \text{ min.}$$

Alactacid Portion

$$\begin{aligned}
 \int_0^{.0833} 2114.54 e^{-(59.13) (.0833)} dt &= 2114.54 \int_0^{.0833} e^{-(59.13) (.0833)} dt \\
 &= \frac{2115.54}{-59.13} e^{-(59.13) (.0833)} \Big|_0^{.0833}
 \end{aligned}$$

APPENDIX D--Continued

$$\begin{aligned}
 &= \begin{bmatrix} 35.76 & e^{-4.93} & -a \\ & -e & \end{bmatrix} \\
 &= \begin{bmatrix} 35.76 & .0072 & -1 \end{bmatrix} \\
 &= 35.76 \begin{bmatrix} -.9928 \end{bmatrix} = 35.50
 \end{aligned}$$

The total alactacid debt is 35.50 cc in .0833 hours, or

$$35.50 \times 60 \text{ min.} = 2130.00 \text{ cc}$$

Total respiratory O₂ debt therefore,

$$\text{Lactacid} = 5683.80 \text{ cc}$$

$$\text{Alactacid} = \underline{2130.00}$$

$$\text{Total} = 7783.80 \text{ cc}$$

The fractionation of any respiratory oxygen debt curve presupposes the presence of excess lactic acid in the blood. This must be established first before using this method. When no excess lactic acid is present in the blood, only one set of constants is sufficient to describe the oxygen debt curve.

FIGURE 3.--FRACTIONATION OF THE OXYGEN DEBT CURVE

APPENDIX E

INDIVIDUAL VALUES OF POWER INPUT OF THE AEROBIC, LACTACID, AND ALACTACID ENERGY COMPONENTS
FOR DIFFERENT WORKLOADS AT 223 METERS AND DURING ACUTE EXPOSURES AT 2286 AND 3810 METERS

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
Aerobic Power - kcal/kg . hr									
A	3.345	3.267	2.875	5.051	6.081	4.375	5.750	6.186	5.303
B	2.942	2.694	3.054	6.254	5.659	5.434	7.433	6.390	6.170
C	3.635	3.445	3.361	7.389	6.230	6.263	8.499	7.914	7.454
E	2.096	1.859	1.941	4.627	5.635	5.573	8.758	7.654	7.790
F	3.448	3.218	3.202	6.918	6.875	6.323	9.507	8.455	7.792
G	4.082	3.648	3.979	7.256	8.296	7.987	10.580	9.815	9.228
H	2.627	2.794	2.728	5.813	5.515	5.200	7.068	6.972	5.794
I	2.195	2.558	2.249	5.999	6.342	6.415	8.871	7.977	7.953
Lactacid Power - kcal/kg . hr									
A	.039	.005	.043	.381	.597	.704	1.358	1.417	1.070
B	.000	.000	.000	.028	.026	.394	.463	.692	1.016
C	.000	.010	.025	.100	.108	.306	.288	.808	1.170
E	.000	.000	.000	.033	.036	.132	.347	.512	.859
F	.000	.018	.018	.000	.023	.094	.165	.552	.861
G	.000	.000	.000	.025	.063	.135	.247	1.099	.851
H	.000	.000	.015	.057	.030	.094	.433	.464	.946
I	.000	.012	.028	.016	.030	.092	.354	.405	.899

APPENDIX E--Continued

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
Alactacid Power - kcal/kg . hr									
A	.036	.044	.061	.198	.134	.481	.930	1.447	1.147
B	.059	.059	.090	.112	.157	.382	.433	.426	.920
C	.104	.046	.066	.132	.110	.226	.194	.957	.888
E	.060	.086	.062	.099	.096	.090	.257	.301	.394
F	.085	.084	.105	.073	.141	.131	.298	.379	.454
G	.059	.089	.064	.104	.096	.111	.283	.989	.718
H	.061	.055	.058	.085	.053	.051	.312	.350	.668
I	.067	.083	.044	.082	.095	.090	.321	.263	.528
Total Power - kcal/kg . hr									
A	3.420	3.316	2.979	5.630	6.812	5.560	8.038	9.051	7.520
B	3.001	2.753	3.144	6.394	5.842	6.210	8.329	7.508	8.106
C	3.739	3.501	3.452	7.621	6.448	6.794	8.981	9.680	9.512
E	2.156	1.945	2.003	4.759	5.767	5.794	9.362	8.470	9.223
F	3.533	3.320	3.325	6.991	7.038	6.548	9.970	9.386	9.107
G	4.023	3.737	4.045	7.385	8.456	8.233	11.110	11.903	10.797
H	2.688	2.849	2.801	5.955	5.597	5.345	7.813	7.787	7.408
I	2.262	2.653	2.321	6.097	6.467	6.597	9.546	8.644	9.380

APPENDIX F

INDIVIDUAL DATA ON THE RELATIVE CONTRIBUTION OF AEROBIC, LACTACID, AND ALACTACID POWER TO THE TOTAL POWER INPUT FOR DIFFERENT SUBMAXIMAL WORKLOADS

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m

Aerobic Power - Per Cent of Total Power

A	97.82	98.53	96.52	89.71	89.27	78.69	71.53	68.35	70.52
B	98.03	97.87	97.15	97.81	96.86	87.50	89.24	85.11	76.12
C	97.23	98.38	97.40	96.95	96.62	92.18	94.63	81.76	78.37
E	97.21	95.59	96.92	97.22	97.71	96.18	93.55	90.40	86.42
F	97.60	96.93	96.30	98.96	97.69	96.57	95.36	90.08	85.56
G	98.58	97.62	98.42	98.25	98.11	98.53	95.23	82.46	85.47
H	97.74	98.07	97.40	97.62	98.53	97.29	90.47	89.54	78.21
I	97.05	96.42	96.89	98.39	98.07	97.24	92.93	92.28	84.79

Lactacid Power - Per Cent of Total Power

A	1.14	.16	1.43	6.76	8.77	12.66	16.90	15.66	14.23
B	.00	.00	.00	.44	.45	6.34	5.56	9.22	12.53
C	.00	.30	.71	1.31	1.67	4.50	3.21	8.35	12.30
E	.00	.00	.00	.69	.63	2.27	3.71	6.05	9.31
F	.00	.54	.54	.00	.32	1.43	1.65	5.88	9.45
G	.00	.00	.00	.34	.75	1.64	2.22	9.23	7.88
H	.00	.00	.52	.96	.53	1.76	5.54	5.96	12.77
I	.00	.46	1.19	.26	.46	1.40	3.71	4.68	9.58

APPENDIX F--Continued

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
Alactacid Power - Per Cent of Total Power									
A	1.04	1.31	2.05	3.53	1.96	8.65	11.57	15.99	15.25
B	1.97	2.13	2.85	1.75	2.69	6.16	5.20	5.67	11.35
C	2.77	1.32	1.89	1.74	1.71	3.32	2.16	9.89	9.33
E	2.79	4.41	3.08	2.09	1.66	1.55	2.74	3.55	4.27
F	2.40	2.54	3.16	1.04	1.99	2.00	2.99	4.04	4.99
G	1.42	2.38	1.58	1.41	1.14	1.35	2.55	8.31	6.65
H	2.26	1.93	2.08	1.42	.94	.95	3.99	4.50	9.02
I	2.95	3.12	1.92	1.35	1.47	1.36	3.36	3.04	5.63

APPENDIX G

INDIVIDUAL MEASUREMENTS FOR PHYSIOLOGICAL VARIABLES DURING SUBMAXIMAL WORK AT 223 METERS, 2286 METERS AND 3810 METERS

ALL VALUES SHOWN FOR 30% AND 60% OF SEA LEVEL AEROBIC CAPACITY ARE THE MEAN VALUES
OBTAINED FROM THE 20TH THROUGH THE 45TH MINUTES OF EXERCISE
ALL 80% VALUES REPRESENT THE MEAN OBTAINED FROM THE 9TH MINUTE OF EXERCISE

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
$\dot{V}_E$ STPD liters/min.									
A	29.98	25.22	21.97	50.45	41.78	42.18	70.62	64.77	67.51
B	30.54	21.60	20.16	51.48	36.91	42.18	66.43	64.77	67.51
C	25.46	18.91	15.27	46.66	36.80	40.58	52.21	53.96	54.84
E	27.41	21.95	18.62	61.02	46.90	51.48	88.00	79.29	89.21
F	39.64	35.58	28.03	60.04	51.51	44.10	74.88	66.44	69.82
G	26.24	20.79	19.41	43.57	42.52	40.67	68.10	64.77	54.87
H	24.90	20.18	18.65	50.56	36.82	35.75	72.43	63.49	67.51
I	23.76	20.79	16.05	40.53	41.99	40.53	72.26	60.65	68.81
$\dot{V}_E$ BTPS liters/min.									
A	37.29	41.20	44.41	62.93	68.45	85.30	87.60	105.80	136.44
B	38.03	35.30	40.74	63.49	60.25	85.30	82.20	105.80	136.44
C	31.55	30.90	30.85	58.90	60.00	82.06	64.90	88.15	110.84
E	34.54	35.85	37.64	75.87	76.61	104.05	107.67	129.52	180.30
F	49.30	58.12	56.65	74.35	84.15	89.14	93.63	108.52	141.12
G	32.62	33.96	39.23	54.61	69.45	82.20	83.84	105.80	110.90
H	30.88	32.96	37.70	62.69	60.15	72.25	91.21	103.71	136.44
I	29.82	33.96	32.44	51.44	68.44	81.91	89.78	99.08	139.08

APPENDIX G--Continued

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m

Tidal Volume BTPS cc

A	1593	1470	1390	2202	2221	2639	2882	2961	3065
B	1494	1552	1405	1825	2032	2639	3162	2961	3065
C	1230	1163	1191	1567	1745	2870	2704	1695	3065
E	1510	1332	1387	3025	2128	2540	2991	3597	3756
F	2257	2157	2187	2986	2722	2920	2926	2713	2822
G	1213	1352	1457	1690	2052	2354	2329	2961	2520
H	1875	1746	1520	2737	2638	1912	3257	3704	3065
I	1428	1487	1622	2250	2232	2414	2805	3096	3161

Frequency, breaths/min.

A	23.50	28.25	34.00	30.50	31.50	32.50	30.00	37.00	46.00
B	25.50	23.33	29.25	36.00	29.75	32.50	26.00	37.00	46.00
C	25.75	26.75	26.00	39.00	36.50	31.50	24.00	52.00	46.00
E	23.00	27.00	27.25	32.75	36.00	41.00	36.00	36.00	48.00
F	22.00	27.00	26.00	25.00	31.00	30.75	32.00	40.00	50.00
G	27.00	25.25	27.00	33.00	34.00	35.00	36.00	37.00	44.00
H	16.50	19.00	27.25	23.00	23.00	26.00	28.00	28.00	46.00
I	21.00	23.00	20.00	23.00	31.00	34.00	32.00	32.00	44.00

APPENDIX G--Continued

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
$\dot{V}CO_2$ cc/min.									
A	879	847	743	1651	1658	1819	2441	2559	2818
B	922	815	850	1650	1565	1819	2790	2589	2818
C	638	656	578	1241	1255	1737	1644	1861	2018
E	814	752	818	2029	1871	2109	2877	3124	3497
F	1106	1272	1116	1962	1881	1890	2538	2850	2926
G	808	758	844	1398	1706	1733	2233	2589	2518
H	818	855	927	1656	1601	1620	2151	2381	2818
I	722	854	799	1626	1733	1825	2854	2729	3131
R									
A	.804	.806	.771	.795	.824	.879	.917	.961	1.033
B	.803	.720	.691	.796	.778	.879	1.003	.961	1.033
C	.669	.730	.676	.745	.851	.898	.902	.919	1.039
E	.759	.771	.816	.875	.850	.927	.932	1.013	1.037
F	.810	.866	.848	.763	.825	.884	.827	.937	1.051
G	.753	.752	.755	.768	.812	.849	.881	.961	1.008
H	.778	.774	.839	.822	.854	.878	.929	.964	1.033
I	.757	.785	.789	.798	.801	.838	.948	.973	1.032

APPENDIX G--Continued

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
Oxygen Pulse cc/beat									
A	9.78	8.90	7.38	13.00	12.35	11.88	15.20	15.20	15.90
B	11.90	10.53	9.75	14.93	12.93	11.88	15.50	15.10	15.90
C	7.48	6.70	5.98	9.38	8.20	8.68	10.60	11.20	15.90
E	10.65	10.00	8.63	13.98	14.40	12.63	18.80	17.50	18.30
F	11.95	11.80	10.83	15.08	13.52	12.30	16.50	17.30	14.10
G	8.60	8.95	7.88	11.70	11.80	11.40	14.60	15.10	14.20
H	9.28	8.90	8.58	11.88	12.20	10.75	12.70	13.40	15.90
I	10.55	9.80	9.00	14.23	13.55	13.15	17.70	16.30	16.90
Heart Rate - beats/min.									
A	112.00	118.00	131.60	160.40	163.60	174.20	175.00	177.00	184.00
B	96.20	107.00	125.40	138.40	154.40	174.20	180.00	177.00	184.00
C	130.40	134.20	142.20	178.60	180.00	173.80	172.00	180.00	184.00
E	99.40	97.20	116.00	167.40	152.80	180.20	164.00	176.00	184.00
F	114.20	120.80	123.20	170.40	168.80	173.60	186.00	176.00	198.00
G	126.40	112.80	142.80	156.40	178.60	179.80	173.00	177.00	176.00
H	113.80	124.80	129.00	169.40	155.00	174.00	182.00	184.00	184.00
I	89.80	111.20	112.80	144.00	159.20	166.40	170.00	172.00	180.00

APPENDIX G--Continued

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
Excess Lactic Acid - mg%									
A	17.30	2.40	14.30	33.70	52.90	51.90	53.50	69.80	31.60
B	0	0	0	10.70	11.70	34.90	55.30	54.50	50.20
C	0	3.50	12.70	44.30	47.70	75.20	82.40	87.60	97.90
E	0	0	0	14.50	15.40	58.40	65.00	70.60	93.00
F	0	8.00	0	0	10.10	41.50	42.10	8.50	84.70
G	0	0	0	11.00	28.60	59.80	48.50	75.70	75.40
H	0	0	6.40	25.40	13.20	41.70	59.70	64.00	63.30
I	0	5.40	12.20	6.90	13.20	40.90	66.30	59.00	88.50
Oxygen Extract - %									
A	3.51	4.01	4.22	3.95	4.65	4.82	3.70	4.11	4.08
B	3.61	4.95	5.72	3.86	5.21	4.82	4.19	4.11	4.08
C	3.59	4.50	5.24	3.38	3.92	4.71	3.42	3.69	3.57
E	3.68	4.23	5.18	3.72	4.55	4.36	3.46	3.90	3.81
F	3.33	4.04	4.56	4.08	4.27	4.73	3.95	4.52	4.03
G	3.89	4.61	5.46	3.98	4.75	4.86	3.63	4.11	4.56
H	4.03	5.22	5.74	3.84	4.94	5.05	3.15	3.86	4.08
I	3.81	5.00	6.05	4.82	4.95	5.20	4.12	4.60	4.44

APPENDIX G--Continued

	30% Max $\dot{V}O_2$			60% Max $\dot{V}O_2$			80% Max $\dot{V}O_2$		
Altitude	223m	2286m	3810m	223m	2286m	3810m	223m	2286m	3810m
Rectal Temperature - °C									
A	37.75	37.00	37.60	38.35	38.25	38.28	37.49	37.48	37.32
B	37.90	37.70	37.61	38.35	38.45	38.28	37.50	37.48	37.32
C	37.45	37.50	37.90	38.50	38.30	38.28	36.95	36.90	36.80
E	37.80	37.65	37.80	38.50	38.45	38.45	37.75	37.80	37.85
F	37.80	37.90	38.10	38.64	38.40	38.40	37.80	37.45	37.30
G	37.50	37.35	37.65	38.10	38.15	38.60	37.20	37.48	37.05
H	37.50	37.90	37.75	38.15	38.00	38.10	37.60	37.60	37.32
I	37.30	37.55	37.50	38.10	38.00	37.85	37.65	37.65	37.60
$\dot{V}_E/\dot{V}O_2$ STPD									
A	29.16	23.98	22.79	24.36	20.74	20.38	26.55	24.12	24.77
B	26.62	19.08	16.38	24.87	18.34	20.38	23.88	24.12	24.77
C	27.02	21.04	17.88	28.06	24.95	27.77	28.64	26.65	28.24
E	25.57	22.51	18.57	26.30	21.32	22.63	28.49	25.71	26.45
F	29.02	24.20	21.27	23.34	22.60	20.63	24.41	21.83	25.07
G	24.43	20.63	17.38	23.91	20.22	19.94	26.87	24.12	21.96
H	23.65	18.82	16.87	25.10	19.65	19.38	31.27	25.71	24.77
I	24.91	19.09	15.81	19.88	19.40	18.61	24.01	21.61	24.77

APPENDIX H

Each of the following tables summarizes the analysis of variance and Duncan multiple range test for each of the energy components. Lines connecting any two or more means indicate they are not significantly different from one another at the 5% level of significance.

Aerobic Power - kcal/kg.hr

Per Cent Aerobic Capacity	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3810m	
30	<u>2.919</u>	<u>2.935</u>	<u>2.924</u>	F (2,14) = 5.61 P < .01
60	<u>6.329</u>	<u>6.164</u>	<u>5.946</u>	
80	8.308	<u>7.671</u>	<u>7.208</u>	

Lactacid Power - kcal/kg.hr

Per Cent Aerobic Capacity	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3810m	
30	<u>.005</u>	<u>.006</u>	<u>.016</u>	F (2,14) = 18.13 P < .01
60	<u>.080</u>	<u>.114</u>	<u>.244</u>	
80	.457	<u>.744</u>	<u>.958</u>	

APPENDIX H--Continued

Alactacid Power - kcal/kg.hr

Per Cent Aerobic Capacity	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3810m	
30	<u>.064</u>	<u>.064</u>	<u>.069</u>	F (2,14) = 8.81 P < .01
60	<u>.111</u>	<u>.110</u>	<u>.195</u>	
80	<u>.378</u>	<u>.639</u>	<u>.715</u>	

Total Power - kcal/kg.hr

Per Cent Aerobic Capacity	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3810m	
30	<u>2.988</u>	<u>3.001</u>	<u>2.918</u>	F (2,14) = 0.43 N.S.
60	<u>6.354</u>	<u>6.553</u>	<u>6.385</u>	
80	<u>9.144</u>	<u>9.054</u>	<u>8.882</u>	

APPENDIX I

Each of the following tables summarizes the analysis of variance and Duncan multiple range test for the per cent contribution of each of the energy components to the total cost of work. Lines connecting any two or more means indicate they are not significantly different from one another at the 5% level of significance.

Relative Contribution of Aerobic Power
to the Total Power Input

Per Cent Aerobic Capacity	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3810m	
30	<u>97.66</u>	<u>97.46</u>	<u>97.12</u>	F (2,14) = 26.93 P < .01
60	<u>96.86</u>	<u>96.61</u>	<u>92.83</u>	
80	<u>90.37</u>	<u>85.00</u>	<u>80.68</u>	

Relative Contribution of Lactacid Power
to the Total Power Input

Per Cent Aerobic Capacity	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3810m	
30	<u>.180</u>	<u>.183</u>	<u>.549</u>	F (2,14) = 28.78 P < .01
60	<u>1.345</u>	<u>1.698</u>	4.000	
80	5.313	8.129	11.006	

APPENDIX I--Continued

Relative Contribution of Alactacid Power
to the Total Power Input

Per Cent Aerobic Capacity	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3810m	
30	<u>2.200</u>	<u>2.393</u>	<u>2.326</u>	F (2,14) = 9.78 P < .01
60	<u>1.791</u>	<u>1.695</u>	3.165	
80	4.320	<u>6.874</u>	<u>8.311</u>	

All percentage data were first normalized using an arcsine transformation.

APPENDIX J

Each of the following tables summarizes the analysis of variance and Duncan multiple range test results for the different variables. Lines connecting any two or more means indicate they are not significantly different from one another at the 5% level of significance.

Work at 30% of Aerobic Capacity

Variable	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3801m	
$\dot{V}_E$ l/min. STPD	28.49	23.13	19.77	F (2,14) = 84.09 P < .01
$\dot{V}_E$ l/min. BTPS	35.50	<u>37.78</u>	<u>39.96</u>	F (2,14) = 8.13 P < .01
f breaths/min.	23.03	<u>24.95</u>	<u>22.09</u>	F (2,14) = 4.49 P < .05
Tidal Volume BTPS, cc	<u>1575</u>	<u>1532</u>	<u>1520</u>	F (2,14) = .597 N.S.
O ₂ Pulse cc/beat	10.02	9.46	8.50	F (2,14) = 33.90 P < .01
Heart Rate beats/min.	<u>110.27</u>	<u>115.75</u>	127.87	F (2,14) = 15.90 P < .01
Rectal Temp. °C	<u>37.62</u>	<u>37.57</u>	<u>37.87</u>	F (2,14) = 1.44 N.S.
O ₂ Extraction Per Cent	3.67	4.57	5.28	F (2,14) = 74.08 P < .01
$\dot{V}_{CO_2}$ cc/min.	<u>.838</u>	<u>.851</u>	<u>.834</u>	F (2,14) = .1505 N.S.

APPENDIX J--Continued

Variable	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3801m	
R	<u>.767</u>	<u>.776</u>	<u>.773</u>	F (2,14) = .1502 N.S.
$\dot{V}_E/\dot{V}_{O_2}$ STPD liters/liter	26.30	21.10	18.37	F (2,14) = 164.40 P < .01
Lactic Acid mg %	<u>2.16</u>	<u>2.41</u>	<u>6.70</u>	F (2,14) = 2.90 N.S.

Work at 60% of Aerobic Capacity

Variable	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3810m	
$\dot{V}_E$ l/min. STPD	50.54	<u>41.90</u>	<u>42.18</u>	F (2,14) = 13.89 P < .01
$\dot{V}_E$ l/min. BTPS	<u>62.93</u>	<u>68.45</u>	85.28	F (2,14) = 39.17 P < .01
f breaths/min	<u>30.28</u>	<u>31.59</u>	<u>32.92</u>	F (2,14) = 1.26 N.S.
Tidal Volume BTPS, cc	<u>2285</u>	<u>2221</u>	<u>2536</u>	F (2,14) = 1.46 N.S.
O ₂ Pulse cc/beat	<u>13.02</u>	<u>12.37</u>	11.58	F (2,14) = 11.02 P < .01
Heart Rate beats/min.	<u>160.62</u>	<u>164.05</u>	174.52	F (2,14) = 5.23 P < .05

APPENDIX J--Continued

Variable	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3810m	
Rectal Temp. °C	<u>38.35</u>	<u>38.25</u>	<u>38.28</u>	F (2,14) = 1.05 N.S.
O ₂ Extraction Per Cent	3.95	<u>4.66</u>	<u>4.82</u>	F (2,14) = 25.21 P < .01
$\dot{V}_{CO_2}$ cc/min.	<u>1652</u>	<u>1659</u>	1819	F (2,14) = 5.24 P < .05
R	.795	.824	.879	F (2,14) = 22.25 P < .01
$\dot{V}_E/\dot{V}_{O_2}$ STPD liters/liter	24.48	<u>20.92</u>	<u>21.21</u>	F (2,14) = 18.36 P < .01
Lactic Acid mg %	<u>18.31</u>	<u>25.00</u>	50.54	F (2,14) = 35.13 P < .01

Work at 80% of Aerobic Capacity

Variable	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3810m	
$\dot{V}_E$ l/min. STPD	<u>70.62</u>	<u>64.76</u>	<u>67.51</u>	F (2,14) = 5.10 P < .05
$\dot{V}_E$ l/min. BTPS	87.60	105.80	136.44	F (2,14) = 81.40 P < .01
f breaths/min.	30.50	37.38	46.25	F (2,14) = 18.62 P < .01

APPENDIX J--Continued

Variable	Altitude			Analysis of Variance for Altitudes
	223m	2286m	3801m	
Tidal Volume BTPS, cc	<u>2282</u>	<u>2961</u>	<u>3065</u>	F (2,14) = .5364 N.S.
O ₂ Pulse cc/beat	<u>15.20</u>	<u>15.10</u>	<u>15.89</u>	F (2,14) = .7019 N.S.
Heart Rate beats/min.	<u>175.25</u>	<u>177.37</u>	184.25	F (2,14) = 8.19 P < .01
Rectal Temp. °C	<u>37.49</u>	<u>37.47</u>	37.32	F (2,14) = 5.56 P < .05
O ₂ Extraction Per Cent	3.70	<u>4.11</u>	<u>4.08</u>	F (2,14) = 9.16 P < .01
$\dot{V}_{CO_2}$ cc/min.	2441	2589	2818	F (2,14) = 15.47 P < .01
R	.917	.961	1.033	F (2,14) = 26.34 P < .01
$\dot{V}_E/\dot{V}_{O_2}$ STPD liters/liter	26.76	<u>24.23</u>	<u>24.84</u>	F (2,14) = 711 P < .01
Lactic Acid mg %	<u>59.10</u>	<u>70.34</u>	<u>73.07</u>	F (2,14) = 2.43 N.S.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Alexander, J. K., L. H. Hartely, M. Modelski, and R. Grover.
"Reduction of Stroke Volume During Exercise in Man Following Ascent to 3100 m. Altitude". J. Appl. Physiol. 23:849-858, 1967.
2. Asmussen, E., W. Döbeln and M. Nielsen. "Blood Lactate and Oxygen Debt After Exhaustive Work at Different Oxygen Tensions". Acta. Physiol. Scand. 15:57-62, 1948.
3. Astrand, P. O. and K. Rodahl. Textbook of Work Physiology. New York: McGraw-Hill Book Co., 1970.
4. Balke, B. "Work Capacity and its Limiting Factors at High Altitude". The Physiological Effects of High Altitude. Edited by W. H. Weike. New York: The Mac Millian Company, 1964.
5. Bay, E., E. S. G. Barron, W. Adams, T. Case, W. C. Halstead and H. T. Richetts. "The Behavior of Blood Lactate and Pyruvate with Exercise at Sea Level and at Altitude". C.A.M. Report No. 344, National Research Council, 1944.
6. Bert, Paul. Barometric Pressure. Translated by M. A. and F. A. Hitchcock. Columbus, Ohio: College Book Company, 1943.
7. Billings, C. E., C. R. Huie and M. F. Foley. "The Effect of Hypoxia on Performance of Muscular Work". Report No. 3. Aviation Medicine Research Laboratory, The Ohio State University, January, 1965.
8. Billings, C. E., J. R. Thomas and C. R. Huie. "The Effect of Hypoxia on Performance of Muscular Work". Report No. 2. Aviation Medicine Research Laboratory, The Ohio State University, August, 1964.
9. Billings, C. E. and R. Bason. "The Physiological Cost of Submaximal Work During Acute Exposure at Moderate Altitudes". In: The Effects of Physical Conditioning and Partial Acclimatization to Hypoxia on Work Tolerance at High Altitudes. Report No. 2002-4. The Ohio State University Research Foundation, June, 1968.

10. Bueding, E. and W. J. Goldfarb. "Blood Changes Following Glucose, Lactate, and Pyruvate Injections in Man". J. Biol. Chem. 147:33-40, 1943.
11. Buskirk, E. R. "Decrease in Physical Working Capacity at High Altitude". Biomedicine of High Terrestrial Elevations. Proceedings of a Symposium held at U. S. Army Research Institute of Environmental Medicine; Natick, Massachusetts, 16-17 October 1967. Edited by A. H. Hegnauer, USARIEM, January, 1969.
12. Buskirk, E. R., J. Kollias, R. F. Akers, E. K. Prokup, and E. P. Reategui. "Maximal Performance at Altitude and on Return From Altitude in Conditioned Runners". J. Appl. Physiol. 23:259-266, 1967.
13. Cerretelli, P. "Lactacid Oxygen Debt in Acute and Chronic Hypoxia". Exercise at Altitude. Edited by R. Margaria. New York: Excerpta Medica Foundation, 1967.
14. Consolazio, C. F., L. O. Matoush and R. A. Nelson. "Energy Metabolism in Maximum and Submaximum Performance at High Altitude". Fed. Proc. 25:1380-1385, 1966.
15. Consolazio, C. F., R. A. Nelson, L. O. Matoush and J. E. Hansen. "Energy Metabolism at High Altitude (3,475 m.)". J. Appl. Physiol. 21:1732-1740, 1966.
16. Cronin, R. F. P. and D. J. MacIntosh. "The Effect of Induced Hypoxia on Oxygen Uptake During Muscular Exercise in Normal Subjects". Canad. J. Biochem. and Physiol. 40:717-726, 1962.
17. Day, J. J. and M. W. Lippett, Jr. "A Long Term Electrode System for Electrocardiography and Impedance Pneumography". Psychophysiology. 1:174-182, 1964.
18. Dejours, P., R. H. Kellogg and N. Pace. "Regulation of Respiration and Heart Rate During Altitude Acclimatization". J. Appl. Physiol. 18:10-18, 1963.
19. Dill, D. B. Life, Heat and Altitude. Cambridge: Harvard University Press, 1938.
20. Dill, D. B., H. T. Edwards, A. Fölling, S. A. Oberg, A. M. Pappenheimer, Jr., and J. H. Talbott. "Adaptations of the Organism to Changes in Oxygen Pressure". J. Physiol. 71:47-63, 1931.
21. Dill, D. B., L. G. Myhre, D. K. Brown, K. Burrus and G. Gehlsen. "Work Capacity in Chronic Exposure to Altitude". J. Appl. Physiol. 23:555-560, 1967.

22. Dill, D. B., L. G. Myhre, E. E. Phillips, Jr., D. K. Brown. "Work Capacity in Acute Exposure to Altitude". J. Appl. Physiol. 21:1168-1176, 1966.
23. Dixon, W. J. BMD Biomedical Computer Programs. Los Angeles: University of California Press, 1967.
24. Durand, J., C. L. Pannier, J. De Lattre, J. P. Martineaud and J. M. Verpillat. "The Cost of the Oxygen Debt at High Altitude". Exercise at Altitude. Edited by R. Margaria. New York: Excerpta Medica Foundation, 1967.
25. Edelman, I. S. and J. Liebman. "Anatomy of Water and Electrolytes". Amer. J. Med. 27:256-277, 1959.
26. Edwards, H. T. "Lactic Acid in Rest and Work at High Altitudes". Amer. J. Physiol. 116:367-375, 1936.
27. Farhi, L. E. and H. Rahm. "Gas Stores of the Body and the Unsteady State". J. Appl. Physiol. 7:472-484, 1954-1955.
28. Favour, C. B. "Lactic Acid, Fitness and Altitude". The International Symposium on the Effects of Altitude on Physical Performance. Chicago: The Athletic Institute, 1967.
29. Freund, J. E., P. E. Livermore and I. Miller. Manual of Experimental Statistics. Edited by Albert A. Bennett. New Jersey: Prentice-Hall, Inc., 1960.
30. Friedman, T. E. and A. C. Ivy. "Work at High Altitude. III. The Production of Lactic and Pyruvic Acid During Work at Ground Level and at Increasing Altitudes to 15,000 Feet". Northwestern University Medical School, Quarterly Bulletin. 21:228-237, 1947.
31. Friedman, T. E., G. E. Haugen and T. C. Kmiecik. III. "The Level of Pyruvic and Lactic Acids and the Lactic Pyruvate Ratio in the Blood of Human Subjects. The Effects of Food, Light Muscular Activity and Anoxia at High Altitude". J. Biol. Chem. 157:673-689, 1945.
32. Grimby, G., N. J. Nilsson and H. Sanne. "Repeated Serial Determinations of 30 Minute Exercise". J. Appl. Physiol. 21:1750-1756, 1966.
33. Hansen, J. E., G. R. Selzer and J. A. Vogel. "Arterial Pyruvate, Lactate, pH and P_{CO_2} During Work at Sea Level and High Altitude". J. Appl. Physiol. 23:523-530, 1967.

34. Hansen, J. E., J. A. Vogel, G. P. Stelter and C. F. Consolazio. "Oxygen Uptake in Man During Exhaustive Work at Sea Level and High Altitude". J. Appl. Physiol. 23:511-522, 1967.
35. Harboe, M. "Lactic Acid Content in Human Venous Blood During Hypoxia at High Altitude". Acta. Physiol. Scand. 40:248-253, 1957.
36. Hartely, L. H., J. K. Alexander, M. Modelski and R. Grover. "Subnormal Cardiac Output at Rest and During Exercise in Residents at 3,100 m. Altitude". J. Appl. Physiol. 23:839-848, 1967.
37. Henry, F. M. and J. C. DeMoor. "Lactic and Alactic Oxygen Consumption in Moderate Exercise of Graded Intensity". J. Appl. Physiol. 8:608-614, 1956.
38. Henry, R. J. Clinical Chemistry: Principles and Techniques. Hoeber Medical Division, Harper and Row, 1964.
39. Hermansen, L. and B. Saltin. "Blood Lactate Concentration During Exercise at Acute Exposure to Altitude". Exercise at Altitude. Edited by R. Margaria. New York: Excerpta Medica Foundation, 1967.
40. Himwich, H. E. "The Role of Lactic Acid in the Living Organism". Yale J. Biol. Med. 4:259-281, 1931-1932.
41. Huckabee, W. E. "Relationships of Pyruvate and Lactate During Anaerobic Metabolism. I. Effects of Infusion of Pyruvate or Glucose and of Hyperventilation". J. Clin. Invest. 37:244-254, 1958.
42. Hultgren, H. N., J. Kelly and H. Miller. "Pulmonary Circulation in Acclimatized Man at High Altitude". J. Appl. Physiol. 20:233-238, 1965.
43. Karlsson, J. and B. Saltin. "Lactate, ATP, and CP in Working Muscles During Exhaustive Exercise in Man". J. Appl. Physiol. 29:598-602, 1970.
44. Klausen, K. "Cardiac Output in Man in Rest and During and After Acclimatization to 3,800 m." J. Appl. Physiol. 21:609-616, 1966.
45. Klausen, K., S. Robinson, E. D. Micahel and L. G. Myhre. "Effect of High Altitude on Maximal Working Capacity". J. Appl. Physiol. 21:1191-1194, 1966.
46. Knuttgen, H. G. "Oxygen Debt, Lactate, Pyruvate, and Excess Lactate After Muscular Work". J. Appl. Physiol. 17:639-644, 1962.

47. Lorentzen, F. V. "Lactic Acid in Blood After Various Combinations of Exercise and Hypoxia". J. Appl. Physiol. 17:661-664, 1962.
48. Lundin, G. and G. Strom. "The Concentration of Blood Lactic Acid in Man During Muscular Work in Relation to the Partial Pressure of Oxygen of the Inspired Air". Acta. Physiol. Scand. 13:253-266, 1947.
49. Margaria, R., H. T. Edwards and D. B. Dill. "The Possible Mechanism of Contracting and Paying the Oxygen Debt and the Role of Lactic Acid in Muscular Contraction". Amer. J. Physiol. 106:689-715, 1933.
50. Margaria, R. "Aerobic and Anaerobic Energy Sources in Muscular Exercise". Exercise at Altitude. Edited by R. Margaria. New York: Excerpta Medica Foundation, 1967.
51. Margaria, R., P. Cerretelli and F. Mangile. "Balance and Kinetics of Anaerobic Energy Release During Strenuous Exercise in Man". J. Appl. Physiol. 19:623-628, 1964.
52. Margaria, R., P. Cerretelli, P. E. Di Prompero, C. Massari and G. Torelli. "Kinetics and Mechanism of Oxygen Debt Contraction in Man". J. Appl. Physiol. 18:371-377, 1963.
53. Moncloa, F., A. Carcelen and L. Beteta. "Physical Exercise Acid-Base Balance and Adrenal Function in Newcomers to High Altitude". J. Appl. Physiol. 28:151-155, 1970.
54. Naimark, A., N. L. Jones and S. Lal. "The Effect of Hypoxia on Gas Exchange and Arterial Lactate and Pyruvate Concentration During Moderate Exercise in Man". Clin. Sci. 28:1-13, 1965.
55. Pugh, L. G. C. E., M. B. Gill, S. Lahiri, J. S. Milledge, M. P. Ward, and J. B. West. "Muscular Exercise at Great Altitudes". J. Appl. Physiol. 19:431-440, 1964.
56. Reeves, J. T., R. F. Grover and J. E. Cohn. "Regulation of Ventilation During Exercise at 10,200 Feet in Athletes Born at Low Altitude". J. Appl. Physiol. 22:546-554, 1967.
57. Saltin, B. and J. Stenberg. "Circulatory Response to Prolonged Severe Exercise". J. Appl. Physiol. 19:833-838, 1964.
58. Schilling, J. A., R. B. Harvey, E. L. Becker, T. Velasquez, G. Wells and B. Balke. "Work Performance at Altitude After Adaptation in Man and Dog". J. Appl. Physiol. 8:381-387, 1955-1956.

59. Stenberg, J., B. Ekblom and R. Messin. "Hemodynamic Response to Work at Simulated Altitude, 4,000 m." J. Appl. Physiol. 21:1589-1594, 1966.
60. Tepperman, J. and H. M. Tepperman. "On the Blood Lactic Acid Response to Measured Exercise in Hypoxic Human Subjects". J. Clin. Inves. 27:176-186, 1948.
61. Terzioglu, M., F. Özer and N. Gokhan. "Variation in Gaseous Content and Acid-Base Equilibrium of Arterial Blood at 1.85 km Altitude". J. Appl. Physiol. 6:423-428, 1953-54.
62. Vogel, J., J. Hansen and C. Harris. "Cardiovascular Response in Man During Exhaustive Work at Sea Level and High Altitude". J. Appl. Physiol. 23:531-539, 1967.
63. Wasserman, K., G. G. Burton and A. L. Van Kessel. "Excess Lactate Concept and Oxygen Debt of Exercise". J. Appl. Physiol. 20:1299-1306, 1965.
64. West, J. B. "Gas Diffusion in the Lung at Altitude". Exercise at Altitude. Edited by R. Margaria. New York: Excerpta Medica Foundation, 1967.