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The effects of mild, obesity-induced arterial hypoxemia on the anaerobic threshold.

Lynne J. Stevensen
University of Windsor

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THE EFFECTS OF MILD, OBESITY-INDUCED ARTERIAL HYPOXEMIA ON THE ANAEROBIC THRESHOLD

by

Lynne J. Stevensen

A Thesis Submitted to the Faculty of Graduate Studies Through the Faculty of Human Kinetics in Partial Fulfillment of the Requirements for the Degree of Master of Human Kinetics at The University of Windsor

Windsor, Ontario, Canada 1978
ACKNOWLEDGEMENTS

The author is indebted to Dr. P. Taylor, Dr. J. Leavitt, Dr. R. Hermiston, Dr. Strauss, M.D., and especially to Dr. F. Cerny for guidance and assistance throughout the investigation.

Special thanks are extended to Pete Lemon and other fellow students in the Faculty of Human Kinetics whose assistance was invaluable.

This paper is dedicated to my mother to whom I owe everything.
ABSTRACT

In seven obese subjects, four of whom developed mild exercise hypoxemia (PaO₂ < 80 mmHg), we studied exercise induced metabolic acidosis: the workload at which it occurred during a progressive exercise test (AT) and the effect of mild hypoxemia on the AT. The mean AT was 243 kpm during normoxic and hyperoxic test conditions. This value falls below those reported by Wasserman, et. al., (J.A.P. 35:236, 1973) for a reference population (275 - 1200 kpm) suggesting a limited physical work capacity. Since the AT of hypoxemic subjects did not increase with 100% oxygen, it is not likely that hypoxia contributed to the early onset of metabolic acidosis. The lack of an experimental effect was thought to be due to the mild degree of arterial hypoxemia and/or the complexity of lactic acid production.
<table>
<thead>
<tr>
<th>TABLE OF CONTENTS</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACKNOWLEDGEMENTS</td>
<td>iii</td>
</tr>
<tr>
<td>ABSTRACT</td>
<td>iv</td>
</tr>
<tr>
<td>LIST OF TABLES</td>
<td>vi</td>
</tr>
<tr>
<td>LIST OF FIGURES</td>
<td>vii</td>
</tr>
<tr>
<td>CHAPTER</td>
<td></td>
</tr>
<tr>
<td>I. INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>Hypothesis</td>
<td>5</td>
</tr>
<tr>
<td>II. METHODS</td>
<td>6</td>
</tr>
<tr>
<td>Subjects</td>
<td>6</td>
</tr>
<tr>
<td>Preliminary Testing</td>
<td>8</td>
</tr>
<tr>
<td>Experimental Testing</td>
<td>10</td>
</tr>
<tr>
<td>Analysis of Data</td>
<td>12</td>
</tr>
<tr>
<td>III. RESULTS</td>
<td>13</td>
</tr>
<tr>
<td>Preliminary Testing</td>
<td>13</td>
</tr>
<tr>
<td>Resting Acid-Base</td>
<td>16</td>
</tr>
<tr>
<td>Exercise Studies</td>
<td>16</td>
</tr>
<tr>
<td>IV. DISCUSSION</td>
<td>29</td>
</tr>
<tr>
<td>V. SUMMARY AND CONCLUSIONS.</td>
<td>38</td>
</tr>
<tr>
<td>Experimental Design</td>
<td>38</td>
</tr>
<tr>
<td>Results and Discussion</td>
<td>38</td>
</tr>
<tr>
<td>Conclusion.</td>
<td>39</td>
</tr>
<tr>
<td>BIBLIOGRAPHY</td>
<td>40</td>
</tr>
<tr>
<td>APPENDIX A</td>
<td>46</td>
</tr>
<tr>
<td>APPENDIX B</td>
<td>48</td>
</tr>
<tr>
<td>APPENDIX C</td>
<td>49</td>
</tr>
<tr>
<td>VITA AUCTORIS</td>
<td>65</td>
</tr>
<tr>
<td>TABLE</td>
<td>PAGE</td>
</tr>
<tr>
<td>-------</td>
<td>------</td>
</tr>
<tr>
<td>1</td>
<td>Physical Characteristics of Control and Experimental Subjects</td>
</tr>
<tr>
<td>2</td>
<td>Resting Pulmonary Functions</td>
</tr>
<tr>
<td>3</td>
<td>Preliminary Testing</td>
</tr>
<tr>
<td>4</td>
<td>Exercise Response Under Normoxic Conditions</td>
</tr>
<tr>
<td>5</td>
<td>Acid-Base Response Under Normoxic Conditions</td>
</tr>
<tr>
<td>6</td>
<td>Exercise Response Under Hyperoxic Conditions</td>
</tr>
<tr>
<td>7</td>
<td>Acid-Base Response Under Hyperoxic Conditions</td>
</tr>
<tr>
<td>8</td>
<td>Exercise Response at the Anaerobic Threshold Under Normoxic and Hyperoxic Conditions</td>
</tr>
<tr>
<td>9</td>
<td>One-Way Analysis of Variance for Comparison of Physical Characteristics of Control and Experimental Groups</td>
</tr>
<tr>
<td>10</td>
<td>Analysis of Variance for Comparison of Exercise Response of Control and Experimental Groups Under Normoxic and Hyperoxic Conditions</td>
</tr>
<tr>
<td>11</td>
<td>Analysis of Variance for Comparison of Acid-Base Response of Control and Experimental Groups Under Normoxic and Hyperoxic Conditions</td>
</tr>
<tr>
<td>12</td>
<td>Analysis of Variance for Comparison of Exercise Response of Control and Experimental Groups at the Anaerobic Threshold Under Normoxic and Hyperoxic Conditions</td>
</tr>
<tr>
<td>13</td>
<td>One-Way Analysis of Variance With Repeated Measures for Comparison of Normoxic and Hyperoxic Exercise Response of Control and Experimental Groups</td>
</tr>
<tr>
<td>14</td>
<td>One-Way Analysis of Variance with Repeated Measures for Comparison of Normoxic and Hyperoxic Exercise Response of Control and Experimental Groups at the Anaerobic Threshold</td>
</tr>
</tbody>
</table>
## LIST OF FIGURES

<table>
<thead>
<tr>
<th>FIGURE</th>
<th>DESCRIPTION</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Response of $\dot{V}O_2$, $\dot{V}CO_2$, and RE to Increasing Levels of Work Under Normoxic Conditions</td>
<td>20</td>
</tr>
<tr>
<td>2</td>
<td>Response of $V_E$, HR and LA to Increasing Levels of Work Under Normoxic and Hyperoxic Conditions</td>
<td>21</td>
</tr>
<tr>
<td>3</td>
<td>Acid-Base Response to Increasing Levels of Work Under Normoxic and Hyperoxic Conditions</td>
<td>22</td>
</tr>
</tbody>
</table>
CHAPTER 1

INTRODUCTION

Studies have shown the work capacity of obese subjects to be significantly less than that of a reference population (14, 26, 50). Most submaximal loads are thus performed at a level requiring a substantial fraction of their maximal aerobic power ($\dot{V}O_2$ max). Since workloads which require 50 - 60 percent $\dot{V}O_2$ max are associated with increased anaerobic metabolism and a subsequent accumulation of blood lactic acid, these individuals incur exercise-induced metabolic acidosis at low levels of work (5, 49, 37).

In the past, obesity has been associated with numerous physiological disturbances capable of impairing cardiorespiratory function which may contribute to the reduced capacity for aerobic work. It has been demonstrated that large fat masses, if distributed on the chest wall and/or abdomen, may interfere with the mechanics of ventilation (7, 16, 25, 54), similar to the chest restriction caused by chest strapping (15). In this case, a rapid shallow breathing pattern is often adapted in an attempt to minimize the cost ($\dot{V}O_2$) of breathing (moving air). Consequences of this type of breathing pattern include uneven distribution of ventilation, atelectasis and/or shunting, all of which may decrease effective surface area for gas exchange (23, 40.
The disturbances in alveolar to arterial gas exchange, in turn, may or may not be reflected in varying degrees of arterial hypoxemia (PaO₂ < 80 mmHg) at rest. During exercise, however, in the face of a fall in mixed venous O₂ saturation and red cell transit time, it is possible that arterial oxygen tensions will fall below expected values (20, 23, 25) even in obese individuals with normal "resting" blood gases. Arterial hypoxemia, if severe enough, could limit O₂ transport thereby interfering with adequate tissue oxygenation.

Although the physiological consequences of an O₂ deficiency are complex, an imbalance between O₂ supply and demand would necessitate increased energy expenditure via anaerobic glycolysis if work is to continue (37, 41, 45, 72). Under these conditions the obese would experience an early onset of metabolic acidosis accounting, in part, for the decreased aerobic power.

It has been suggested that by identifying the work load at which an individual shifts from the aerobic to anaerobic (oxidative to glycolytic) systems during a progressive exercise test, one could indirectly obtain an estimate of the maximal aerobic power. Knowing that the anaerobic threshold (AT) is reached at about 50 to 60 percent of VO₂ max, one can associate a low AT, in terms of workload and/or VO₂, with a low VO₂ max (70). Thus, assessment of this AT, defined as the load just below that at which metabolic acidosis occurs, would give an indication of whether or not aerobic power is limited without pushing subjects to heavy loads.
In the past, the primary methods of detecting exercise-induced metabolic acidosis relied on measuring changes in blood acid-base status or lactic acid, necessitating arterial or venous sampling. More recently, Wasserman and coworkers (70) have cited evidence that alterations in respiratory gas exchange occur concurrently with the onset of metabolic acidosis, allowing non-invasive detection of the AT. The response of ventilatory parameters to changes in the acid-base status associated with lactic acidosis are secondary to a leftward shift in the bicarbonate buffering system

\[
\text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^- \]

The increased \( \text{CO}_2 \) reaching the lungs provides an additional ventilatory stimulus which becomes manifest in increases in not only \( \dot{V}_E \), but in the volume of \( \text{CO}_2 (\dot{V}\text{CO}_2) \) eliminated, and in the respiratory gas exchange ratio (RE) as \( \dot{V}\text{CO}_2 \) increases out of proportion to increases in \( \dot{V}_O_2 \) (70, 72). As a result of these physiological interrelationships, it has become evident that the AT can be identified by the point of either a non-linear increase in 1) \( \dot{V}_E \), 2) \( \dot{V}\text{CO}_2 \), and/or 3) RE as work rate is increased during an incremental exercise test (70).

Although the use of ventilatory parameters to detect the onset of metabolic acidosis allows for more widespread use of the AT test, its application may be limited due to several problems. One problem involves the frequently observed pulmonary patient with abnormal ventilatory control. In these patients, ventilation may increase with no associated increase in lactic acid. In addition, a theoretical question arises when considering the physiological basis on which the

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The concept of the AT was developed. For many years, it was customary to attribute lactic acid production to anaerobic glycolysis secondary to tissue hypoxia (37, 75). However, considerable evidence has now accumulated that the onset and control of anaerobic metabolism is more complex than can be explained completely by inadequate tissue oxygenation (41, 45, 75). If metabolic acidosis is dependent on factors other than oxygen supply, the AT concept becomes less simplistic; the appearance or non-appearance of an AT could be determined by many complex factors and the AT might lack the specificity required to justify its use in assessing physical work capacity.

From the above discussion, it appears that there is a need for more study of and information concerning the concept and measurement of the AT. By determining the effects of arterial hypoxemia on the AT, it was felt that the role of hypoxia in anaerobic metabolism as well as the usefulness of the AT test in specific circumstances might be ascertained. Since severe obesity may be associated with arterial hypoxemia, these individuals provide a model for the study of the problem. The purpose of this study was to assess the AT of obese subjects and compare these values with results from a reference population. More importantly, the study was designed to compare the AT of each subject during 1) room air (FIO₂ = .208) and 2) high oxygen (FIO₂ = 1.00) breathing to see if the removal of even mild hypoxemia would result in an increase in the AT.
Hypothesis

It was hypothesized that the AT of obese subjects would fall below values assessed in a reference population. Wasserman and co-workers (70) have identified 275 kpm as the lower limit of AT in their group (N = 85), and thus the AT of obese subjects was expected to fall below this value. Since concern was given to the AT of obese subjects regardless of their PaO2, all seven subjects were grouped, the mean AT calculated and compared to the above reference values.

It was further hypothesized that the AT measured in obese, hypoxemic (PaO2 < 80 mmHg) subjects during room air breathing would be lower than values obtained while subjects inspired 100% O2. If, during a progressive exercise test, the onset of metabolic acidosis is premature due to hypoxemia-induced tissue hypoxia, correcting for the hypoxemia would delay anaerobiosis, except in the presence of large right-to-left shunts. An increase in the AT during high O2 breathing would indeed suggest that under normoxic exercise conditions arterial hypoxemia was causing inadequate tissue oxygenation resulting in an early production of acidosis. In this case, the observed hypoxemia could be implicated as a possible contributing factor in the decreased aerobic work capacity.
CHAPTER II

METHODS

It was hypothesized that 1) the AT of obese subjects would be lower than values assessed in a reference population, and 2) the AT of mildly hypoxemic subjects would be higher during a progressive exercise test performed under hyperoxic as compared to normoxic conditions.

To test the hypothesis, the AT's of seven obese subjects were determined using an incremental work test in which the initial work rate consisted of four minutes of pedaling on an unloaded (0 kpm) calibrated Monark Bicycle Ergometer (Quinton Model #850); following which, the work rates were increased 100 kpm every two minutes. Subjects were studied on two separate occasions, the first for preliminary testing and at least a week later for experimental purposes. On day two, the subjects performed two exercise tests; the first under normoxic conditions, the second while inspiring 100% oxygen.

Subjects

Six female and one male obese (♀ > 30%, ♂ > 25% body fat) volunteer subjects were studied at rest and during exercise tests of progressively increasing work-loads breathing ambient air and 100% O₂. The physical characteristics of both groups are listed in Table 1. Of the seven subjects, four developed
### TABLE 1

**PHYSICAL CHARACTERISTICS OF CONTROL AND EXPERIMENTAL SUBJECTS**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Body Weight (kg)</th>
<th>Body Fat (%)</th>
<th>Fat Weight (kg)</th>
<th>Fat free Weight (kg)</th>
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<tbody>
<tr>
<td><strong>Control Group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PN</td>
<td>32.0</td>
<td>163.0</td>
<td>96.0</td>
<td>50.2</td>
<td>48.0</td>
<td>48.0</td>
</tr>
<tr>
<td>DD</td>
<td>50.0</td>
<td>175.3</td>
<td>106.1</td>
<td>33.6</td>
<td>67.0</td>
<td>39.2</td>
</tr>
<tr>
<td>AG</td>
<td>23.0</td>
<td>168.0</td>
<td>91.0</td>
<td>46.5</td>
<td>42.3</td>
<td>48.7</td>
</tr>
<tr>
<td>Mean</td>
<td>35.0</td>
<td>168.7</td>
<td>97.7</td>
<td>43.4</td>
<td>52.4</td>
<td>45.3</td>
</tr>
<tr>
<td>S.D.</td>
<td>13.8</td>
<td>6.0</td>
<td>7.6</td>
<td>8.7</td>
<td>12.9</td>
<td>5.3</td>
</tr>
<tr>
<td><strong>Experimental Group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BC</td>
<td>32.0</td>
<td>178.0</td>
<td>132.0</td>
<td>54.0</td>
<td>71.0</td>
<td>61.0</td>
</tr>
<tr>
<td>JK</td>
<td>37.0</td>
<td>165.0</td>
<td>137.3</td>
<td>58.0</td>
<td>79.6</td>
<td>57.7</td>
</tr>
<tr>
<td>JP</td>
<td>33.0</td>
<td>170.0</td>
<td>110.7</td>
<td>48.0</td>
<td>53.0</td>
<td>58.0</td>
</tr>
<tr>
<td>JH</td>
<td>48.0</td>
<td>168.0</td>
<td>121.0</td>
<td>48.3</td>
<td>58.0</td>
<td>63.0</td>
</tr>
<tr>
<td>Mean</td>
<td>37.5</td>
<td>170.3</td>
<td>125.3*</td>
<td>54.1</td>
<td>65.4</td>
<td>59.8*</td>
</tr>
<tr>
<td>S.D.</td>
<td>7.6</td>
<td>5.6</td>
<td>11.8</td>
<td>4.8</td>
<td>12.1</td>
<td>2.5</td>
</tr>
</tbody>
</table>

* Significant difference from control group P < 0.05
# Significant difference from control group P < 0.01
mild hypoxemia \( \text{PaO}_2 < 80 \text{ mmHg} \), during the exercise.

**Day One -- Preliminary Testing**

Prior to testing, all subjects were informed of testing procedures and asked to read and sign a prepared informed consent (Appendix A). Preliminary tests were then administered. These included pulmonary functions, underwater weighing and an incremental exercise test. The exercise test allowed an initial evaluation of subjects and, being similar to tests to be performed on day two also served to familiarize subjects with the experimental testing procedures.

**Pulmonary Function Testing**

Pulmonary function tests using a 13.5 Liter Collins Spirometer (Model #P-1300) were performed in duplicate, the best effort being selected, corrected to BTPS and expressed as a percentage of the documented reference standards by Anderson (4), Baldwin (8), Grimby (38), Kory (46) and Morris (53).* The lung volumes which were measured included inspiratory capacity (IC), expiratory reserve volume (ERV) and vital capacity (VC). Using gas dilution methods, four subjects were tested for functional residual capacity (FRC) allowing calculation of residual volume (RV) and total lung capacity (TLC). In addition, resting tidal volume \( V_T \), frequency \( f \) and expired minute ventilation \( \dot{V}_E \) were determined. The dynamic ventilatory tests administered included forced expiratory volume in one second as a percent of FVC (FEV\(_1\)%), and maximal ventilatory volume (MVV).

*These were selected because the population was most similar to this population.

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Body Composition (Percent Body Fat)

Body volume was determined using an underwater weighing technique which included correcting for residual air (12, 30). Knowing the specific gravity of the body, proportionate masses of fat and nonfat components based on the formula established by Brozek (Total fat = $\frac{4.570 - 4.12}{\text{Body Density}}$) were then calculated (12).

Exercise Test

Following the underwater weighing, a standard incremental exercise test was administered to all subjects. The test consisted of continuous pedaling on a calibrated bicycle ergometer at 50 revolutions per minute. After four minutes of unloaded (0 kpm) pedaling, work rates were increased by 100 kpm every minute until the highest tolerable load was reached. Maximum work capacity was defined as that load at which the subject could no longer maintain the required pedaling frequency.

While exercising, subjects breathed through a low resistance two-way valve ($V_D = 50$ ml) allowing collection of expired air in a 350 Liter Collins Tissot (Model #P-1800). Minute ventilation was recorded, corrected for BTPS and plotted against workloads. The point at which the $V_E$-workload curve became non-linear (where $V_E$ began to increase disproportionately to increases in load) was identified as the AT (70).

The electrocardiogram (ECG) (C5 - C5R) was continuously monitored on an oscilloscope. Heart rate (HR) and blood pressure (BP) were recorded during each minute of rest, exercise and recovery. Blood pressures were taken 30 seconds into each minute, heart rates the final 5 - 10 seconds.
Day Two — Experimental Testing

On the second day of testing, subjects performed two standard incremental exercise tests with an arterial catheter in a radial artery. The first test was done while breathing room air ($F_{O_2} = 0.208$) and the second while inspiring humidified 100% $O_2$. These tests were identical to the preliminary exercise test except that subjects pedaled for two rather than one minute at each workload and did not continue to volitional fatigue. The AT was identified from an increase in ventilation, as described earlier, and in blood lactic acid levels during the test.

Schedule

Prior to testing a number 25 angiocatheter was inserted percutaneously under a local anesthetic ($\sim 0.5$ of 1% cc of lidocaine) into a radial artery for blood sampling. After a rest period, test one was begun and proceeded until subjects exceeded their predetermined AT (Day One). Subjects then breathed 100% humidified $O_2$ at rest for 15 minutes to insure nitrogen washout of the lungs. While continuing high $O_2$ breathing, subjects then repeated the exercise test, this time exercising to maximal levels.

Measurements

During all stages of both tests, including rest and recovery, ventilatory, acid-base and circulatory measurements were made. As on day one, expired gas was collected for determination of minute ventilation. During the final minute of each stage, a two liter mixed sample was collected and
immediately analyzed for \( O_2 \) and \( CO_2 \) tensions using Godart Rapox and Capnograph Gas Analyzers, calibrated with Gallenkampfed gases. Along with \( \dot{V}_E \), \( O_2 \) and \( CO_2 \) were used to calculate \( \dot{V}CO_2 \), \( \dot{VC}_O_2 \) and RE (Appendix B).

Simultaneous with the collection of expired gas samples, about 7 cc of arterial blood was drawn under anaerobic conditions and immediately analyzed for pH, \( PaCO_2 \) and \( PO_2 \) with an Ultramicro \( pH/PCO_2/PO_2 \) blood gas system (IL Model #113). \( HCO_3^- \) was calculated using a Severinghaus blood gas calculator (65). In addition, a portion of each sample was deproteinized using 9% TCA and subsequently analyzed for lactic acid (L.A.) concentrations using enzymatic techniques (34). Hematocrit (H) and hemoglobin (Hb) were also determined, the latter using a Coleman Junior Spectrophotometer and the cyanmethemoglobin technique.

Heart rate was recorded the final 10 seconds of every minute of exercise and recovery on a Hewlett Packard Recorder (Model #1500B) by means of a 3 lead ECG (C5 – C5R). The ECG was constantly monitored on an oscilloscope. Blood pressure was recorded during the last 30 seconds of rest and recovery periods and the first minute of each work stage.

The size of right-to-left shunts were determined from the response of \( PaO_2 \) to 100% \( O_2 \) inhalation at rest. Each 20 mmHg of \( PO_2 \) below 670 mmHg was considered to be equivalent to a 1% shunt (42).
Analysis of Data

To analyze for significant differences in the AT and exercise response between test conditions a one-way ANOVA with repeated measures using a Canon Programmable Calculator (Model XX-310) was selected with each subject acting as his own control. The XX-310 was also used to perform a one-way ANOVA for comparison between groups and to determine means, standard deviations and standard error.
CHAPTER III

RESULTS

To determine if obesity induced hypoxemia precipitates tissue hypoxia and thus restricts work capacity, seven obese subjects, four of whom developed mild exercise hypoxemia, were studied. Subjects performed two standard incremental work tests; the first while breathing room air, the second while inspiring 100% O₂. A comparison of the results revealed 1) that the AT was decreased in obese subjects as compared to reference values and 2) that no change occurred as a result of breathing 100% O₂ in either the control or experimental group.

Preliminary Testing

The obese subjects were divided into two groups. Subjects whose resting and/or exercise PaO₂ fell below 80 mmHg were considered mildly hypoxemic and placed in the experimental group. The control group consisted of those subjects whose PaO₂ remained at or above 80 mmHg. The experimental group was significantly heavier (P < 0.05) and had a greater lean body mass (P < 0.01) than the control subjects (Table 1).

The individual and group results from the pulmonary function and exercise tests performed on day one are listed in Table 2 and 3. Mean values for IC, ERV, VC, FRC and TLC in obese subjects were reduced, the latter three being 91, 91 and 90% of predicted values, respectively. In contrast,
<table>
<thead>
<tr>
<th></th>
<th>IC ml</th>
<th>ERV ml</th>
<th>VC ml</th>
<th>FRC ml</th>
<th>RV ml</th>
<th>TLC ml</th>
<th>FRC/TLC %</th>
<th>( V_T ) ml</th>
<th>( V_E ) l/min</th>
<th>FEV (_1) %</th>
<th>MVV ml/min</th>
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<tr>
<td>% Predicted</td>
<td></td>
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<td>2158</td>
<td>1919</td>
<td>5565</td>
<td>39%</td>
<td>600</td>
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<td>12.1</td>
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<tr>
<td>% Predicted</td>
<td>81%</td>
<td>32%</td>
<td>122%</td>
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<td>2782</td>
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<td>1810</td>
<td>1374</td>
<td>5476</td>
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<td>837</td>
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<td>103%</td>
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<td>769</td>
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<td>5052</td>
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<tr>
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<td>2073</td>
<td>709</td>
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<td>942</td>
<td>4597</td>
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<td>800</td>
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<td>Test</td>
<td>Right-to-Left Shunt</td>
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<td>%</td>
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<tr>
<td>Mean</td>
<td>700 kpm</td>
<td>267 kpm</td>
<td>5.8</td>
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<tr>
<td>S.D.</td>
<td>216 kpm</td>
<td>153 kpm</td>
<td>1.4</td>
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<table>
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<th>Exercise</th>
<th>Test</th>
<th>Right-to-Left Shunt</th>
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<tbody>
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<td></td>
<td>Max Workload</td>
<td>AT</td>
<td>%</td>
</tr>
<tr>
<td>BC</td>
<td>1000 kpm</td>
<td>300 kpm</td>
<td>7.5</td>
</tr>
<tr>
<td>JK</td>
<td>500 kpm</td>
<td>200 kpm</td>
<td>13.0</td>
</tr>
<tr>
<td>JP</td>
<td>700 kpm</td>
<td>200 kpm</td>
<td>7.0</td>
</tr>
<tr>
<td>JH</td>
<td>500 kpm</td>
<td>200 kpm</td>
<td>7.5</td>
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<tr>
<td>Mean</td>
<td>675 kpm</td>
<td>225 kpm</td>
<td>7.8</td>
</tr>
<tr>
<td>S.D.</td>
<td>236 kpm</td>
<td>50 kpm</td>
<td>4.1</td>
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</table>
\( \dot{V}_E \), and \( f \) (especially in three subjects) were higher than normally expected for a reference population (19). ERV and \( \dot{V}_E \) showed the greatest variance from reference values; ERV being only about 50\% of norm (\( \bar{x} = 560 \text{ ml} \) vs 1200 ml), \( \dot{V}_E \) being almost twice that of reference values (\( \bar{x} = 11.9 \) vs 6.0 L/min).

The mean workload reached at peak exercise was 700 and 675 kpm for the control and experimental groups, respectively (\( P > 0.05 \)). The mean AT of nonhypoxemic subjects was 267 kpm compared to a mean of 225 for hypoxemic subjects (\( P > 0.05 \)).

**Resting Arterial Acid-Base**

The group means for resting, exercise and recovery pH, \( \text{HCO}_3^- \), \( \text{PaCO}_2 \) and \( \text{PO}_2 \) under normoxic conditions are presented in Table 4. Mean values for \( \text{PaO}_2 \) were 88.3 and 85.6 mmHg for control and experimental groups, respectively (\( P > 0.05 \)). As with \( \text{PaO}_2 \), group differences in mean resting values for \( \text{PaCO}_2 \), pH and \( \text{HCO}_3^- \) were not significant (\( P > 0.05 \)). From the response of \( \text{PaO}_2 \) to hyperoxic conditions mean right-to-left shunts of 5.83 and 7.75\% were calculated for the control and experimental group in that order (\( P > 0.05 \)). A shunt of 13\% was calculated for JK, while BC, JH and PN had shunts of 7.5\% (Table 3).

**Exercise Studies**

**Group Comparisons Under Normoxic (\( \text{FIO}_2 = .208 \)) Conditions**

The results of the progressive exercise test are shown in Table 4 and 5 and Figures 1 - 3. For the most part, differences between hypoxemic and control groups were small in magnitude and were not statistically significant (\( P > 0.05 \)).
<table>
<thead>
<tr>
<th>Pre - Exercise</th>
<th>Workload kpm</th>
<th>Post - Exercise 4 - 5 Min.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>100</td>
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<tr>
<td>pH Control</td>
<td>7.45±0.03</td>
<td>7.44±0.03</td>
</tr>
<tr>
<td>Exp.1</td>
<td>7.46±0.03</td>
<td>7.46±0.01</td>
</tr>
<tr>
<td>HCO3⁻, mEq/l</td>
<td>25.1±4.9</td>
<td>25.7±3.2</td>
</tr>
<tr>
<td>Control</td>
<td>28.0±3.6</td>
<td>28.1±3.6</td>
</tr>
<tr>
<td>Exp.1</td>
<td>25.6±3.4</td>
<td>25.1±4.3</td>
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<tr>
<td>PaCO₂, mmHg</td>
<td>35.6±6.2</td>
<td>36.5±6.2</td>
</tr>
<tr>
<td>Control</td>
<td>35.9±4.7</td>
<td>37.7±2.7</td>
</tr>
<tr>
<td>Exp.1</td>
<td>39.6±8.1</td>
<td>39.2±9.7</td>
</tr>
<tr>
<td>PaO₂, mmHg</td>
<td>85.4±2.6</td>
<td>84.7±2.8</td>
</tr>
<tr>
<td>Control</td>
<td>79.6±7.5</td>
<td>75.5±7.6</td>
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<tr>
<td>Exp.1</td>
<td>88.3±7.6</td>
<td>85.4±12.2</td>
</tr>
</tbody>
</table>

* P < 0.05

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However, from visual examination of the plotted means (Fig. 1-3), the following observations were noted:

1. Cardiorespiratory Response (Table 5, Figures 1 and 2). At rest and over most submaximal levels of increasing work, $\dot{V}_E$, $\dot{V}_{CO_2}$, $\dot{V}_{O_2}$, RE and HR in the experimental group remained above mean control values. None of the variables differed significantly between groups at any time during rest, exercise or recovery. At 0 kpm, the value for RE in experimental subjects was 1.08 suggesting hyperventilation.

2. Acid-base Response (Table 4, Figures 2 and 3). Similarly, group differences in LA, pH (except at 4-5 min) and $HCO_3^-$ were not significant but values were slightly elevated in hypoxemic subjects. Nor were group differences in $PaO_2$ and $PaCO_2$ significant ($P > 0.05$). $PaO_2$, however, was lower in the experimental group over all stages of test one. With increasing work, the mean $PaO_2$ of hypoxemic and non-hypoxemic subjects declined until the AT was reached at which point it began to rise. Mean values for $PaO_2$ at the AT were 87.3 and 73.8 mmHg for control and experimental groups, respectively. $PaCO_2$ fluctuated throughout the test, the lowest values being observed at 200 kpm in the experimental group and at 0 kpm in the control group. A fall in $HCO_3^-$ and a rise in pH were observed as $PaCO_2$ decreased which indicated that the subjects were hyperventilating. In most subjects the decrease in $PaCO_2$ was followed by a subsequent increase in $PaO_2$; however, in PN, JH, JP and BC, either $PaO_2$ did not rise or $PaCO_2$ rose again at which time $PaO_2$ continued to decrease.
<table>
<thead>
<tr>
<th></th>
<th>Pre-Exercise</th>
<th>Workload</th>
<th>kpm</th>
<th>400</th>
<th>Post - Exercise 4 - 5 Min</th>
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<tbody>
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<td><strong>$V_e$, 1/min</strong></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Control</td>
<td>8.8 ± 2.0</td>
<td>10.8 ± 1.4</td>
<td>13.1 ± 4.1</td>
<td>19.2 ± 1.6</td>
<td>23.6 ± 2.8</td>
</tr>
<tr>
<td>Exptl.</td>
<td>9.2 ± 2.0</td>
<td>10.8 ± 1.4</td>
<td>13.1 ± 4.1</td>
<td>19.2 ± 1.6</td>
<td>23.6 ± 2.8</td>
</tr>
<tr>
<td><strong>$V_O_2$, 1/min</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Control</td>
<td>.37 ± .08</td>
<td>.49 ± .10</td>
<td>.67 ± .18</td>
<td>1.01 ± .01</td>
<td>1.07 ± .04</td>
</tr>
<tr>
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<td>.53 ± .10</td>
<td>.63 ± .11</td>
<td>.75 ± .11</td>
<td>.92 ± .09</td>
</tr>
<tr>
<td><strong>$VCO_2$, 1/min</strong></td>
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<tr>
<td>Control</td>
<td>.36 ± .03</td>
<td>.59 ± .24</td>
<td>.85 ± .23</td>
<td>1.02 ± .12</td>
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<td>.59 ± .24</td>
<td>.85 ± .23</td>
<td>1.02 ± .12</td>
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<tr>
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<td>.89 ± .17</td>
<td>.92 ± .15</td>
<td>.92 ± .17</td>
<td>.96 ± .29</td>
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<td><strong>HR, beats/min</strong></td>
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<tr>
<td>Control</td>
<td>73 ± 20</td>
<td>89 ± 20</td>
<td>96 ± 18</td>
<td>113 ± 19</td>
<td>117 ± 17</td>
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<tr>
<td>Exptl.</td>
<td>85 ± 19</td>
<td>99 ± 16</td>
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<td>122 ± 17</td>
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<td><strong>LA, mg%</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>9.9 ± 1.2</td>
<td>9.2 ± 2.8</td>
<td>10.6 ± 4.5</td>
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<td>16.1 ± 7.5</td>
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<td>10.6 ± 4.5</td>
<td>10.4 ± 3.9</td>
<td>15.2 ± 2.8</td>
<td>20.2 ± 0.0</td>
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* $P < 0.05$
Figure 1. Response of $\dot{V}O_2$, $\dot{V}CO_2$, and RE to increasing levels of work under normoxic conditions.

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Figure 2. Response of VE, HR and LA to increasing levels of work under normoxic and hyperoxic conditions.
Figure 3. Acid-Base response to increasing levels of work under normoxic and hyperoxic conditions.
3. Anaerobic Threshold. Figures 2 and 3 illustrate changes in arterial lactate, pH and $\text{HCO}_3^-$ during the progressive exercise test. The nonlinear increase in blood lactic acid levels, which indicates the onset of metabolic acidosis, occurred between 200 and 300 kpm in both groups. A concomitant drop in pH and $\text{HCO}_3^-$ can be seen at the same workload.

Ventilatory determinants signalled the onset of metabolic acidosis at the same work levels indicated from the lactate changes. As demonstrated in Figures 1 and 2, the AT can be visualized from the nonlinear increase in $\dot{V}_E$, $\dot{V}\text{CO}_2$ and RE between 200 and 300 kpm.

When statistically analyzed, the mean AT of hypoxemic subjects breathing room air was 225 kpm for the control group ($P > 0.05$). The mean of all seven subjects was 243 kpm which fell below the 275 kpm identified by Wasserman (70) as the lower limit of "normal" for a reference population.

**Group Comparisons Under Hyperoxic ($\text{FIO}_2 = 1.00$) Conditions.**

The results of the resting studies and the exercise test performed under hyperoxic conditions are listed in Tables 6 and 7. These results are also shown in Figures 2 and 3 with test one data. None of these variables differed significantly between groups at any time during $\text{O}_2$ inhalation.

1. Cardiorespiratory response. Mean values for $\dot{V}_E$ and HR were similar to those of test one, the differences between groups being of similar magnitude and direction as during the first test.

2. Acid-base response. No group differences in LA levels were seen during high oxygen breathing, however higher levels
of $\text{HCO}_3^-$ and pH and lower levels of $\text{PaCO}_2$ and $\text{PaO}_2$ were measured in the experimental group. In both groups, mean $\text{PaCO}_2$ fell with the onset of exercise and then proceeded to rise. A subsequent fall in $\text{PaCO}_2$ was noted in the control group at the highest workload. The mean $\text{PaO}_2$ with 100% $\text{O}_2$ inhalation was greater in the control group than in the experimental group, the difference between groups being as large as 80 mmHg at 300 kpm.

3. Anaerobic Threshold. From observation of Figures 2 and 3 the AT can be identified from changes in both $\dot{V}_E$ and acid-base parameters. A nonlinear increase in LA and a decrease in $\text{HCO}_3^-$ and pH occurred between 200 and 300 kpm in both groups. Statistically, the mean AT of control and experimental groups, which was 267 and 225 kpm respectively, did not differ. The mean of all seven subjects was 243 kpm.

Within Group Comparisons: Normoxia ($\text{FIO}_2 = .208$) vs Hyperoxia ($\text{FIO}_2 = 1.00$). With the exception of $\text{PaO}_2$, differences in the exercise response to normoxic and hyperoxic conditions were not significantly different ($P > 0.05$).

1. Cardiorespiratory and acid-base response. Mean values for $\dot{V}_E$, HR, and LA were similar during tests performed under normoxic and hyperoxic conditions in both the control and experimental groups. The response of pH, $\text{PaCO}_2$ and $\text{HCO}_3^-$ during test one and two were also similar, the differences being insignificant ($P > 0.05$). $\text{PaCO}_2$ levels, however, were slightly higher during test two, the differences being larger in the control group. In addition, $\text{PaCO}_2$ responded in a less erratic fashion during the repeat test, the reason being that the drop in $\text{PaCO}_2$ was less severe and was restricted to the onset of


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<th>300</th>
<th>400</th>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>7.6 ± 1.9</td>
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<td>11.8 ± 5.9</td>
<td>14.6 ± 2.1</td>
<td>17.8 ± 2.1</td>
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<td>10.0 ± 2.9</td>
<td>15.5 ± 5.8</td>
<td>18.2 ± 8.2</td>
<td>18.9 ± 3.5</td>
<td>22.3 ± 3.5</td>
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<tr>
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</tr>
<tr>
<td>Control</td>
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<td>80.7 ± 15</td>
<td>83 ± 18</td>
<td>92 ± 20</td>
<td>101 ± 19</td>
</tr>
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<td>91 ± 17</td>
<td>94 ± 17</td>
<td>97 ± 14</td>
<td>103 ± 13</td>
</tr>
<tr>
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<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>10.9 ± 1.7</td>
<td>10.3 ± 2.0</td>
<td>10.2 ± 1.9</td>
<td>12.4 ± 0.2</td>
<td>16.1 ± 1.0</td>
</tr>
<tr>
<td>Exptl.</td>
<td>11.1 ± 4.8</td>
<td>10.5 ± 2.9</td>
<td>10.5 ± 2.4</td>
<td>11.3 ± 2.6</td>
<td>14.8 ± 4.2</td>
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*P < 0.05
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<td><strong>ACID-BASE RESPONSE UNDER HYPEROXIC CONDITIONS</strong></td>
<td></td>
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<td></td>
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<td></td>
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<tr>
<td>pH</td>
<td>7.42 ± 0.20</td>
<td>7.44 ± 0.03</td>
<td>7.48 ± 0.01</td>
<td>7.46 ± 0.01</td>
<td>7.43 ± 0.05</td>
<td>7.35 ± 0.06</td>
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<td>Control</td>
<td>7.42 ± 0.20</td>
<td>7.44 ± 0.03</td>
<td>7.48 ± 0.01</td>
<td>7.46 ± 0.01</td>
<td>7.43 ± 0.05</td>
<td>7.35 ± 0.06</td>
</tr>
<tr>
<td>Expt.</td>
<td>24.7 ± 7.7</td>
<td>25.7 ± 8.3</td>
<td>25.1 ± 2.6</td>
<td>25.1 ± 2.6</td>
<td>25.7 ± 8.7</td>
<td>24.8 ± 6.6</td>
</tr>
<tr>
<td>HCO₃⁻, mEq/l</td>
<td>27.1 ± 2.1</td>
<td>26.2 ± 2.1</td>
<td>26.6 ± 2.8</td>
<td>36.9 ± 10.6</td>
<td>38.5 ± 7.1</td>
<td>41.4 ± 8.1</td>
</tr>
<tr>
<td>Control</td>
<td>27.1 ± 2.1</td>
<td>26.2 ± 2.1</td>
<td>26.6 ± 2.8</td>
<td>36.9 ± 10.6</td>
<td>38.5 ± 7.1</td>
<td>41.4 ± 8.1</td>
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<td>Expt.</td>
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<td>40.2 ± 7.7</td>
<td>41.4 ± 8.1</td>
<td>38.5 ± 7.1</td>
<td>41.4 ± 8.1</td>
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<tr>
<td>PaCO₂, mmHg</td>
<td>37.4 ± 2.5</td>
<td>36.9 ± 4.1</td>
<td>34.9 ± 4.6</td>
<td>36.9 ± 4.1</td>
<td>34.9 ± 4.6</td>
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<td>36.9 ± 4.1</td>
<td>34.9 ± 4.6</td>
<td>36.9 ± 4.1</td>
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<tr>
<td>Expt.</td>
<td>566 ± 123</td>
<td>576 ± 100</td>
<td>566 ± 53</td>
<td>544 ± 66</td>
<td>623 ± 75</td>
<td>612 ± 47</td>
</tr>
<tr>
<td>PaO₂, mmHg</td>
<td>544 ± 123</td>
<td>536 ± 64</td>
<td>542 ± 72</td>
<td>544 ± 66</td>
<td>612 ± 47</td>
<td>590 ± 79</td>
</tr>
</tbody>
</table>

* p < 0.05

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exercise. This was especially true in the control group. As expected, the differences between PaO$_2$ levels of test one and two were significant (P<0.05). In both tests PaO$_2$ fell until the AT was reached and then rose slowly until the test was terminated.

2. Anaerobic Threshold. The AT data of control and experimental groups during room air and high oxygen breathing studies are presented in Table 8. The mean AT in the hypoxemic group was at 225 kpm both before and after oxygen inhalation, while for the control group it was 267 kpm under both conditions. Consequently, there was no significant difference in the mean AT of all subjects between test one and test two (P<0.05). The mean AT (N = 7) was 243 kpm before and after O$_2$ breathing. The mean PaO$_2$ of the control group at the AT was 87.3 mmHg during test one while the mean PaO$_2$ of the experimental group was 73.8 mmHg. This was the only measurement which differed significantly between groups at the AT (P<0.05).
<table>
<thead>
<tr>
<th></th>
<th>AT</th>
<th>( \dot{V}O_2 )</th>
<th>( \dot{V}E )</th>
<th>HR</th>
<th>LA</th>
<th>PaO(_2)</th>
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</thead>
<tbody>
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<td>l/min</td>
<td>l/min</td>
<td>beats/min</td>
<td>mg%</td>
<td>mmHg</td>
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<td><strong>FIO(_2) = .208</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>267±153</td>
<td>.82±.36</td>
<td>17.2±6.7</td>
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<td>11.7±8.2</td>
<td>87.3±2.0</td>
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<td>.75±.09</td>
<td>17.4±2.6</td>
<td>114±5</td>
<td>11.2±2.8</td>
<td>73.8±5.1*</td>
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<td><strong>FIO(_2) = 1.00</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>267±153</td>
<td>16.3±5.9</td>
<td>97±6</td>
<td>6.9±2.1</td>
<td>580±80</td>
<td>(N = 2)</td>
</tr>
<tr>
<td>Exptl.</td>
<td>225±125</td>
<td>19.3±5.8</td>
<td>102±11</td>
<td>11.9±2.7</td>
<td>544±72</td>
<td></td>
</tr>
</tbody>
</table>

\*P < 0.05.
CHAPTER IV
DISCUSSION

The results of this study demonstrate that seven obese subjects had AT's (\(\bar{x} = 243\) kpm) which were below the lower limit reported for normal subjects (275 kpm) (70). This early onset of anaerobic metabolism may account for part of their decrease in total work capacity. In addition, removal of mild arterial hypoxemia in four of these subjects had no effect on the AT indicating that this degree of arterial desaturation was insufficient to cause marked tissue hypoxia.

Earlier studies by Dempsey (23) and others (14, 36, 50) have found that the aerobic power of obese subjects is decreased and that the obese person performs routine tasks with a physiological cost much greater than normal resulting in an extremely narrow "cost-capacity" margin (25, 26). It has been suggested that the greater the body weight, the greater the energy expended for a given weight supported task (3, 26, 49). Indeed, investigators have shown \(\dot{V}O_2\), HR, BP and LA for given submaximal treadmill loads to be greatly increased in obese subjects compared to normal weight subjects of the same age and sex (14, 25, 26, 50). However, one would not expect an increased body mass to interfere with energy expenditure when the work performed is weight supporting. In this study, however, the experimental group, who were significantly heavier than the control group, had a slightly greater \(\dot{V}O_2\) while performing
identical intensities of external work on the bicycle ergometer. This is in agreement with Dempsey et. al. (25) who have found the grossly obese subject's VO₂ per unit of workload on the bike to be markedly increased. The authors suggest that in the grossly obese muscular work and energy expenditure are not completely independent of body mass. They propose that the increased postural muscle activity and body movement which accompany alternate leg extensions probably account for the increased cost during a weight supported task such as cycling.

Slightly higher levels of lactic acid, heart rate and ventilation were also observed in the experimental group suggesting that they were performing at greater relative intensities of work than the controls. Assuming that the onset of metabolic acidosis occurred at about the same percentage of VO₂ max in both groups, the lower AT in the experimental group tends to support this. The increased levels of VE, VCO₂ and VO₂ reflect an increased respiratory effort on the part of the experimental subjects, which is to be expected, since the VO₂ and therefore, the need for gas exchange was greater in these subjects.

While the increased lactic acid levels are to be expected if the hypoxemic group worked at a relatively greater severity of work, one would predict a lower, not higher level of HCO₃⁻ and pH. The discrepancy probably reflects the alkalotic status imposed by a mild hyperventilation which is best seen in the experimental group at lower workloads (Figure 3). The concomitant drop in PaCO₂ and rise in VE and RE lend support to this observation. Two subjects, AG and JK appeared to be
hyperventilating at rest as well as during exercise.

Confirming previous reports (7, 9, 23, 25), mean PaO$_2$ of these subjects dropped during mild to moderate exercise and fell below 80 mmHg in the experimental group. It is likely that the mild degree of hyperventilation seen in these subjects was due to stimulation of the peripheral chemoreceptors secondary to arterial hypoxemia. If effective, the increased $\dot{V}_E$ should have elicited an increase in PaO$_2$ except in the presence of large right-to-left shunts (42). Although PaO$_2$ of three subjects responded as expected, in subjects with shunts $\geq 7.5\%$, hyperventilation did not manifest an increase in PaO$_2$. Conversely, O$_2$ tensions remained low or continued to decline as the test proceeded.

Similarly, these subjects did not respond as effectively to high O$_2$ breathing as subjects with normal amounts of shunting. While the PaCO$_2$ levels of both groups were slightly higher and less erratic during test two, it was more pronounced in the control group (shunt $< 5\%$). It seems that although 100% O$_2$ inhalation did diminish the need to hyperventilate in both groups (except at the very onset of exercise), a higher level of $\dot{V}_E$ was required on the part of hypoxemic subjects with large shunts to maintain arterial blood gases.

It appears that the effectiveness of both hyperventilation and oxygen breathing in maintaining arterial blood gases depends, at least to some extent, on the degree of right to left shunting. A significant amount of mixed venous blood must have been passing under ventilated alveoli accounting for the desaturation of arterial blood in spite of compensatory mechanisms.
From a review of the literature (7, 9, 16, 25, 54), it appears that the above mentioned anomalies are secondary to disturbances in the mechanics of breathing; the main abnormality being a rapid shallow breathing pattern close to residual volume. The resting lung volumes of most subjects were in accord with these findings; i.e., ventilation was elevated; expiratory reserve volume was considerably reduced and three subjects with shunts > 7.5% had breathing frequencies well above average.

As a result of the reduced ERV, FRC/TLC ratios were low when compared to a reference population (37% vs 54%) (56). Rorvick (56) who reported a ratio of 40% in obese subjects, found a positive relationship between PaO2 and the ratio of FRC/TLC. The relationship can be explained by the research of Sutherland (68) who found that small airway closure and air trapping began at a FRC/TLC of 46% and that at RV, 50% of the lung units were closed. Small airway closure in turn, would result in regional hypoventilation secondary to inequalities in ventilation to perfusion distribution. It is likely therefore, that shunting accounts for the low levels of PaO2. Farebrother, et al. (32) reported a positive correlation between airway closure and PaO2 which supports this hypothesis.

Other researchers (9, 16, 23, 40, 42) have reported obesity to be associated with physiological shunts as high as 12% and have, for the most part, attributed them to uneven ventilation distribution and/or atelectasis. Dempsey and co-workers (23) have found the diffusion capacity of obese individuals to be below normal and supports the above hypothesis by suggesting
that the reduction in alveolar capillary interface for gas exchange is secondary to non-uniform ventilation distribution rather than a true diffusion gradient abnormality.

In view of the above discussion, it is not surprising that mild hypoxemia was observed in a number of obese subjects during exercise. It is interesting to note that in all subjects PaO₂ steadily declined until the AT was reached at which point it began to rise. Milic-Emili (52) in his research showed that preferential distribution of ventilation to lower lung zones occurred at 40% VT/VC. It may be that the increase in ventilation associated with the AT caused an increase in tidal volume great enough to open closed airways. In this case, enhanced gas exchange could account for the rise in PaO₂ observed beyond the anaerobic threshold. The PaO₂ of the experimental group was significantly lower than the control group at the AT suggesting a more severe impairment in the former group.

The rapid, shallow breathing pattern adapted by obese subjects, both at rest and during exercise, is probably an attempt to avoid the increased work of breathing and respiratory effort (7, 9, 25). The increased cost however, was not severe enough to precipitate hypoventilation and any accompanying hypercapnia under normoxic conditions. Subjects managed to increase their ventilation sufficiently to maintain relatively normal (or low in the case of hyperventilation) arterial CO₂ tensions.

In view of the above discussion, it is not surprising that the working capacity of obese subjects was limited. Although
VO₂ max was not measured directly, subjects incurred metabolic acidosis at very low levels of work (AT = 243 kpm) suggesting a decreased total work capacity. In 85 normal subjects between 17 and 91 years of age Wasserman and co-workers (70) found that the lower limit of normal was 275 kpm (VO₂ = 1 L/min.), while values for very fit adults were as high as 1200 kpm. Most of the subjects studied had a VO₂ of less than 0.8 L/min. at the AT. Thus, it would seem that grossly obese individuals cannot exercise to the level of VO₂ needed for walking at a brisk pace (VO₂ = 1.5 L/min) or performing daily tasks (VO₂ = 1.2 L/min) without developing a lactic acidosis (5).

For the most part, researchers reporting a negative association between relative fatness and max VO₂ have attributed the decrease in work capacity entirely to the handicap of an "inert non-contributory load" (14, 25, 26). It was not known whether or not the development of arterial hypoxemia during exercise in obese persons could contribute to the decreased capacity by prematurely inducing acidosis. As with the added fat weight, arterial hypoxemia could potentially interfere with overall maximal circulatory and respiratory function which would be reflected in a decreased total work capacity.

Peripheral hypoxemia, if severe enough could decrease O₂ tensions to a point where the limited O₂ transport would effect a decrease tissue oxygen tension at the cellular level in working muscle. Since oxidative capacity is dependent on the availability of O₂, any deficit would place an increased demand on anaerobic glycolysis to provide energy for the resynthesis of ATP if work is to continue (34, 37, 67). This shift in
primary energy production from oxidative to glycolytic metabolic pathways is reflected in an increase in lactic acid, marking the onset of metabolic acidosis. It is this assumption, together with the fact that metabolic acidosis occurs at approximately 50 - 60% max $\dot{VO}_2$ (5, 17, 34), that leads to the use of the AT as a clinical tool in the evaluation of work capacity. It seems safe to assume that if arterial hypoxemia does induce tissue hypoxia, the resultant increase in anaerobiosis will cause an early onset of metabolic acidosis and the AT will be detected at low workloads.

The AT did not appear to be affected by the degree of hypoxemia developed during exercise in these subjects. There was no change in the AT during a repeat study breathing 100% $O_2$ which corrected the hypoxemia, increasing arterial $O_2$ tensions to above 500 mmHg in six of the seven subjects. Thus, this degree of acute hypoxemia ($PaO_2 < 80$ mmHg), having no visible effect on the AT, could not be cited as a precursor to tissue hypoxia and in turn, the early onset of metabolic acidosis during a progressive exercise test.

Exercise induced metabolic acidosis was identified using both direct (LA) and indirect ($\dot{V}_E$, $\dot{V}CO_2$ and RE) measurements. In almost all cases the AT's, when detected from alterations in ventilatory gas exchange, were identical to those identified using arterial lactate and acid-base changes. Detection of the AT using multiple criteria and the fact that under similar conditions Davis (22) reports excellent reliability suggests that changes in respiratory gas exchange can be used as reliable
indicators of the AT. For this reason, it was felt that the lack of an experimental effect was not due to error in detection of the AT, but rather to 1) the relatively mild hypoxemia ($\bar{x} = 74.0 \pm 5$) and/or 2) the possibility that lactate production under these circumstances is not entirely dependent on $O_2$ supply.

One possible reason why the AT did not increase with $O_2$ inhalation could be that the degree of hypoxemia observed was not severe enough to limit $O_2$ transport and thus induce tissue hypoxia. The effect of a mild degree of acute hypoxemia on tissue oxygenation and therefore glycolytic (anaerobic) dependence is not known. As the critical $PvO_2$ for most tissues is around 25 mmHg (35, 42, 45, 63), it is not felt that this was reached even in the cells with the greatest diffusion distance.

As to the second possibility, if the degree of hypoxemia is severe enough to cause tissue hypoxia, an increased lactic acid production during exercise would be postulated unless lactic acid production is a more complex series of reactions and is not solely dependent on the availability of oxygen. For many years, it was customary to attribute LA production during exercise to anaerobic glycolysis secondary to inadequate tissue oxygenation (38, 45, 75). Recent evidence however, suggests that glycolysis is probably due to an imbalance between the glycolytic and oxidative capacity of the cell and in some cases lactate may be produced in spite of a relatively high $O_2$ content and tension in venous blood (38, 42, 45) and in others, with apparently low venous oxygen tension, no lactate is formed (24).
Both possibilities, the mild degree of hypoxemia and the complexity of LA production may have contributed to the lack of an experimental effect.

From the present results, it can be concluded that the concept of the AT is more complex than the theoretical basis that has been presented.
CHAPTER V

SUMMARY AND CONCLUSIONS

Experimental Design

It was hypothesized 1) that the AT of obese subjects would fall below values in a reference population and 2) that removal of obesity induced-hypoxemia would result in an increase in the AT.

To test this hypothesis, the AT was determined in seven obese subjects using a standard progressive work test which consisted of 100 kpm increments every two minutes before and after 100% $\text{O}_2$ inhalation.

Results and Discussion

The mean AT of obese subjects fell below values assessed in a reference population indicating a limited work capacity. Metabolic acidosis was incurred at work loads requiring less than a mean of $0.77 \text{ L} \text{ O}_2/\text{min}$.

Results of the analysis of variance of the data for between and within conditions indicate that there were no significant differences between either 1) the mean AT of the control and experimental groups or 2) the AT of experimental subjects determined under normoxic and hyperoxic conditions.

Obese subjects incurred metabolic acidosis at very low levels of work indicating that these individuals were limited in their capacity to do work and functioned with a narrow "cost-capacity" margin. The decreased capacity is probably due
to the effect of an added load on the overall circulatory and respiratory function. In view of the high cost of performing even light tasks, it is not surprising that obese persons often avoid physical activity, contributing in part to the decreased total work capacity.

The AT of hypoxemic subjects did not increase after the removal of obesity induced arterial hypoxemia. Thus, it does not appear that the degree of hypoxemia developed in these subjects was severe enough to cause an early onset of metabolic acidosis during a progressive exercise test breathing room air. Consequently, tissue hypoxia could not be inferred nor could the observed degree of acute, mild arterial hypoxemia be implicated as a factor limiting the aerobic power of obese subjects studied.

Conclusion

As was hypothesized, the AT of obese subjects fell below those assessed in a reference population. An increase in the AT of hypoxemic subjects during high oxygen breathing, however, was not seen as hypothesized indicating the need for further research concerning the mechanisms regulating anaerobic glycolysis, including arterial hypoxemia and those factors affecting the ability of the obese individual to perform daily activity.
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INFORMED CONSENT FOR PARTICIPATION IN THE STUDY: "OBESITY-INDUCED ARTERIAL HYPOXEMIA AND TISSUE HYPOXIA"

In order to participate in this study, I hereby consent, voluntarily, to physiological tests by a physician and exercise specialists at the University of Windsor. It is my understanding that I will participate in the following series of tests and measurements: 1) standing height and weight, 2) pulmonary function tests to determine resting lung volumes and dynamic lung functions, 3) underwater weighing to determine percent body fat and 4) three exercise stress tests on the bicycle ergometer. I understand that work rates will be increased until a maximum load which can be tolerated is reached or until it is considered advisable to stop the test. I further understand that during the final exercise test I will be inspiring 100% oxygen. My electrocardiogram and blood pressure will be recorded, expired gases will be collected and arterial blood samples will be withdrawn at rest, during the tests and in recovery.

The results of these tests will provide information regarding the oxygen homeostasis in my blood and any disorders, such as hypoxemia, that may affect tissue oxygenation. I
understand fully what is involved in this study and the attendant risks and by my signature, I freely consent to participate.

Signed: ____________________________

Witness: ____________________________

Date: ____________________________
SAMPLE CALCULATIONS FOR MEASUREMENT OF 

\( \dot{V}_E, \dot{V}_{O_2}, \dot{V}_{CO_2} \)

Measurements (Hypothetical)

Time = 66 sec  \( FEO_2 = 0.147 \)  \( PB = 752 \) mm Hg
Volume = 7250 ml  \( FE_{CO_2} = 0.0533 \)  \( PH_2O = 22 \) mm Hg
\( FIO_2 = 0.209 \)  \( T = 24^\circ C \)

Calculations

Step 1  \( F_{IN2} = 1 - F_{IO2} = 1 - 0.209 = 0.791 \)

Step 2  \( F_{EN2} = 1 - (FEO2 + FE_{CO2} + \frac{PH_2O}{PB}) = 1 - (0.147 + 0.0533 + \frac{22}{752}) = 0.770 \)

Step 3  \( \dot{V}_{ATPS} = \frac{Volume}{Time} \times 60 = \frac{7250 \text{ ml}}{66 \text{ sec}} \times 60 \text{ sec/min} = 6590 \text{ ml/min} \)

Step 4  \( \dot{V}_{STPD} = \dot{V}_E = \dot{V}_{ATPS} \times \frac{PB - PH_2O}{760 \text{ mm Hg}} \times \frac{273^\circ C}{273^\circ C + TOC} = 6590 \text{ ml/min} \times \frac{752 - 22}{760} \times 0.273 + 24 = 5818 \text{ ml/min} \)

Step 5  \( \dot{V}_I = \dot{V}_E \times \frac{F_{EN2}}{F_{IN2}} = 5818 \text{ ml/min} \times \frac{0.770}{0.791} = 5664 \text{ ml/min} \)

Step 6  \( \dot{V}_{O2} = (\dot{V}_I \times F_{IO2}) - (\dot{V}_E \times F_{EO2}) = (5664 \text{ ml/min} \times 0.209) - (5818 \text{ ml/min} \times 0.147) = 329 \text{ ml/min} \)

Step 7  \( \dot{V}_{CO2} = \dot{V}_E \times F_{IO2} = 5818 \text{ ml/min} \times 0.0533 = 310 \text{ ml/min} \)

Step 8  \( R = \frac{\dot{V}_{CO2}}{\dot{V}_{O2}} = \frac{310 \text{ ml/min}}{329 \text{ ml/min}} = 0.94 \)
### TABLE 9
ONE-WAY ANALYSIS OF VARIANCE FOR COMPARISON OF PHYSICAL CHARACTERISTICS OF CONTROL AND EXPERIMENTAL GROUPS

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TABLE 10 - continued

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TABLE 11 - continued

ANALYSIS OF VARIANCE FOR COMPARISON OF
ACID-BASE RESPONSE OF CONTROL EXPERIMENTAL
GROUPS UNDER NORMOXIC AND HYPOXIC CONDITIONS

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TABLE 11 - continued

ANALYSIS OF VARIANCE FOR COMPARISON OF
ACID-BASE RESPONSE OF CONTROL EXPERIMENTAL
GROUPS UNDER NORMOXIC AND HYPEROXIC CONDITIONS

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### TABLE 12

**ANALYSIS OF VARIANCE FOR COMPARISON OF EXERCISE RESPONSE OF CONTROL AND EXPERIMENTAL GROUPS AT THE ANAEROBIC THRESHOLD UNDER NORMOXIC AND HYPOXIC CONDITIONS**

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**TABLE 13**

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TABLE 13 - continued

ONE-WAY ANALYSIS OF VARIANCE WITH REPEATED MEASURES
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RESPONSE OF CONTROL AND EXPERIMENTAL GROUPS

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TABLE 13 - continued

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### TABLE 13 - continued

**ONE-WAY ANALYSIS OF VARIANCE WITH REPEATED MEASURES**

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TABLE 13 - continued

ONE-WAY ANALYSIS OF VARIANCE WITH REPEATED MEASURES
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TABLE 13 - continued

ONE-WAY ANALYSIS OF VARIANCE WITH REPEATED MEASURES
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RESPONSE OF CONTROL AND EXPERIMENTAL GROUPS

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### TABLE 13 - continued

ONE-WAY ANALYSIS OF VARIANCE WITH REPEATED MEASURES FOR COMPARISON OF NORMOXIC AND HYPOXIC EXERCISE RESPONSE OF CONTROL AND EXPERIMENTAL GROUPS

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*P < 0.05

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TABLE 14
ONE-WAY ANALYSIS OF VARIANCE WITH REPEATED MEASURES FOR COMPARISON OF NORMOXIC AND HYPEROXIC EXERCISE RESPONSE OF CONTROL AND EXPERIMENTAL GROUPS AT THE ANAEROBIC THRESHOLD

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VITA AUCTORIS

Birthplace: Nicollet, Minnesota, U.S.A.

Birthdate: February 4, 1951

Educational Background:

1969 Nicollet Public. #507 N.S.S.G.D.
Nicollet, Minnesota

1973 Southwest University, B.A.
Marshall, Minnesota

1973-77 University of Windsor, Graduate Research
Windsor, Ontario

Professional Societies: American College of Sports Medicine
(ACSM)