THE UNIVERSITY OF ALBERTA

# CARDIORESPIRATORY FITNESS IN WHEELCHAIR ATHLETES WITH CEREBRAL PALSY

BY

LEONA HOLLAND

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science.

DEPARTMENT OF PHYSICAL EDUCATION AND SPORT STUDIES

Edmonton, Alberta
FALL 1991

ISBN 0-315-70077-7

UNIVERSITY OF ALBERTA

RELEASE FORM

NAME OF AUTHOR: Leona Joy Holland

TITLE: Cardiorespiratory Fitness in Wheelchair Athletes with Cerebral Palsy

DEGREE: Master of Science

YEAR THIS DEGREE GRANTED: Fall, 1991



Leona Holland
(Students's Signature)

278-51112 Range Road 222
Sherwood Park, Alberta
T8C 1G9

Date: October, 1991

UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled Cardiorespiratory Fitness in Wheelchair Athletes with Cerebral Palsy in partial fulfillment of the requirements for the degree of Master of Science.

(Dr. Robert Steadward)

(Dr. Yagesh Bhambhani)

(Dr. Stewart Petersen)

Date: October 8, 1991

**ABSTRACT**

Two research studies were conducted to examine cardiorespiratory fitness parameters in elite male class 3 and 4 athletes with cerebral palsy (CP). The purpose of the first study was to examine validity and reliability of the wheelchair ergometer (WE) during testing of maximal oxygen uptake ($\dot{V}O_2max$) and other physiological responses, by comparing these results to those collected on the cycle ergometer (CE). The second investigation was undertaken to further examine the purpose outlined in the initial study, as well as to investigate validity and reliability of lactate threshold (LT) and ventilatory threshold (VT) on the WE and CE. Researchers have determined that both $\dot{V}O_2max$ and anaerobic threshold are highly relevant variables in endurance sports such as track (400m and longer).

Eleven subjects attempted two graded exercise tests to volitional fatigue on each of the two modes. The protocol was discontinuous in nature and subjects were required to exercise for two minutes and rest for one minute. The WE test was initiated at a velocity of 5 kilometers per hour (kmh) and increased by 2 kmh every work bout. Power output on the CE began at 30 watts and 60 rpm, and increased by 30 watts every work period. Blood samples for lactate concentrations were drawn pre-exercise, during each rest period, and three minutes post-exercise.

Blood lactate, and $\dot{V}_E/\dot{V}O_2$ and $\dot{V}_E/\dot{V}CO_2$ values were graphed

against exercise stages for the WE tests only since six of the eleven subjects did not have adequate hip and/or knee flexion to pedal the CE. Two expert investigators who were blind to the study subjectively determined the visual "break away" points for VT and LT. Despite some concern as to whether or not the evaluators accurately followed the criteria for AT identification, data from these two evaluators was deemed correct, and subsequently analyzed. Pearson product-moment correlations and two-way analyses of variance with repeated measures over two trials were used to examine the validity and reliability of physiological responses at $\dot{V}O_2max$ on both modes, and at LT and VT as identified by the expert investigators on the WE.

In the first study, reliability coefficients for $\dot{V}O_2max$ during the two exercise modes were high (0.92 and 0.89 for the CE and WE respectively), although the validity coefficients for this variable were poor (0.31 and -0.24 for trials one and two respectively, n=4). Since subjects who use their wheelchairs to perform tasks of daily life seemed to perform better on the WE, and those who walk (with or without aids) did better on the CE, it is recommended that the testing mode be specific to the athlete's primay mode of ambulation unless the goal is to establish standards for class 3 and 4 athletes with CP; in this case, the WE should be utilized since all subjects could use this mode.

The expert investigators were not able to identify VT and

LT break away points for all the WE tests. In an attempt to alleviate this problem, researchers may need to implement a protocol with small work increments (i.e., 1 kmh), and either a continuous protocol or a discontinuous protocol with longer exercise bouts (i.e., four to six minutes). VT may be the preferred method to identify anaerobic threshold since it was identified more frequently than LT, and reliability between trials and investigators was slightly higher. However, additional research in the identification of AT for wheelchair athletes with CP is recommended before either LT or VT be used for exercise prescription.

**ACKNOWLEDGEMENT**

I would like to express my sincere appreciation to a number of individuals who have contributed to my graduate experience and particularly to the completion of this work:

Dr. Robert Steadward for his guidance and generosity in granting me work time to complete this document.

Dr. Yagesh Bhambhani for many things- his assistance in collecting the data, his generosity in allowing me to use his lab, his patience especially in the area of statistics, his endless corrections, and his ongoing direction and support.

Dr. Stewart Petersen for his suggestions as a member of my supervisory committee.

Ewen Nelson, the Rick Hansen Centre's computer expert, for being patient with me and coming to my rescue many, many times.

Gord Bell and Gord Sleivert, the "expert investigators", for identifying the lactate and ventilatory thresholds for the subjects.

To Barbara Campbell, for her flavorful and most appropriate editorial suggestions.

To the eleven subjects who agreed to participate in these studies, some of whom travelled a long distance- it would never have been possible without your hard work!

To the Central Research Fund for making this experience financially feasible.

# TABLE OF CONTENTS

## LIST OF TABLES

## LIST OF FIGURES

# CHAPTER I

## Introduction and Review of Literature

Until 1978, there was no contention to the popular belief of educators, medical practitioners, and family members of persons with cerebral palsy (CP), which affirmed that physical activity was detrimental to individuals with CP, as it increased spasticity and decreased flexibility (McCubbin & Shasby, 1985; Jones, 1988). As a consequence, this view discouraged potential athletes with CP from fitness and competition training and represented one reason for the lack of respective research data (Jones, 1988).

The year 1978 brought about a significant transformation insofar as fitness and training techniques for persons with CP were concerned. Many persons with CP previously relegated to relatively sedentary lifestyles began their trek to physical activity and never looked back. It was then The Cerebral Palsy Sports and Recreation Association (CP-ISRA) was founded, fulfilling dreams of apsiring athletes and elevating sports for persons with CP from obscurity to the reality of participation in ever-increasing numbers, from the 1980 Paralympics in The Netherlands to the present time. By comparison, athletes with spinal cord injuries have been competing at the international level since 1952 (Guttmann, 1976). Today, coaches, adapted physical education scholars, and fitness experts precribe the same strategies and training principles used by the able-bodied population and/or persons with other physical disabilities for many athletes with CP. Whether or not the application of these particular strategies to persons with CP is the most appropriate when attempting to attain peak performance, introduces a realm of interesting research possibities.

Due to the multifarious nature of the disability and associated confounding physical characteristics, researchers encounter mounting obstacles with respect to a further lack of data collection on individuals with CP. Hence the extreme difficulty of recruiting adequate sample numbers of subjects with similar attributes.

This chapter provides information on the condition of CP so that the reader will better understand the nature of the disability and its corresponding characteristics. Following this background, the chapter will begin to explore the previously undiscovered frontier of cardiorespiratory fitness parameters as they relate to the persons with CP, based on existing information obtained from material on the non-disabled population. Outlined at the end of this chapter are the purposes of this thesis, along with several hypotheses. Two studies which were conducted to complete this thesis are recapitulated in Chapters II and III. Finally, Chapter IV embraces comments specifically pertaining to cardiorespiratory fitness parameters in athletes with CP, based on research findings. It is anticipated that information gained from the two studies which were conducted and subsequently presented in this thesis, will be beneficial to athletes with CP and their coaches, as well as fitness enthusiasts and rehabilitation therapists, as they design and activate training and therapy programs for individuals with CP.

**Cerebral Palsy**

Detailed definitions of the condition of cerebral palsy are presented in order to provide a better understanding of the diverse disability prior to discussion related to cardiorespiratory fitness.

The literal translation of the term "cerebral palsy" is "brain paralyzed" (Jones, 1988). It is a persistent, non-changing disorder of movement, posture, and balance due to damage to the brain that occurs before, during, or immediately following birth. Technically, this separates persons with CP from disorders caused by traumatic injury such as cerebral vascular accidents (strokes) or other insults sustained to the brain, inasmuch as many of the physical signs are similar (Dunn, 1988).

**Topographical and Physiological Classification**

The distribution and extent of brain damage varies from person to person; therefore, while similarities in patterns exist, no two persons with CP have exactly the same motor patterns (Jones, 1988). In order to help organize the diagnosis of CP into more manageable terms, a classification system has been devised. Cerebral palsy is usually classified by its clinical manifestation and physical signs displayed by the individual. Different types of CP are distinguished by what the classifier **sees** (topographical classification) and the degree of muscle tone **felt** (physiological classification) (Dunn, 1988).

(1) Traditional topographical classification involves identification by the affected extremities (their anatomical location). For example, the legs are more involved in diplegics; three limbs are affected in triplegics; all four limbs are involved in quadriplegics (Sherill, 1986).

(2) Muscle tone refers to muscle "tenseness" and a muscle's state of readiness for action. Normal postural tone forms the background for normal movement and varies according to an individual's state of rest or exertion. Under normal circumstances, postural (muscle) tone never falls so low that an individual is unable to maintain him or herself against the force of gravity, nor does it rise so high as to prevent movement through range. An individual without brain damage is able to experience a variation of tone but this variation is always within normal limits; thus, normal postural tone never interferes with function. A person with CP has variations in tone which cannot be altered (corrected) by the individual. Abnormal tone severely affects anti-gravity control, grading of movement, postural adaptations, and can completely block movement (Dunn, 1988).

A person who has "spastic" muscle tone has hypertonic muscle tension and primitive reflexes, or acquired abnormal reflexes. Five reflexes frequently influence movement patterns, especially in a person who is significantly affected by CP. A brief description of the five reflexes follows (Bobath, 1976).

(a) Asymmetrical Tonic Neck Reflex.

In the asymmetrical tonic neck reflex, when the head is turned to one side, extensor tone predominates in the limbs on the side to which the face is turned, and flexor tone predominates in the muscles on the opposing side. Domination of this reflex occludes use of the hands in the midline or in bringing a grasped object to the mouth.

(b) Symmetrical Tonic Neck Reflex.

During the symmetrical tonic neck reflex, flexion of the upper extremities and extension of the lower extremities occur when the head is moved toward the chest. When the head is moved toward the back, it causes extension of the upper extremities and flexion of the lower extremities.

(c) Positive Support Reaction.

In the positive support reaction, stimulus to the feet results in extension of the hips and knees, and plantar flexion of the feet. Retention of this reflex greatly affects the ability to stand.

(d) Tonic Labyrinthine Reflex.

A person with the tonic labyrinthine reflex has dominating flexor tone when lying on the stomach, and dominating extensor tone when lying on the back. Retention of this reflex prevents achieving a stable sitting posture and developing independent sitting balance.

(e) Moro and Startle Reflexes.

The moro and startle reflexes both result in a delay in starting the desired motor action. The moro reflex occurs when there is a loud noise, sudden jarring, or the head is dropped backwards 20 to 30 degrees. This causes the limbs to spread apart and then come together in an embraced position. The startle reflex is believed to become more predominant as the moro reflex declines with age. While the moro reflex is initially movement of the extensors, the startle reflex is primarily a rapid flexion response. It is not normal for an adult to have retained the moro reflex, but the startle reflex is present in all adults. A person with CP may have a more exaggerated or more sensitive (easier to illicit) startle reflex.

Tenseness and difficult, inaccurate voluntary movements result from the retention of these reflexes. Examples of other types of muscle tone include "rigidity" (severe form of spasticity), "athetosis" (writhing movements), and "tremor" (vibratory movements).

Tone is affected by temperature, pain, emotions, posture/body position, movement, and fatigue. Tone increases with cold, pain, and emotions. A person with ataxia and non-tension athetoidism often has underlying low tone. Heat from a hot pool for example, can accentuate this. Quick, short, repetitive movements performed by someone with spasticity will serve to increase tone and thus, decrease range of movement. Some rhythmical movements are used by physical therapists to decrease tone. Fatigue influences all performances. For example, the ataxic athlete is usually more uncoordinated (Dunn, 1988).

The description of an athlete with CP therefore, might read "spastic diplegic" referring to the presence of spasticity (resistance to movement) primarily in the legs. It does not indicate the degree of spasticity, precise distribution of the spasticity, resulting deformities, if any, or loss of range of movement.

Damage to the central nervous system determines affected motor output, topographical areas, and muscle tone. The developing child attempts to do as many activities as possible which sometimes means using the abnormal patterns available, or developing movements to compensate. Movement becomes limited to these few patterns; this in turn becomes "habit". Habitual movements over time, can lead to joint contractures and in some persons, to structural deformities. Frequently, a person with severe CP may undergo surgery to reduce contractures which, in turn, further affect movement patterns (Bleck, 1982).

<u>**Sports Classification**</u>

In an attempt to promote fairness amongst competitors, sports classification groups together athletes with similar physical disabilities. Eight classes exist within the Cerebral Palsy International Sports and Recreation Association's Classification System, and athletes undergo a functional test to determine the class in which they will compete (CP-ISRA Classification and Sports Rule Manual, 1990). Athletes in classes 1 through 4 compete in wheelchairs, while competitors in classes 5 to 8 use their lower body to ambulate. Following, are brief descriptions of the 8 sport classes for CP athletes (Shephard, 1990).

Class 1 competitors have severe spasticity in all four limbs and contractures are often present. Movement is very slow. There is no fine motor dexterity and great difficulty with gross motor tasks. Increased spasticity levels with movement and poor functional muscle strength and coordination, necessitate the use of an electric wheelchair for mobility.

Class 2 participants are severe athetoid quadriplegics characterized by a prevalence of jerky, uncontrolled movements. If the upper body is less involved than the lower limbs, the athlete propels the wheelchair with the hands and arms; if the lower body is more functional, the feet and legs are used to move the wheelchair.

Class 3 athletes are moderate to minimal quadriplegics or triplegics in wheelchairs, or severe hemiplegics in wheelchairs. The quadriplegics have moderate to very minimal involvement in their limbs. Tri- or hemiplegics have good functional use of the dominant arm but severe lower extremity function. They generally possess fair to good functional strength and range of motion.

Class 4 competitors are spastic diplegics with good upper extremity strength and control. They show some reflex distractions during upper extremity tests but this does not constitute quadriplegia. They also compete in wheelchairs.

Class 5 participants are moderate to severe spastic diplegics who may use canes, crutches, or other aids for ambulation. A scissor-gait is often present, and functional inhibitions are obvious while walking or trying to run. There is little or no involvement in the upper extremities.

Class 6 athletes have athetosis in all four limbs, but walk without assistive devices. Obvious balance and coordination difficulties exist due to the upper body involvement. Quite often, the faster the competitor moves, the easier movement becomes because the athetosis is not as evident as when the athlete is trying slower, more controlled movements.

Class 7 competitors are hemiplegics who have good functional range of motion and control on the non-affected side. There is often a limp present during ambulation because there is muscle imbalance between the right and left sides of the body.

Class 8 participants are minimally affected diplegics, monoplegics, or quadriplegics. There may be a slight loss of function due to incoordination, although range of motion and symmetry are good.

## Cardiorespiratory Parameters

There is almost no information available which pertains to cardiorespiratory fitness parameters in persons with CP. Thus, these variables as they relate to the non-disabled population are presented below.

Maintenance of life depends on efficient operation of the body at the cellular level. Each cell needs a ready supply of oxygen and food while carbon dioxide and other waste products must be carried away from it. Adequate functioning of the circulatory and respiratory systems is needed for these life-sustaining services. The cardiovascular system (heart and blood vessels) keeps blood circulating throughout the body. The respiratory system (lungs and air passages) removes carbon dioxide and replaces it with fresh oxygen. Because of their dependence on each other, the two systems are often referred to jointly as the cardiorespiratory system (Getchell, 1976).

### Maximal Aerobic Power

Maximal aerobic power is defined as the maximal rate at which oxygen can be consumed by the body per minute. It represents the greatest difference between the rate at which inspired oxygen enters the lungs and the rate the expired oxygen leaves the lungs. Therefore, in order to measure maximal oxygen uptake one must know the amount of oxygen inspired and the amount expired; the difference between these two values is the amount of oxygen that has been taken up and used by the electron transport system of the mitochondria to produce energy for active tissues (Lamb, 1984).

While it is true that a person's maximal oxygen uptake ($\dot{V}O_2max$) reflects the maximal functional capacity of the cardiovascular system, and maximal functional capacity of the cardiovascular system is usually the most important determinant of performance in physical activity of an aerobic nature, it is not true that a person with excellent maximal oxygen uptake is necessarily an outstanding endurance performer (Getchell, 1976). Many other factors such as motivation and technique play a role in endurance performance. Thus, while $\dot{V}O_2max$ test results can be used to determine the physiological potential to become a good endurance performer, they cannot be used to accurately predict whether or not the subject will become a champion. An important use of the $\dot{V}O_2max$ test from a health standpoint is its use in the detection of cardiovascular disease, and in the assessment of one's capacity for exercise prior to the undertaking of an exercise program (Astrand & Rodahl, 1986).

While $\dot{V}O_2max$ is the most commonly measured element of aerobic power, there are other related characteristics for which alternative methods of quantification can be more descriptive in specific applications. For example, in sports that depend on the highest rate of energy release over a period of about one to four minutes, the time that is taken to achieve $\dot{V}O_2max$ is critically important. Thus, the 400m runner and 100m swimmer can take advantage of being able to quickly supplement an anaerobic release of energy with an aerobic one. As performance time increases, the ability to maintain $\dot{V}O_2max$ at maximum levels as indicated by time becomes progressively more important. An approach for quantifying the endurance ability of performances that extend beyond ten minutes is to measure the length of time that a performance can be maintained at lactate threshold (LT) intensity or at some level between LT and $\dot{V}O_2max$. Thus, for "aerobic endurance" testing, oxygen utilization is measured rather than cardiac output (Thoden, 1991).

**Units of Maximal Oxygen Uptake Measurement**

Because oxygen is used by all body tissues, a larger individual has a greater oxygen uptake than a smaller one both at rest and during exercise. Thus, when body weight is not supported during the test (e.g., running), it is appropriate to record oxygen uptake values on the basis of body weight, typically in terms of milliliters of oxygen per kilogram of body weight (ml/kg/min). When expressed in this fashion, average maximal oxygen uptake values for college men and women might be about 48 and 40 ml/kg/min respectively (Fox et al. 1988).

For sports such as rowing, in which total work output is important and the subject is not weight-bearing, $\dot{V}O_2max$ is usually reported as an absolute volume per minute (l/min) (Thoden, 1991).

**Reliability of Maximal Oxygen Uptake**

Davis et al. (1976) studied anaerobic threshold and maximal aerobic power for three modes of exercise in able-bodied college-age males. Test-retest correlation coefficients for $\dot{V}O_2max$ (l/minute) for arm cranking, leg cycling, and treadmill running modes were 0.92, 0.94, and 0.96 respectively.

Bar-Or & Zwiren (1975) examined the reliability and validity of a continuous progressive arm cranking test on 41 non-disabled males. The reliability coefficient of $\dot{V}O_2max$ was 0.94.

Thoden (1991) $\dot{V}O_2max$ tested fifteen able-bodied subjects in a test-retest experiment using the treadmill. There were no significant differences from test to retest, and the reliability coefficient was 0.95.

Katch et al. (1982) conducted 8 to 20 $\dot{V}O_2max$ treadmill tests on four trained females and one trained male over a 2 to 4 week period (80 tests total). Results revealed that biological variation and technological error amounted to +/- 5.6%. Biological variability accounted for 90% or more of this variability, while technological error accounted for less than 10%. Causes for biological variation in $\dot{V}O_2max$ are not known according to Katch et al. (1982), although it is most likely a consequence of the tremendously complex response patterns to any given stimulus. Thus, attempting to single out one or perhaps two major factors contributing to biological variability is difficult. Certainly motivation would contribute to the calculated biological variability. However, there is no way to quantify this motivational effect other than to acknowledge its presence (Katch et al., 1982).

**Physiological Factors that Determine Maximal Oxygen Uptake**

Several physiological factors (central and peripheral) influence $\dot{V}O_2max$, as indicated by Lamb (1984).

(1) Central Factors.

The heart, lungs, and blood vessels must be functioning adequately so that oxygen inhaled into the lungs is delivered to the blood.

(2) Peripheral Factors.

(a) The process of oxygen delivery to the tissues by red blood cells must be normal; that is, there must be normal heart function, blood volume, red blood cell count and hemoglobin concentration. As well, the blood vessels must be able to shift blood from non-working tissues to working muscles where the oxygen demand is greatest.

(b) The tissues, especially muscles, must have normal capacity to use the oxygen delivered to them. Thus, they must have normal energy metabolism and mitachondrial function. Some factors that influence the ability of muscles to utilize oxygen include the type of exercise (i.e., continuous or intermittent, brief or prolonged, light or heavy in relation to $\dot{V}O_2max$), state of physical training, diet (i.e., high or low in carbohydrates), and state of health (i.e., certain pathological conditions such as diabetes affecting the choice of fuels). Lundberg (1976) postulated that lower $\dot{V}O_2max$ and reduced net mechanical efficiency during physical exercise in persons with CP, could be due to a reduction in localized muscle blood flow of the spastic muscle groups. Thus, removal of lactate is inhibited with elevated levels of spasticity.

(c) A simple blood test can determine whether the characteristics of the blood are normal.

Heart function is reflected by factors determining cardiac output. Both the ability of the circulatory system to transfer blood from inactive to active regions and of the tissues to extract oxygen from the blood are reflected by the difference in content of oxygen between arterial and venous blood [arteriovenous $O_2$ difference, $(a-v)O_2$ diff]. A person who can shift most of his or her blood to working muscles during exercise will have a large arteriovenous oxygen difference because active muscles will be able to extract more oxygen from the blood than inactive tissues of the body. Likewise, one whose muscles have highly active mitochondria will be able to utilize oxygen from the blood supply quite readily (Astrand & Rodahl, 1986).

Assuming that lungs and blood characteristics are normal, maximal oxygen uptake is a function of maximal cardiac output and maximal arteriovenous oxygen difference, as indicated by the Fick equation: $\dot{V}O_2max = Q_{max} \times max\ (a-v)O_2$ diff. Hence, cardiac output represents the amount of blood potentially available for oxygen delivery to active tissues each minute, and arteriovenous $O_2$ difference represents the degree to which the oxygen contained in the blood pumped out of the heart is used by exercising and non-exercising tissues for aerobic energy metabolism (Astrand & Rodahl, 1986).

**Training Effects**

The effects of physical training can be classified into biochemical changes and cardiorespiratory (systemic) changes according to Fox et al. (1988).

(1) Biochemical Changes

There are three major aerobic adaptations at the biochemical level, that occur as a result of training.

(a) An increased myoglobin content appears to occur in those muscles involved in the training program (Pattengale & Holloszy, 1967). Since myoglobin facilitates oxygen transport, oxygen uptake in exercised muscle is enhanced.

(b) There is an increase in the capacity of the muscle cell to oxidize carbohydrate due to a rise in the number and size of the mitochondria in skeletal muscle fibers (Gollnick & King, 1969), and an increase in the level of activity or concentration of enzymes involved in the Krebs cycle and electron transport system (Benzi et al., 1975). As well, there is also an increase in the amount of glycogen stored in the muscle following training (Holloszy, 1967).

(c) The breakdown (oxidation) of fat to carbon dioxided and water with adenosine triphosphate (ATP) production in the presence of oxygen is increased (Morgan, 1971). This effect is sometimes referred to as "glycogen sparing", and is a definite advantage because fat serves as a major source of fuel for skeletal muscle during endurance exercise. This change is related to a greater release of fatty acids from adipose tissue and increased activity of the enzymes involved in the activation, transport, and breakdown of fatty acids.

The changes noted above do not occur to the same degree in the red or slow-twitch, and white or fast-twitch fibers, since responses are specific.

(a) In the case of aerobic changes, it is fairly well agreed that the aerobic potential of skeletal muscle following training is increased equally in both red and white fibers (Baldwin et al., 1972).

(b) Changes in the glycolytic capacity appear to be greater in fast-twitch fibers (Fink et al., 1975).

(c) Red fibers occupy a greater area of the muscle inendurance athletes than do white fibers, while white fibers are larger in weight-lifters and sprinters (Gollnick et al., 1972). This information implies a selective hypertrophy dependent upon the kind of training and/or physical activities performed.

(2) Cardiorespiratory Changes

Changes to the cardiorespiratory system are demonstrable under resting conditions, and during submaximal and maximal exercise.

There are five main changes resulting from training that are apparent at rest (Fox et al., 1988).

(a) Using echocardiography, cardiac hypertrophy of endurance athletes is characterized by a large ventricular cavity and a normal thickness of the ventricular wall. Thus, the volume of blood that fills the ventricle during diastole and stroke volume capabilities are greater than those of the non-endurance athlete. The cardiac hypertrophy of non-endurance athletes (e.g., wrestlers, shot putters), who engage in high resistance or isometric types of activities, is characterized by a normal-sized ventricular cavity and a thicker ventricular wall. Thus, their stroke volume capabilities are no different from those who do not participate in physical activities (Morganroth et al., 1975).

(b) Information about resting bradycardia (decreased heart rate) points out that training bradycardia is dependent upon a long time period (maybe years) of intensive training, and the magnitude of decrease in resting heart rate produced by training is less when the level of fitness is greater (Frick et al., 1967). Apparently, the magnitude of bradycardia is the same in endurance and non-endurance athletes. Evidence indicates that the resting bradycardia due to physical training is a result of a parasympathetic inhibition (Frick et al., 1967).

(c) Resting stroke volume of athletes or trained subjects (especially in endurance athletes) is higher than that of their non-athletic counterparts. As mentioned earlier, endurance athletes have an increased ventricular cavity, thus allowing more blood to fill the ventricle during diastole resulting in a larger stroke volume. Another contributing factor to an increased resting stroke volume following training is an increased myocardial contractility (Morganroth, 1975).

(d) Both the total blood volume and hemoglobin increase with training (Oscai et al., 1968).

(e) Hypertrophy of skeletal muscle resulting from long-term endurance training is accompanied by an increase in capillary density (Hermansen & Wachtlova, 1971). The supply of oxygen and other nutrients to, and the removal of waste products from the muscle are all enhanced because there are more capillaries per fiber.

Several changes in the functioning of the oxygen transport and related systems following training are evidenced during steady-state, submaximal exercise.

(a) Oxygen consumption during exercise at a given submaximal workload is the same (Fox et al., 1975) or slightly lower (Fox et al., 1975b) before as compared to after training due to an increase in mechanical efficiency (skill) acquired.

(b) Training causes a decrease in the accumulation of lactic acid during a given submaximal exercise (Karlsson et al., 1972), thus allowing the athlete to maintain a high intensity throughout the event without experiencing early fatigue. This effect occurs because training enhances the oxygen supply to active muscle cells so that the exercise may be performed to a greater extent aerobically.

(c) During submaximal exercise at a given load or $\dot{V}O_2$, the cardiac output of trained subjects is sometimes slightly lower than, and sometimes the same as that of untrained subjects (Fox et al., 1975b), depending on the type, intensity, and duration of training programs conducted. Fox et al. (1975b) point out that stroke volume and/or $(a-v)O_2$ difference appear to be sensitive to training intensity rather than to either frequency or duration.

(d) Stroke volume is increased during submaximal exercise at a given workload following training (Saltin et al., 1968), again, due primarily to the increased size of the ventricular cavity (and thus the greater amount of blood filling the cavity).

(e) A consistent and pronounced change associated with training is a decreased heart rate during submaximal exercise following training (Saltin et al., 1969). As in the case of the resting bradycardia, this decrease is most pronounced in comparisons of non-athletic subjects and highly trained athletes. A slower beating heart is more efficient, requiring less oxygen than a faster beating heart at the same cardiac output level. Although resting bradycardia is caused by a parasympathetic inhibition, bradycardia during exercise is caused by a decreased sympathetic drive due to either an effect directly on the heart itself, or an indirect effect resulting from alterations in the trained skeletal muscles (Clausen et al., 1970).

(f) Blood flow per kilogram of working muscle is lower in trained than in untrained individuals at the same absolute submaximal workload. The working muscles compensate for lower blood flow in the trained state by extracting more oxygen (Holloszy, 1973).

Several physiological changes are necessary to bring about a training effect to increase maximal working capacity.

(a) Effects of training on the amount of oxygen that can be consumed per minute during maximal exercise has been studied extensively; there is little doubt that it is increased with training for both persons with physical disabilities (Davis, 1991; Lundberg, 1984), and able-bodied individuals (Frick et al., 1970). The magnitude of increase in $\dot{V}O_2max$ is highest in athletes who compete and train for endurance types of activities. However, an average improvement of between 5 and 20 percent can be anticipated for college age male or female students following 8 to 12 weeks of training (Lamb, 1984). $\dot{V}O_2max$ is considered by most physiologists to be the single most accurate measure of endurance fitness. An increase in this component is brought about by an increased oxygen delivery to the working muscles through an increased cardiac output, and an increased oxygen extraction from the blood by the skeletal muscles (Getchel, 1976).

(b) The maximal cardiac output increases with training, and the magnitude of change is similar to that of the $\dot{V}O_2max$ since the two are directly related. (The former is a factor in determining the latter.) Since maximal heart rate is either unchanged or slightly decreased following training, increased cardiac output following training is entirely due to an increase in stroke volume (Ekblom & Hermansen, 1968).

(c) Increase in maximal stroke volume resulting from training is related to cardiac hypertrophy and increased myocardial contractility described earlier. A larger ventricular volume coupled with an increased force of contraction allows for a maximal output of blood with each beat. The single most important feature that distinguishes the athlete who has been training for several years from the sedentary person who has been training for only a few months is the magnitude of stroke volume (Saltin, 1969). In other words, stroke volume is a major determinant of the magnitude of the Q and thus of the $\dot{V}O_2max$.

(d) The maximal attainable heart rate is either unchanged or decreases slightly following training, probably due to an increased heart volume due to cardiac hypertrophy, and a decreased sympathetic drive (Saltin & Astrand, 1967).

(e) Training induces an increase in the glycolytic capacity (lactic acid production) which provides additional ATP energy thereby improving performance or working capacity of activities that rely heavily on this system for energy (Fox et al., 1988).

(f) Blood flow per kilogram of muscle is no different for the trained or untrained individual, even though blood flow to the total working musculature is indeed greater during maximal exercise following training. This seemingly contradiction can be explained by pointing out that total muscle mass required to perform work following training is also greater, and thus, the flow per kilogram remains constant (Saltin, 1969).

In addition to the circulatory changes discussed above, several respiratory changes also occur as a result of physical training.

(a) Maximal minute ventilation (not a limiting factor for the $\dot{V}O_2max$) is increased following training due to increases in both tidal volume and breathing frequency (DeJours, 1966).

(b) Training causes an increased ventilatory efficiency (the amount of air ventilated at the same oxygen consumption level is lower than in untrained individuals) which allows more blood to move to the working skeletal muscles and less to the respiratory muscles (Astrand & Rodahl, 1986). This is indicated by a reduced $\dot{V}_E/\dot{V}O_2$ return.

(c) The various lung volumes measured under resting conditions (with the exception of tidal volume) are larger in trained than in untrained individuals which can be attributed to the fact that training results in improved pulmonary function and therefore in larger lung volumes (Astrand & Rodahl, 1986).

(d) Athletes tend to have larger diffusion capacities at rest and during exercise than do non-athletes because larger lung volumes of athletes provide a greater alveolar-capillary surface area (Kaufmann et al., 1974).

**Factors Influencing Training Effects**

The effects of training are influenced by many factors, which are presented below.

(1) With both continuous (Davies and Knibbs, 1971; Faria,1970) and interval (Cohen and Fox, 1975; Fox et al., 1975) type training, intensity of exercise sessions is important in guaranteeing maximal gains in fitness. Intensity is relative, and thus, what is intense for one person may be easy for another. The gains in $\dot{V}O_2max$ are inversely related to initial $\dot{V}O_2max$ levels, irrespective of the intensity of the training program. The American College of Sports Medicine (ACSM) (1990) recommends that intensity of training equal 60-90% of maximum heart rate or 50-85% of maximum oxygen uptake or maximum of heart rate reserve. Pollock (1973) points out that responses to the same type of aerobic training are equal for both men and women.

(2) Most studies reveal that frequency and duration of training have some effect on the magnitude of training results (Davies and Knibbs, 1971; Knuttgen et al., 1973; Gibbons et al., 1983), although precise amounts are unknown. ACSM (1990) recommends the following: (1) frequency of training: 3-5 days per week, and (2) duration of training: 20-60 minutes of continuous aerobic

activity. Duration is dependent on intensity of the activity; thus, lower intensity activity should be conducted over a longer period of time. Lower to moderate intensity activity of longer duration is recommended for the non-athletic adult.

(3) The specificity of training effects is important in three capacities.

(a) Training effects are specific to the type of exercise performed during the program (Pechar et al., 1974). Thus, athletes who train utilizing the bicycle are more likely to improve their ability to work on the bike than on the treadmill.

(b) Training effects are specific to muscle groups used during the exercises (Fox et al., 1975). Therefore, the magnitude of post-training changes will be greater when the exercise is performed with the trained rather than the untrained muscle groups (limbs).

(c) Training effects are specific to the type of training program used. For example, a group of sprinters is more likely to increase the capacity of the ATP-PC system, while an endurance group is apt to have a greater improvement in the oxidative energy system (Fox , 1975).

(4) It has been estimated through testing of identical twins (Klissouras, 1972), that $\dot{V}O_2max$ is 93.4 percent genetically determined. In the same manner, the capacity

of the lactic acid system and maximal heart rate have been found to be genetically determined to the extent of 81.4 and 85.9 percent respectively (Klissouras et al., 1972).

(5) The preferred mode of exercise training will utilize large muscle groups in order to promote the greatest improvement in $\dot{V}O_2max$. ACSM (1990) suggests that the chosen mode of exercise be any activity that: (a) uses large muscle groups, (b) can be maintained continuously, and (c) is rhythmical and aerobic in nature.

**The Determination of Maximal Aerobic Power**

There are several modes used to appraise maximal oxygen uptake in the non-disabled population including treadmill, cycle ergometer (CE), stepping, rowing, skiing, and swimming. Values of $\dot{V}O_2max$ measured on an inclined treadmill are usually 5 to 15 percent higher than those obtained on either the CE or step bench (McArdle and Magel, 1970), likely due to the smaller muscle mass being used and localized fatigue experienced on the CE and stepping tests. This fatigue would occur prior to maximally stressing the circulatory and respiratory systems, thus leading to the lesser $\dot{V}O_2max$.

For persons with lower limb impairment, the arm crank and more recently, the wheelchair ergometer (WE) have been utilized. On the WE, the subject's racing or daily wheelchair is mounted on a set of rollers, and the subject wheels "on the spot". The WE appears to be the preferred mode for skilled wheelers because it is specific to the movement the subjects perform daily.

Either a continuous or discontinuous protocol may be implemented, since similar results are obtained (McArdle et al., 1973). Generally, the discontinuous test requires more time which may be a distinct disadvantage for both subjects and tester.

Additional factors to be considered in determining $\dot{V}O_2max$ are exercise posture, muscle mass used in the exercise, exercise intensity, exercise duration, mechanical efficiency for the task, and the motivation of the subject (Rowell, 1974). Posture must be upright because the highest oxygen uptakes observed in a horizontal position while pedalling a cycle ergometer or during swimming are almost always 5 to 29 percent less than the highest uptakes for the same subjects on the treadmill (Magel et al., 1975).

Because the increased activity of skeletal muscles accounts for most of the increased oxygen uptake during exercise, it is obvious that large muscles must be used if maximal oxygen uptake is to be attained.

Both exercise intensity and duration must be great enough to elicit a near-maximal response of the cardiovascular system if $\dot{V}O_2max$ is to be attained. For example, a minimum of three or four minutes of running on the treadmill is required to achieve $\dot{V}O_2max$, whereas treadmill walking up progressively steeper grades may require 20 minutes or longer to elicit a maximal response (Nagle, 1973). Intensity of the workload is increased progressively in tests of maximal oxygen uptake, so that eventually the intensity must reach a level sufficient to bring about a maximal response. The workload may be increased in a variety of manners including increasing the velocity of the exercise, adding tension or resistance, increasing the grade, or a combination of these methods. Ettinger's study (1989) demonstrated that for young, healthy, physically active males using the CE, 60 and 80 rpm can be used interchangeably in submaximal testing over a wide range of power outputs. Similarly, Gueli & Shephard (1977) found that heart rate and oxygen uptake were closely related to pedalling rates with a plateau of optimum efficiency of cycling at rates of 60 to 85 rpm.

As well, an effective test of $\dot{V}O_2max$ should not depend on the skill or motivational levels of the subject. For example, a high-lesion quadriplegic may be more accurately tested on the arm crank because poor wheeling skills may hinder the ability to reach a maximal response on the wheelchair ergometer.

**Criteria for Maximal Aerobic Power Attainment**

The primary criterion for determining that maximal aerobic power has been attained is a levelling off or decrease in $\dot{V}O_2$ with increasing workload. Because it is known that oxygen uptake increases linearly with increasing workloads up to the maximal rate of oxygen uptake, a plateau of oxygen uptake with an increasing workload is a clear indication that the subject has achieved maximum. Other criteria are volitional exhaustion, achievement of maximal or near maximal heart rate (which is indicative of maximum cardiac output), a respiratory exchange ratio greater than unity (due to the buffering of lactic acid which causes large quantities of carbon dioxide to be released), and a blood lactate level above 100 mg. percent.

**Anaerobic Threshold**

The power output or oxygen uptake at which blood levels of lactic acid begin to rise significantly above normal resting levels (approximately 10 mg % or 1.1 mmol/litre) during exercise of increasing intensity, has been termed the anaerobic threshold (AT). At rest and low exercise intensities, the rate of blood and muscle lactate production and removal are equal; therefore, lactate concentration does not rise. At some particular exercise intensity, which varies among subjects, both lactate production and removal begin to increase. As the work rate increases still further, lactate production surpasses the removal, and muscle and blood lactate concentration rise (MacDougall, 1977).

Wasserman et al. (1964) felt that this particular exercise intensity corresponded to the AT because an analysis of blood samples obtained during testing revealed that there was a concomitant increase in blood lactate concentration, and decreases in blood bicarbonate and blood pH. Investigators offered the following explanation for their conclusion. Lactic acid that forms in the muscle cell during the graded exercise test diffuses across the cell membrane into the blood where it is buffered predominantly by the bicarbonate buffer system according to Equation 1.

$CH_3CHOHCOOH + NaHCO_3 \rightarrow CH_3CHOHCOONa + H_2CO_3$ (Equation 1)

Lactic Acid + Sodium Bicarbonate --> Sodium Lactate + Carbonic Acid

The carbonic acid that is formed is highly volatile and dissociates into water and carbon dioxide as shown in Equation 2, thereby increasing the partial pressure of carbon dioxide in the blood.

$H_2CO_3 \rightarrow H_2O + CO_2$ (Equation 2)

Carbonic Acid --> Water + Carbon Dioxide

A non-linear increase observed in ventilation volume may be an attempt by the respiratory centre to compensate for metabolic acidosis that results from the buffering of lactate in the blood. Non-linear increase in the volume of carbon dioxide produced is due to the combined effect of hyperventilation and higher levels of carbon dioxide (from lactate) in the blood. The non-linear increase in the respiratory exchange ratio, the ratio between the carbon dioxide produced and oxygen consumed (measured at the lungs), is a consequence of the non-linear increase in the carbon dioxide production. The fact that there is no change in the value of the respiratory exchange ratio as long as the bicarbonate concentration is constant only reinforces the investigator's suspicion that changes observed in the respiratory gas exchange variables are due to changes occurring within the blood. Based on these observations, Wasserman et al. (1973) defined the AT as the power output or oxygen consumption at which ventilation volume, volume of carbon dioxide produced, and the respiratory exchange ratio increase non-linearly during a graded exercise test.

<u>**Controversy Regarding Use of the Term "Anaerobic Threshold"**</u>

The term "anaerobic threshold" has been the target of considerable controversy with the accuracy of the words "anaerobic" and "threshold" being questioned (Walsh & Banister, 1988). Hill et al. (1924) and Wasserman and McIlroy (1964) believe that the increase in blood lactate is inextricably linked to the onset of local muscle hypoxia at a certain work rate. Some investigators have challenged the idea that muscle hypoxia is the cause of increase in blood lactate at a particular work rate (Holloszy, 1973; Whipp and Mahler, 1980). They emphasize that blood lactate concentration is the net result of lactate production and removal, whereby the rise may not necessarily indicate the onset of increased production by exercising muscle. Rather, it could have occurred much earlier but may not have caused an increased blood lactate concentration because lactate removal had also increased. It is known that non-exercising muscle (Essen et al., 1975), and some internal organs (Wahren et al., 1975; Whipp, 1983) can metabolize lactate. The debate over the "anaerobic" portion of the term has caused some investigators to use a less mechanistic descriptor (e.g., lactate threshold).

Use of the word "threshold" has also been challenged. Some researchers believe that during graded exercise, blood lactate begins to increase during initial work rates (Davis &

Gass, 1981). Thus, they do not find evidence for a "threshold" or a range of work rates where blood lactate remains at its resting value.

**Detection of Anaerobic Threshold using the Ventilatory Threshold Method**

One non-invasive method by which to identify the anaerobic threshold, involves identification of the ventilatory threshold (VT). This technique requires monitoring a variable that decreases or remains relatively unchanged over a number of work rates before it begins to increase (break point). The process is described below.

Above AT, the volume of oxygen consumed ($\dot{V}O_2$) remains relatively linear while the volume of carbon dioxide produced ($\dot{V}CO_2$) is accelerated as a consequence of bicarbonate buffering of lactic acid. This accelerated $\dot{V}CO_2$ increase is usually accompanied by a parallel increase in the volume of expired air ($\dot{V}_E$) thereby keeping partial pressure of $CO_2$ in arterial blood ($PaCO_2$) and partial pressure of expired tidal carbon dioxide ($P_{ET}CO_2$) relatively constant (Davis, 1985). Because the rate of $\dot{V}O_2$ increase remains linear while the $\dot{V}_E$ and $\dot{V}CO_2$ increases accelerate, the ventilatory equivalent for $O_2$ ($\dot{V}_E/\dot{V}O_2$), which is decreasing or remains unchanged below AT, starts to increase above AT without an increase in the ventilatory equivalent for $CO_2$ ($\dot{V}_E/\dot{V}CO_2$). Because this phenomenon represents hyperventilation with respect to oxygen, partial pressure of expired tidal oxygen ($P_{ET}O_2$) starts its increase at AT. In contrast, $P_{ET}CO_2$ does not decrease above the AT for about 2 minutes because of the close tracking of $\dot{V}_E$ to $\dot{V}CO_2$ (isocapnic buffering). Due to the fact that $\dot{V}_E/\dot{V}O_2$ increases without a simultaneous increase in $\dot{V}_E/\dot{V}CO_2$ and $P_{ET}O_2$ increases without a reciprocal decrease in $P_{ET}CO_2$ only when metabolic acidosis occurs without respiratory compensation, these observations are specific gas exchange indicators of AT. It discriminates between changes in ventilation secondary to metabolic acidosis from other causes of increase in ventilation (e.g., pain, anxiety, and hypoxic increase in ventilatory drive), since those mechanisms cause reciprocal changes in $P_{ET}O_2$ and $P_{ET}CO_2$ and a simultaneous increase in $\dot{V}_E/\dot{V}CO_2$ and $\dot{V}_E/\dot{V}O_2$ rather than isocapnia at the transition point. Respiratory exchange ratio (R), which usually increases slowly as work rate is incremented, increases more rapidly at AT (Wasserman, 1986).

This method is relatively easy to use to detect AT in subjects whose ventilatory control mechanism responds appropriately to the increase in $CO_2$ from buffering. Unfortunately, some normal subjects have insensitive chemoreceptors that cause $\dot{V}_E$ to lag behind the $\dot{V}CO_2$. Also the control mechanism of patients with obstructive lung disease may allow $PaCO_2$ to rise. Thus, the ventilatory response may not increase proportionally with the increase in $CO_2$ output at AT (Wasserman, 1986).

In summary, AT located by changes in ventilatory exchange is identified by the following criteria: (1) the $\dot{V}_E/\dot{V}O_2$ curve, having been flat or decreasing, begins to rise while the

$\dot{V}_E/\dot{V}CO_2$ curve remains constant or decreases, (2) the $P_{ET}CO_2$ work rate curve is slowly rising or constant, but the $P_{ET}O_2$ work rate curve, having been declining or flat, begins to rise, (3) the slope of the R work rate curve, having been flat or rising slowly, becomes more positive.

Wasserman et al. (1986) have shown that for "rapid" incremental exercise tests, $\dot{V}_E$ and $\dot{V}CO_2$ increase at the same rate for a few work rates beyond anaerobic threshold. This is evident by the fact that $\dot{V}_E/\dot{V}O_2$ does not increase at anaerobic threshold but remains stable implying that arterial $PCO_2$ is unchanged in this region where buffering of lactic acid is occurring (isocapnic buffering). Thus, the criterion of systematic increase in $\dot{V}_E/\dot{V}O_2$ without a concomitant increase in $\dot{V}_E/\dot{V}CO_2$ is the most specific gas exchange method for detection of anaerobic threshold. Caiozzo et al. (1982) compared several gas exchange indices of anaerobic threshold detection and found that indeed, ventilatory equivalent criterion yielded the best agreement with blood lactate estimates of anaerobic threshold.

Isocapnic buffering does not occur when the increment duration is long (e.g., 4 minutes). During these so-called "steady-state" incremental exercise tests, both ventilatory equivalents begin to increase at the same $\dot{V}O_2$. As the duration of the work rate increment is shortened, the region of isocapnic buffering becomes larger, exceeding 1 liter per $min^{-1}$ of $\dot{V}O_2$ above anaerobic threshold for rapid incremental exercise tests. Why this difference in the gas exchange response exists between slow and fast incremental exercise test is currently not known.

**Factors Affecting the Accuracy of Ventilatory Threshold**

During a graded exercise test, there are several factors that could affect the accuracy of power output or oxygen uptake at AT when it is detected by the respiratory gas exchange criteria described above (Bhambhani, 1982).

(1) Blood Temperature.

An increase in blood temperature during exercise will slowly result in excess carbon dioxide being ventilated without a change in the pressure of arterial or alveolar carbon dioxide because it increases the dissociation constant of carbonic acid and decreases the solubility of carbon dioxide (Naimark et al., 1964). Since the changes are slow and small, breath by breath measurements can be used to identify this effect (Bhambhani, 1982).

(2) Oxymyoglobin Stores.

Oxymyoglobin, which has limited stores in humans (i.e., 200 ml on the average), is utilized during exercise but is not reflected in oxygen uptake measured at the lungs even though it results in the production of carbon dioxide which acts as a stimulus for the ventilatory centre. Its overall effect during exercise is minimal because amounts are so limited (Naimark et al., 1964).

(3) Substrate Concentration.

No change is observed in the oxygen consumption at AT when blood glucose levels are elevated, but when blood born free fatty acid levels are high, the $O_2$ is significantly increased (Ivy et al., 1979). The effect of glycogen depletion on the AT is controversial, as both a ten percent decrease in $O_2$ has been reported (Wiswell et al, 1980), while Hughes et al. (1982) found no significant differences.

(4) Mode of Exercise.

The mode of exercise seems to influence both the values of $\dot{V}O_2$max and oxygen consumption at AT. Davis et al. (1976) reported significantly lower values for arm cranking compared to leg cycling or treadmill walk-running. When these values were expressed as a percentage of respective maximum oxygen uptakes for the three modes of exercise, significantly lower values were once again observed for arm work compared to leg exercises which were not significantly different from each other. These investigators attributed the lower values during arm cranking to: (a) smaller muscle mass being used, (b) lower training level of the arms compared to the legs, (c) differences in motor unit recruitment patterns between the arms and legs, and (4) possible differences in the fibre type distribution between arms and legs.

(5) Duration of Exercise at Each Intensity.

Too short a duration at each intensity of a graded exercise test (i.e., less than four minutes), would lead to an overestimation of power output and oxygen consumption at AT because of: (1) utilization of stored phosphagens and oxymyoglobin in the muscles which transiently support the energy requirements for the first few seconds at each intensity (Clode and Campbell, 1969), and (2) delay in diffusion of lactic acid from the muscle into the blood which would result in lactate concentration measured at a particular intensity being due to conditions that existed at the previous intensity (Stamford, et al., 1978). Too long a duration at each intensity would unnecessarily prolong the duration of the test with the individual being subjected to undue stress (Wasserman et al., 1973).

**Detection of Anaerobic Threshold using the Lactate Threshold Method**

Blood analysis is an invasive technique used to determine lactate threshold (LT). Thus, the point at which the lactic acid level significantly breaks away is termed "lactate threshold".

Lactate concentration increases in the blood during exercise secondary to increased muscle lactate production, while the blood level depends on the balance between the rate of increased production and increased removal. Muscle lactate can increase because of two potential mechanisms according to Wasserman (1986): (1) glycolysis increases so rapidly that the mitochondria cannot utilize pyruvate rapidly enough to prevent its elevation in the cytosol, which in turn results in lactate increase by mass action (low mitochondrial/glycolytic capacity), and (2) mitochondrial membrane shuttle, which normally reoxidizes reduced cytosolic NAD and transfers protons and electrons to mitochondrial co-enzymes for eventual combination with oxygen becomes rate limited. This causes a change in cell redox state, thus converting pyruvate to lactate and accelerating glycolysis, providing more substrate for this reaction, as well as giving the mitochondria fuel.

The first hypothesis assumes that accelerated glycolysis causes a primary increase in pyruvate which overwhelms the mitochondrial capability to utilize it, forcing lactate to rise (Holloszy and Booth, 1976). Another similar hypothesis is that lactate increases are caused by the sequential contraction of fiber types, in which type I fibers (high mitochondrial/glycolytic capacity) are the predominant contracting fibers at low and moderate work rates, and type II fibers (low mitochondrial/glycoytic capacity) contract proportionally more at higher work rates. Since no change in redox state is implied, pyruvate must increase before or with lactate (i.e., a mass action effect).

Donovan and Brooks (1983) postulated that increased blood lactate concentration results from reduced lactate clearance, but this hypothesis also requires that pyruvate increase when lactate increases. Pyruvate was not measured in any of the studies purported to support hypothesis 1.

To distinguish between the two hypotheses (i.e., lactate increase by mass action versus an increase accompanying a change in cell redox state), Wasserman et al. (1986), simultaneously studied arterial lactate and pyruvate during an exercise test in which work rate was incremented each minute to the subject's maximum. Also, changes in lactate and pyruvate were measured at the transition from exercise to recovery when the $O_2$ requirement is abruptly reduced. The studies, coupled with reports from other researchers (Idstrom et al., 1985; Ketal et al., 1985) showed that the increase in lactate concentration during exercise leads to a metabolic acidosis, accelerated rate of glycogen depletion in the muscle and reduction in energy charge, and important changes in ventilatory and gas exchange dynamics. While small changes in lactate occur at low work rates, these are proportional to changes in pyruvate (i.e., compatible with accelerated glycolysis without a change in redox state). Major changes in lactate at and above the lactate threshold, in contrast, appear to be primarily determined by $O_2$ availability and change in cell redox.

Conflicting information has been published concerning the possibility that lactate anaerobic threshold may be influenced by the rate of increase in work rate. Hughson and Green (1982) reported that slow ramp tests yielded lower anaerobic threshold values than those found for fast ramp testing. Conversely, Wasserman et al. (1973) and Yoshida (1984) found similar anaerobic threshold values during exercise testing for both 1 and 4-minute increment durations (the increment size was the same for both tests).

**Validity of Lactate and Ventilatory Thresholds**

Numerous studies have examined the validity of the VT method of anaerobic threshold. Several teams of researchers (Caiozzo et al., 1982; Davis et al., 1976; Yoshida et al., 1981) have found close agreement between AT determined by the non-invasive VT technique and by blood lactate. For example, in the investigation conducted by Caiozzo et al. (1982), the systematic increase in $\dot{V}_E/\dot{V}O_2$ without a concomitant increase in $\dot{V}_E/\dot{V}CO_2$ yielded an anaerobic threshold highly correlated (r=0.93) with those determined from the break point in blood lactate in 16 subjects.

Meanwhile, other researchers have reported results that are not as encouraging, although Davis (1985) pointed out that the investigators failed to carefully follow the detection criteria (Davis, 1984) or report their results accurately (Powers et al., 1984).

Many researchers who have reported high correlations between LT and VT agree that LT consistently occurs some time after VT. For example, Posner et al. (1987) found that LT occurred a mean 2.3 minutes after VT. As a result, VT occurred at 47% of $\dot{V}O_2$max and LT at 61%. Yeh et al. (1983) attributed this delay in LT to the fact that the rise in venous lactate lags behind the rise in arterial lactate by 1.5 minutes. Venous lactate is influenced by a number of mechanisms including lactate metabolism in working and non-working muscle and other body tissues (Hughs et al., 1982) encountered in the transit from muscle to sampling site. Posner et al. (1987)

pointed out that LT occurs at different times during exercise depending on where one is sampling (systemic arterial, venous, or drawn from blood flowing from specific muscle groups).

Keyser et al. (1989) gathered data on cardiovascular responses occurring at AT during intermittent CE and arm cranking in eight healthy college students. AT was determined by plotting expired minute volume on oxygen uptake. Analysis of variance revealed AT was similar for arm and cycle tests. Absolute $\dot{V}O_2$ at AT was higher for cycle than for arm cranking exercise ($p<.05$). Heart rate was higher for CE than for arm work ($p<.05$).

Nikolic & Todorovic (1984) determined AT in 19 female and 41 male physical education students during incremental arm and leg exercise on a CE and arm crank respectively. AT values were determined from the non-linear increase in $\dot{V}_E$. The $\dot{V}_E$ at AT was lower during arm rather than leg exercise in both groups. AT during leg exercise showed a correlation of 0.38 for $\dot{V}O_2max$ and 0.62 for HR at AT for males. Similar correlations were obtained during arm exercise.

When Davis et al. (1976) examined anaerobic threshold (gas exchange method) and maximal aerobic power for arm cranking, leg cycling, and treadmill walking/running, no significant difference was found between AT mean values for leg cycling and treadmill exercising. Meanwhile, the mean AT for arm cranking was significantly lower than for the two lower-body testing modes. Analysis of the relationship between AT and $\dot{V}O_2max$ resulted in significant coefficients of 0.60, 0.52, and 0.70 for arm cranking, leg cycling, and treadmill exercising respectively.

**Reliability of Lactate and Ventilatory Thresholds**

(1) Inter-Tester Reliability

Posner et al. (1987) reported close correlation of VT measurements made by three evaluators (r=0.94); this finding was similar to other studies including those conducted by Davis et al. (1979), Caiozzo et al. (1982), and Powers et al (1984).

(2) Test-Retest Reliability

Posner et al. (1987) tested thirty elderly male subjects (mean age = 71 years) twice, and reported high correlation (r= 0.97) between the two trials. Aunola and Rusko (1984), engaged 33 men, aged 20 to 50 years, to complete two exercise tests on a cycle ergometer. The VT reproducibility was high (r=0.95) during the 2 minute incremental exercise test. In another study using the cycle ergometer, Nemoto and Miyashita (1980) tested 20 non-athletes and 10 athletes (aged 20.9-31.5 years). The test-retest correlation coefficient for VT was 0.78. Test-retest coefficients of 0.77, 0.74, and 0.72 were calculated by Davis et al. (1976) for AT (gas exchange method) for arm crank, CE, and treadmill walking/running respectively.

**Physiological Factors Influencing the Anaerobic Threshold**

Three physiological factors influence the anaerobic threshold, as outlined by Bhambhani (1982).

(1) Fiber Type.

Conflicting results have been reported with respect to there being a significant correlation between the percentage of slow twitch fibre types and maximum oxygen uptake. For example, Rusko et al (1980) observed no significant correlations between percentage of slow twitch fibres in the vastus lateralis muscle and $\dot{V}O_2max$ at AT expressed as ml/kg/min or as a percentage of maximum oxygen uptake. This was unexpected because these fibres are characterized by a high oxidative and low glycolytic capacity; therefore, a positive correlation was anticipated. Meanwhile, Wenger and Reed (1976) and Ivy et al. (1980) in fact reported positive correlations.

(2) Enzyme Activities.

Evidence to support the hypothesis that an increase in activity of oxidative enzymes would have a tendency to delay the production of lactic acid and thereby increase oxygen uptake at AT, is controversial. Green et al. (1979) did not observe a significant correlation between oxygen consumption at AT expressed as a percentage of $\dot{V}O_2max$ and succinate dehydrogenase activity. Meanwhile, Rusko et al. (1980) obtained significant correlations between these variables. These investigators did however, report a negative correlation between total activity of the enzyme lactate dehydrogenase which is predominant in type IIb skeletal muscle fibres and responsible for the conversion of pyruvate to lactate in the final step of the glycolytic pathway. This correlation was expected because high activity in this enzyme should result in a faster accumulation of lactic acid, consequently resulting in a lower oxygen consumption at AT or vice versa.

(3) Menstrual Cycle.

Stephenson et al. (1980) discovered no significant differences between measurements of oxygen consumption at AT and $\dot{V}O_2max$ on different cycle days.

**The Influence of Physical Training on Anaerobic Threshold**

It is known that AT can be elevated through training and that successful long-distance athletes have significantly higher thresholds than do persons who do not train for long-distance events. In trained individuals, the anaerobic threshold may not be observed until the subject exercises to a level that is 10-20 times the resting metabolic rate. In both absolute terms and expressed as a percentage of $\dot{V}O_2max$, AT is high in the endurance-trained athlete (i.e., 70 to 80% of $\dot{V}O_2max$). In sedentary able-bodied subjects however, the anaerobic threshold might occur at about 4 times the resting metabolic rate, or 50 to 60% of $\dot{V}O_2max$ (Davis, 1985).

It is generally accepted that an athlete's AT is a very critical factor in determining the potential for sustaining prolonged physical exercise at a high percentage of $\dot{V}O_2max$ (Wasserman, 1986). Powers et al. (1984), using nine runners showed that anaerobic threshold correlated highly (r=0.94) with 10 km racing times. Kumagai et al. (1982) compared five and ten kilometer times to AT and $\dot{V}O_2max$ in seventeen runners. They found correlations of 0.95 and 0.84 respectively, for race pace versus AT. Corresponding correlations of $\dot{V}O_2max$ were 0.65 and 0.67 respectively.

According to Bhambhani (1982), the effect of physical training on AT can be studied in two ways- via an indirect method or a direct technique. The indirect method looks at the relationship between the oxygen consumption at AT and maximum oxygen consumption uptake. As many investigators have reported (Getchell, 1976; Gibbons et al. 1983), physically fit persons have higher maximum oxygen uptakes than unfit individuals as a result of their ability to transport, deliver, and utilize oxygen more efficiently. In theory, these factors should delay the onset of lactic acid production and thereby result in a higher oxygen consumption at AT in fit persons. Therefore, a significant positive correlation between oxygen consumption at AT and $\dot{V}O_2max$ should be evident. Several studies have examined this relationship and support this line of reasoning to some extent.

The direct technique requires observation of changes in AT as a result of training. Three studies, which are briefly described below, outline the effects of training programs on AT.

Following training, Hill et al. (1987) reported that the adjusted means for trained subjects were significantly greater than for controls for $\dot{V}O_2max$ (6%), $\dot{V}O_2$ (23%), and % $\dot{V}O_2max$ (13%). AT was detected from changes in ventilatory exchange

(VT). Exercising subjects completed 18 interval training sessions (five x five minutes of cycling at 90 to 100% $\dot{V}O_2max$) and eight continuous training sessions (40 minutes of running or cycling) in six weeks.

Henritze et al. (1985) identified post-training improvements in their investigation which appealed to thirty-three college women. Blood lactate was analyzed to identify AT. Post-testing for subjects who trained above LT five days per week for twelve weeks revealed significantly higher $\dot{V}O_2max$ (13%), $\dot{V}O_2$ lactate threshold (47%), and $\dot{V}O_2$ lactate threshold/$\dot{V}O_2max$ (33%) values as compared to the control group.

Smith and O'Donnell (1984) recruited six healthy males to participate in a 36 week training program. The subjects undertook a running program during which they started at an average of 20 and progressed to 73 kilometers per week. After the initial sessions, subjects were advised to train at close to the maximum pace that they could sustain for about an hour. Significant increases were seen in $\dot{V}O_2max$ of 13.6%, AT (32.3%), and AT as a % of $\dot{V}O_2max$ (17%). AT was determined indirectly by following changes in ventilation and respiratory gas exchange.

**Significance of the Anaerobic Threshold**

A high AT is required by a long duration endurance athlete because it allows the athlete to work at a higher intensity for longer periods of time at lower oxygen cost. Three reasons, as identified by Bhambhani (1982), will be discussed.

(1) Ability to Perform at a Higher Exercise Intensity.

Cross-sectional (Hermansen and Saltin, 1967) and longitudinal studies (Ekblom, 1969) have shown that blood lactate accumulation at the same submaximal work rate is reduced after endurance training. Several investigations also suggest that AT is increased after training relative to $\dot{V}O_2max$ (Hermansen et al., 1967; Williams et al., 1968). Davis et al. (1979) showed that after 9 weeks of endurance training AT improved 44% in terms of absolute $\dot{V}O_2$ and 15% relative to $\dot{V}O_2max$ in previously sedentary middle-aged men. As well, Costill (1970) observed that trained individuals were able to work closer to their AT than untrained persons. Thus, the athlete with the higher AT should be able to perform at a higher intensity than the competitor with a lower AT.

(2) Ability to Perform for Prolonged Periods.

One reason for the close relationship between AT and endurance performance relates to the rate of muscle glycogen breakdown. Because long-term, high-intensity exercise results in, and is perhaps ultimately limited by muscle glycogen depletion (Karlsson, 1971), exercise just below AT results in a much slower reduction of muscle glycogen stores than exercise above AT and therefore is tolerable for much longer periods of time. This is because glycogen is used at a rate that is 18-19 times faster during anaerobic glycolysis compared to oxidative phosphorylation for the same energy yield. Boyd et al. (1974) have demonstrated that elevations in blood lactate concentration inhibit lipolysis in exercising persons and thus force obligatory carbohydrate utilization.

(3) Ability to Perform at a Lower Oxygen Cost.

At exercise intensities above AT, ventilation volume increases non-linearly due to additional stimulus to the respiratory centre as a result of increased carbon dioxide production (Wasserman et al., 1967), thereby increasing the $\dot{V}_E/\dot{V}O_2$ ratio. An increase in this ratio indicates that oxygen cost of ventilation is increased, perhaps making less oxygen available to the muscles that are directly involved in the exercise. Therefore, in order to continue working at the same intensity it will be necessary to increase oxygen consumption, thereby increasing the total energy cost of the exercise (MacDougall, 1977). Thus, if two individuals are exercising at an intensity which is higher than AT of one and lower than that of the other, then mechanical efficiency in terms of energy cost will be greater in the latter subject.

## **Summary**

The condition of cerebral palsy was discussed, as well as some physiological responses that can be monitored during graded exercise testing. Unfortunately, information related to these parameters on persons with cerebral palsy is almost non-existent. Knowledge gained in these areas may have some important implications for fitness program prescription of athletes with cerebral palsy, or individuals with CP who are physically active for their general well-being.

**Statement of Purpose**

In consideration of the potential importance of cardiorespiratory fitness and associated parameters, an initial study was designed:

(1) To investigate validity of $\dot{V}O_2$max during wheelchair ergometry in athletes [illegible] CP by comparing these values with those obtained [illegible] cycle ergometry, and secondly

(2) To investigate the test-retest reliability of $\dot{V}O_2$max and other physiological measurements, including heart rate, expired ventilation volume, respiratory exchange ratio, oxygen pulse, ventilatory equivalent for oxygen, lactate, and rating of perceived exertion, during both these modes of exercise.

A second project was undertaken with a larger sample size:

(1) To investigate validity and reliability of the ventilatory threshold and lactate threshold methods in identifying anaerobic threshold during wheelchair and cycle ergometry, and

(2) To further investigate validity and test-retest reliability of $\dot{V}O_2$max and other physiological measurements in athletes with CP during wheelchair ergometry and cycle ergometry.

**Validity and Reliability**

The goals of this thesis, as stated above, are primarily related to the concepts of validity and reliability. It is important therefore, to outline definitions of these two concepts as they are applied in the following chapters; this will ensure that the reader fully understands the methods, discussion, and conclusions presented by the author.

(1) Validity.

The criteria for the WE to be deemed a valid mode for measuring $\dot{V}O_2max$ was two-fold: (1) Using an ANOVA, $\dot{V}O_2max$ mean values for two trials were not to be significantly different from those collected on the WE since the CE was labelled the "gold standard", and (2) Using, the Pearson product-moment correlation, $\dot{V}O_2max$ scores on the WE would have to be statistically correlated with $\dot{V}O_2max$ scores on the CE over both trials. Since the sample size for this thesis was small, correlation coefficients had to be close to 1.00. For example, for pools of 4, 6, and 11 subjects, 'r' values of 0.95, 0.81, and 0.67 respectively were required for statistical significance. The reader must realize that research referred to in this thesis usually had larger sample sizes (because most included non-disabled subjects). Hence, smaller 'r' values were required to detect statistical significance for those investigators. When the same 'r' values are calculated for studies using large and small sample sizes, the reader must exercise caution during the interpretation of the results because there is greater uncertainty in the results for the small subject pool.

In the second study, the VT and LT methods would be considered valid techniques to identify anaerobic threshold if scores for each of the two methods were significantly correlated, using the Pearson product-moment correlation.

(2) Reliability.

Test-retest reliability and inter-tester reliability were investigated in this thesis, using the Pearson product-moment correlation. Test-retest reliability was evident when scores over two trials were statistically correlated. Inter-tester reliability was evident when the evaluators assigned threshold values that were statistically correlated.

**"Maximal Oxygen Uptake" versus "Peak Oxygen Uptake"**

A distinction is made by some researchers between "maximal oxygen uptake" ($\dot{V}O_2max$) and "peak oxygen uptake" ($p\dot{V}O_2$). $\dot{V}O_2max$ is considered to be the highest oxygen uptake score possible, and requires use of the largest muscle groups in the body. In the absence of a plateau or when the large muscle groups of the body (i.e., legs) are not used in the exercise, one cannot be certain that the highest or peak oxygen uptake is indeed the subjects's maximal oxygen uptake. In this thesis, the term "maximal oxygen uptake" is used, whatever mode or muscle groups are used.

## Hypotheses

The following hypotheses were examined in the two studies:

(1) With the cycle ergometer as criterion reference, the wheelchair ergometer would be considered a valid and reliable mode of measurement of $\dot{V}O_2max$ in wheelchair athletes with cerebral palsy.

(2) The wheelchair and cycle ergometers would be reliable modes to measure additional physiological responses including heart rate, expired ventilation volume, respiratory exchange ratio, oxygen pulse, ventilatory equivalent for oxygen, lactate, and rating of perceived exertion in wheelchair athletes with cerebral palsy.

(3) No significant differences would be observed between lactate threshold for two trials on the cycle ergometer and wheelchair ergometer.

(4) No significant differences would be observed between ventilatory threshold for two trials on the cycle ergometer and wheelchair ergometer.

(5) No significant differences would be observed between two expert evaluators in the identification of the lactate and ventilatory thresholds.

(6) Significant differences would be observed between lactate and ventilatory thresholds.

**References**

American College of Sports Medicine (1990). The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults. Medicine and Science in Sports and Exercise, 22,265-274.

Astrand, P., & Rodahl, K. (1986). Textbook of work physiology; physiological bases of exercise (3rd ed.). New York, New York: McGraw-Hill Book Company.

Aunola, S., & Rusko, H. (1984). Reproducibility of aerobic and anaerobic thresholds in 20-50 year old men. European Journal of Applied Physiology and Occupational Physiology, 53, 260-266.

Bar-Or, O., & Zwiren, D. (1975). Maximal oxygen consumption test during arm exercise- reliability and validity. Journal of Applied Physiology, 38(3), 424-426.

Baldwin, K., Winder, W., Terjung, R., & Holloszy, J. (1972). Glycolytic capacity of red, white, and intermediate muscle: adaptive response to running. Medicine and Science of Sports and Exercise, 4, 50.

Benzi, G., Panceri, P. DeBernardi, M., Villa, R., Arcelli, E., d'Angelo, L., Arrigoni, E., & Berte, F. (1975). Mitochondrial enzymatic adaptation of skeletal muscle to endurance training. Journal of Applied Physiology, 38(4), 565-569.

Bhambhani, Y. (1982). Ventilatory thresholds during a graded exercise test: the effects of three training intensities in males. Unpublished doctoral dissertation, University of Alberta, Edmonton.

Bleck, E.E. (1982). Cerebral palsy. In Bleck, E.E. & Nagel, A.N. (Eds.), Physically handicapped children: a medical atlas for teachers (2nd ed.). New York, New York: Grune & Stratton.

Bobath, B. (1976). Abnormal postural reflex activity caused by brain lesions (2nd ed.). London, England: William Heinemann Medical Books Limited.

Boyd, A.E., Glamber, S.R., Mager, M., & Lebovity, H.E. (1974). Lactate inhibition of lipolysis in exercising man. Metabolism, 23, 531-542.

Boyer, J., & Lasch, F. (1970). Exercise therapy in hypertensive men. Journal of the American Medical Association, 211, 1668-1671.

Buskirk, E., Iampietro, P., & Bass, D. (1958). Work performance and dehydration; effects of physical condition and heat acclimatization. Journal of Applied Physiology, 12, 189-194.

Caiozzo, V.J., Davis, J.A., Ellis, J.F., Azus, J.l., Vandagriff, R., Prietto, C.A., & McMaster, W.C. (1982). A comparison of gas exchange indices used to detect the anaerobic threshold. Journal of Applied Physiology, 53, 1184-1189.

Clausen, J., Trap-Jensen, J., & Lassen, N. (1970). The effects of training on the heart rate during arm and leg exercise. Scandinavian Journal of Clinical and Laboratory Investigations, 26, 295-301.

Clode, M., & Campbell, E.J.M. (1969). The relationship between gas exchange and changes in blood lactate concentrations during exercise. Clinical Science, 37, 263-272.

Cohen, K., & Fox, E. (1975). Intensity and distance of interval training programs and metabolic changes in females. Unpublished manuscript.

Costill, D.L. (1970). Metabolic responses during distance running. Journal of Applied Physiology, 28, 251-255.

CP-ISRA (1990). Classification and sports rules manual (5th ed.). The Netherlands: Author.

Davies, C., & Knibbs, A. (1971). The training stimulus: the effects of intensity, duration and frequency of effort on maximum aerobic power output. Internationale Zeitschrift fur Angewandte Physiologie, 29, 299-305.

Davis, G., Plyley, M.J., & Shephard, R.J. (1991). Gains of cardiorespiratory fitness with arm-crank training in spinally disabled men. Canadian Journal of Sports Sciences, 16(1), 64-72.

Davis, J.A., & Gass, G.C. (1981). The anaerobic threshold as determined before and during lactic acidosis. European Journal of Applied Physiology and Occupational Physiology, 47, 141-149.

Davis, J.A. (1984). Validation and determination of the anaerobic threshold (Letter to the Editor). Journal of Applied Physiology, 57, 611.

Davis, J.A. (1985). Anaerobic threshold: review of the concept and directions for future research. Medicine and Science in Sports and Exercise, 17(1), 6-18.

Davis, J.A., Frank, M.H., Whipp, B.J., & Wasserman, K. (1979). Anaerobic threshold alterations caused by endurance training in middle-aged men. Journal of Applied Physiology, 46, 1039-1046.

Davis, J.A., Vodak, P., Wilmore, J.H., Vodak, J., & Kurtz, P. (1976). Anaerobic threshold and maximal aerobic power for three modes of exercise. Journal of Applied Physiology, 41, 97-108.

DeJours, P. (1966). Respiration. New York, New York: Oxford University Press.

Donovan, C.M., & Brooks, G.A. (1983). Endurance training affects lactate clearance, not lactate production. American Journal of Physiology, 244, E83-E92.

Dunn, J. (1988). Cerebral palsy, the condition. In Coaches manual. Ontario: Ontario Cerebral Palsy Sports Association.

Ekblom, B. (1969). Effect of physical training on oxygen transport system in man. Acta Physiologica Scandanavica Supplementum, 328.

Ekblom, B., & Hermansen, L. (1968). Cardiac output in athletes. Journal of Applied Physiology, 25(5), 619-625.

Eriksson, B., Gollnick, P., & Saltin, B. (1970). Muscle metabolism and enzyme activities after training in boys 11-13 years old. Acta Physiologica Scandinavica, (87), 485-497.

Essen B.B., Pernow, P.D., Gollnick, P.D. & Saltin, B. (1975). Muscle glycogen content and lactate uptake in exercising muscles. In H. Herald & J.R. Poortmans (Eds.), Metabolic adaptations to prolonged physical exercise (pp. 1-134). Basel: Birkhauser.

Ettinger, A.V. (1989). Cardiovascular and perceived exertion responses to cycling velocities. Unpublished master's thesis, University of Alberta, Edmonton.

Faria, I. (1970). Cardiovascular response to exercise as influence by training of various intensities. Research Quarterly, 41, 44-50.

Fink, W., Costill, D., Daniels, J., Pollock, M., & Saltin, B. (1975). Muscle fiber composition and enzyme activities in male and female athletes. Physiologist, 18(3), 213.

Fox, E. (1975). Differences in metabolic alterations with sprint versus endurance interval training programs. In Howald, H., & Poortmans, J. (Eds.), Metabolic adaption to prolonged physical exercise. Basel, Switzerland: Birkhauser Verlag.

Fox, E., Bartel, R., Billings, C., O'Brien, R., Bason, R., & Mathews, D. (1975). Frequency and duration of interval training programs and changes in aerobic power. Journal of Applied Physiology, 38(3), 481-484.

Fox, E.L., Bowers, R.W., & Foss, M.L. (1988). The Physiological Basis of Physical Education and Athletics. Dubuque, Iowa: Wm. C. Brown Publishers.

Fox, E., McKenzie, D., & Cohen, L. (1975). Specificity of training: metabolic and circulatory responses. Medicine and Science in Sports, 7(1), 83.

Frick, M., Elovainio, R., & Somer, T. (1967). The mechanism of bradycardia evoked by physical training. Cardiologia, 51, 46-54.

Frick, M., Sjogren, A., Persasalo, J., & Pajunen, S. (1970). Cardiovascular dimensions and moderate physical training in young men. Journal of Applied Physiology, 29(4), 452-455.

Getchell, B. (1976). Physical fitness, a way of life. New York, New York: John Wiley & Sons, Inc.

Gibbons, E.S., Jessup, G.T., Wells, T.D., & Werthmann, D.A. (1983). Effects of various training intensity levels on anaerobic threshold and aerobic capacity in females. Journal of Sports Medicine, 23, 315-318.

Gollnick, P., Armstrong, R., Saltin, B., Saubert, C., Sembrowich, W., & Shepherd, R. (1973). Effect of training on enzyme activity and fiber composition of human skeletal muscle. Journal of Applied Physiology, 34(1), 107-111.

Gollnick, P., Armstrong, R., Saubert, C., Piehl, L., & Saltin, B. (1972). Enzyme activity and fiber composition in skeletal muscle of untrained and trained men. Journal of Applied Physiology, 33(3), 312-319.

Green, H., Daub, R., Painter, D., Houston, M., & Thomson, J. (1979). Anaerobic threshold and muscle fiber type, area and oxidative enzyme activity during graded cycling. Medicine and Science in Sports, 11(113). Abstract.

Gueli, D. & Shephard, R.J. (1977). Pedal frequency in bicycle ergometry. Canadian Journal of Applied Sports Science, 1, 137-141.

Guttmann, L. (1976). Spinal cord injuries; comprehensive management and research. Oxford, England: Blackwell Scientific Publications.

Henritze, J., Weltman, A., Schurrer, R.L., & Barlow, K. (1985). Effects of training at and above the lactate threshold on the lactate threshold and maximal oxygen uptake. European Journal of Applied Physiology and Occupational Physiology, 54, 84-88.

Hermansen, L., & Wachtlova, M. (1971). Capillary density of skeletal muscle in well-trained and untrained men. Journal of Applied Physiology, 30(6), 860-863.

Hermansen, L., Hultman, E., & Saltin, B. (1967). Muscle glycogen during prolonged severe exercise. Acta Physiologica Scandanavica, 71, 129-139.

Hill A.V., Long, C.N.H., & Lupton, H. (1924). Muscular exercise, lactic acid, and the supply and utilization of oxygen. Par VI- the oxygen debt at the end of exercise. Proceedings of the Royal Society of Medicine, Series B97, 127-137.

Hill, D.W., Cureton, K.J., Grisham, S.C., & Collins, M.A. (1987). Effect of training on the rating of perceived exertion at the ventilatory threshold. European Journal of Applied Physiology and Occupational Physiology, 56, 206-211.

Holloszy, J. (1967). Effects of exercise on mitochondrial oxygen uptake and respiratory enzyme activity in skeletal muscle. Journal of Biological Chemistry, 242, 2278-2282.

Holloszy, J. (1973). Biochemical adaptations to exercise: aerobic metabolism. In Wilmore, J., (Ed.), Exercise and sports sciences reviews (pp 45-71). New York, New York: Academic Press.

Holloszy, J.O., & Booth, F.W. (1976). Biochemical adaptations to exercise training in muscle. Annual Review of Physiology, 38, 273-291.

Hughes, E.F., Turner, S.C. & Brooks, G.A. (1982). Effects of glycogen depletion and pedaling speed on "anaerobic threshold". Journal of Applied Physiology, 52, 1598-1607.

Hughson, R.L., & Green, H.J. (1982). Blood acid-base and lactate relationships studied by ramp work tests. Medicine and Science in Sports and Exercise, 14, 297-302.

Idstrom, J.P., Harihara Subramanian, V., Chance, B., Schersten, T., & Bylund-Felenius, A.C. (1985). Oxygen dependence of energy metabolism in contracting and recovering rat skeletal muscle. American Journal of Physiology, 248, H40-H48.

Ivy, J.L., Costill, D.L., Essig, D.A., Lower, R.W., & Van Handel, P.J. (1979). The relationship of blood lactate to the anaerobic threshold and hyperventilation. Medicine and Science in Sports, 11, 79. Abstract.

Ivy, J.L., Withers, R.T., Van Handel, P.J., Elger, D.H., & Costill, D.L. (1980). Muscle respiratory capacity and fiber type determinants of the lactate threshold. Journal of Applied Physiology, 48, 523-527.

Jones, J.A. (Ed.). (1988). Training guide to cerebral palsy sports (3rd ed.). Champaign, Illinois: Human Kinetics Books.

Karlsson, J. (1971). Lactate and phosphagen concentrations in working muscle of man. Acta Physiologica Scandanavica Supplementum, 358, 1-72.

Karlsson, J., Nordesjo, L., Jorfeldt, L., & Saltin, B. (1972). Muscle lactate, ATP, and CP levels during exercise after physical training in man. Journal of Applied Physiology, 33(2), 199-203.

Katch, V.L., Sady, S.S., & Freedson, P. (1982). Biological variability in maximum aerobic power. Medicine and Science in Sports and Exercise, 14(1), 21-25.

Kaufmann, D., Swenson, E., Fencl, J., & Lucas, A. (1974). Pulmonary function of marathon runners. Medicine and Science in Sports, 6(2), 114-117.

Ketal, L., Kreit, J., Simon, R., & Grum, C. (1985). Relation of plasma hypoxanthine to anaerobic threshold. Clinical Research, 33, 2. Abstract.

Keyser, R.E., Mor, D., & Andres, F.F. (1989). Cardiovascular responses and anaerobic threshold for bicycle and arm ergometer exercise. Archives of Physical Medicine and Rehabilitation, 70, 687-691.

Kilbom, A. (1971). Physical training with submaximal intensities in women. I. Reaction to exercise and orthostasis. Scandinavian Journal of Clinical and Laboratory Investigations, 28, 141-161.

Klissouras, V. (1972). Genetic limit of functional adaptability. Internationale Zeitschrift fur Angewandte Physiologie, 30, 85-94.

Knuttgen, H., Nordesjo, L., Ollander, B., & Saltin, B. (1973). Physical conditioning through interval training with young male adults. Medicine and Science in Sports, 5, 220-226.

Kumagai, S., Tanaka, K., Matsura, Y., Matsuzaka, A., Hirakoba, K., & Asano, K. (1982). Relationships of the anaerobic threshold with the 5 km, 10 km, and 10 mile races. European Journal of Applied Physiology, 49, 13-23.

Lamb, D.R. (1984). Physiology of exercise; responses and adaptations (2nd ed.). New York, New York: Macmillan Publishing Co., Inc.

Lundberg, A. (1984). Longitudinal study of physical working capacity of young people with cerebral palsy. Developmental Medicine and Child Neurology, 26, 328-334.

MacDougall, J.D. (1977). The anaerobic threshold- its significance to the endurance athlete. Canadian Journal of Applied Sports Sciences, 2, 137-140.

Magel, J.R., Foglia, G.F., McArdle, W.D., Gutin, B., Pechar, G.S., & Katch, F.I. (1975). Specificity of swim training on maximum oxygen uptake. Journal of Applied Physiology, 38, 151-155.

Mann, G., Garrett, H., Farhi, A., Murray, H., & Billings, F. (1969). Exercise to prevent coronary heart disease. American Journal of Medicine, 46, 12-27.

Mathews, D.K. & Fox E.L. (1976). The physiological basis of physical education and athletes. Philadelphia, Pennsylvania: W.B. Saunders Company.

McArdle, W., & Magel, J. (1970). Physical work capacity and maximum oxygen uptake in treadmill and bicycle exercise. Medicine and Science in Sports, 2(3), 118-123.

McArdle, W., Katch, F., & Pechar, G. (1973). Comparison of continuous and discontinuous treadmill and bicycle tests for max$\dot{V}O_2$. Medicine and Science in Sports, 5(3), 156-160.

McCubbin, J.A, & Shasby, G.B. (1985). Effects of isokinetic exercise on adolescents with cerebral palsy. Adapted Physical Activity Quarterly, 2, 56-64.

MacDougall, J.D. (1977). The anaerobic threshold: its significance for the endurance athlete, Canadian Journal of Applied Sports Sciences, 2, 137-140.

Morgan, T., Cobb, L., Short, F., Ross, R., & Gunn, D. (1971). Effects of long-term exercise on human muscle mitochondria. In Pernow, B., & Saltin, B. (Eds.), Muscle metabolism during exercise. New York, New York: Plenum Press.

Morganroth, J., Maron, B., Henry, W., & Epstein, S. (1975). Comparative left ventricular dimensions in trained athletes. Annals of Internal Medicine, 82, 521-524.

Nagle, F.J. (1973). Physiological assessment of maximal performance. In J. Wilmore (Ed.), Exercise and sport sciences reviews: Vol. 1, (pp. 313-338). New York, New York: Academic Press.

Naimark, A., Wasserman, K., & McIlroy, M.B. (1964). Continuous measurement of ventilatory exchange ratio during exercise. Journal of Applied Physiology, 19, 644-652.

Nemoto, I., & Miyashita, M. (1980). Aerobic and anaerobic threshold of Japanese male adults. Journal of Human Ergology, 9, 183-189.

Nikolic, Z. & Todorovic, B. (1984). Anaerobic threshold during arm and leg exercises and cardiorespiratory fitness test in a group of male and female students. International Journal of Sports Medicine, 5, 330-335.

Oscai, L., Williames, B., & Hertig, B. (1968). Effect of exercise on blood volume. Journal of Applied Physiology, 24(5), 622-624.

Pattengale, P., & Holloszy, J. (1967). Augmentation of skeletal muscle myoglobin by a program of treadmill running. American Journal of Physiology, 213, 783-785.

Pechar, G., McArdle, W., Katch, F., Magel, J., & DeLuca, J. (1974). Specificity of cardiorespiratory adaptation to bicycle and treadmill training. Journal of Applied Physiology, 36(6), 753-756.

Pollock, M. (1973). The quantification of endurance training programs. In Wilmore, J. (Ed.), Exercise and sport sciences reviews: Vol. 1., New York, New York: Academic Press.

Posner, J.D., Gorman, K.M., Klein, S., & Cline, C.J. (1987). Ventilatory threshold: measurement and variation with age. Journal of Applied Physiology, 63(4), 1519-1525.

Powers, S.K., Dodd, S., & Garner, R. (1984). Precision of ventilatory and gas exchange alterations as a predictor of the anaerobic threshold. European Journal of Applied Physiology and Occupational Physiology, 52, 173-177.

Reinhard, U., Juller, P.H., & Schmulling, R.M. (1979). Determination of anaerobic threshold by the ventilation equivalent in normal individuals. Respiration, 38, 36-42.

Rowell, L. (1974). Human cardiovascular adjustments to exercise and thermal stress. Physiology Review, 54(1), 75-159.

Rusko, H., Rahkila, P., & Karvinen, E. (1980). Anaerobic threshold, skeletal muscle enzymes and fiber composition in young female cross country skiers. Acta Physiologica Scandanavica, 108, 263-268.

Saltin, B. (1969). Physiological effects of physical training. Medicine and Science in Sports, 1(1), 50-56.

Saltin, B. (1986). Physiological adaptation of physical conditioning: old problems revisited. Acta Medica Scandinavica (Suppl. 711), 11-24.

Saltin, B., & Astrand, P. (1967). Maximal oxygen uptake in athletes. Journal of Applied Physiology, 23, 353-358.

Saltin, B., Blomqvist, G., Mitchell, J., Johnson, R., Wildenthal, K., & Chapman, C. (1968). Response to exercise after bedrest and after training. Circulation (Suppl. 7).

Saltin, B., Hartley, L., Kilbom, A., & Astrand, I. (1969). Physical training in sedentary middle-aged and older men. II. Oxygen uptake, heart rate and blood lactate concentrations at submaximal and maximal exercise. Scandinavian Journal of Clinical and Laboratory Investigations, 24, 323-334.

Shepard, R.J. (1990). Fitness in special populations. Champaign, Illinois: Human Kinetics Books.

Sherrill, C. (1986). Adapted Physical Education; a multidisciplinary Approach (3rd ed.). Wm. C. Brown Publishers: Dubuque, Iowa.

Smith, D.A. & O'Donnell, T.V. (1984). The time course during 36 weeks' endurance training of changes in $VO_2$max. and anaerobic threshold as determined with a new computerized method. Clinical Science, 67, 229-236.

Stamford, B.A., Weltman, A. & Fulco, C. (1978). Anaerobic threshold and cardiovascular responses during one versus two-legged cycling. Research Quarterly, 49, 351-362.

Stephenson, L.A., Lokla, M.A., & Wilderson, J.E. (1980). Anaerobic threshold, work capacity, and perceived exertion during the menstrual cycle. Medicine and Science in Sports and Exercise, 12, 87. Abstract.

Thoden, J.S. (1991). Testing aerobic power. In MacDougall, J.D., Wenger, H.A., & Green, H.J. (Eds.), Physiological Testing of the High-Performance Athlete (2nd ed.). Human Kinetics Books: Champaign, Illinois.

Wahren, J., Hagenfeld, L., & Felig, P. (1975). Glucose and free fatty acid utilization in exercise: studies in normal and diabetic man. Israel Journal of Medical Sciences, 11, 551-559.

Walsh, M.L. & Banister, E.W. (1988). Possible mechanisms of the anaerobic threshold; a review. Sports Medicine, 5, 269-302.

Wasserman, K. (1986). Anaerobiosis, lactate, and gas exchange during exercise: the issues. Federation Proceedings, 45, 2904-2909.

Wasserman, K., & McIlroy, M.B. (1964). Detecting the threshold of anaerobic metabolism in cardiac patients during exercise. American Journal of Cardiology, 14, 844-852.

Wasserman, K., Beaver, W.L., & Whipp, B.J. (1986). Mechanisms and patterns of blood lactate increase during exercise in man. Medicine and Science in Sports and Exercise, 18(3), 344-352.

Wasserman, K., Van Kessel, A.L., & Burton, G.G. (1967). Interaction of physiological mechanisms during exercise. Journal of Applied Physiology, 22, 71-85.

Wasserman, L., Whipp, B.J., Koyal, S.N., & Beaver, W.L. (1973). Anaerobic threshold and respiratory gas exchange during exercise. Journal of Applied Physiology, 35, 236-243.

Wenger, H.A., & Reed, A.T. (1976). Metabolic factors associated with muscular fatigue during aerobic and anaerobic work. Canadian Journal of Applied Sports Sciences, 1, 43-48.

Whipp, B.J. (1983). Exercise hyperventilation in patients with McArdle's disease (Letter to the Editor). Journal of Applied Physiology, 55, 1638-1639.

Whipp, B.J., & Mahler, M. (1980). Dynamics of pulmonary gas exchange during exercise. In J.B. West (Ed.), Pulmonary gas exchange: Vol II (pp. 33-96). New York, New York: Academic Press.

Williams, C.G., DuRaan, A.J.N., Von Rahden, M.J.F., & Wyndham, C.H. (1968). The capacity for endurance work in highly trained men. Internationale Zeitschefift for Angewandte Physiologie Einschliesslich Arbeits Physiologie, 26, 141-149.

Wilmore, J., Royce, J., Girandola, R., & Katch, F. (1970). Body composition changes with a 10-week program of jogging. Medicine and Science in Sports, 2(3), 113-117.

Wiswell, R.A., Girandola, N.R., Bulbulian, R., & Simard, C. (1980). The effect of two hours of running on anaerobic threshold. Medicine and Science in Sports and Exercise, 12(36). Abstract.

Yeh, M.P., Gardner, R.M., Adams, T.D,, Yanowitz, F.G., & Crapo, R.O. (1983). "Anaerobic threshold": problems of determination and validation. Journal of Applied Physiology, 55, 1178-1186.

Yoshida, T. (1984). Effect of exercise duration during incremental exercise on the determination of anaerobic threshold and the onset of blood lactate accumulation. European Journal of Applied Physiology and Occupational Physiology, 53, 196-199.

Yoshida, T.A., Nagata, M., Muro, M., Takeuchi, N., & Suda, Y. (1981). The validity of anaerobic threshold determination by Douglas bag method compared with arterial blood lactate concentration. European Journal of Applied Physiology, 46, 423-430.

# CHAPTER II

## Validity and Reliability of the Maximal Aerobic Power in Cerebral Palsied Wheelchair Athletes[1]

### Introduction

Recently, the physically disabled population has become more aware of the benefits of physical fitness. This is manifested by the increasing number of disabled people, including those with cerebral palsy (CP), who are participating in group fitness classes or individual training programs (Cooper et al., 1986). Fitness testing, in turn, has become an important component with which to monitor progress. Many researchers have investigated maximal aerobic power ($\dot{V}O_2max$) during wheelchair ergometry (WE) in individuals with a variety of physical disabilities (Glaser, 1989), but relatively few studies (Lundberg, 1978; Lundberg, 1976; Lundberg, 1975) have evaluated persons with CP. As a result, no valid and reliable test protocol has been established for evaluating the $\dot{V}O_2max$ of these subjects.

In the able-bodied population, the most valid $\dot{V}O_2max$ test protocols are performed with lower extremities using a cycle ergometer (CE) or treadmill (Astrand & Rodahl, 1986). While many cerebral palsied individuals can use their legs to perform tests on these apparatus, several confounding factors

---

1 A version of this chapter has been accepted for future publication: Bhambhani, Y., Holland, L., and Steadward, R. Archives of Physical Medicine and Rehabilitation.

may prevent accurate measurement of $\dot{V}O_2max$ utilizing these exercise modes. For example, Lundberg (1978) points out that balance problems are likely to occur on the treadmill for persons with CP. An additional concern is the fact that some individuals with CP have high levels of muscle spasticity which prevent sufficient flexion of the knee and/or hip to pedal the cycle ergometer. A third confounding factor arises when one considers that localized muscular factors also have a significant influence on the $\dot{V}O_2max$, and hence, individuals with CP who are wheelchair ambulatory may perform better when utilizing upper body muscle groups during testing (Sawka, 1986). It is evident, therefore, that there is a need to establish a valid $\dot{V}O_2max$ test protocol for individuals with CP.

The purposes of this study were to: (1) investigate the validity of $\dot{V}O_2max$ during WE in CP athletes by comparing these values with those obtained during CE, and (2) examine the test-retest reliability of the $\dot{V}O_2max$ and other physiological measurements during both these modes of exercise. The CE was selected as criterion reference because its validity and reliability have been well established in able-bodied subjects (Astrand & Rodahl, 1986). The WE was utilized because: (1) this exercise pattern was more specific to the subjects' requirements in performing activities of daily living and/or their competitive sports which stressed the upper, not lower body, and (2) factors such as balance and increased spasticity in the leg extensor muscles would not likely inhibit performance, and perhaps $\dot{V}O_2max$, on the WE.

## Methods

### Subjects

Six adult males with spastic CP provided their written, informed consent to participate in this study. All were affiliated with the Alberta Cerebral Palsy Sports Association and were current or former athletes competing at the national or international level. The participants were either class 3 or class 4 wheelchair athletes according to the Cerebral Palsy International Sport and Recreation Association's (CP-ISRA) classification system (Cerebral Palsy International Sports and Recreation Association, 1990). Pertinent characteristics of the subjects are provided in Table II-1 and their clinical status is described below.

Subject 1 was classified as a diplegic with minimal upper body involvement. He was able to ambulate with canes and had good symmetry in the upper body. Subject 2 was a hemiplegic who used a one arm drive wheelchair during his daily activities and competition. Even in his less involved right side, he had a high degree of muscle spasticity. Subject 3 was a wheelchair dependent diplegic who had a high level of hip and leg extension, and during intense physical activity, experienced a high degree of spasticity. Subject 4 was a spastic quadriplegic with a weaker and less flexible right side. He had significant contractures in his hamstrings, and was able to ambulate effectively with canes. Subject 5 was a spastic diplegic with very weak lower extremities and used a heelchair at all times. His upper body strength was asymmetrical, with the right side being dominant. Subject 6 was a spastic diplegic with a limited range of motion in his hip flexors and adductors. Despite this, he was able to ambulate without any aids. Of these six subjects, the first two had slight l[illegible]ng disorders, but were able to comprehend the instructions pertaining to the exercise testing procedures. Since all the subjects were elite athletes required to perform maximally in national and/or international competitions, they were aware of the demands of maximal physical activity.

**Testing Sessions**

Each subject attempted four graded exercise tests (two on the CE and two on the WE) in random order over a two week period. The tests were scheduled at approximately the same time of day on all occasions with at least 48 hours between sessions. While all six subjects completed the two WE tests, only four were able to perform the two CE tests. Subjects 2 and 3 were unable to flex their knees and/or hips sufficiently to pedal the CE.

**Wheelchair and Cycle Ergometry Tests**

The CE tests were conducted on a Uniwork cycle ergometer, with the subject's feet strapped with tensor bandages to the pedals of the CE to prevent eversion due to muscle spasticity. (The strapping did not function like toe clips which allow the subject to "pull up" with his legs.) The protocol was discontinuous in nature, and required two minutes of work followed by one minute of rest. During the rest, a finger tip blood sample was collected. The test was initiated at a power output of 30 Watts and 60 revolutions per minute (rpm); thereafter, power output was increased by 30 Watts until volitional fatigue or $\dot{V}O_2max$ was attained. Criteria for determining $\dot{V}O_2max$ was a levelling off or a decrease in the oxygen consumption with increasing power output (Astrand & Rodahl, 1986). Subjects were given verbal encouragement throughout the tests. During the last 30 seconds at each power output, the overall rating of perceived exertion (RPE) was monitored using the Borg scale (Borg, 1970). This scale has been reported to be an accurate method of evaluating the perception of physical effort in CP individuals during CE (Birk & Mossing, 1980).

The WE tests were conducted with subjects wheeling their personal wheelchairs mounted on a set of custom designed frictionless rollers with side mounted flywheels, as described by Eriksson et al. (1988). This apparatus was modified in the following manner: reflective tape was fastened on the rim of the flywheel, so that a signal could be picked up by an optical sensor which was mounted a few millimeters away. The sensor was interfaced with an analog/digital board placed in a micro- computer, to record the number of rpm of the rollers. A computer program then calculated the velocity of wheeling (in kilometers per hour, kmh) from rpm data and circumference of the rollers. This information was updated every five seconds and displayed as a speedometer to provide visual feedback to the subjects. The WE test was initiated at a velocity of 5 kmh, and was increased by 2 kmh until volitional fatigue or $\dot{V}O_2max$ was attained. RPE was recorded during the last 30 seconds at each of the wheeling velocities.

Metabolic measurements were continuously monitored and recorded every 30 seconds during exercise tests using an automated metabolic measurement cart. The exercise electrocardiogram was monitored with leads in the $CM_5$ position, and heart rate (HR) was interfaced with the metabolic cart. In addition to $\dot{V}O_2max$ and HR, the following variables were examined at maximal exercise intensity: expired ventilation volume ($\dot{V}_E$, l/min); respiratory exchange ratio (RER), calculated as the ratio between $\dot{V}CO_2$ produced and $\dot{V}O_2$ consumed; oxygen pulse ($O_2$ pulse, ml/beat), calculated as the ratio between $\dot{V}O_2$ in ml/min and HR; and the ventilatory equivalent for oxygen ($\dot{V}_E/\dot{V}O_2$ ratio).

**Blood Lactate Measurements**

Lactate concentrations of whole blood were determined from arterialized blood samples which were drawn from a pre-warmed finger tip prior to and three minutes after the cessation of each exercise test. Blood samples were analyzed using a lactate analyzer that was calibrated according to specifications outlined by the manufacturer. The difference between pre and post exercise concentrations were used for analysis.

**Statistical Analysis**

Pearson product-moment correlations were used to examine the validity and reliability of $\dot{V}O_2max$ during the two modes of exercise. The reliability of other physiological responses identified above were also examined using this procedure. As well, a two-way analysis of variance with repeated measures was used to compare the mean values of all these variables between the two trials for each of the two exercise modes. Percentage differences between trials on each mode were calculated for each variable in the following manner: {(Mean of Trial 1 - Mean of Trial 2)/Mean of Trial 1} x 100. All results were considered to be significant at the .05 level of confidence.

## Results and Discussion

### Results

The correlation coefficients obtained for $\dot{V}O_2max$ between the two modes of exercise during two test trials are summarized in Table II-2. The means, standard deviations, percentage differences, and reliability coefficients of physiological responses monitored during the exercise tests are presented in Table II-3. Results of analyses of variance for various physiological responses monitored during the exercise tests indicated that there were no significant differences between the mean values of the two modes of exercise for each trial, and between the mean values of the two trials for each mode of exercise.

### Comparison with Previous Studies

Relatively few studies have examined physiological responses of CP subjects during exercise. Information that is available has been obtained using the CE as the exercise mode. Current $\dot{V}O_2max$ values during the two CE tests, 40.7 ± 5.6 and 39.7 ± 6.4 ml/kg/min), are slightly lower than those reported by Lundberg (1976) (45 ± 3 ml/kg/min) on five young males of a similar age with spastic diplegia. This was despite the fact that the CP subjects in the current study attained a 3% higher maximal HR. In comparison with five age matched controls, Lundberg (1976) also reported that the CP subjects had: (1) a 12% lower $\dot{V}O_2max$, (2) an increased degree of ventilatory stress during maximal exercise as evidenced by a 13% higher $\dot{V}_E/\dot{V}O_2$ ratio, (3) an approximately 50% decrement in physical work capacity at a HR of 170 beats/min, (4) an overall reduction of 50% in the net mechanical efficiency during submaximal bicycle exercise, and (5) an equivalent blood lactate concentration during maximal exercise. Lundberg (1976) postulated that lower $\dot{V}O_2max$ and decrement in net mechanical efficiency could be due to a reduction in localized muscle blood flow of leg extensor muscles in the CP subjects.

A review of numerous studies that have compared cardiovascular responses during upper and lower body exercise in able-bodied subjects (Sawka, 1986) indicates that the average $\dot{V}O_2max$ and maximum HR values during upper body exercise in the form of arm cranking (AC) are approximately 73% and 96% of values respectively observed during lower body exercise in the form of CE. However, these values can actually range between 36% and 89% for the $\dot{V}O_2max$, and between 92% and 102% for maximum HR. Higher percentages are generally observed in individuals who train their upper body muscle groups. A comparison of these variables during WE and CE for the CP subjects in this study indicated that $\dot{V}O_2max$ during WE was 97% of that observed during CE, while the corresponding value for HR was approximately 94%. Since there seems to be no significant difference in $\dot{V}O_2max$ between WE and AC in both able-bodied and wheelchair dependent subjects, the higher percentage of $\dot{V}O_2max$ observed during upper body exercise in the current study is most likely due to training status of the subjects, since all were current or former athletes who competed at the national or international level.

**of the Physiological Responses**

The primary purpose of this study was to determine the validity of $\dot{V}O_2max$ during WE using CE as the criterion reference value. Validity coefficients observed for $\dot{V}O_2max$ between these two exercise modes were very low (Table II-2), suggesting that WE was not a suitable exercise mode for evaluating cardiovascular fitness of CP subjects. These observations are in conflict with reports on able-bodied subjects (Sawka, 1986) where most of the studies indicate significant correlation coefficients for $\dot{V}O_2max$ observed during upper and lower body exercise.

Despite poor validity coefficients, results of the analysis of variance revealed no significant differences between WE and CE on both the test trials for mean values of $\dot{V}O_2max$. Examination of individual values illustrated in Figure II-1, however, revealed that with the exception of subject 1 who performed consistently on each trial during both modes of exercise, there were large intra-individual differences in the remaining three subjects between the two modes of exercise. Subjects 4, 5, and 6 had differences of 5.7%, 20.6% and 46.1% respectively when mean values of the two trials were combined for each mode. It is also interesting to note that of these three subjects, the two who performed better on the CE ambulated with canes or did not require any ambulatory aides, while the third who did better on the WE was wheelchair ambulatory. Thus, the limited data from this study seems to suggest that the exercise mode selected for evaluating cardiorespiratory fitness of subjects with CP should be specific to their primary mode of ambulation. The observed intra-individual differences between the two exercise modes could not be explained on the basis of the clinical status of the subjects.

**Reliability of the Physiological Responses**

The secondary purpose of this study was to examine test-retest reliability of physiological responses during maximal exercise on the WE and CE. Results in Table II-3 indicated significant reliability coefficients for $\dot{V}O_2max$, $\dot{V}_E$, $O_2$ pulse, and RPE during WE, whereas those obtained for HR, RER, $\dot{V}_E/\dot{V}O_2$ ratio, and blood lactate were not significant. In a recent study by Bhambhani et al. (in press) on individuals with spinal cord injury, investigators reported significant test-retest reliability coefficients for peak values of all the above variables during WE, with the exception of blood lactates which were not examined in that study. Glaser et al. (1977) also reported significant reliability coefficients for $\dot{V}O_2$, RER, $\dot{V}_E$, and HR during submaximal WE in 10 able-bodied subjects. Low reliability of maximal HR during WE in the current study could be attributed to variations in upper body movement between the two test trials, since it was not stabilized during testing. It should be noted, however, that the difference between the mean values of the two trials was only 0.5%. Poor reliability of blood lactate concentrations could have been due to variations in spasticity of the exercising muscles in CP subjects on different test days, which might have affected the rate of diffusion of lactate from the muscle into blood, thereby rendering it an unreliable measurement. Although reliability coefficients for most of the variables examined were higher during CE (see Figure II-2 for scattergram of $\dot{V}O_2max$), none of them were statistically significant. This is because of the smaller number of subjects who were able to complete the CE test {note: for the WE test (n = 6), an 'r' value of 0.81 was required for statistical significance at the .05 level, whereas for the CE test (n = 4), an 'r' value of 0.95 was required}. It is suggested, therefore, that further studies using larger sample sizes be conducted to confirm the reliability of physiological measurements during CE in subjects with CP.

The limited number of subjects evaluated caused difficulty in establishing reliability of some of the physiological responses monitored during the two modes of exercise in the current study; however, the results do have some implications for individuals interested in promoting cardiovascular fitness of persons with CP. Generally, $\dot{V}O_2max$ is considered to be the best indicator of cardiorespiratory fitness because it is directly related to maximal cardiac output (Astrand, 1986). The fact that $\dot{V}O_2max$ is a reliable measurement in CP subjects enables coaches and therapists to objectively monitor progress as a result of training programs that are designed to promote cardiovascular fitness. Such programs, however, are usually prescribed on the basis of a target HR which is expressed either as a percentage of maximum HR or as a percentage of maximum HR reserve (American College of Sports Medicine, 1990); therefore, it is imperative that reliability of maximum HR be satisfactorily established in these subjects prior to the implementation of training programs.

**Conclusion**

Limited evidence from the current study indicates that CE may not be the most suitable exercise mode for evaluating cardiorespiratory fitness of athletes within CP class 3 or class 4 because some of them lack sufficient knee and/or hip flexion to pedal the CE. The poor correlation coefficients obtained for $\dot{V}O_2max$ between CE and WE suggest that the latter mode of exercise may not be a valid measure of the individual's $\dot{V}O_2max$ when the CE value is used as criterion reference. Since those subjects who were wheelchair ambulatory seemed to perform better on the WE, and the one who did not require any aides for ambulation performed better on the CE, it is recommended that the exercise mode selected for evaluating these subjects be specific to their primary mode of ambulation. The accuracy of $\dot{V}O_2max$ measurement is not compromised during either of these exercise modes since its reliability seems to be quite high.

**References**

American College of Sports Medicine (1990). The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults. Medicine and Science in Sports and Exercise, 22, 265-274.

Astrand, P.O. & Rodahl, K. (1986). Textbook of work physiology; physiological bases of exercise. (3rd ed.). New York, New York: McGraw-Hill Book Company.

Bhambhani, Y., Eriksson, P., & Steadward, R. (in press). Reliability of peak physiological responses during wheelchair ergometry in individuals with spinal cord injury. Archives of Physical Medicine and Rehabilitation.

Birk, T. & Mossing, M. (1980). Relationship of perceived exertion to heart rate and ventilation in active teenagers with cerebral palsy. Adapted Physical Activity Quarterly, 5, 165-169.

Borg, G. (1970). Perceived exertion as an indicator of somatic stress. Scandinavian Journal of Rehabilition and Medicine, 2, 92-98.

Cerebral Palsy International Sports and Recreation Association (1990). Classification and sports rules manual, (5th ed.). The Netherlands; Author.

Cooper, M.A.& Sherill, C. (1986). Attitudes toward physical activity of elite cerebral palsied athletes. Adapted Physical Activity Quarterly, 3, 14-21.

Eriksson, P., Lofstrom, L., & Ekblom, B. (1988). Aerobic power during maximal exercise in untrained and well-trained persons with quadriplegia and paraplegia. Scandinavian Journal of Rehabilitation and Medicine, 20, 141-147.

Glaser, R.M. (1989). Arm exercise training for wheelchair users. Medicine and Science in Sports and Exercise, 21, S149-157.

Glaser, R., Ginger, L., & Laubach, L. (1977). Validity and reliability of wheelchair ergometry. Physiologist, 20(34).

Glaser, R.M., Sawka, M.N., Brune, M.F., & Wilde, S.W. (1980). Physiological responses to maximal effort wheelchair and arm crank ergometry. *Journal of Applied Physiology*, *48*, 1060-1064.

Intermountain Thoracic Society (1980). *Manual of uniform procedures for clinical pulmonary function testing*, (2nd ed.).

Lundberg, A. (1975). Mechanical efficiency in bicycle ergometer work of young adults in cerebral palsy. *Developmental Medicine and Child Neurology*, *17*, 434-439.

Lundberg, A. (1976). Oxygen consumption in relation to work load in students with cerebral palsy. *Journal of Applied Physiology*, *40*, 873-875.

Lundberg, A. (1978). Maximal aerobic capacity of young people with cerebral palsy. *Developmental Medicine and Child Neurology*, *20*, 205-210.

Lundberg, A. (1984). Longitudinal study of physical working capacity of young people with cerebral palsy. *Developmental Medicine and Child Neurology*, *26*, 328-334.

Sawka, M.N. (1986). Physiology of upper body exercise. In K.B. Pandolf (Ed.), *Exercise and Sport Science Reviews* (pp 175-211). New York, New York: Macmillan Press.

Table II-1

Characteristics of Cerebral Palsied Subjects (n=6)

| Subject | Age (yr) | Height (cm) | Weight (kg) | CP-ISRA Class | Mode of Ambulation |
|---|---|---|---|---|---|
| 1 | 23 | 175 | 78.3 | 4 | canes |
| 2 | 26 | 173 | 61.4 | 3 | wheelchair |
| 3 | 29 | 168 | 63.0 | 4 | wheelchair |
| 4 | 24 | 157 | 55.3 | 3 | canes |
| 5 | 28 | 166 | 54.5 | 4 | wheelchair |
| 6 | 19 | 169 | 62.8 | 4 | none |
| Mean | 24.8 | 167.9 | 62.6 | | |
| SD | 3.7 | 6.4 | 8.6 | | |

Table II-2

Validity Coefficients for the Maximal Aerobic Power during Wheelchair and Cycle Ergometry in Cerebral Palsied Subjects (n=4)*

| | Wheelchair One | Wheelchair Two |
|---|---|---|
| Cycle One | 0.31 | 0.11 |
| Cycle Two | -0.06 | -0.24 |

*none of the correlation coefficients were significant at the .05 level.

Table II-3

Reliability of the Maximum Physiological Responses during Wheelchair (n=6) and Cycle Ergometry (n=4) in Cerebral Palsied Subjects (standard deviations in parentheses)

| Variable | Mode | Trial One | Trial Two | r | % Diff |
|---|---|---|---|---|---|
| $\dot{V}O_2$max | CE | 2.51 (.45) | 2.40 (.43) | .93 | 4.4 |
| l/min | WE | 2.34 (.46) | 2.43 (.37) | .89* | -3.8 |
| | % Diff | 6.8 | -1.3 | | |
| $\dot{V}O_2$max | CE | 40.7 (5.6) | 39.7 (6.4) | .92 | 2.5 |
| ml/kg/min | WE | 38.6 (8.3) | 39.2 (6.9) | .89* | -1.6 |
| | % Diff | 5.2 | 1.3 | | |
| HR | CE | 197 (5.3) | 198 (6.2) | .95 | -0.5 |
| beats/min | WE | 187 (8.2) | 186 (6.1) | .78 | -0.5 |
| | % Diff | 5.1 | 6.1 | | |
| $\dot{V}_E$ | CE | 139.0 (28.1) | 146.2 (35.6) | .74 | -5.2 |
| l/min | WE | 112.8 (32.2) | 116.1 (33.6) | .96* | -2.9 |
| | % Diff | 18.8 | 20.6 | | |
| RER | CE | 1.10 (.14) | 1.32 (.14) | .94 | -20.0 |
| | WE | 1.28 (.08) | 1.21 (.05) | .08 | 5.5 |
| | % Diff | -16.4 | 8.3 | | |
| $O_2$ pulse | CE | 12.7 (2.2) | 12.4 (1.9) | .93 | 2.4 |
| ml/beat | WE | 12.5 (2.0) | 12.9 (1.6) | .88* | -3.2 |
| | % Diff | 1.6 | -4.0 | | |
| $\dot{V}_E/\dot{V}O_2$ | CE | 55.5 (4.3) | 59.2 (5.5) | .65 | -6.7 |
| ratio | WE | 47.5 (7.2) | 47.0 (7.5) | .73 | 1.1 |
| | % Diff | 14.4 | 20.6 | | |
| Lactate | CE | 12.1 (2.4) | 11.1 (0.9) | .34 | 8.3 |
| mmol/l | WE | 9.7 (3.1) | 10.2 (1.8) | .65 | -5.2 |
| | % Diff | 19.8 | 8.1 | | |
| RPE | CE | 19.6 (.3) | 19.4 (.4) | .96* | 1.0 |
| | WE | 19.3 (.4) | 19.2 (.3) | .93* | 0.5 |
| | % Diff | 1.5 | 1.0 | | |

*denotes significant correlation between the two test trials for the same exercise mode.

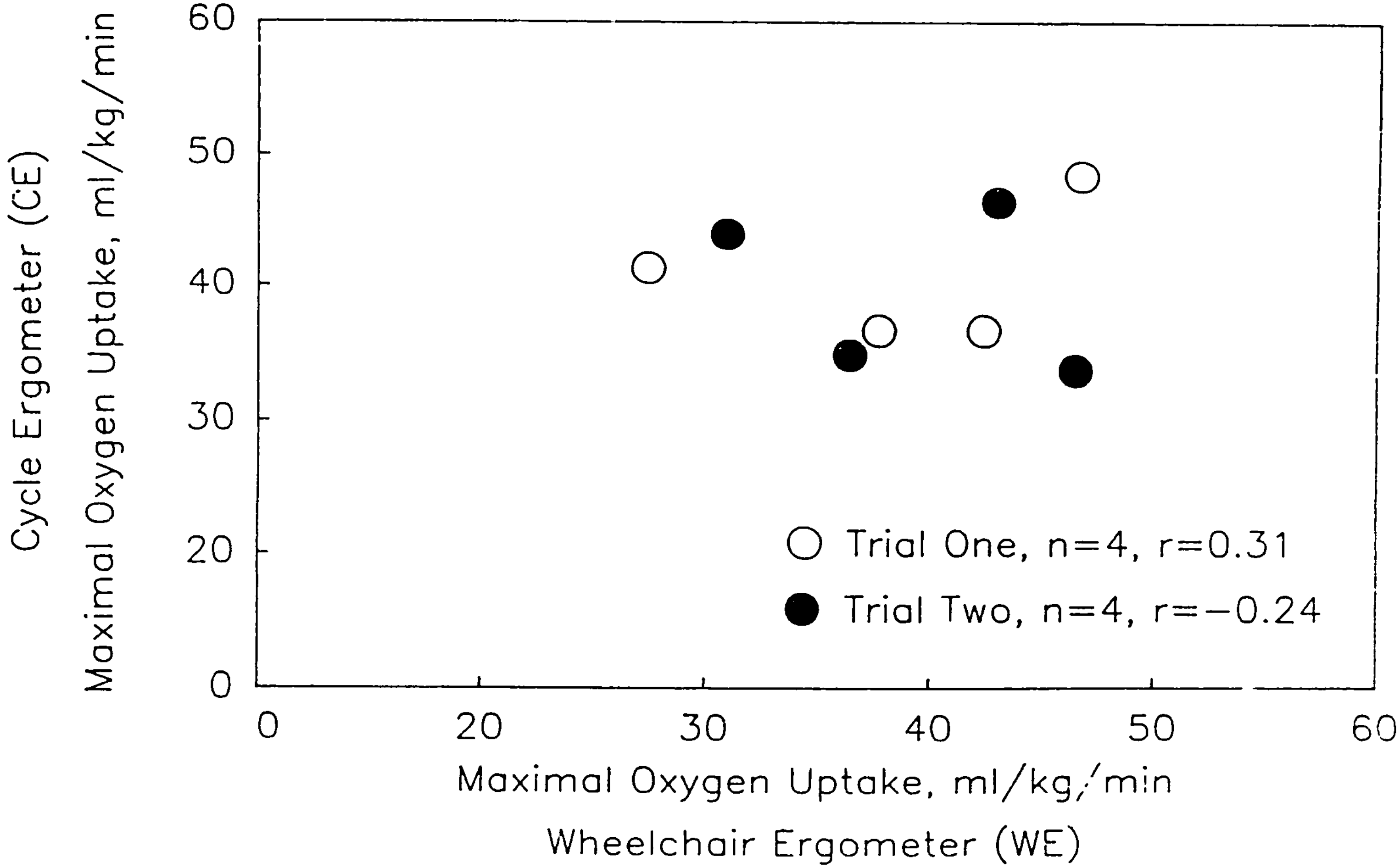


Figure II-1 Validity of the Maxixmal Aerobic Power in Cerebral Palsied Subjects

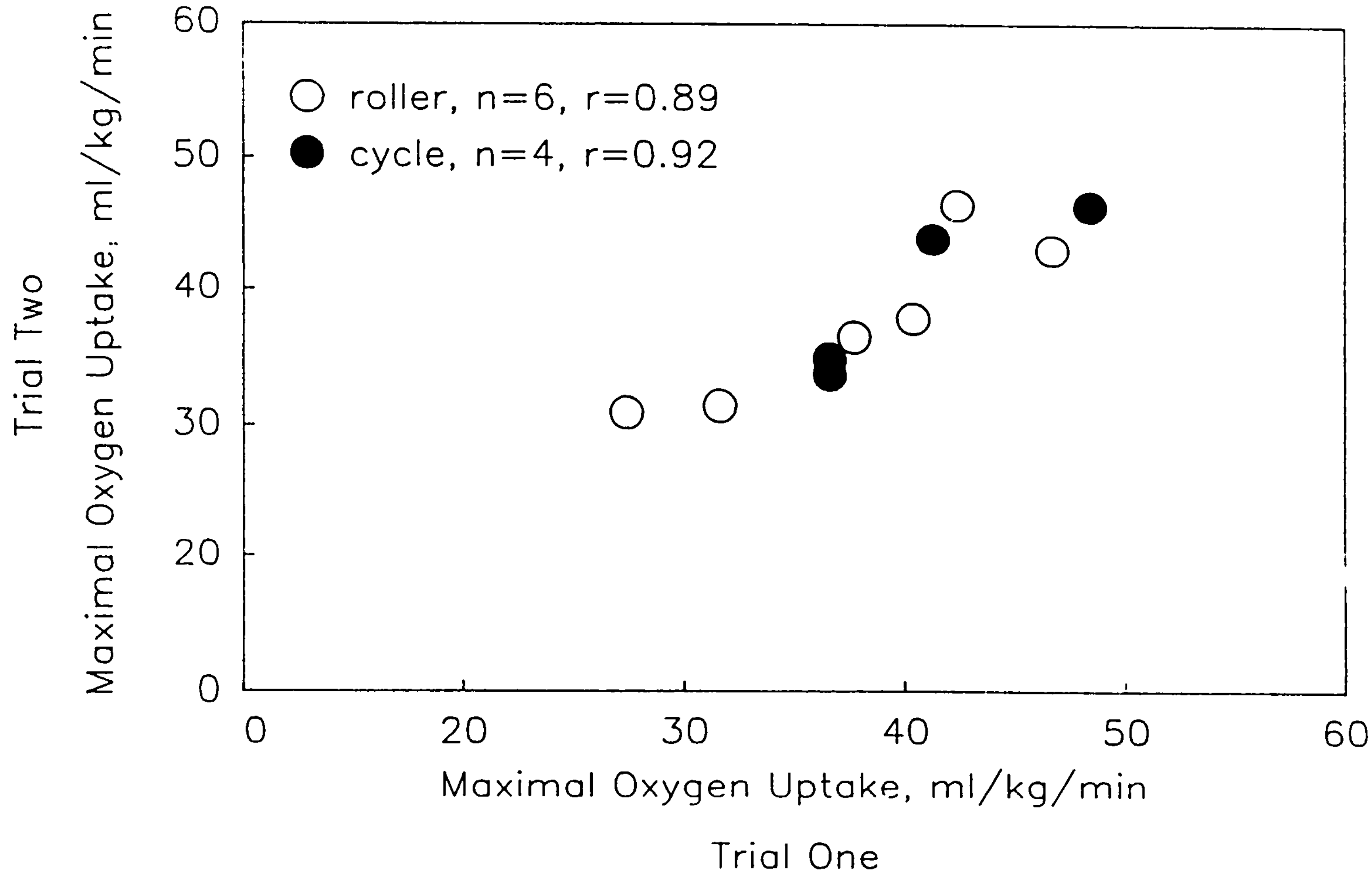


**Figure II-2** Reliability of the Maximal Aerobic Power during Cycle and Wheelchair Ergometry in Cerebral Palsied Subjects

# CHAPTER III

## Athletes with Cerebral Palsy: Reliability of Physiological Responses during Exercise

### Introduction

Few studies related to physical activity and/or physical fitness for persons with cerebral palsy (CP) have been conducted (McCubbin and Shasby, 1985). Persons with CP who exercise regularly to improve their general health or to pursue competitive opportunities, must rely primarily on training principles and knowledge that have evolved from the able-bodied population or from other disability groups (e.g., spinal cord injured). As a result, methods to determine cardiorespiratory parameters such as anaerobic threshold (AT), have not been identified or proven valid and reliable in persons with CP.

MacDougall (1977) noted that in long-duration endurance events (e.g., 400m or more on the track), the ability of an athlete to sustain exercise at a high percentage of $\dot{V}O_2max$ may be equal in importance to the actual $\dot{V}O_2max$. Theoretically, measurement of the anaerobic threshold, or that point where blood lactate begins to accumulate, should provide an indication of this ability. When exercise intensity exceeds this level, endurance time is reduced because of such factors as increased acidity in muscle, and a diminished capacity to mobilize lipids and therefore to spare muscle glycogen

(MacDougall, 1977). Powers et al. (1984), using nine runners showed that anaerobic threshold correlated highly (r=0.94) with 10 kilometer racing times. Kumagai et al. (1982) compared five and ten kilometer times to AT and $\dot{V}O_2max$ in seventeen runners. They found correlations of 0.95 and 0,84 respectively, for race pace versus AT.

One technique used to identify AT involves identification of the ventilatory threshold (VT). Criteria for this method is as follows: (1) the $\dot{V}_E/\dot{V}O_2$ curve, having been flat or decreasing, begins to rise whie the $\dot{V}_E/\dot{V}CO_2$ curve remains constant or decreases, (2) the $P_{ET}CO_2$ work rate curve is slowly rising or constant, but the $P_{ET}O2$ work rate curve, having been declining or flat, begins to rise, (3), the slope of the R work rate curve, having been flat or rising slowly, becomes more positive (Wasserman, 1986).

Blood analysis is an invasive technique used to determine lactate threshold (LT). Thus, the point at which the lactic acid level significantly breaks away is termed "lactate threshold".

The previous chapter in this thesis, outlines an investigation conducted to examine validity and reliability of $\dot{V}O_2max$ testing on the wheelchair ergometer (WE) in class 3 and 4 male CP athletes. Compared to the cycle ergometer (CE), a popular mode for the able-bodied population, the WE was not found to be a valid testing mode, although both CE and WE were extremely reliable (r=0.93 and 0.89 for $\dot{V}O_2max$ on the CE and WE respectively). However, two of the six subjects were unable to use the CE due to inadequate knee and/or hip flexion, which reduced the sample size to four. It is very difficult to investigate the validity of $\dot{V}O_2max$ utilizing the WE when results are compared to data from such a small sample size (on the CE). There is a need therefore, to further examine validity and reliability of the WE, especially since it appears to be the preferred testing mode (as all the subjects could utilize it).

The purposes of this study were to: (1) investigate validity and reliability of the ventilatory threshold (VT) and lactate threshold (LT) methods in identifying anaerobic threshold during WE and CE, and (2) further examine validity and test-retest reliability of $\dot{V}O_2max$ and other physiological measurements in athletes with CP during WE and CE.

## Methods

### Subjects

Eleven adult males with spastic CP provided written informed consent to participate in this study. All subjects were current or former athletes competing at the national or international level. Participants were either class 3 or class 4 wheelchair athletes according to the Cerebral Palsy International Sport and Recreation Association's (CP-ISRA) classification system. Some pertinent characteristics of the subjects are given in Table III-1. Note that six of the eleven subjects were the same athletes who participated in the study described in chapter II.

## Testing Sessions

Each athlete attempted four graded exercise tests (two on the CE and two on the WE) in random order with at least 48 hours of recovery between tests. All subjects were able to complete two tests on the WE, while only five athletes (nos. 1,4,5,6,and 8) were able to flex their knees and/or hips enough to pedal the CE. Tests were scheduled at approximately the same time of day on all occasions to control for possible diurnal variations in the physiological responses being monitored.

## Wheelchair and Cycle Ergometery Tests

WE testing was conducted with subjects sitting in their personal wheelchairs mounted on a set of custom designed frictionless rollers with side mounted flywheels, as described by Eriksson et al. (1988). This instrument was modified in the following manner: reflective tape was fastened on the rim of the flywheel, so that a signal could be picked up by an optical sensor which was mounted a few millimeters away. The sensor was interfaced with an analog/digital board placed in a micro computer, so that the revolutions per minute (rpm) of the rollers could be recorded. A computer program then calculated the velocity of wheeling (in kilometers per hour, kmh) from rpm data and circumference of the rollers. This information was updated every five seconds and displayed as a speedometer on the computer monitor to provide visual feedback to the subjects.

A discontinuous protocol was implemented whereby two minutes of exercise was followed by one minute of rest. During the rest period a finger tip blood sample was drawn. The WE test was initiated at a velocity of 5 kmh and increased by 2 kmh every work bout until volitional fatigue or $\dot{V}O_2$max was attained. Criteria for determining $\dot{V}O_2$max was a levelling off or decrease in oxygen consumption with increasing velocity of wheeling. Subjects were given verbal encouragement throughout the tests.

The CE tests were carried out on an electically braked Uniwork cycle ergometer (Quinton Instruments, Corival 400), with the subject's feet strapped to the pedals of the CE to prevent ankle eversion due to muscle spasticity. The strapping did not permit the subject to "pull up" with his legs as toe-clips allow. The CE test was initiated at a power output of 30 watts and 60 rpm. Thereafter, power output was increased by 30 watts every work bout until volitional fatigue or $\dot{V}O_2$max was attained.

Metabolic measurements were continuously monitored and recorded every 30 seconds during exercise tests using an automated metabolic measurement cart (MMC Horizon, Sensormedics). This system measured the volume of mixed expired air, then analyzed a sample of dried gas for its oxygen and carbon dioxide concentrations. These analyzers were calibrated with commercially available precision gases prior to and after each test. Absolute $\dot{V}O_2$ (l/min) and carbon dioxide production ($\dot{V}CO_2$, l/min) were then calculated from these data using the Haldane transformation, with help of a computerized software package available with the instrument. The exercise electrocardiogram was monitored with leads in the $CM_5$ position, and HR was interfaced with the metabolic cart. The highest $\dot{V}O_2$ value recorded during the test was considered to be maximal oxygen consumption ($\dot{V}O_2max$). In addition to $\dot{V}O_2max$ and HR, the following variables were examined: $\dot{V}_E$, l/min; respiratory exchange ratio (RER); oxygen pulse ($O_2$ pulse), ml/beat, calculated as the ratio between absolute $\dot{V}O_2$ and the HR; ventilatory equivalent for oxygen ($\dot{V}_E/\dot{V}O_2$ ratio); tidal frequency, br/min; and tidal vol, l/br.

**Lactate Threshold**

Lactate concentrations were determined from arterialized blood samples drawn from a pre-warmed finger tip prior to, during the one minute rest periods, and three minutes following cessation of each exercise test. Blood samples were analyzed using a lactate analyzer (Lactate Analyzer 640, Medilogic Limited) that was calibrated according to specifications outlined by the manufacturer. All blood lactate values were graphed against exercise stage for each subject. Two expert investigators who were blind to the study, attempted to identify the lactate thresholds. LT was determined subjectively as the visual point at which the lactic acid level significantly "broke away". Mean values of physiological responses monitored during this study were calculated at LT as identified by each of the two investigators.

**Ventilatory Threshold**

The values of the ventilatory equivalents for oxygen ($\dot{V}_E/\dot{V}O_2$) and carbon dioxide ($\dot{V}_E/\dot{V}CO_2$) were averaged for the last minute of each work bout, and plotted against exercise stage. The same two investigators who determined lactate thresholds also visually identified ventilatory thresholds. VT was named as the point at which $\dot{V}_E/\dot{V}CO_2$ remained linear while $\dot{V}_E/\dot{V}O_2$ abruptly increased (Davis, 1985). Mean values of physiological responses monitored during this study were also calculated at ventilatory threshold as identified by each of the two investigators.

**Statistical Analysis**

Pearson product-moment correlations were used to examine reliability of: (1) physiological responses monitored at $\dot{V}O_2max$ during the WE and CE tests, and (2) physiological responses monitored on the WE at the lactate and ventilatory thresholds as identified by two evaluators. LT and VT were not identified during the CE tests because only five subjects were able to use this mode. A two-way analysis of variance with repeated measures was used to compare mean values of monitored variables on the WE: (1) at $\dot{V}O_2max$ between two trials for each of the two exercise modes, and (2) at VT and LT between two trials for each of the two investigators. Percentage differences between trials on each mode, and trials for each investigator were also calculated for each variable in the following manner: {(Mean of Trial 1 - Mean of Trial 2)/Mean of Trial 1} x 100. All results were considered to be significant at the .05 level of confidence. Pearson product-moment correlations were also used to examine validity of the lactate and ventilatory thresholds as identified by two evaluators, and reliability of LT and VT over two trials. Correlations to examine validity of physiological responses on the WE were not calculated because only five of the eleven subjects could utilize the CE; therefore, the sample size was too small for an accurate comparison.

## Results and Discussion

### Results

Means, standard deviations, and percentage differences between two trials for maximal values of physiological responses monitored during WE and CE are presented in Table III-2. Also included in this table are reliability coefficients for each variable for the two test trials and modes. Tables III-3 and III-4 outline means, standard deviations, and percentage differences between the two WE trials for the monitored physiological responses at the lactate and ventilatory thresholds respectively, as identified by two investigators.

Validity coefficients between lactate threshold and ventilatory threshold were -0.32 and 0.76 (n=5) for evaluator 1 on trials 1 and 2 respectively, and -0.53 and -0.67 (n=3) for evaluator 2 on the same consecutive trials ($p<.05$). A significant correlation was noted only for evaluator 1 during trial 2. Reliability coefficients for LT and VT were -0.45 (n=7) and 0.79 (n=7), and -0.18 (n=5) and 0.65 (n=7) for evaluators 1 and 2 respectively ($p<.05$). Evaluator 1 had the only significant correlation for VT.

**Comparison of Maximal Responses with Previous Studies**

The reliability coefficient for $\dot{V}O_2max$ (l/min) on the WE (Table III-2) remained the same (r=0.89) in this study (n=11) compared to the earlier investigation presented in chapter II (n=6). Thus, use of this mode maintained a high level of reliability. Therefore, it may be the preferred apparatus to measure cardiorespiratory fitness in class 3 and 4 CP athletes due to the ease with which it can be used.

There was only one additional subject in the second study who could pedal the CE (n=5); therefore, no calculation of validity was carried out between the WE and CE. Although the reliability coefficient for $\dot{V}O_2max$ on the CE was lower in the second investigation with the slightly larger sample size, (r=0.93 and 0.87 for the first and second studies respectively), the correlation was significant only in the most recent study. Thus, for athletes who are able to pedal the CE, it is a reliable $\dot{V}O_2max$ testing mode.

Reliability coefficients for other physiological responses at $\dot{V}O_2max$ monitored in this study (Table III-2), were all significant on the WE except for RER. These results were therefore, more consistent than in the first study where HR, RER, $\dot{V}_E/\dot{V}O_2$ ratio, and lactate were not significantly correlated. These results are likely due to the larger sample size in the second study, and provide additional support for use of the WE during graded exercise testing to maximum.

The WE results of the study outlined in chapter II revealed a correlation coefficient of 0.65 for lactate (not significantly correlated), while the value was 0.68 in the subsequent investigation (a significant correlation probably due to the larger sample size). Inconsistent diffusion of lactate from trial one to trial two was likely due to the day-to-day variation of spasticity experienced by persons with CP.

No variables except $\dot{V}O_2max$ were significantly correlated on the CE although results of the analysis of variance revealed no significant differences between means except for RER. Again, this poor reliability was likely due to the small sample size.

Mean values for the physiological responses were similar in the two studies for both modes and trials. This is not surprising considering that data from the six subjects in the first study were combined with scores from an additional five athletes in the subsequent investigation. Thus, when these scores were compared to those collected by Lundberg (1976), similar differences occurred as documented in the first study.

**Validity of Lactate and Ventilatory Thresholds**

Although WE data from the eleven subjects were made available to both evaluators, evaluator 1 could identify seven lactate and ventilatory thresholds, while evaluator 2 could accurately determine only five lactate and seven ventilatory thresholds. Evaluators were asked to determine LT and VT only if they could identify it with confidence. Some data was erratic and it was impossible to determine thresholds. However, when the author and additional expert evaluators reviewed the thresholds that were identified by the two initial evaluators, there was concern expressed as to whether or not the two evaluators accurately followed the identification criteria. Despite both evaluators determining "questionable" break away points for some tests, the author can only assume that these points are correct. If additional experts were asked to re-assess the thresholds, perhaps different AT scores would be assigned, but the data would be biased. Thus, the data provided by the two initial evaluators is taken to be correct, but the reader should apply the results with caution.

Figures III-1a and III-2a reveal VT, while Figures III-1b and III-2b indicate LT for Subject 10 during trials one and two respectively. Note that the two evaluators identified the same points for both thresholds. Figures III-3 and III-4 outline the same information for Subject 9, although the evaluators did not deem VT to be the same point for either trial. Figures III-5a and III-5b reveal the VT and LT respectively for Subject 1 during trial one. Neither evaluator was able to identify AT for this subject.

Difficulties in identifying the thresholds may have developed for two reasons. (1) The protocol was discontinuous in nature to provide time to draw finger-tip blood samples for determination of blood lactate. It appears that the first minute of each two-minute exercise bout may have acted only as a "catch-up" period, thereby diminishing opportunity to attain steady-state at the higher work intensity. This in turn did not allow for obvious lactate and ventilatory "break points" to be located by the evaluators. A continuous protocol with an alternate method to draw blood samples, or a discontinuous protocol with longer exercise bouts (taking caution to prevent premature muscular fatigue) may be preferred testing methods. Researchers have implemented a wide variety of work bout lengths including 4 to 6 minutes (Keul et al., 1979; Mader et al., 1976) to allow tissue lactate to be well reflected in blood. Shorter periods, ranging from a continuous increase in loading (Whipp & Davis, 1979) to 30-second intervals (Davis et al., 1976) have been used to gain sharp changes in lactate and to restrict its uptake from blood. Thoden (1991) suggests work load increments be at least 2 minutes in duration, recognizing that an increased confidence in the steady-state nature of blood lactate will be achieved by longer increments. (2) Wheeling speed was increased by 2 kmh every work bout which provided for reliable $\dot{V}O_2$max scores, but may have been t-o large an increment to again, identify either of the two threshold break points. Perhaps a one kmh increment would be more appropriate. Most investigators agree that the work increment should be relatively small to precisely identify anaerobic threshold (Thoden, 1991). For example on the CE, Thoden (1991) suggests about a 15 watt increment be used with 2 to 3 watt increases or decreases for larger and smaller subjects.

Validity coefficients between LT and VT for trials 1 and 2 were -0.32 and 0.76, and -0.53 and -0.67 for evaluators 1 and 2 respectively. A significant correlation was noted only for VT for evaluator 1. All the coefficients in the current study were low compared to results reported by Aunola and Rusko (1984) in which the correlation coefficient for $\dot{V}T$ and LT was 0.81 for able-bodied men between the ages of 20 and 50 years who were exercising on the CE. Caiozzo et al. (1982) and Davis et al. (1976) also reported high coefficients (r=0.95) in their leg-pedalling studies with non-disabled subjects. It appears that additional testing (incorporating the above mentioned suggestions), should be undertaken to more accurately determine validity of LT and VT identification in athletes with CP.

A similar trend occurred in this study as found in investigations with non-disabled subjects, as reported by Yeh et al. (1983) and Posner et al. (1987) in terms of LT lagging behind VT. Thus, LT usually occurred at a higher percentage of $\dot{V}O_2max$ than VT for both trials according to both evaluators. Simon et al. (1983) noted that LT and VT were similar for constant load cycle ergomertry, but during incremental ergometry, VT preceded LT.

In addition to low to good validity coefficients, examination of LT and VT values revealed large intra-individual differences when comparing the two techniques for detecting AT. The largest discrepancy was observed by evaluator 2 who indicated that VT for Subject 7 during the first WE test occurred at 75.4% of his $\dot{V}O_2$max, while LT occurred at 55.4%. Meanwhile according to this same evaluator, only one subject (no. 10), had the same VT and LT points (60.8% of $\dot{V}O_2$max). Evaluator 1's largest difference occurred in Subject 6 during the first WE test where 25.9% of $\dot{V}O_2$max was identified as LT, and 76.6% the VT. Two important questions arise from these results, which require further investigation. (1) Can VT and/or LT be used for exercise prescription and, (2) can these thresholds be correlated with endurance performances of these athletes?

In the study conducted by Aunola and Rusko (1984) on able-bodied subjects, range for LT was 67% to 87% of $\dot{V}O_2$max, with the mean AT at 77% of maximal oxygen uptake (for the fit group) and 76% (for the less fit group). Reproducibility of AT was equal among subjects below or over 35 years of age. In the current study, LT range was 25.9% to 91.4% (mean = 63.3%) for evaluator 1, and 60.8% to 85.4% (mean = 66.4%) for evaluator 2. The greater range exhibited by the subjects with CP may again, have been due to the discontinuous protocol with short work bouts and large graded increments, and/or the variations in spasticity of the exercising muscles in the subjects on different test days; might have affected the rate of diffusion of lactate from the muscle into the blood.

Lower mean values for AT in the CP subjects may be due to the fact that the upper limbs were used during the exercise. Davis et al. (1976) calculated the VTs for arm cranking, leg cycling, and treadmill walk/running to be at 46.5 +/-8.9, 63.8

+/- 9.0, and 58.6 +/- 5.8% of $\dot{V}O_2max$ respectively. They attributed the lower score for the upper body mode to three factors: (1) subjects were not used to upper body exercise; therefore, specific training adaptations were not induced in these muscles, (2) there were differences in recruitment patterns of motor units during the arm crank tests versus the leg tests, and (3) distribution of fast and slow twitch muscle fibers and possibly their recruitment patterns may have differed significantly between the arms and legs of the subjects. Not all subjects who participated in this study used a wheelchair except during training workouts and competition, and therefore reasons quoted above for a lower AT may also apply to these subjects. Thus, the lower mean anaerobic threshold means identified in this study may be due to the fact that upper body muscles were utilized during testing, and may not be due to the condition of CP.

**Reliability of Lactate and Ventilatory Thresholds**

Tables III-3 and III-4 outline means, standard deviations, and percentage differences between two WE trials for monitored physiological responses at the lactate and ventilatory thresholds respectively, as identified by the two investigators. Reliability coefficients which are also included in these tables were varied. Reliability coefficients for % $\dot{V}O_2max$ at LT were -0.45 and -0.18 for investigators 1 and 2 respectively, and 0.79 ($p<.05$) and 0.65 for for % $\dot{V}O_2max$ at VT. Although evaluator 1's VT data was significantly correlated, it was much lower than the 0.95 coefficient reported by Aunola and Rusko (1984).

LT reliability coefficients were low according to both evaluators in the current investigation. Aunola and Rusko (1984) reported a rest-retest correlation coefficient of only 0.52 in a study they conducted with 33 non-disabled males, aged 20 to 50 years. Thus, it appears that LT results are not reliable despite the ANOVA revealing no significant differences between means.

Again, examination of the LT and VT values revealed large intra-individual differences when comparing: (1) results for the two trials, and (2) results for the two evaluators.

(1) Differences between Trials. Lactate and ventilatory thresholds were compared between trials for each of the two evaluators. Subject 7 had the largest % $\dot{V}O_2max$ at VT difference according to both evaluators (14.1%). Subject 6 had the greatest % $\dot{V}O_2max$ at LT difference (65.4%) according to evaluator 1, while the second evaluator chose Subject 9 (24.1%). 1.9% (for Subject 4) was the smallest LT difference for evaluator 1, while Subject 10 displayed the smallest LT variation between trials (3.9%) according to evaluator 2. Both evaluators agreed that Subject 8 experienced the smallest amount of VT variation (2.3%) between trials. Thus, intra-individual differences between trials appeared to be less for VT than for LT. This tendency may again be due to day-to-day fluctuations of spasticity levels experienced by subjects.

(2) Differences between Evaluators. When comparing %

$\dot{V}O_2max$ at VT for two evaluators, the only difference appeared for Subject 4, who on the first WE assessment according to evaluator 1 scored 43.3% of his $\dot{V}O_2max$ while evaluator 2 calculated it to be 64.2%. On the subsequent WE test for the same subject, evaluator 1 labelled VT at 40.5%, while evaluator 2 determined it to be at 61.4% of $\dot{V}O_2max$. The only discrepancy between evaluators for % $\dot{V}O_2max$ at LT identification was for Subject 7 on both WE tests. Evaluator 1 indicated LT to be 75.4% and 98.2% of $\dot{V}O_2max$, while evaluator 2 deemed it to be at 55.4% and 67.7% for the first and second tests respectively. Thus, in the current study, the evaluators did not agree on two out of the fourteen tests (14%) in which VT was identified (seven subjects over two trials), and two out of ten tests (20%) in which LT was determined. In the investigation conducted by Powers et al. (1984), evaluators differed in their choice of LT and VT on approximately 20% of the tests. Thus, a similar level of agreement between evaluators was exhibited during the study conducted by Powers et al. (1984) and the current investigation.

**Conclusion**

Evidence from the current study indicates that WE serves as a reliable and functional mode for evaluating maximal cardiorespiratory fitness parameters of class 3 and 4 athletes with CP. While the CE provides consistent $\dot{V}O_2max$ scores (l/min), it is appropriate only for persons who have adequate hip and/or knee flexion (to be able to pedal the CE);

typically these individuals do not utilize a wheelchair to carry out daily activities.

Validity of VT and LT techniques to identify AT remains questionable in athletes with CP who are exercising on the WE, for several reasons: (1) neither evaluator was able to identify thresholds for all eleven subjects, (2) coefficients were only poor to good, and lower than reports from other investigators who have studied the same area with other populations, and (3) intra-individual differences were large; LT for example, ranged from 25.9% to 91.3% of $\dot{V}O_2$max. A continuous protocol with small work increments or a discontinuous protocol with longer work bouts may alleviate some of the problems experienced in this investigation. As well, extreme care must be taken to ensure that the evaluators who determine the break away points, carefully follow the criteria for threshold identification .

The VT method appears to be the preferred technique by which to identify AT, since reliability between trials and evaluators was higher. Also, this method is preferred because of its ease of administration. For example, during the LT technique, a discontinuous protocol is necessary unless an alternate method of blood collection is utilized. As well, the varying levels of muscle spasticity appear to alter LT due to the reduced blood flow in localized muscle.

**References**

Aunola, S., & Rusko, H. (1984). Reprcducibility of aerobic andanaerobic thresholds in 20-50 year old men. European Journal of Applied Physiology, 53, 260-266.

Caiozzo, V.J., Davis, J.A., Ellis, J.F., Azus, J.L., Vandagriff, R., Prietto, C.A., & McMaster, W.C. (1982). A comparison of gas exchange indices used to detect the anaerobic threshold. Journal of Applied Physiology, 53, 1184-1189.

Davis, J.A. (1985). Anaerobic threshold: review of the concept and directions for future research. Medicine and Science in Sports and Exercise, 17,(1), 6-18.

Davis, J.A., Vodak, P., Wilmore, J.H., Vodak, J., & Kurtz, P. (1976). Anaerobic threshold and maximal aerobic power for three modes of exercise. Journal of Applied Physiology, 41, 97-108.

Eriksson, P., Lofstrom, L., & Ekblom, B. (1988). Aerobic power during maximal exercise in untrained and well-trained persons with quadriplegia and paraplegia. Scandinavian Journal of Rehabilitation and Medicine, 20, 141-147.

Keul, J., Suison, G., Berg, A., Dickhuth, H.H., Hoesttler, I., & Kuebel, R. (1979). Bestimmung der individullen anaerobic schwelle zur leistungbewertung und trainingsgestaltun [Determination of the individual anaerobic threshold in the assessment of efficiency and in the designing of training]. Dtsch A fuer Sportmedizin, 7, 212-218.

Kumagai, S., Tanaka, K., Matsura, Y., Matsuzaka, A., Hirakoba, K., & Asano, K. (1982). Relationships of the anaerobic threshold with the 5 km, 10 km, and 10 mile races. European Journal of Applied Physiology, 49, 13-23.

Lundberg, A. (1976). Oxygen consumption in relation to work load in students with cerebral palsy. Journal of Applied Physiology, 40, 873-875.

MacDougall, J.D. (1977). The anaerobic threshold- its significance to the endurance athlete. Canadian Journal of Applied Sports Sciences, 2, 137-140.

Mader, A., Leisen, H., Heck, H., Phillippi, H., Rost, R., Schwerch, P., & Hollurann, W. (1976). Zur beurteiling der sportartspezifischen audauer-leistungsfahigkeit in labor [On the judging of sport-specific endurance efficiency in the laboratory]. Sportarzt Sportmedizin, 4, 80-88; 5, 109-112.

McCubbin, J.A, & Shasby, G.B. (1985). Effects of isokinetic exercise on adolescents with cerebral palsy. Adapted Physical Activity Quarterly. 2, 56-64.

Posner, J.D., Gorman, K.M., Klein, S., & Cline, C.J. (1987). Ventilatory threshold: measurement and variation with age. Journal of Applied Physiology, 63(4), 1519-1525.

Powers, S.K., Dodd, S., & Garner, R. (1984). Precision of ventilatory and gas exchange alterations as a predictor of the anaerobic threshold. European Journal of Applied Physiology, 52, 173-177.

Simon, J., Young, J.L., Blood, D.K. Segal, K.R., & Case R.B. (1983). Plasma lactate and ventilation threshold in trained and untrained cylists. Journal of Applied Physiology, 54, 13-17.

Thoden, J.S. (1991). Testing aerobic power. In MacDougall, J.D., Wenger, H.A., & Green, H.J. (Eds.), Physiological Testing of the High-Performance Athlete (2nd ed.). Human Kinetics Books: Champaign, Illinois.

Wasserman, K. (1986). Anaerobiosis, lactate, and gas exchange during exercise: the issues. Federation Proceedings, 45, 2904-2909.

Whipp, B.J. & Davis, J.A. (1979). Chemoreceptors and exercise hyperpnea. Medicine and Science in Sports, 11, 204-212.

Yeh, M.P., Gardner, R.M., Adams, T.D,, Yanowitz, F.G., & Crapo, R.O. (1983). "Anaerobic threshold": problems of determination and validation. Journal of Applied Physiology, 55, 1178-1186.

Table III-1

Characteristics of Subjects with Cerebral Palsy (n=11)

| Subject | Age (yr) | Height (cm) | Weight (kg) | CP-ISRA Class | Mode of Ambulation |
|---|---|---|---|---|---|
| 1 | 23 | 175 | 78.3 | 4 | canes |
| 2 | 26 | 173 | 61.4 | 3 | wheelchair |
| 3 | 29 | 168 | 63.0 | 4 | wheelchair |
| 4 | 24 | 157 | 55.3 | 3 | canes |
| 5 | 28 | 166 | 54.5 | 4 | wheelchair |
| 6 | 19 | 169 | 62.8 | 4 | none |
| 7 | 33 | 173 | 77.7 | 4 | canes |
| 8 | 19 | 177 | 60.6 | 4 | crutches |
| 9 | 24 | 173 | 66.6 | 3 | crutches |
| 10 | 23 | 178 | 63.0 | 4 | wheelchair |
| 11 | 28 | 170 | 70.0 | 3 | wheelchair |
| Mean | 25 | 171 | 64.8 | | |
| SD | 4.3 | 5.9 | 7.8 | | |

Table III-2

Reliability of the Maximum Physiological Responses during Wheelchair (n=11) and Cycle Ergometry (n=5) in Elite Athletes with Cerebral Palsy (standard deviations in parentheses)

| Variable | Mode | Trial One | Trial Two | r | % Diff |
|---|---|---|---|---|---|
| $\dot{V}O_2$max | CE | 2.49 (.39) | 2.52 (.38) | .87* | -1.20 |
| l/min | WE | 2.36 (.40) | 2.43 (.32) | .89* | -2.97 |
| | % Diff | 5.2 | 3.6 | | |
| $\dot{V}O_2$max | CE | 40.6 (4.8) | 40.6 (5.9) | .85 | 0.00 |
| ml/kg/min | WE | 37.1 (7.4) | 37.6 (5.7) | .92* | -1.30 |
| | % Diff | 8.6 | 7.4 | | |
| HR | CE | 196 (2.4) | 197 (5.8) | .74 | 0.50 |
| beats/min | WE | 188 (8.0) | 191 (7.2) | .82* | -1.60 |
| | % Diff | 4.1 | 3.0 | | |
| $\dot{V}_E$ | CE | 127.8 (34.7) | 148.6 (26.5) | .36 | -16.30 |
| l/min | WE | 121.2 (35.2) | 125.4 (29.8) | .92* | -3.50 |
| | % Diff | 5.2 | 15.6 | | |
| RER | CE | 1.16 (.14) | 1.33 (.13) | .68 | -14.70 |
| | WE | 1.29 (.15) | 1.29 (.10) | -.13 | 0.00 |
| | % Diff | -11.2 | 3.0 | | |
| $O_2$ pulse | CE | 12.4 (2.0) | 12.7 (1.8) | .65 | -2.40 |
| ml/beat | WE | 12.5 (2.0) | 12.6 (1.5) | .87* | -0.80 |
| | % Diff | -0.8 | 0.8 | | |
| $\dot{V}_E/\dot{V}O_2$ ratio | CE | 51.2 (10.1) | 59.1 (4.7) | .28 | -15.40 |
| | WE | 50.7 (10.0) | 51.2 (7.2) | .87* | -1.00 |
| | % Diff | 1.0 | 13.4 | | |
| Lactate | CE | 11.0 (3.2) | 10.2 (2.1) | .79 | 7.30 |
| mmol/l | WE | 8.4 (2.6) | 9.9 (2.1) | .68* | 17.90 |
| | % Diff | 23.6 | 2.9 | | |
| Tidal freq | CE | 55.3 (10.4) | 64.4 (07.2) | -.10 | -16.50 |
| br/min | WE | 55.9 (17.7) | 59.9 (13.5) | .94* | 0.00 |
| | % Diff | -8.3 | 7.0 | | |
| Tidal vol | CE | 2.25 (.42) | 2.30 (.22) | .69 | -2.20 |
| l/br | WE | 2.12 (.51) | 2.12 (.49) | .88* | 0.00 |
| | % Diff | 5.8 | 7.8 | | |

* denotes significant correlation between the two test trials for the same exercise mode

Table III-3

Reliability of the Physiological Responses at Lactate Threshold during Wheelchair Ergometry in Elite Athletes with Cerebral Palsy (standard deviations in parentheses)
n=7 for evaluator #1; n=5 for evaluator #2

| Variable | Evaluator | Trial One | Trial Two | r | % Diff |
|---|---|---|---|---|---|
| $\dot{V}O_2$ | 1 | 1.48 (.54) | 1.82 (.39) | .17 | -22.97 |
| l/min | 2 | 1.46 (.42) | 1.74 (.24) | -.20 | -19.18 |
| | % Diff | 1.35 | 6.67 | | |
| $\dot{V}O_2$ | 1 | 23.1 (8.3) | 26.9 (3.9) | -.08 | -16.45 |
| ml/kg/min | 2 | 23.6 (5.6) | 24.6 (3.7) | .23 | -4.24 |
| | % Diff | -2.16 | 8.55 | | |
| $\%\dot{V}O_2max$ | 1 | 63.3 (18.5) | 76.0 (13.3) | -.45 | 2.96 |
| | 2 | 66.4 (11.5) | 69.8 (9.2) | -.18 | 16.67 |
| | % Diff | -4.90 | 8.16 | | |
| HR | 1 | 152 (25.9) | 167 (16.0) | .74 | -9.87 |
| beats/min | 2 | 151 (36.8) | 167 (18.3) | .92* | -10.60 |
| | % Diff | 0.66 | 0.00 | | |
| $\dot{V}_E$ | 1 | 57.3 (25.5) | 49.3 (38.8) | .30 | 13.96 |
| l/min | 2 | 60.9 (22.6) | 53.8 (18.1) | .13 | 11.66 |
| | % Diff | -6.28 | -9.13 | | |
| RER | 1 | 1.05 (.09) | 1.14 (.12) | .34 | -8.57 |
| | 2 | 1.04 (.10) | 1.13 (.07) | .11 | -8.65 |
| | % Diff | 0.95 | 0.88 | | |
| $O_2$ pulse | 1 | 10.3 (3.3) | 11.0 (2.3) | .24 | -6.80 |
| ml/beat | 2 | 10.6 (1.7) | 10.6 (2.2) | .23 | 0.00 |
| | % Diff | -2.91 | 3.64 | | |
| $\dot{V}_E/\dot{V}O_2$ ratio | 1 | 35.6 (7.5) | 41.5 (12.5) | .66 | -16.57 |
| | 2 | 38.1 (7.2) | 42.7 ( 7.7) | .64 | -12.07 |
| | % Diff | -7.02 | -2.89 | | |
| Lactate | 1 | 3.3 (2.9) | 2.5 (1.6) | .92* | 24.24 |
| mmol/l | 2 | 3.3 (1.9) | 2.8 (1.3) | .77 | 15.15 |
| | % Diff | 0.00 | -12.0 | | |
| Tidal freq | 1 | 38.6 (14.9) | 43.7 (17.2) | .80* | -13.21 |
| br/min | 2 | 42.4 (15.5) | 47.1 (16.6) | .79 | -11.08 |
| | % Diff | -9.84 | -7.78 | | |
| Tidal vol | 1 | 1.45 (.49) | 1.87 (.67) | .34 | -28.97 |
| l/br | 2 | 1.44 (.35) | 1.70 (.49) | .32 | -18.06 |
| | % Diff | 0.69 | 9.09 | | |

* denotes significant correlation between the two test trials for the same evaluator

Table III-4

Reliability of Physiological Responses at Ventilatory Threshold during Wheelchair Ergometry (n=7) in Elite Athletes with Cerebral Palsy (standard deviations in parentheses)

| Variable | Evaluator | Trial One | Trial Two | r | % Diff |
|---|---|---|---|---|---|
| $\dot{V}O_2$ l/min | 1 | 1.44 (.28) | 1.41 (.26) | .56 | 2.08 |
| | 2 | 1.51 (.24) | 1.46 (.19) | .19 | 3.31 |
| | % Diff | -4.86 | -3.55 | | |
| $\dot{V}O_2$ ml/kg/min | 1 | 23.6 (3.0) | 22.6 (4.1) | .27 | 4.24 |
| | 2 | 25.0 (3.4) | 23.9 (3.6) | .14 | 4.40 |
| | % Diff | -5.93 | -5.75 | | |
| $\%\dot{V}O_2max$ | 1 | 61.9 (12.0) | 58.1 (13.0) | .79* | 6.14 |
| | 2 | 64.9 (8.8) | 61.1 (10.5) | .65 | 5.86 |
| | % Diff | -4.85 | -5.16 | | |
| HR beats/min | 1 | 142.0 (17.5) | 138 (16.8) | .82* | 3.16 |
| | 2 | 146.0(12.7) | 142 (13.1) | .67 | 2.95 |
| | % Diff | -2.46 | -2.68 | | |
| $\dot{V}_E$ l/min | 1 | 48.4 (13.5) | 45.6 (12.2) | .85* | 5.79 |
| | 2 | 51.0 ( 9.7) | 48.0 ( 8.9) | .71 | 5.88 |
| | % Diff | -5.37 | -5.26 | | |
| RER | 1 | 1.02 (.18) | 1.00 (.05) | .55 | 1.96 |
| | 2 | 1.04 (.16) | 1.01 (.04) | .29 | 2.88 |
| | % Diff | -1.96 | -1.00 | | |
| $O_2$ pulse ml/beat | 1 | 10.8 (1.6) | 10.2 (1.3) | .77* | 5.56 |
| | 2 | 11.0 (1.6) | 10.5 (1.0) | .78* | 4.55 |
| | % Diff | -1.85 | -2.94 | | |
| $\dot{V}_E/\dot{V}O_2$ ratio | 1 | 32.7 (6.0) | 32.2 (5.8) | .59 | 1.53 |
| | 2 | 33.2 (5.1) | 32.6 (5.5) | .51 | 1.81 |
| | % Diff | -1.53 | -1.24 | | |
| Lactate mmol/l | 1 | 2.5 (1.2) | 2.1 (1.3) | .95* | 16.0 |
| | 2 | 2.5 (1.2) | 2.1 (1.3) | .99* | 16.0 |
| | % Diff | 0.00 | 0.00 | | |
| Tidal freq br/min | 1 | 31.2 (8.7) | 30.6 (7.8) | .92* | 1.92 |
| | 2 | 32.2 (8.5) | 31.6 (7.5) | .92* | 1.86 |
| | % Diff | -3.21 | -3.27 | | |
| Tidal vol l/br | 1 | 1.60 (.43) | 1.52 (.40) | .78* | 5.00 |
| | 2 | 1.65 (.37) | 1.56 (.35) | .70 | 5.45 |
| | % Diff | -3.13 | -2.63 | | |

* denotes significant correlation between the two test trials for the same evaluator

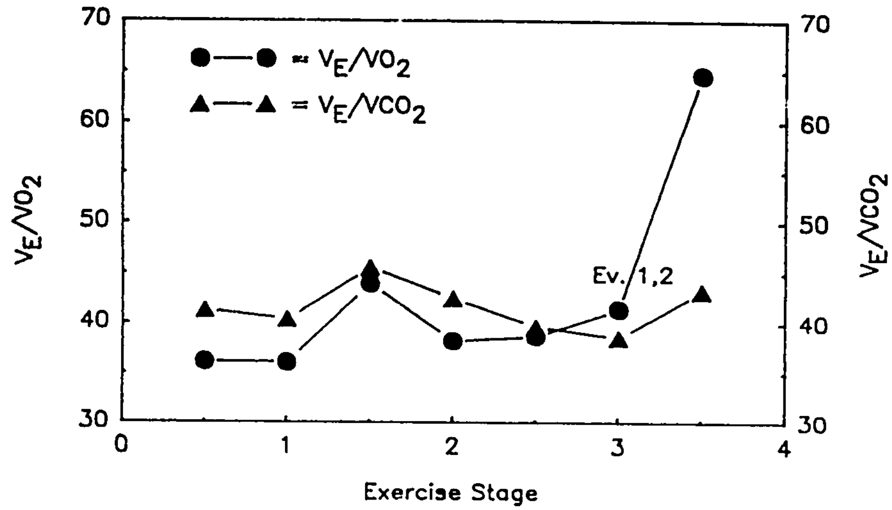


Figure III-1a Ventilatory Threshold of a Typical Subject (no. 10) during Trial One on the Wheelchair Ergometer, as Identified by Two Evaluators

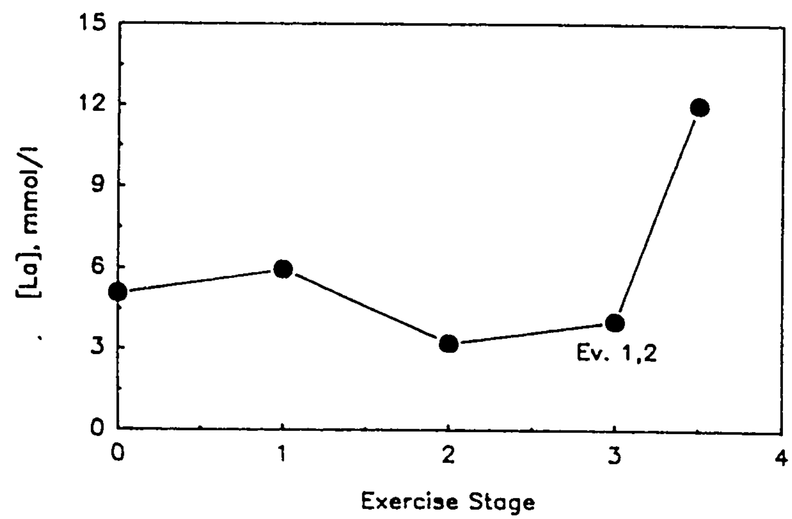


Figure III-1b Lactate Threshold of a Typical Subject (no. 10) during Trial One on the Wheelchair Ergometer, as Identified by Two Evaluators

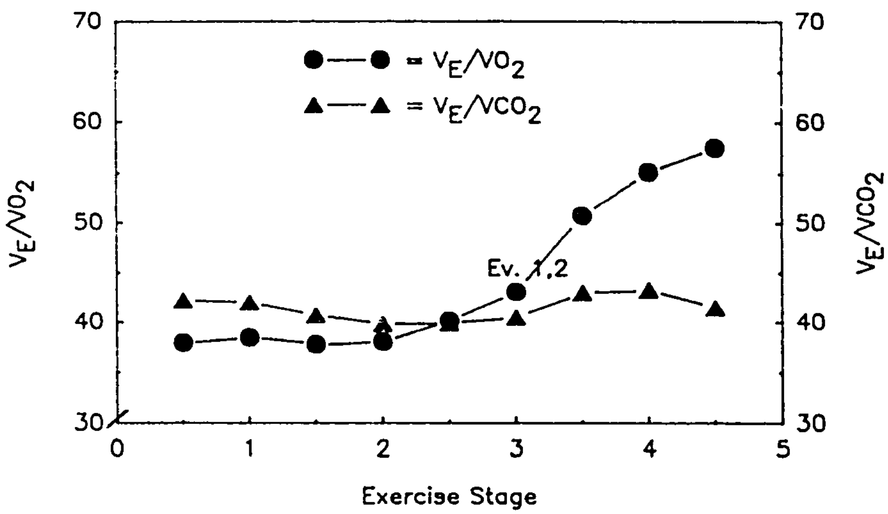


Figure III-2a Ventilatory Threshold of a Typical Subject (no. 10) during Trial Two on the Wheelchair Ergometer, as Identified by Two Evaluators

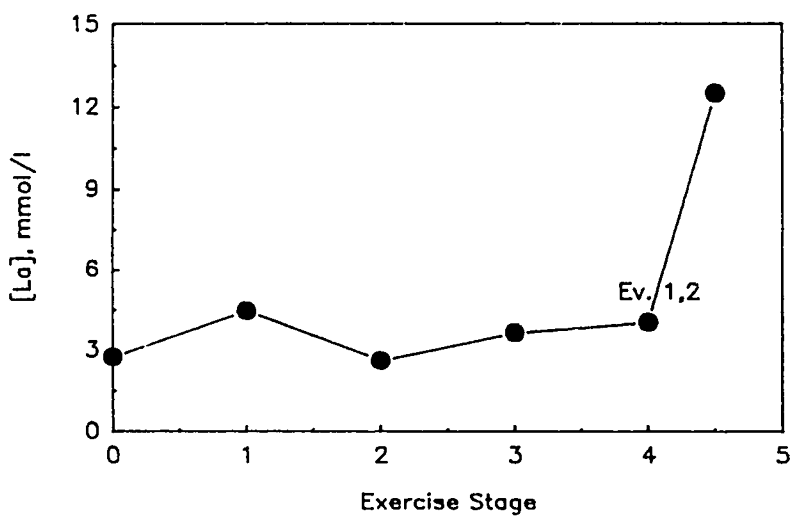


Figure III-2b Lactate Threshold of a Typical Subject (no. 10) during Trial Two on the Wheelchair Ergometer, as Identified by Two Evaluators

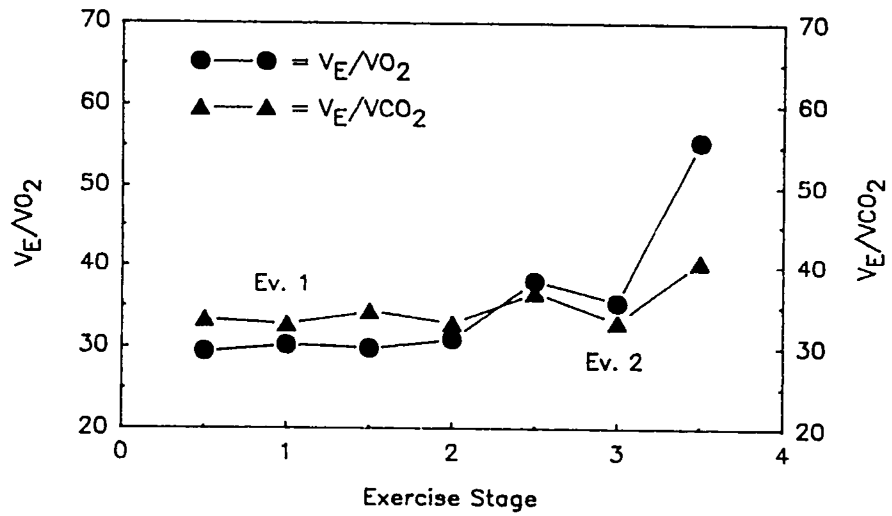


Figure III-3a Ventilatory Threshold of an Atypical Subject (no. 9) during Trial One on the Wheelchair Ergometer, as Identified by Two Evaluators

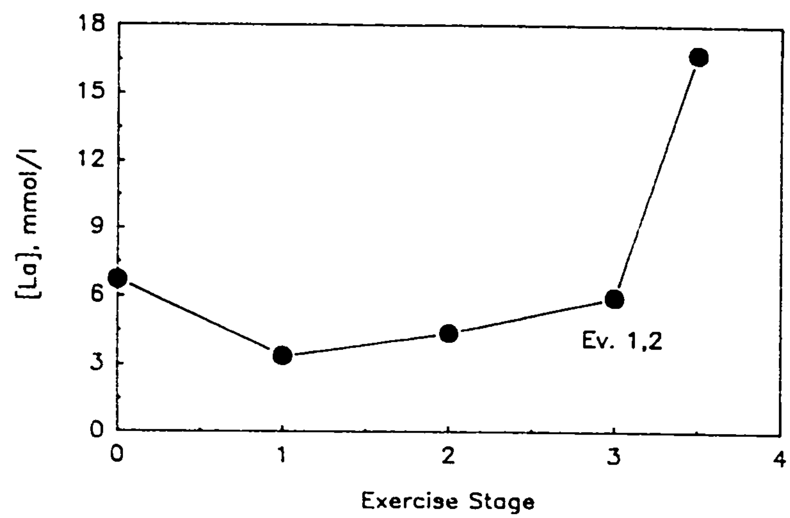


Figure III-3b Lactate Threshold of an Atypical Subject (no. 9) during Trial One on the Wheelchair Ergometer, as Identified by Two Evaluators

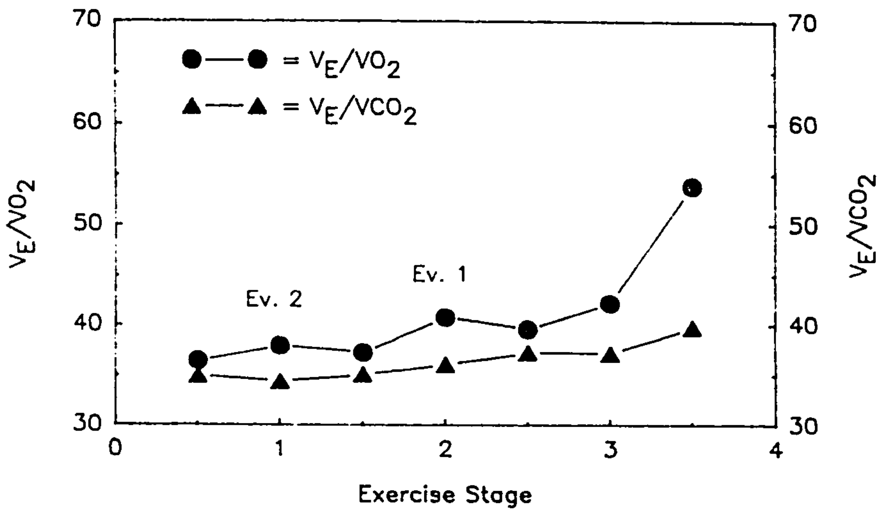


Figure III-4a Ventilatory Threshold of an Atypical Subject (no. 9) during Trial Two on the Wheelchair Ergometer, as Identified by Two Evaluators

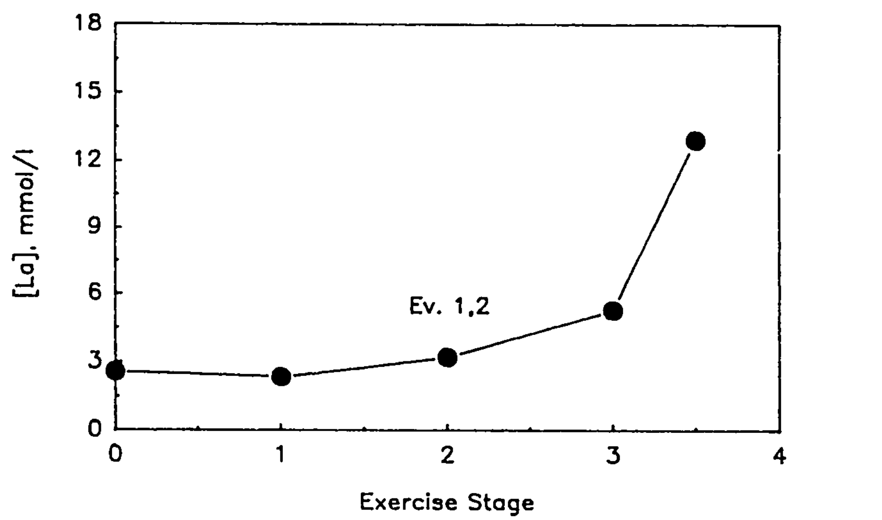


Figure III-4b Lactate Threshold of an Atypical Subject (no. 9) during Trial Two on the Wheelchair Ergometer, as Identified by Two Evaluators

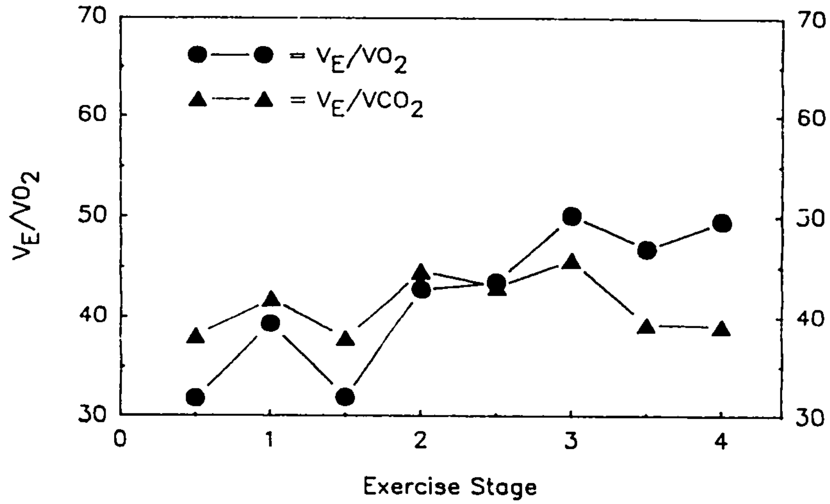


Figure III-5a $V_E/VO_2$ and $V_E/VCO_2$ Graphed over Time for an Atypical Subject (no. 1) during Trial One on the Wheelchair Ergometer in which no Ventilatory Threshold could be Identified by Two Evaluators

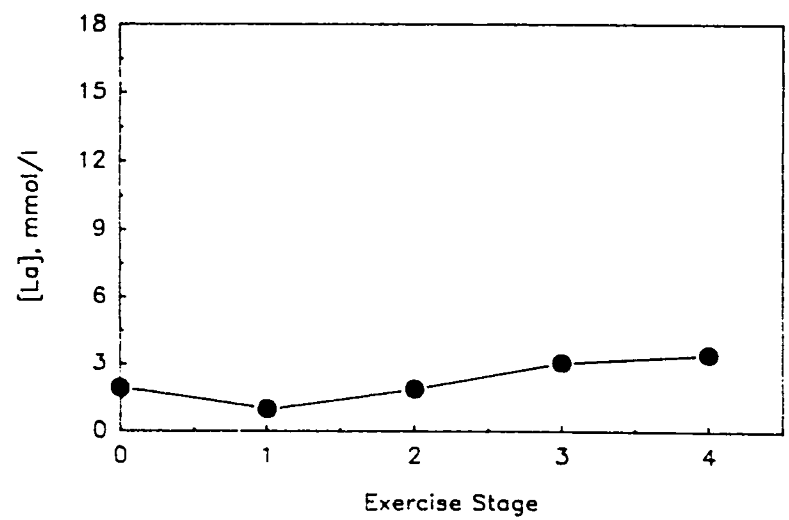


Figure III-5b Lactate Concentration Graphed over Time for an Atypical Subject (no. 1) during Trial One on the Wheelchair Ergometer in which no Lactate Threshold could be Identified by Two Evaluators

# CHAPTER IV

## General Discussion

This chapter will address the hypotheses that were proposed in chapter I. Thereafter, suggestions will be put forward for coaches and therapists, specific to cardiorespiratory fitness parameters in athletes with CP. Ultimately, recommendations will be made for further investigations.

**Hypotheses**

The first hypothesis proposed that the WE would be considered a valid and reliable mode with which to measure $\dot{V}O_2max$ when compared to the CE. Low validity coefficients suggest that the WE is not a valid mode with which to measure $\dot{V}O_2max$ because results were not significantly correlated to those measured on the CE. One must recall however, that the ANOVA revealed no significant differences between the means. As well, one must question whether the CE should be considered the "gold standard" for $\dot{V}O_2max$ testing for wheelchair athletes with CP since more than half the subjects were unable to pedal the CE due to insufficient hip and/or knee flexion. In this study, the CE was the chosen mode of comparison because to date, it has been the only mode used to test persons with CP; as well, it is a valid and reliable mode for the non-disabled population. Typically, those individuals who do not use a wheelchair to carry out their tasks of daily living were able to pedal the CE. It appears therefore, that the coach or therapist must learn how the subject ambulates on a day-to-day basis prior to determining the $\dot{V}O_2max$ testing mode. A guideline for the coach/therapist to follow is that wheelchair ambulatory people are likely best measured on the WE while individuals who frequently walk (with or without aids) may be tested on the CE. Accuracy of $\dot{V}O_2max$ measurement will not be compromised during either of the exercise modes since reliability is high for both. In order to provide consistency in terms of establishing $\dot{V}O_2max$ standards and/or norms for class 3 and 4 athletes with CP, coaches and therapists should conduct all tests on the WE so that a large pool of subjects can be compared.

One may further question the process of validating the WE in this study because results from upper body and lower limb exercises were compared, and one would naturally expect results from the CE to be higher because a larger muscle mass was recruited to carry out the exercise. It must be understood however, that athletes in class 3 or 4 have minimal to severe lower limb impairment and little upper body involvement. Therefore the upper body may have more functional muscle than the lower limbs.

The reader may also question comparative methods used to make the graded exercise tests progressively difficult; in the WE test, speed of the exercise was increased while in the CE assessment, resistance was elevated. Can these two methods can be compared? Research data on the CE indicates that optimal results are gathered at speeds of 60 to 85 rpm (Ettinger, 1989; Gueli & Shephard, 1977); therefore, tension must be added in order to increase the difficulty of the task. Unilateral motion of cycling aids in the maintenance of momentum since force is continually applied by at least one leg. Meanwhile, speed on the WE is increased to elevate task difficulty because the wheelchair slows down so much during the recovery portion of stroke if resistance is high, and much of the momentum is lost. This is due to the bilateral nature of the wheelchair stroke. As well, the style of the stroke must be changed dramatically if tension is added (i.e., shorter push phase); as a consequence, the concept of specificity is ignored.

The second hypothesis suggested that both modes would provide reliable results for all physiological responses monitored at $\dot{V}O_2max$ in this study. The WE provided consistent data for all variables (except RER) while the CE was reliable only for $\dot{V}O_2max$, likely due to the small number of athletes who could pedal the CE. For both modes, the ANOVA did not reveal any significant differences. Thus, additional testing must be carried out on the CE in order to provide more accurate results on this mode. Meanwhile, WE results can be considered reliable for testing of $\dot{V}O_2max$ (absolute and relative), HR, $\dot{V}_E$, $O_2$ pulse, $\dot{V}_E/\dot{V}O_2$ ratio, lactate, tidal frequency, and tidal volume.

The third hypothesis implied that no significant differences between LT would be observed between the two trials on both the CE and WE. Since the sample size for the CE was only five, this mode was not considered. For the twelve lactate thresholds identified by the evaluators, reliability was poor (r=-0.45 and -0.18 for evaluators 1 and 2 respectively), even though the ANOVA did not indicate significant differences between trials. As well, intra-individual differences between trials were large. Thus, an alternate method to identify AT should be considered.

The fourth hypothesis stated that no significant differences between VT would be observed for the two modes and trials. Again, results on the CE were not considered due to the small sample size. Despite no significant differences revealed by the ANOVA, reliability coefficients for the two evaluators indicated poor (r=0.65) to good consistency (r=0.79) for the seven subjects in which VT was identified. Smaller intra-individual differences combined with better reliability suggest that this may be the preferred method to determine anaerobic threshold. As well, VT is implemented with greater ease.

The fifth hypothesis proposed that no significant differences would be observed between evaluators in the identification of anaerobic threshold. Evaluators differed 14% of the time when determining VT and 20% for LT. These differences are similar to those documented by other researchers (Powers et al., 1984). Smaller variation utilizing the VT method adds support for implementing this technique over the LT method to determine AT.

The final hypothesis indicated that significant differences would be observed between lactate and ventilatory thresholds. Validity coefficients between LT and VT were low; for evaluator 1 during trials 1 and 2 respectively, the coefficients were -0.32 and 0.76; for evaluator 2 they were -0.53 and -0.67 for the same two trials. These results were considerably lower than the 0.93 validity coefficient reported by Caiozzo et al. (1982). LT lagged behind VT in the current study which was a similar finding reported by Yeh et al. (1983) and Posner et al. (1987); thus, LT usually occurred at a higher percentage of $\dot{V}O_2max$.

**Conclusion**

Results from the two studies presented in this thesis suggest that coaches and therapists should feel confident that the WE will provide reliable maximal data when testing class 3 or 4 individuals with CP. In order to establish standards for $\dot{V}O_2max$, the WE should be the chosen mode so that a large subject pool can be recruited. However, if the individual ambulates on a daily basis without a wheelchair and standards are of no importance, the CE may be the preferred testing mode.

If the coach or therapist is interested in collecting data pertaining to $\dot{V}O_2max$ and other physiological responses typically monitored during this type of testing (i.e., heart rate, $\dot{V}_E$, $\dot{V}_E/\dot{V}O_2$), a discontinuous protocol with 2 kmh increments is appropriate. However, if the objective is to gather information about anaerobic threshold, a continuous protocol with small increments or a discontinuous protocol with long exercise bouts is suggested. As well, implementing the gas exchange method (specifically VT), is preferred as it appears to be more reliable than the LT method and it is performed with greater ease since blood samples are not required.

**Future Research**

The two investigations described in this thesis have provided insight into the area of cardiorespiratory fitness for wheelchair athletes with cerebral palsy. However, further research related to this area of fitness is vital before new questions about cardiorespiratory fitness in this population are investigated. Three questions of paramount importance come to mind. (1) Can VT and/or LT accurately and reliably be used to identify AT in wheelchair athletes with CP? Results from this thesis indicate that VT is the preferred method, but further investigation is required before a coach or therapist can confidently implement either technique. Likely the LT method is not appropriate due to the varying levels of spasticity experienced by persons with CP. As well, it is important that evaluators closely follow the criteria used to identify break away points for both the VT and LT methods. (2) Can VT and LT be used for exercise prescription? Many researchers (Henritze et al., 1985; Smith & O'Donnell, 1984) have demonstrated that training just above anaerobic threshold elevates the threshold, allowing the athlete to exercise at a higher percentage of $\dot{V}O_2max$ without concomitant acculmuation of blood lactate. Therefore, if an athlete's VT or LT is identified, and training is carried out just above this threshold, will optimal training results be achieved? Obviously, the length of the event will determine whether or not the athlete can exercise at such a high intensity. Presently, the longest sanctioned track event for class 3 and 4 athletes with CP is 1500m. World records for the 1500m and 800m are 7:01.30 and 2:59.41, and 4:21.67 and 2:12.49 for class 3 and 4 respectively. Thus, AT could potentially be an important variable for these athletes to use. However, until additional knowledge is gathered about AT, the coach may prefer to prescribe a training program based on $\dot{V}O_2max$ performance. (3) Is endurance performance significantly correlated with VT and LT? Thus, if a class 4 athlete competes just above anaerobic threshold in the 1500m race, will peak performance be accomplished? In order to answer questions 2 and 3, the investigator must ensure that the athlete is well acquainted with exercising at precise intensities so that training and competition are carried out as required, and appropriate findings can be documented.

**References**

Caiozzo, V.J., Davis, J.A., Ellis, J.F., Azus, J.L., Vandagriff, R., Prietto, C.A., & McMaster, W.C. (1982). A comparison of gas exchange indices used to detect the anaerobic threshold. Journal of Applied Physiology, 53, 1184-1189.

Ettinger, A.V. (1989). Cardiovascular and perceived exertion responses to cycling velocities. Unpublished master's thesis, University of Alberta, Edmonton.

Gueli, D. & Shephard, R.J. (1977). Pedal frequency in bicycle ergometry. Canadian Journal of Applied Sports Science, 1, 137-141.

Henritze, J., Weltman, A., Schurrer, R.L., & Barlow, K. (1985). Effects of training at and above the lactate threshold on the lactate threshold and maximal oxygen uptake. European Journal of Applied Physiology and Occupational Physiology, 54, 84-88.

Posner, J.D., Gorman, K.M., Klein, S., & Cline, C.J. (1987). Ventilatory threshold: measurement and variation with age. Journal of Applied Physiology, 63(4), 1519-1525.

Powers, S.K., Dodd, S., & Garner, R. (1984). Precision of ventilatory and gas exchange alterations as a predictor of tne anaerobic threshold. European Journal of Applied Physiology and Occupational Physiology, 52, 173-177.

Smith, D.A. & O'Donnell, T.V. (1984). The time course during 36 weeks' endurance training of changes in $VO_2max$. and anaerobic threshold as determined with a new computerized method. Clinical Science, 67, 229-236.

Yeh, M.P., Gardner, R.M., Adams, T.D,, Yanowitz, F.G., & Crapo, R.O. (1983). "Anaerobic threshold": problems of determination and validation. Journal of Applied Physiology, 55, 1178-1186.