

**ADAPTATIONS OF ENERGY SYSTEMS
IN COMPETITIVE SWIMMING:**
A Physiological Evaluation of a NCAA
Division III Training Program

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Table of Contents

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|--------------------------|----|
| I. Abstract | 1 |
| II. Introduction | 2 |
| III. Materials & Methods | 11 |
| IV. Results | 13 |
| V. Discussion | 28 |
| VI. Summary | 39 |
| VII. Appendix | 41 |
| VIII. Bibliography | 52 |

Abstract

The training effects of a 21-week swim season on anaerobic threshold (AT), maximum oxygen consumption (mVO_2), and plasma lactate were studied with six members of the Washington and Lee University swim team as subjects. The season was subdivided into discrete training phases, and percentages of energy systems utilized in training each week were calculated. Swimming tests, measuring plasma lactate, and treadmill tests, measuring AT and mVO_2 , were performed throughout the season to determine if the expected adaptations were occurring. One increase in mVO_2 was observed, two increases in AT were observed, and five of the subjects had increased lactate levels, while only four swimmers exhibited increasing velocity in the tests. Five physiological adaptations were scrutinized based on these test results. It is concluded that the training program is satisfactory. Recommendations for optimization of the program are made.

Introduction

Much research has been performed evaluating different training strategies of elite NCAA Division I athletes; however, few studies have been performed using Division III college athletes. NCAA Divisions I and III differ not only in level of competition, but also in overall objectives. In general, Division I institutions place greater emphasis on benefits to the team and the institution, while Division III institutions emphasize benefits to the individual (NCAA News, 1978). Thus, regimens optimal for Division I teams may not be appropriate for a Division III team such as Washington and Lee. This project was undertaken to determine the optimal training program for sprinters, middle distance, and distance swimmers at the Division III level. An overall plan was proposed for the 1991-92 season for the Washington and Lee swim team, and physiological tests were performed to determine if the swimmers responded to the plan as expected. Swimmers from each of the three training groups were chosen to be subjects in the anaerobic threshold, maxVO₂, and plasma lactate testing.

In devising a training program, one must take into account the development and interaction of several physiological systems. These systems include the cardiovascular system carrying oxygen and fuel to the muscles, the muscular system translating energy into forces, and the various energy systems providing the fuel to run the muscles. These fuels include ATP (adenosine triphosphate), CP (creatine phosphate), glycogen, and fats, which must all eventually be converted to ATP to be used by the muscles. The mechanisms for

converting the various substances to ATP differ greatly, and can be considered as three phases: (1) ATP/CP reaction, (2) anaerobic metabolism, and (3) aerobic metabolism.

The simplest mechanism is the relationship of CP to ATP, which allows muscles to contract at maximum speed by replacing ATP as soon as it is used (Troup, 1990). CP is stored in the muscle cells, and when it is hydrolyzed it releases 12 kcal/mol of free energy with endproducts of creatine and inorganic phosphate (P_i). The ATP is continuously resynthesized from ADP (adenosine diphosphate) and P_i (inorganic phosphate) by the energy liberated during the breakdown of stored CP. Since the muscle stores of ATP and CP are very small (20 mmol/kg muscle), the amount of energy from this system is limited and its usefulness lies in its rapid availability (Strauss, 1984). Due to the limited amount of stored CP and ATP, this mechanism provides the majority of energy in approximately the first 10 seconds of exercise, then ATP must be provided by glycolysis (Troup, 1990).

Glycolysis is an anaerobic pathway since oxygen is not a direct participant in this pathway. However, the rate of glycolysis is influenced by the availability of oxygen. It was Pasteur who first noted that oxygen retards glycolysis (Stryer, 1989). Oxygen debt in muscle conversely accelerates glycolysis to produce ATP without the oxygen requiring aid of the tricarboxylic acid cycle and subsequent electron transport, although these methods are more efficient. The endproduct of glycolysis is ultimately lactate, a major contributor to muscle fatigue. This endproduct diffuses from muscle to be eventually converted by the

liver to glucose via gluconeogenesis. The synthesis of lactate from glucose and its eventual reconversion back to glucose is known as the Cori cycle. It and glycolysis are detailed in biochemistry texts, such as Darnell et al. (1990). The anaerobic metabolism is important because it provides a rapid source of energy, along with the ATP/CP reaction, lasting from approximately 10-40 seconds in exercise (Troup, 1990). Lactate production rises to a maximum level from 2-7 minutes then declines to approximately zero in 10-20 minutes during moderate exercise. A detailed discussion of lactate production can be found in Stainsby (1986).

During aerobic metabolism, glycogen is converted to carbon dioxide and water ultimately, and it yields much more ATP than anaerobic metabolism. This process occurs in the mitochondria of muscle cells in the presence of oxygen, and no fatigue byproducts are formed. Fats and proteins can also be broken down aerobically to carbon dioxide and water with energy being released. This mechanism of ATP production is used under resting conditions and also during prolonged, endurance-type activities. The large sustained output is possible only when early fatigue is avoided, and large amounts of glycogen, fats, and oxygen are available. The aerobic phase of exercise is anything over approximately 40 seconds (Troup, 1990).

Another method of ATP production is via lipid metabolism, which is an energy source in daily training, because trained athletes rely more upon fat catabolism than glycogen catabolism (Troup, 1990). Fats are an abundant source of energy; one mole of a C_{16} fatty acid can produce 136 moles of ATP. The process of fatty

acid oxidation can be examined in detail in Maglischo (1982). The initiation of fatty acid catabolism requires the release of human growth hormone (Paxton, 1986), and as with most endocrine processes, is slow and rather long lasting. Therefore, it comes into play only during endurance exercise, generally beyond 15-20 minutes (Troup, 1990). Lipid metabolism is also important in preventing muscle glycogen depletion from day to day.

Although these metabolic pathways are distinct from one another, they are interrelated in such a way that an energy continuum exists relating the way in which ATP is made available and the type of activity being performed. Any one system usually contributes more during a given activity; however, both aerobic and anaerobic processes contribute certain amounts of ATP continually. The contribution of the energy supplying processes during exercise depends on several factors (Maglischo, 1982). The first factor is pace, because faster speeds require rapid muscle contractions; therefore, the body must rely on the faster ATP/CP and anaerobic reactions for energy. The second factor is the ability to consume oxygen, because, for example, a swimmer consuming more oxygen during a race will be able to oxidize more NADH, $FADH_2$, and pyruvate in the mitochondria and thereby decrease anaerobic glycolysis. Stroke efficiency in swimming is also a factor, because more efficient strokes require less effort; therefore, the total energy requirement is decreased. Proper training results in the increased ability to store energy for later release, and in the ability to release energy in a more efficient fashion during exercise.

In order to achieve these proper training results, U.S. Swimming (1990) has categorized training methods into aerobic and anaerobic energy systems to allow coaches and swimmers to know which system they should utilize. The energy system utilized is a result of the swim set and the way it is performed. The aerobic system can be further divided into three categories, and the anaerobic system can be divided into four categories. In the aerobic systems, A1 is Anaerobic 1, A2 is Aerobic 2, and AT 1,2 is Anaerobic Threshold. In the anaerobic systems, AOV is Aerobic Overload, mVO2 is Max Oxygen Consumption, LP1 is Lactate Peak, and ALP2 is Alactic Peak. Refer to Table 1 and Table 2 for descriptions of each energy system.

Aerobic and anaerobic energy systems are used in various combinations throughout the season to optimize performance. U.S. Swimming (1990) has also divided the athletic season into phases, and all energy systems are not used in every phase. Each phase should last from 3-6 weeks if possible, to allow for physiological adaptations to occur. The phases are further broken into microcycles lasting a week to allow for changes to be made in the training schedule due to uncontrollable circumstances. The phases are referred to as preparatory, competitive, and transitional.

The season begins with a preparatory phase, which places an emphasis on overall conditioning to raise the base level of preparation and then changes to emphasize the specific needs of the swimmer in relation to their unique competitive events. The first part of the phase consists of a general and gradual increase in volume (yardage) at a fairly low intensity along with improving AT

Table 1. Characteristics of Aerobic Energy Systems

- | | |
|-----------|--|
| A1 | <ul style="list-style-type: none"> • Warm-up swims and recovery swims • Removal of lactate • 70% of mVO₂ • Prevents soreness and cramps • Work 0 to 30 minutes/swim 500 to 2000 yards • Varied R.I. (rest interval) |
| <hr/> | |
| A2 | <ul style="list-style-type: none"> • Warm-up pace with slightly higher intensity • Base training 70 to 80% of mVO₂ • Up to 83% speed • Increase HR (heart rate) and flexibility • Work 0 to 60 minutes/swim 500 to 4000 yards • 10 to 30 seconds R.I. |
| <hr/> | |
| AT | <ul style="list-style-type: none"> • Equal production and removal of lactate • 70 to 75% of mVO₂ • Work 10 to 60 minutes/swim 800 to 4000 yards • AT1 - endurance/short rest, 5 to 10 seconds R.I. • AT2 - middle distance development, longer rest, 10 to 30 seconds R.I. |

Table 2. Characteristics of Anaerobic Energy Systems

- AOV**
- 90% of mVO₂
 - High intensity with short rest
 - Enhance lactate tolerance
 - Majority of yardage for middle distance swimmers
 - Work 10-30 minutes/ swim 500-2500 yards
 - Work to rest ratio is 1:1/4
-

- mVO₂**
- 90% effort
 - Improve maximum endurance capacity
 - High intensity swim with adequate rest interval
 - Amount increases later in the season
 - Work 5-20 minutes/ swim 500-2000 yards
 - Work to rest ratio is 1:1/2-1
-

- LP1**
- Training paces result in high lactate concentration
 - Improves maximum lactate value
 - Amount increases later in season
 - Race economy
 - Work 5-15 minutes/ swim 200-1200 yards
 - Work to rest ratio is 1:2+
-

- ALP2**
- ATP/CP efficiency
 - High intensity work of short duration
 - Improve power
 - 100/200 yard pace work
 - Work 5-20 minutes/ swim 50-500 yards
 - Work to rest ratio is 1:1+

and increasing the mVO_2 . Swimmers must also concentrate on gaining muscular strength and improving stroke mechanics. When more specificity is needed, the intensity increases to more closely mimic the intensity of racing; however, the yardage will only increase gradually since the emphasis is on specific events. The total preparatory phase will be approximately 2/3 of the total season.

Competition is the next phase, and it consists of the training regimen taking place during scheduled meets, culminating at the Championship meet. In this phase, swimmers must maintain general fitness while increasing performance levels in subsequent meets. The training volume will decrease slightly to allow for more rest during high quality sets. The swimmers are also maintaining AT and mVO_2 , while developing the anaerobic components of their races through more repeat swims at race pace or faster (Maglischo, 1982).

The transitional phase is a systematic recovery period from the stress of heavy training, in which the overall fitness level may drop. This phase occurs when swimmers decrease volume and intensity over breaks, and during the taper, a rest period. Transition occurs during cross-training or during non-specific training, such as the first part of the preparatory phase. The taper maintains the adaptations of training accomplished during the earlier phases, but also allows for recovery so the swimmer can perform at higher levels of mVO_2 in meets. The taper is 2-5 weeks of reduced work with increased quality expected, and varies depending on the length of the season.

Swimmers should use the phases with varying combinations of

energy systems to maximize the training potential of each system. Five important adaptations must occur: (1) improved AT, (2) increased lactate tolerance, (3) delayed fatigue, (4) improved mVO₂, and (5) improved ATP/CP reaction. These adaptations were observed in the physiological tests performed throughout the season.

Materials & Methods

Six swimmers from the Washington and Lee swim team were chosen to participate in this study to determine the type of adaptations actually occurring during the competitive season. Using a treadmill, breath analyzer, and ECG at the VMI physiology lab, Anaerobic Threshold and maxVO₂ were measured on three dates. The tests were performed pre-season, mid-season, and immediate post-season. The subjects stood still for 4 minutes to obtain baseline data, then walked slowly for 2 minutes before beginning to run at the set pace. Males ran at a constant speed of 6.5 mph, and the females ran 5.5 mph, but both groups had a 1% incline each minute. The subjects were instructed to run to exhaustion, then walk out for a sufficient recovery. Swimming tests were performed 4 times during the season in order to test blood lactate levels. The tests were October 11, November 14, January 5, and sometime in March depending on the specific Championship meet for the individual. The time between tests differs somewhat because of breaks in school; however, they are still at an appropriate time to measure adaptations. The swimmers warmed up before the test, then began the test with 4X100 yard freestyle on 2:30 with heart rate kept below 140 beats per minute. A fifth 100 yard swim was done at max effort and was followed by a recovery swim. After a full recovery, a 200 yard freestyle and 500 yard freestyle were performed and followed by full recoveries. Velocity was recorded for each maximum effort, and blood lactate concentration and heart rate (HR) were also determined immediately following each maximum effort swim. Lancets were used to puncture finger capillaries and 10 ul

capillary tubes were used to collect the blood. The lactate concentration was estimated using a plasma lactate colorimetric assay (Sigma #735-10). Heart rate was determined by each swimmer taking his/her own pulse. Surveys of the swimmers were also conducted to obtain some additional background information concerning training before and during the season.

Results

Blood lactate: Table 3 indicates the overall changes in the blood lactate levels, but more specific fluctuations during the season are detailed in Figures 1-6, and Tables 1,2, and 3 of the Appendix. Two of the three females had decreasing velocity in all swims, while one female showed a velocity increase in all swims. Cofield had decreasing lactate values in all swims, and Dudley showed increasing lactate values for all swims. Also, Fisher showed increasing lactate in the 100 yard swim; however, her lactate values decreased in the 200 and 500 yard swims. On the other hand, all male subjects had increasing velocities along with increasing lactate values. The greatest velocity increase for all males was present in the 100 yard swim, and Pearson showed the greatest lactate increase in the 200 yard swim. Rowe and Brown exhibited the greatest lactate increase in the 500 yard swim.

Treadmill: Table 4 indicates the overall changes in the treadmill tests, but finer fluctuations during the season can be examined in Figures 7-11 and Tables 4 and 5 of the Appendix. Dudley and Brown were the only subjects to show an increase in the maximum time; however, Dudley, Pearson, and Rowe showed increases in the anaerobic threshold time. Pearson showed the only significant increase in the anaerobic threshold, as is seen in a 24% increase of maxVO₂. Brown exhibits the only significant increase in maxVO₂, with an increase of 576 ml/min (15%).

Workout Analysis: Tables 6, 7, and 8 of the Appendix indicate the actual yardage and percentage of each week's training spent utilizing specific energy systems. The low percentages of AN1 and

AN2 indicate that very little high intensity sprint training occurred throughout the season, and a greater emphasis was placed on aerobic training.

| OVERALL CHANGES IN BLOOD LACTATE TESTS | | | | | | |
|---|-------------|---------|-------------|---------|-------------|---------|
| | 100 yd swim | | 200 yd swim | | 500 yd swim | |
| | Velocity | Lactate | Velocity | Lactate | Velocity | Lactate |
| Cofield | -.04 | -3.47 | -.07 | -1.66 | -.07 | +.34 |
| Dudley | +.09 | +2.61 | +.06 | +.93 | +.04 | +5.64 |
| Fisher | -.02 | +1.19 | 0 | -2.76 | -.01 | +1.39 |
| Pearson | +.14 | +.67 | +.02 | +5.72 | +.02 | +3.93 |
| Rowe | +.14 | +3.47 | +.09 | +4.21 | +.07 | +5.20 |
| Brown | +.14 | +3.75 | +.14 | +3.48 | +.10 | +7.47 |

Table 3. Results showing the overall changes throughout the season. Velocity is measured in m/sec, and lactate is measured in mmol/l. The values were calculated by subtracting the initial value from the final value. The raw data can be found in the Appendix under Tables 1, 2, and 3.

OVERALL CHANGES IN
TREADMILL TESTS

| | Anaerobic Threshold | | | | Maximum | | |
|---------|---------------------|--------|-------|-------|---------|--------|-------|
| | VO2 | VO2/KG | %mVO2 | Time | VO2 | VO2/KG | Time |
| Dudley | -66 | +1 | 0 | +1:40 | -84 | +1.3 | +2:30 |
| Fisher | -373 | -5.6 | +1% | -1:10 | -499 | -7.6 | -:30 |
| Pearson | +319 | +5.9 | +24% | +3:10 | -805 | -9.3 | -1:30 |
| Rowe | -195 | -4.1 | -6% | +:05 | +144 | 0 | -:30 |
| Brown | +193 | +2.9 | -5% | -:10 | +576 | +8.1 | +1:30 |

Table 4. Results showing overall changes in treadmill tests throughout the season. VO2 is measured in ml/min and time is in minutes. The values were calculated by subtracting the initial value from the final value. The raw data can be found in the Appendix under Tables 4 and 5.

STACEY COFIELD LACTATE/VELOCITY

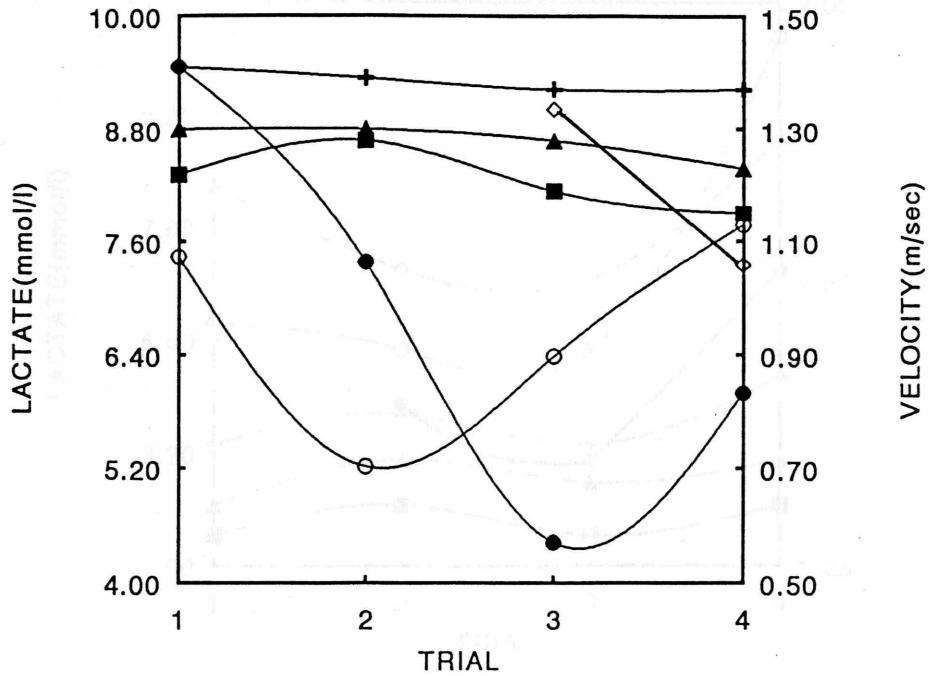


Figure 1. Fluctuations in lactate and velocity observed throughout the season. The symbols are: dark circle- 100 yard lactate, open diamond- 200 yard lactate, open circle- 500 yard lactate, cross- 100 yard velocity, dark triangle- 200 yard velocity, and dark square- 500 yard velocity. Greater fluctuations are present in the lactate values than the velocity.

CLAIRE DUDLEY LACTATE/VELOCITY

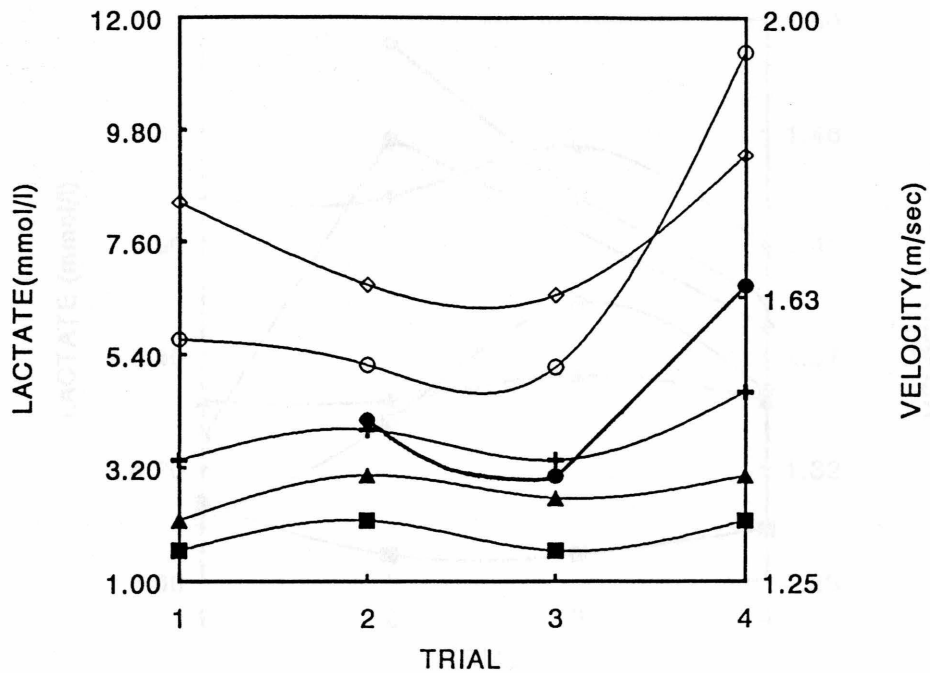


Figure 2. Fluctuations in lactate and velocity observed throughout the season. The symbols are: dark circle- 100 yard lactate, open diamond- 200 yard lactate, open circle- 500 yard lactate, cross- 100 yard velocity, dark triangle- 200 yard velocity, and dark square- 500 yard velocity. Lactate values show a greater increase than velocity.

SUSAN FISHER LACTATE/VELOCITY

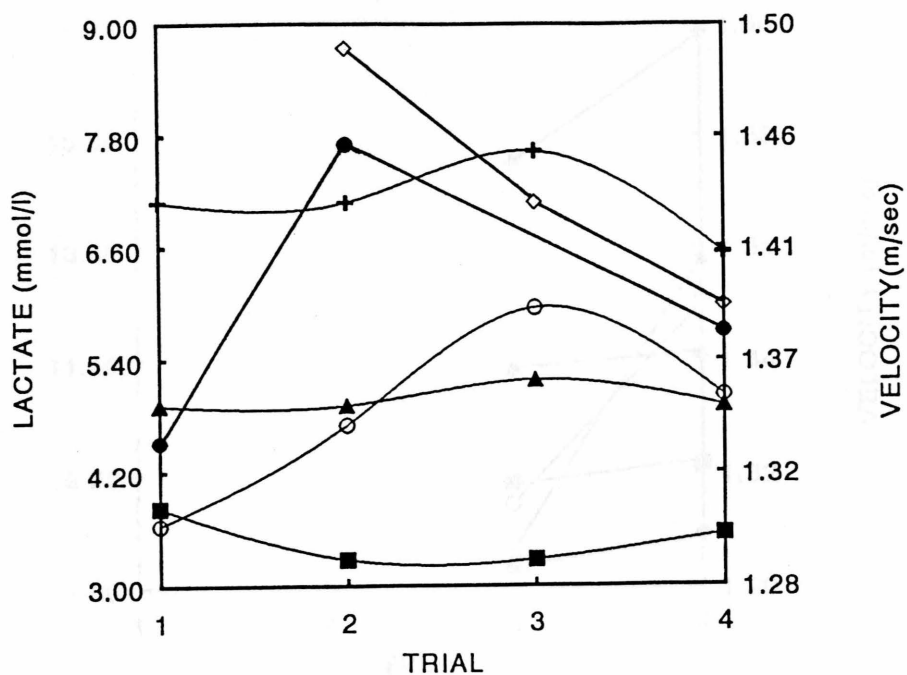


Figure 3. Fluctuations in lactate and velocity observed throughout the season. The symbols are: dark circle- 100 yard lactate, open diamond- 200 yard lactate, open circle- 500 yard lactate, cross- 100 yard velocity, dark triangle- 200 yard velocity, and dark square- 500 yard velocity. Great fluctuations are present in lactate and velocity.

ANDREW PEARSON LACTATE/VELOCITY

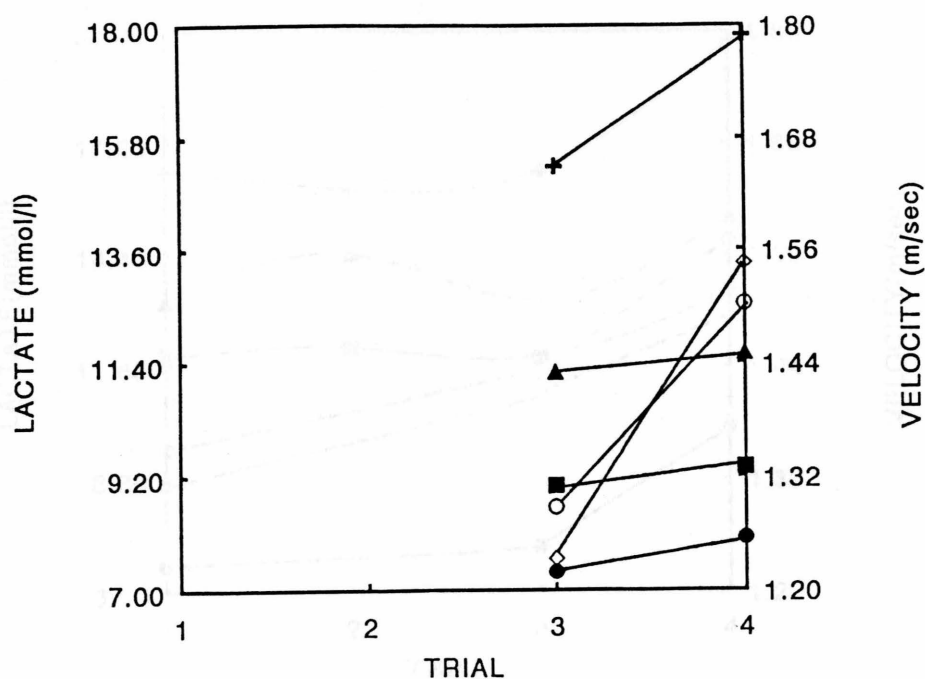


Figure 4. Fluctuations in lactate and velocity observed throughout the season. The symbols are: dark circle- 100 yard lactate, open diamond- 200 yard lactate, open circle- 500 yard lactate, cross- 100 yard velocity, dark triangle- 200 yard velocity, and dark square- 500 yard velocity. Increases are present in lactate and velocity from the third to the fourth trial. Not present for first two tests due to water polo season.

JOHN ROWE LACTATE/VELOCITY

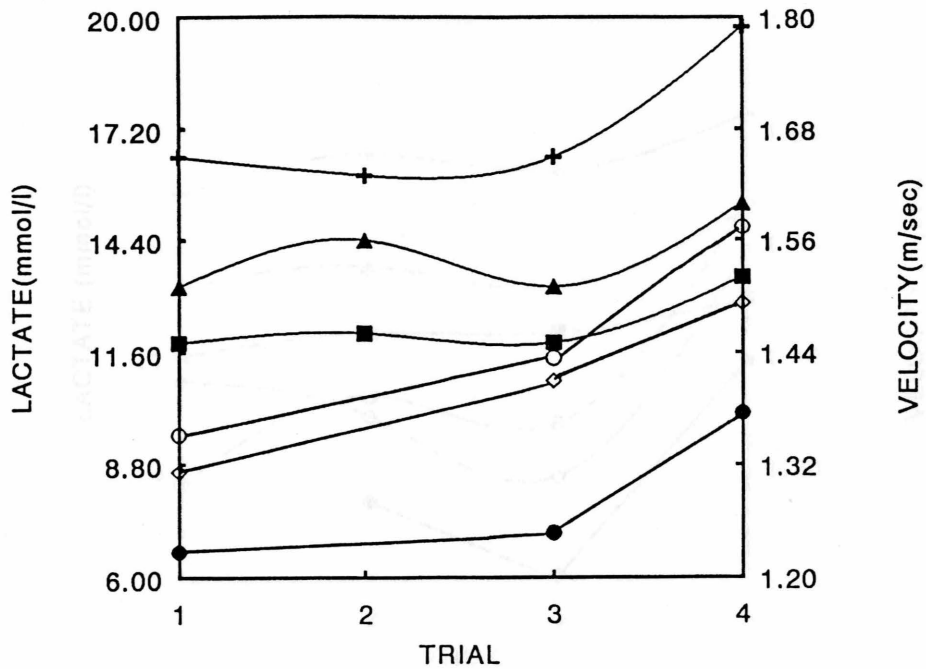


Figure 5. Fluctuations in lactate and velocity observed throughout the season. The symbols are: dark circle- 100 yard lactate, open diamond- 200 yard lactate, open circle- 500 yard lactate, cross- 100 yard velocity, dark triangle- 200 yard velocity, and dark square- 500 yard velocity. Lactate and velocity show increases throughout the season.

DOUG BROWN LACTATE/VELOCITY

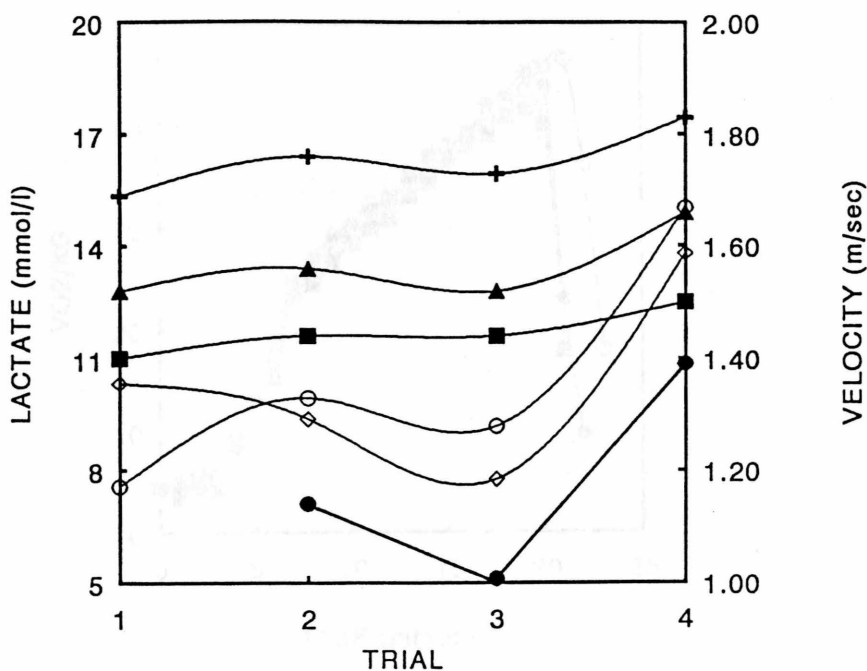


Figure 6. Fluctuations in lactate and velocity observed throughout the season. The symbols are: dark circle- 100 yard lactate, open diamond- 200 yard lactate, open circle- 500 yard lactate, cross- 100 yard velocity, dark triangle- 200 yard velocity, and dark square- 500 yard velocity. A greater increase is present in the lactate values than in the velocity.

CLAIRE DUDLEY TREADMILL VO₂/KG

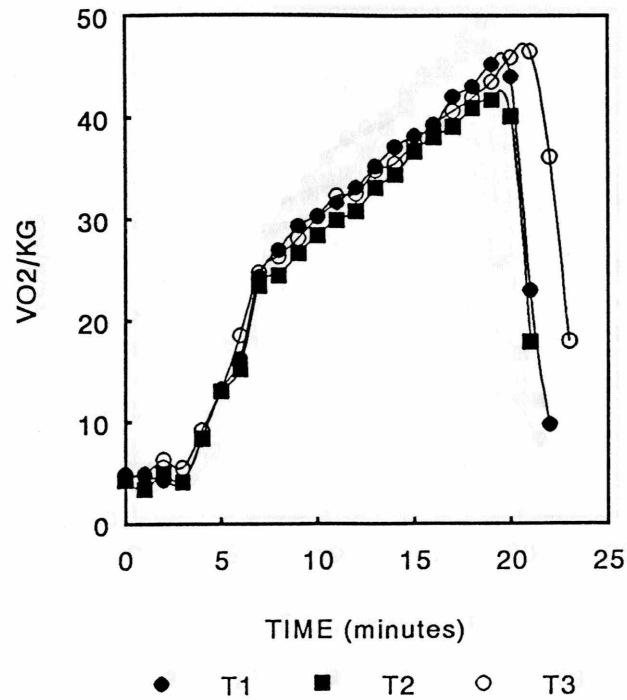


Figure 7. Results showing VO₂/KG curves for the three treadmill tests. A slight increase is present from the first to the third test.

SUSAN FISHER TREADMILL VO₂/KG

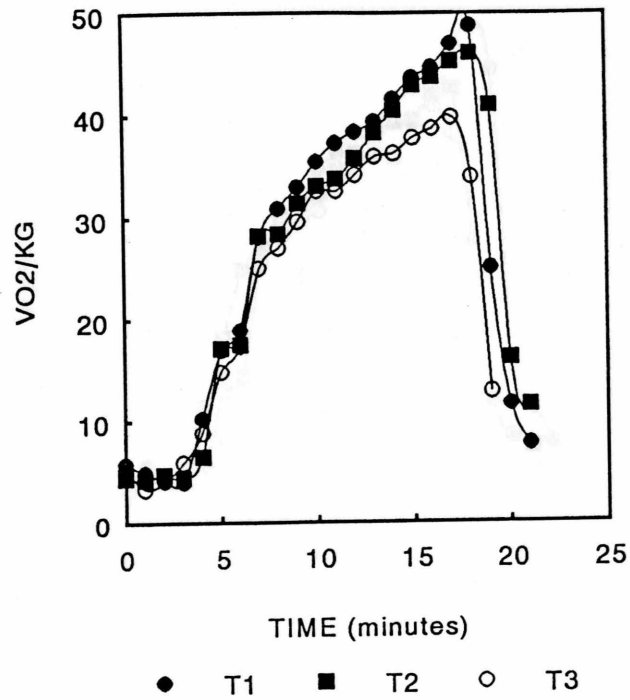


Figure 8. Results showing VO₂/KG curves for the three treadmill tests. A notable decrease is present between the first and the third test.

ANDREW PEARSON TREADMILL VO2/KG

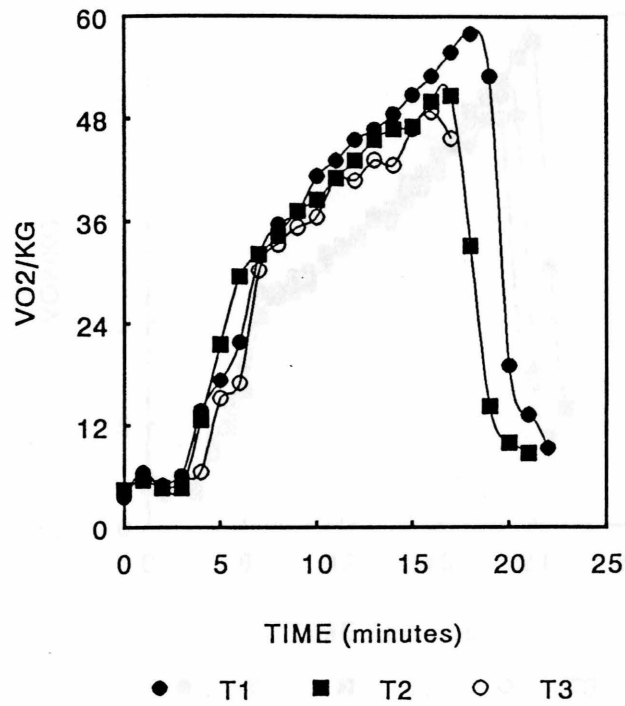


Figure 9. Results showing VO2/KG curves for the three treadmill tests. A notable decrease is present between the first and the third test.

JOHN ROWE TREADMILL

VO₂/KG

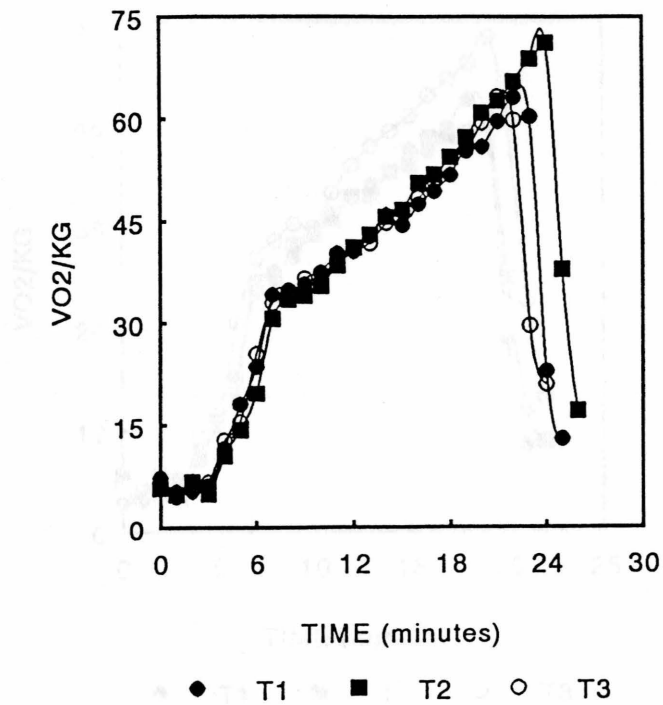


Figure 10. Results showing VO₂/KG curves for the three treadmill tests. An increase is present between the first and second test, but the values decrease between the second and third test.

DOUG BROWN TREADMILL VO₂/KG

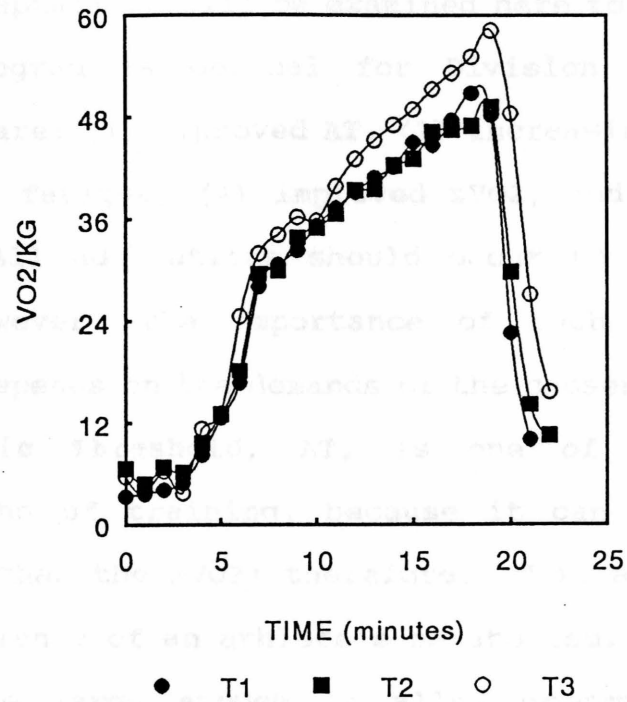


Figure 11. Results showing VO₂/KG curves for the three treadmill tests. An increase exists between the first and the third test.

Discussion

Since more data were generated than is necessary to fulfill the purpose of this thesis, only the most relevant observations will be examined. However, all data are included in the Appendix to allow for further examination and study in the future. Five important adaptations will be examined here to estimate whether our training program is optimal for Division III swimmers. The adaptations are: (1) improved AT, (2) increasing lactate tolerance, (3) delaying fatigue, (4) improved mVO_2 , and (5) improved ATP/CP reaction. All adaptations should occur to some extent in each swimmer; however, the importance of each adaptation for the individual depends on the demands of the chosen athletic specialty.

Anaerobic Threshold, AT, is one of the most important considerations of training, because it can be more effectively conditioned than the mVO_2 ; therefore, AT is also a good indicator of the efficiency of an athlete's metabolism. The change in AT is likely to be large enough to allow progress to be monitored throughout the season, even though a change in mVO_2 may not be measurable. The AT is the point at which anaerobic metabolism is producing lactate at such a rate that clearance capabilities are exceeded and blood lactate levels increase sharply (Nye, 1987). Based on that definition, "lactate threshold" may be a more appropriate term than "anaerobic threshold," because many muscle fibers may still be aerobic during the transition (Sharkey, 1990). Although anaerobic metabolism occurs before the threshold is reached, lactate in the muscles does not immediately increase the plasma lactate concentration. This is due to the lactate being

metabolized in working muscles and diffusing into adjacent resting muscle fibers rather than into the blood. The plasma lactate concentration is also kept low by the increasing efficiency of aerobic metabolism, which reduces the need for anaerobic metabolism. Lactate is further removed from the blood by the heart, liver, and other muscles as soon as plasma levels peak (Maglischo, 1982).

AT training plays an especially significant role when one swims distances of 400 yards or more. The rate of lactate removal from working muscles can be improved by training at speeds slightly beyond the present AT. This type of training should optimally provide lactate excess to stimulate faster removal rates without interference from severe acidosis. Based on this hypothesis, Maglischo (1982) suggests that one should train at rates which produce plasma lactate concentrations slightly in excess of 4 mmol/L. This concentration represents a significant increase of plasma lactate above normal levels of 1-2 mmol/L. The increased concentration indicates that lactate is being produced faster than it can be metabolized, with the excess amount being poured into the blood. This also signifies that aerobic metabolism is operating at near-maximum to maximum capacity.

Anaerobic capacity is, however, an individual entity, since the stores of phosphocreatine and the extent to which lactate can accumulate are limited (Medbo et al., 1988). For this reason, blood lactate tests run throughout the season are helpful in determining the levels of conditioning in each individual. Another method, suggested by Kindermann (1979), is to monitor heart rate

while measuring lactate levels in order to find the heart rate corresponding to AT. This would allow the swimmers to use heart rate to more closely monitor the energy system being utilized during workouts. A combination of both methods would be ideal, because the lactate tests indicate when repeat times or intervals need to be changed due to adaptations causing faster velocities, while the heart rate measurements allow for daily monitoring. The results in Table 3 indicate the great variability in lactate values. The treadmill results (Table 4) indicate that Pearson and Brown show the only increase in $\dot{V}O_2$ values at the AT, and Pearson shows a notable increase in the $\%m\dot{V}O_2$. These results suggest that the portion of the training program responsible for overloading and improving anaerobic capacity is optimal for these individuals. The AT results for the other individuals, however, suggest that the anaerobic training may not be optimal for the entire team; therefore, other adaptations must be examined to determine what changes need to be made in the program.

Improving the AT is the most important adaptation for distance swimmers, because it reflects an increase in $m\dot{V}O_2$, an increase in lactate removal, and a decrease in lactate production. Well-trained endurance athletes reach AT when working at 85-90% of $m\dot{V}O_2$. The values obtained from the subjects in this study are somewhat lower than these values, but this is most likely due to the difference between elite athletes and Division III athletes. Athletes training for endurance events should optimally produce less lactate, because they tend to have more slow twitch fibers which have a greater capacity for aerobic metabolism (Maglischo,

1982). A comparison between Pearson and Brown shows that Brown produces more lactate; however, this is not necessarily detrimental to his swimming, because an increased lactate tolerance could help offset this problem.

Increasing lactate tolerance is thus another important adaptation in training, which permits energy to be supplied anaerobically during races so that maximum speeds can be maintained for long periods. Lactate tolerance is improved through improved buffering, increased M-LDH activity, and an increased tolerance to the pain of acidosis (Maglischo, 1982). Improved buffering capacity reduces the acidotic effect of lactate, preventing drastic pH decreases during exercise. The buffer systems can act immediately at the onset of exercise to prevent pH drops. However, the value of buffering to overall performance is still uncertain. For example, an improved pain tolerance also aids in maintaining a fast pace in spite of declining pH in muscles. Therefore, swimmers with higher pain tolerance can supply more energy anaerobically despite increasing acidosis, as is seen by Brown and Dudley in the final swim test.

Increased lactate tolerance is especially important in 100 and 200 yard swims, because this exercise period is too short to allow sufficient amounts of oxygen to be consumed to prevent reliance on glycolysis. These swims, therefore, cause a rapid accumulation of lactate. Paradoxically, to improve lactate tolerance, lactate production must be caused to increase with training. Such increased production is evident in five of the six subjects tested in this study. An increase in blood lactate after training

indicates that the swimmer is appropriately capable of producing more lactate during a swim, representing an enhanced glycolytic rate; therefore, faster speeds are maintained longer.

At the biochemical level, enhanced glycolytic rates are reflected in increased activities of key glycolytic enzymes, such as hexokinase, phosphorylase, and M-LDH (Maglischo, 1982). The most significantly increased enzyme activities have been seen after high intensity sprint training while some decrease has been found after endurance training. High intensity sprint training results in a large oxygen deficit because the generated power outputs exceed maximal aerobic capacity, causing an increase in glycolytic and oxidative enzyme activities (Jacobs et al., 1987).

Delaying fatigue caused by the accumulation of lactate is another essential adaptation to training. This delay is achieved by reducing the amount of lactate accumulation through a reduced rate of lactate production. According to Maglischo (1982), the reduced production is accomplished through an increased $\dot{V}O_2$. The increased $\dot{V}O_2$ allows for more pyruvate and electron pairs to enter the mitochondria and be used in subsequent aerobic metabolism. Lactate production is also reduced since pyruvate, the substrate for LDH, can be removed from working muscles when it is aminated to form alanine. The alanine then diffuses into the blood and is converted to glucose in the liver. An increase in alanine has been observed in human plasma during exercise, supporting this hypothesis. The conversion of pyruvate to alanine is estimated to reduce lactate formation by 35-60% in trained athletes.

Fatigue can also be delayed by increasing lactate removal,

because greater removal delays the pH reduction in the muscles. As has been mentioned, lactate diffuses into adjacent resting fibers within the same muscle to be metabolized for energy. However, some lactate diffuses into the bloodstream and is transported to other resting muscles, the heart, and the liver to be metabolized (Maglischo, 1982). Improvement in the lactate removal mechanisms could be important in decreasing much lactate accumulation in long sprints and middle distance swims. Enhancing removal of lactate is most effective in delaying fatigue in races under 500 yards. The removal mechanisms are dependent on the increased liver enzyme activity and on the amount of blood flowing through the muscles. Any increase in blood to the muscles should improve the rate of removal, because lactate transport from muscle is dependent upon free diffusion.

Donovan and Brooks (1983) suggested that training in an animal model affects lactate clearance rather than lactate production. Their studies revealed equivalent lactate production in trained and untrained animals; however, the trained animals exhibited greater clearance of lactate from the blood. In trained animals there was improved glucose homeostasis resulting from increased gluconeogenesis and reduced lactate oxidation during hard exercise. The bulk of lactate removal, through oxidation, is probably metabolized at sites of high blood flow, which include cardiac and red skeletal muscle. Whatever mechanism is used, it appears that raising the blood lactate level in effect forces the lactate to be removed. All results indicate that substantial lactate oxidation occurs during or immediately following exercise.

An improvement in $m\dot{V}O_2$ is another important adaptation to be achieved during training. Results from $m\dot{V}O_2$ tests represent an individual's maximum ability to utilize oxygen for the purpose of supplying energy (Troup, 1990). An increased $m\dot{V}O_2$ probably results from an improved transportation of oxygen by the circulatory system and an increased extraction and utilization of oxygen by the muscles. The possibility of increasing $m\dot{V}O_2$ is based on the idea that the respiratory system itself does not limit the $\dot{V}O_2$, because humans can exhale nearly half of the oxygen inhaled during strenuous exercise. Cook and Brynteson (1973), however, found no improvement in $m\dot{V}O_2$ during a season of swimming and attributed this to the fact that the swimmers were initially in excellent condition and high maximal oxygen uptake was attained during summer training. The absence of a significant increase in four of the subjects suggests that they were already in good condition before the season began. Brown, who trained very little during the summer, showed the greatest increase in $m\dot{V}O_2$, while the other subjects, who trained harder over the summer, showed little or no increase in $m\dot{V}O_2$. In order to change the $m\dot{V}O_2$, training affects the circulatory system, capillary density, blood flow, and blood volume (Maglischo, 1982).

In the circulatory system, when blood passes through tissues it gives up oxygen and takes up lactic acid and carbon dioxide. Cardiac output, the amount of blood pumped by the heart every minute, is approximately 2.5-3.6 L/min at rest and increases to over 30 L/min during strenuous exercise. In one study, cardiac output increased 18% with training. Training maximum cardiac

output is best accomplished with long swims and short rest interval repeats at moderate speed (EN1 and mVO2). This type of training is believed to increase the total number of capillaries surrounding the muscle fibers, which is important because when blood reaches the muscle fibers it diffuses from the capillaries. It may be, however, that some nonfunctional capillaries become functional.

Blood flow is also affected by training, through a greater amount of blood being sent to the muscles during exercise. Approximately 15-20% of the blood goes to muscles during rest; however, during exercise 85-90% of the blood goes to the muscles. This increase occurs because arteries supplying the muscles dilate while other arteries get smaller. At submaximal training, some studies have shown that blood flow decreases, which could mean that fewer muscle fibers are needed to perform the same amount of work, or that there is an increased oxygen extraction by the muscles. With more oxygen being extracted by the muscles and fewer fibers working, less blood is required. Studies have reported contradictory results of blood flow increase or decrease depending on the type of training performed. For instance, with the great amount of A1 and A2 training during the season, the blood flow could have decreased keeping the mVO2 from increasing.

Blood volume and red blood cell (RBC) count are also affected by training, because trained athletes tend to have more total blood volume and a greater RBC count. Moderate to high intensity middle distance swims with short rest intervals (mVO2) are best to improve these effects. These swims would create great oxygen demand, which would require more RBC to carry the oxygen, and may also produce

hypoxia causing a training increase in blood volume and RBC count. With the low percentages (<15%) of mVO_2 training in the program, these adaptations probably did not occur at an optimal level.

Various training methods can be used to achieve an improved mVO_2 . Maglischo (1982) has recommended the best way to improve mVO_2 is to use exercise periods of 3-5 minutes at 80-90% effort. The body requires 2-3 minutes to adjust to the increased oxygen demand and to begin providing it at a maximum rate. This delayed effect occurs because the need for oxygen must be created before the oxygen transporting mechanisms can be stimulated to maximum performance. The rest interval here is unimportant, because the effect on the body occurs within the swim. To obtain the optimal effect, the mVO_2 must be reached and held until the end of the set. A short rest interval does not allow for complete recovery between repeats; hence, each swim begins with the VO_2 more elevated than the previous swim, and overload of the system occurs.

According to Kindermann et al. (1979), the optimal load for endurance training which will increase the mVO_2 should be in the range of intensity to produce 4 mmol/L lactate. These conditions stimulate oxidative metabolism in skeletal muscle cells, with very little use being made of mechanisms that would lead to lactate production. If training occurs at a lower level than this, the high volume load of the heart will lead to improvement in the cardiovascular system, but the stimulus is insufficient to achieve adaptations in muscle cells. These results indicate that training at a lower intensity will maintain the state of conditioning, while training at the 4 mmol/L stage will increase exercise capacity.

Results from the present study support this hypothesis, because although the intensity was not high enough to increase mVO_2 , a steady level of conditioning was maintained.

Heart rate (HR) can also be used to determine levels of training which will increase the mVO_2 . HR is linearly related to the exercise intensity and is easier to monitor than mVO_2 . The average maximum HR, with a 10 bpm (beat per minute) standard deviation, can be estimated by subtracting the individual's age from 220 (Strauss, 1984). For lower conditioning, the average HR can be estimated by the equation: $(60\% \text{ max HR} - \text{Resting HR}) + \text{Resting HR}$. For higher conditioning, the average HR can be estimated from the equation: $(80\% \text{ max HR} - \text{Resting HR}) + \text{Resting HR}$. Interval training at less than maximum HR is designed to increase AT and mVO_2 , and training at maximum HR should increase lactate tolerance (Maglischo, 1982). This is the type of HR monitoring that can be used in combination with the lactate tests discussed above. According to Counsilman (1977), exercise with a HR below 120 bpm is 100% aerobic work. A HR of 120-150 bpm represents 90-95% aerobic work, 150-160 bpm is 65-85% aerobic work, 165-180 bpm is 50-65% aerobic work, and anything over 180 bpm represents over 50% anaerobic work. Based on studies performed by Costill et al. (1988), the exercising HR should decrease as a result of training, allowing a swimmer to perform the same work with less effort and to perform faster swims with a similar HR (Nye, 1987). An increased HR during rest or exercise would be a good indicator of overtraining (Kirwin et al, 1988), because this indicates that the effort must be increased to perform the same work as before.

Improved ATP/CP efficiency is the most important adaptation for sprinters; however, they must have a conditioning base with an established AT and mVO₂ before concentrating on speed work. The efficiency of this reaction partially depends on the concentration of ATP and CP in the muscles (Maglischo, 1982). Muscles contract at maximum speed until the CP supply is partially depleted, and they continue to contract at near maximum speed until the muscles are nearly depleted. Maglischo (1982) has reported a 39% increase in CP and a 25% increase in ATP after using endurance training programs. In some studies, an increase in CP was shown, but no ATP increase was present; therefore, it seems likely that certain training methods are able to increase the CP content of muscles.

Enzyme activity also affects the efficiency of the ATP/CP reaction (Maglischo, 1982). ATPase controls the rate of energy release by ATP, MK (Myokinase) regulates the breakdown of ADP to AMP, and CPK (Creatine Phosphokinase) regulates the replacement of ATP by CP. Training causes increased enzyme activity, which may increase the rate of energy release from ATP and the rate of replacement from CP. If increased enzyme activity occurs, a greater speed is possible for a longer period of time. Increased enzyme activity is best accomplished through short repeat swims at maximum speed, in excess of 95% race pace. The rest interval should be long enough to allow near complete replacement of the CP supply. If CP is not restored, anaerobic metabolism will become the major energy source, and lactate will accumulate. Excess lactate will reduce speed, and the ATP/CP reaction will not be stimulated (Maglischo, 1982). Race pace training improves the

interaction of metabolic processes, and it may provide a means of reducing the contributions of anaerobic energy sources during races. The energy systems most emphasized during this type of training are AN1 and AN2 (LP1 and ALP2).

Since our training program consisted of extremely low percentages of sprint training, one may conclude that an insufficient stimulus was present for the ATP/CP reaction. This would cause the swimmers to show little increased performance in sprint events; however, Pearson showed an increase in velocity in the 100 yard swim and in meet performance. This condition of increased sprint performance without emphasis on AN1 and AN2 training contradicts the above ideas; although, studies by Hickson et al. (1976) suggest that similar enzyme adaptations occur with endurance as well as sprint training. An unexpected result of his study was that glycolytic enzyme activity does not increase with sprint training, despite the dependence on glycolysis during sprinting. The adaptive changes in sprint-trained muscles were in the same direction as the adaptations in endurance-trained muscles; although, they were smaller in magnitude. The aerobic training for Pearson, therefore, may have been at a high enough intensity to stimulate the necessary enzymatic changes.

Summary

All the adaptations examined are indicators of conditioning and should be used to inform the coach and the swimmer if changes need to be made in the training program. The results from the tests performed throughout the season suggest that the training program is satisfactory; however, it could be enhanced to lead to

better performance. The first necessary change is to increase the amount of high intensity sprint training and $\dot{m}V_{O_2}$ training. Too much emphasis was placed on aerobic training, and the intensity of the work was not high enough to stimulate the above adaptations to an optimal level. Optimal training regimens differ among individuals; however, a well-planned program with appropriate emphasis placed on each energy system will prove to be successful for the majority of swimmers. Another consideration is pre-season training, as mentioned concerning $\dot{m}V_{O_2}$. Some pre-season training is important to achieve a base conditioning level; however, high intensity training year-round can be detrimental to performance. If the swimmers begin the season with a fairly strong aerobic base, more consideration can be given to specific needs of the individual. For instance, sprinters can concentrate more on $\dot{m}V_{O_2}$ and lactate tolerance training. The conclusions reached in this study should provide some guidelines for future seasons and for monitoring conditioning levels of the swimmers more closely.

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APPENDIX

SUBJECT PERFORMANCE COMPARISON
100 YARD TRIALS

| TEST | PARAMETER | Susan (M) | Doug (D) | Stacey(S) | Andrew(S) | Claire(M) | John (M) |
|------|------------------|-----------|----------|-----------|-----------|-----------|----------|
| 1 | Velocity (m/sec) | 1.43 | 1.69 | 1.41 | --- | 1.41 | 1.65 |
| | Lactate (mmol/L) | 4.51 | --- | 9.46 | --- | --- | 6.62 |
| | Heart Rate(min) | 170 | 200 | 160 | --- | 180 | 200 |
| 2 | Velocity (m/sec) | 1.43 | 1.76 | 1.39 | --- | 1.45 | 1.63 |
| | Lactate (mmol/L) | 7.71 | 7.10 | 7.38 | --- | 4.12 | --- |
| | Heart Rate(min) | 180 | 220 | 160 | --- | 180 | 200 |
| 3 | Velocity (m/sec) | 1.45 | 1.73 | 1.37 | 1.65 | 1.41 | 1.65 |
| | Lactate (mmol/L) | 2.64 | 5.11 | 4.42 | 7.35 | 3.04 | 7.10 |
| | Heart Rate(min) | 180 | 220 | 180 | 190 | 180 | 220 |
| 4 | Velocity (m/sec) | 1.41 | 1.83 | 1.37 | 1.79 | 1.50 | 1.79 |
| | Lactate (mmol/L) | 5.70 | 10.85 | 5.99 | 8.02 | 6.73 | 10.09 |
| | Heart Rate(min) | 180 | 210 | 170 | 200 | 170 | 210 |

Table 1. Results from sprint trials. Lactate was determined as explained in the Materials and Methods section. Heart rate was taken by the subjects themselves for a six second period immediately after the trial. S=sprinter, M=middle distance, D=distance swimmer.

| SUBJECT PERFORMANCE COMPARISON 200 YARD TRIALS | | | | | | | |
|---|------------------|-----------|----------|-----------|-----------|-----------|----------|
| TEST | PARAMETER | Susan (M) | Doug (D) | Stacey(S) | Andrew(S) | Claire(M) | John (M) |
| 1 | Velocity (m/sec) | 1.35 | 1.52 | 1.30 | --- | 1.33 | 1.51 |
| | Lactate (mmol/L) | 2.39 | 10.32 | --- | --- | 8.38 | 8.61 |
| | Heart Rate(min) | 180 | 190 | 160 | --- | 170 | 190 |
| 2 | Velocity (m/sec) | 1.35 | 1.56 | 1.30 | --- | 1.39 | 1.56 |
| | Lactate (mmol/L) | 8.74 | 9.36 | --- | --- | 6.75 | --- |
| | Heart Rate(min) | 180 | 200 | 170 | --- | 160 | 200 |
| 3 | Velocity (m/sec) | 1.36 | 1.52 | 1.28 | 1.43 | 1.36 | 1.51 |
| | Lactate (mmol/L) | 7.09 | 7.76 | 9.01 | 7.61 | 6.55 | 10.88 |
| | Heart Rate(min) | 170 | 190 | 170 | 180 | 170 | 220 |
| 4 | Velocity (m/sec) | 1.35 | 1.66 | 1.23 | 1.45 | 1.39 | 1.60 |
| | Lactate (mmol/L) | 5.98 | 13.80 | 7.35 | 13.33 | 9.31 | 12.82 |
| | Heart Rate(min) | 160 | 210 | 180 | 190 | 170 | 210 |

Table 2. Results from middle distance trials. Lactate was determined as explained in the Materials and Methods section. Heart rate was taken by the subjects themselves for a six second period immediately after the trial. S=sprinter, M=middle distance, D=distance swimmer.

| SUBJECT PERFORMANCE COMPARISON 500 YARD TRIALS | | | | | | | |
|---|------------------|-----------|----------|-----------|-----------|-----------|----------|
| TEST | PARAMETER | Susan (M) | Doug (D) | Stacey(S) | Andrew(S) | Claire(M) | John (M) |
| 1 | Velocity (m/sec) | 1.31 | 1.40 | 1.22 | --- | 1.29 | 1.45 |
| | Lactate (mmol/L) | 3.63 | 7.56 | 7.44 | --- | 5.69 | 9.53 |
| | Heart Rate(min) | 180 | 200 | 160 | --- | 190 | 200 |
| 2 | Velocity (m/sec) | 1.29 | 1.44 | 1.28 | --- | 1.33 | 1.46 |
| | Lactate (mmol/L) | 4.70 | 9.92 | 5.22 | --- | 5.19 | --- |
| | Heart Rate(min) | 190 | 200 | 180 | --- | 175 | 200 |
| 3 | Velocity (m/sec) | 1.29 | 1.44 | 1.19 | 1.31 | 1.29 | 1.45 |
| | Lactate (mmol/L) | 5.95 | 9.17 | 6.38 | 8.61 | 5.15 | 11.45 |
| | Heart Rate(min) | 180 | 220 | 160 | 180 | 170 | 220 |
| 4 | Velocity (m/sec) | 1.30 | 1.50 | 1.15 | 1.33 | 1.33 | 1.52 |
| | Lactate (mmol/L) | 5.02 | 15.03 | 7.78 | 12.54 | 11.33 | 14.73 |
| | Heart Rate(min) | 170 | 210 | 160 | 180 | 180 | 190 |

Table 3. Results from distance trials. Lactate was determined as explained in the Materials and Methods section. Heart rate was taken by the subjects themselves for a six second period immediately after the trial. S=sprinter, M=middle distance, D=distance swimmer.

Table 4. Results from the treadmill tests for females. The overall changes are presented in the Results section. The breath-by-breath parameters were averaged over thirty second intervals.

**TREADMILL TEST RESULTS
FEMALES**

| | SUSAN FISHER | | | CLAIRE DUDLEY | | |
|--------|--------------|--------|--------|---------------|--------|--------|
| | TEST 1 | TEST 2 | TEST 3 | TEST 1 | TEST 2 | TEST 3 |
| BASE | | | | | | |
| VO2 | 317 | 308 | 315 | 326 | 268 | 296 |
| VCO2 | 408 | 379 | 368 | 328 | 245 | 301 |
| VO2/KG | 5.2 | 5.0 | 5.3 | 5.2 | 4.6 | 5.1 |
| VE | 14.6 | 13.5 | 12.8 | 13.1 | 9.3 | 11.5 |
| R | 1.29 | 1.23 | 1.17 | 1.01 | .91 | 1.01 |
| HR | 87 | 82 | 89 | 81 | 78 | 76 |
| AT | | | | | | |
| VO2 | 2344 | 2525 | 1971 | 2136 | 1686 | 2070 |
| VCO2 | 2413 | 2488 | 2476 | 2141 | 1771 | 2330 |
| VO2/KG | 38.5 | 40.8 | 32.9 | 34.3 | 28.8 | 35.3 |
| VE | 58.1 | 63.0 | 60.2 | 58.7 | 50.1 | 66.5 |
| R | 1.03 | .99 | 1.26 | 1.00 | 1.05 | 1.13 |
| HR | 178 | 170 | 174 | 164 | 164 | 164 |
| %mVO2 | 81% | 86% | 82% | 76% | 68% | 76% |
| %mHR | 94% | 91% | 92% | 87% | 89% | 88% |
| TIME | 12:15 | 13:30 | 11:05 | 12:30 | 10:50 | 14:10 |
| MAX | | | | | | |
| VO2 | 2892 | 2925 | 2393 | 2810 | 2491 | 2726 |
| VCO2 | 3403 | 3459 | 3248 | 3551 | 3035 | 3673 |
| VO2/KG | 47.5 | 47.7 | 39.9 | 45.2 | 42.5 | 46.5 |
| VE | 92 | 89 | 78.7 | 108.9 | 95.2 | 114.0 |
| R | 1.18 | 1.18 | 1.36 | 1.27 | 1.22 | 1.35 |
| HR | 189 | 187 | 189 | 188 | 184 | 186 |
| TIME | 18:00 | 18:30 | 17:30 | 19:30 | 19:30 | 22:00 |

Table 5. Results from the treadmill tests for males. The overall changes are presented in the Results section. The breath-by-breath parameters were averaged over thirty second intervals.

**TREADMILL TEST RESULTS
MALES**

| | ANDREW PEARSON | | | JOHN ROWE | | | DOUG BROWN | | |
|--------|----------------|--------|--------|-----------|--------|--------|------------|--------|--------|
| | TEST 1 | TEST 2 | TEST 3 | TEST 1 | TEST 2 | TEST 3 | TEST 1 | TEST 2 | TEST 3 |
| BASE | | | | | | | | | |
| VO2 | 284 | 314 | 248 | 483 | 374 | 440 | 229 | 533 | 445 |
| VCO2 | 319 | 311 | 289 | 526 | 409 | 510 | 238 | 523 | 404 |
| VO2/KG | 3.8 | 4.5 | 3.5 | 7.1 | 5.5 | 6.2 | 3.0 | 6.9 | 5.8 |
| VE | 11.5 | 13.0 | 9.3 | 19.7 | 15.2 | 18.4 | 11.9 | 20.0 | 16.7 |
| R | 1.12 | 0.99 | 1.17 | 1.09 | 1.09 | 1.16 | 1.04 | 0.98 | 0.91 |
| HR | 95 | 100 | 82 | 71 | 82 | 76 | 67 | 67 | 61 |
| AT | | | | | | | | | |
| VO2 | 2787 | 2234 | 3106 | 2906 | 3044 | 2711 | 2799 | 2421 | 2992 |
| VCO2 | 3171 | 2508 | 4193 | 3571 | 3414 | 2894 | 3559 | 2779 | 3230 |
| VO2/KG | 37.6 | 32.1 | 43.5 | 42.6 | 44.3 | 38.5 | 36.0 | 31.1 | 38.9 |
| VE | 71.1 | 73.0 | 73.4 | 104.2 | 89.4 | 74.8 | 85.2 | 69.5 | 83.7 |
| R | 1.14 | 1.12 | 1.35 | 1.23 | 1.12 | 1.07 | 1.27 | 1.15 | 1.08 |
| HR | 173 | 160 | 184 | 141 | 153 | 143 | 168 | 149 | 160 |
| %mVO2 | 65% | 64% | 89% | 67% | 64% | 61% | 72% | 63% | 67% |
| %mHR | 84% | 78% | 92% | 73% | 80% | 75% | 89% | 80% | 81% |
| TIME | 9:30 | 7:00 | 12:40 | 11:25 | 13:40 | 11:30 | 11:20 | 9:55 | 11:10 |
| MAX | | | | | | | | | |
| VO2 | 4280 | 3509 | 3475 | 4311 | 4789 | 4455 | 3910 | 3849 | 4486 |
| VCO2 | 5758 | 4497 | 5029 | 5927 | 6356 | 5860 | 6249 | 5607 | 5507 |
| VO2/KG | 58.0 | 50.5 | 48.7 | 63.2 | 69.8 | 63.2 | 50.3 | 49.5 | 58.4 |
| VE | 138.5 | 122.0 | 122.0 | 184.6 | 185.3 | 172.2 | 164.4 | 168.0 | 160.4 |
| R | 1.34 | 1.28 | 1.45 | 1.38 | 1.32 | 1.32 | 1.59 | 1.45 | 1.27 |
| HR | 206 | 206 | 200 | 193 | 192 | 191 | 189 | 187 | 184 |
| TIME | 18:30 | 17:00 | 16:00 | 22:30 | 24:00 | 22:00 | 18:30 | 19:00 | 20:00 |

TABLE 6
ENERGY SYSTEMS TRAINED IN
SPRINTERS

| DATE | A1 | % | A2 | % | EN1 | % | mVO2 | % | AN1 | % | AN2 | % | TOTAL |
|----------|--------|----|--------|----|--------|----|-------|----|-------|---|-------|---|--------|
| 9/23/91 | 2,000 | 22 | 4,900 | 53 | 1,700 | 18 | 600 | 7 | 0 | 0 | 0 | 0 | 9,200 |
| 9/30/91 | 7,625 | 34 | 1,650 | 7 | 6,700 | 30 | 4,500 | 20 | 200 | 1 | 1,575 | 7 | 22,250 |
| 10/07/91 | 3,400 | 20 | 3,400 | 20 | 6,600 | 39 | 2,900 | 17 | 0 | 0 | 600 | 4 | 16,900 |
| 10/14/91 | 3,000 | 33 | 2,100 | 23 | 2,700 | 29 | 800 | 9 | 200 | 2 | 400 | 4 | 9,200 |
| 10/21/91 | 2,700 | 10 | 7,650 | 30 | 10,850 | 42 | 4,050 | 16 | 550 | 2 | 0 | 0 | 25,800 |
| 10/28/91 | 2,450 | 22 | 2,950 | 26 | 5,050 | 45 | 300 | 3 | 150 | 1 | 250 | 2 | 11,150 |
| 11/04/91 | 1,900 | 18 | 2,500 | 23 | 6,300 | 59 | 0 | 0 | 0 | 0 | 0 | 0 | 10,700 |
| 11/11/91 | 3,150 | 12 | 10,100 | 39 | 9,100 | 35 | 1,200 | 5 | 800 | 3 | 1,600 | 6 | 25,950 |
| 11/18/91 | 7,100 | 36 | 3,900 | 20 | 6,450 | 33 | 1,200 | 6 | 700 | 4 | 450 | 2 | 19,800 |
| 11/25/91 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 12/02/91 | 2,900 | 12 | 8,500 | 35 | 12,525 | 51 | 500 | 2 | 150 | 1 | 0 | 0 | 24,575 |
| 12/09/91 | 2,200 | 14 | 5,500 | 34 | 6,800 | 43 | 1,500 | 9 | 0 | 0 | 0 | 0 | 16,000 |
| 12/16/91 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 12/23/91 | 3,300 | 17 | 5,800 | 30 | 9,950 | 51 | 300 | 2 | 0 | 0 | 0 | 0 | 19,350 |
| 12/30/91 | 11,700 | 35 | 7,250 | 22 | 13,425 | 40 | 1,050 | 3 | 0 | 0 | 0 | 0 | 33,425 |
| 1/06/92 | 2,500 | 8 | 12,600 | 39 | 12,200 | 38 | 2,100 | 7 | 2,450 | 8 | 175 | 1 | 32,025 |
| 1/13/92 | 4,100 | 29 | 6,800 | 48 | 2,600 | 18 | 150 | 1 | 500 | 4 | 100 | 1 | 14,250 |
| 1/20/92 | 7,800 | 43 | 4,250 | 23 | 4,425 | 24 | 575 | 3 | 400 | 2 | 850 | 5 | 18,300 |
| 1/27/92 | 3,000 | 17 | 5,800 | 32 | 6,750 | 37 | 1,600 | 9 | 0 | 0 | 950 | 5 | 18,100 |
| 2/03/92 | 7,900 | 27 | 6,400 | 22 | 11,800 | 41 | 1,350 | 5 | 200 | 1 | 1,100 | 4 | 28,750 |
| 2/10/92 | 1,200 | 32 | 1,000 | 27 | 0 | 0 | 1,200 | 32 | 300 | 8 | 0 | 0 | 3,700 |
| 2/17/92 | 2,800 | 17 | 11,150 | 67 | 1,900 | 11 | 250 | 1 | 350 | 2 | 300 | 2 | 16,750 |
| TOTAL % | 22 | | 30 | | 37 | | 7 | | 2 | | 2 | | 100 |

Table 6. Results of workout analysis showing actual yardage and percentage of each energy system trained. A description of the energy systems can be found in the Introduction. EN1 is AT1,2, mVO2 is AOV and mVO2, AN1 is LP1, and AN2 is ALP2. The weeks with no values are during breaks from school.

TABLE 7
ENERGY SYSTEMS TRAINED IN
MIDDLE DISTANCE SWIMMERS

| DATE | A1 | % | A2 | % | EN1 | % | mVO2 | % | AN1 | % | AN2 | % | TOTAL |
|----------|--------|----|--------|----|--------|----|-------|----|-------|---|-------|----|--------|
| 9/23/91 | 2,000 | 20 | 4,900 | 50 | 2,300 | 23 | 600 | 6 | 0 | 0 | 0 | 0 | 9,200 |
| 9/30/91 | 7,625 | 34 | 1,650 | 7 | 6,700 | 30 | 4,500 | 20 | 200 | 1 | 1,575 | 7 | 22,250 |
| 10/07/91 | 3,400 | 21 | 3,175 | 20 | 6,000 | 38 | 2,900 | 18 | 0 | 0 | 500 | 3 | 15,975 |
| 10/14/91 | 3,000 | 29 | 2,900 | 28 | 1,600 | 16 | 2,800 | 27 | 0 | 0 | 0 | 0 | 10,300 |
| 10/21/91 | 2,700 | 10 | 7,650 | 30 | 10,850 | 42 | 4,050 | 16 | 550 | 2 | 0 | 0 | 25,800 |
| 10/28/91 | 2,450 | 21 | 3,600 | 30 | 4,200 | 35 | 1,450 | 12 | 0 | 0 | 250 | 2 | 11,950 |
| 11/04/91 | 1,900 | 17 | 2,500 | 22 | 6,800 | 61 | 0 | 0 | 0 | 0 | 0 | 0 | 11,200 |
| 11/11/91 | 3,150 | 10 | 10,700 | 34 | 14,050 | 44 | 3,450 | 11 | 0 | 0 | 250 | 1 | 31,600 |
| 11/18/91 | 4,100 | 29 | 3,700 | 27 | 5,800 | 42 | 0 | 0 | 0 | 0 | 300 | 2 | 13,900 |
| 11/25/91 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 12/02/91 | 2,900 | 11 | 8,600 | 33 | 12,700 | 48 | 1,900 | 7 | 200 | 1 | 0 | 0 | 26,300 |
| 12/09/91 | 2,600 | 11 | 7,100 | 30 | 11,700 | 50 | 2,100 | 9 | 0 | 0 | 0 | 0 | 23,500 |
| 12/16/91 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 12/23/91 | 3,300 | 17 | 5,800 | 30 | 9,950 | 51 | 300 | 2 | 0 | 0 | 0 | 0 | 19,350 |
| 12/30/91 | 14,600 | 30 | 11,950 | 25 | 20,400 | 42 | 1,500 | 3 | 250 | 1 | 0 | 0 | 48,700 |
| 1/06/92 | 2,600 | 9 | 10,650 | 37 | 12,000 | 42 | 2,100 | 7 | 900 | 3 | 175 | 1 | 28,425 |
| 1/13/92 | 2,800 | 24 | 4,675 | 40 | 1,875 | 16 | 1,600 | 14 | 200 | 2 | 0 | 0 | 11,550 |
| 1/20/92 | 7,100 | 34 | 6,200 | 30 | 4,825 | 23 | 1,800 | 9 | 600 | 3 | 350 | 2 | 20,875 |
| 1/27/92 | 5,700 | 17 | 8,000 | 24 | 10,050 | 31 | 4,800 | 15 | 2,000 | 6 | 2,400 | 7 | 32,950 |
| 2/03/92 | 4,300 | 38 | 3,000 | 26 | 1,400 | 12 | 1,500 | 13 | 100 | 1 | 1,100 | 10 | 11,400 |
| 2/10/92 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 2/17/92 | 1,700 | 5 | 18,850 | 55 | 8,000 | 23 | 1,950 | 6 | 2,250 | 7 | 1,500 | 4 | 34,250 |
| TOTAL % | 19 | | 31 | | 37 | | 10 | | 2 | | 2 | | 100 |

Table 7. Results from workout analysis showing actual yardage and percentage of each energy system trained. The weeks with no values are during breaks from school. Descriptions of each energy system can be found in the Introduction. EN1 is AT1,2, mVO2 is AOV and mVO2, AN1 is LP1, and AN2 is ALP2.

TABLE 8
ENERGY SYSTEMS TRAINED IN
DISTANCE SWIMMERS

| DATE | A1 | % | A2 | % | EN1 | % | mVO2 | % | AN1 | % | AN2 | % | TOTAL |
|----------|--------|----|--------|----|--------|----|-------|----|-------|---|-------|---|--------|
| 9/23/91 | 2,000 | 20 | 4,900 | 48 | 2,700 | 26 | 600 | 6 | 0 | 0 | 0 | 0 | 10,200 |
| 9/30/91 | 7,625 | 34 | 1,650 | 7 | 6,700 | 30 | 4,500 | 20 | 200 | 1 | 1,575 | 7 | 22,250 |
| 10/07/91 | 3,400 | 21 | 3,175 | 20 | 6,000 | 38 | 2,900 | 18 | 0 | 0 | 500 | 3 | 15,975 |
| 10/14/91 | 2,900 | 24 | 2,100 | 18 | 3,500 | 29 | 3,200 | 27 | 300 | 3 | 0 | 0 | 12,000 |
| 10/21/91 | 4,000 | 12 | 9,450 | 28 | 12,850 | 38 | 7,250 | 21 | 550 | 2 | 0 | 0 | 34,100 |
| 10/28/91 | 2,450 | 20 | 3,600 | 29 | 4,650 | 38 | 1,250 | 10 | 150 | 1 | 250 | 2 | 12,350 |
| 11/04/91 | 6,100 | 21 | 8,050 | 28 | 12,500 | 43 | 2,300 | 8 | 0 | 0 | 0 | 0 | 28,950 |
| 11/11/91 | 3,150 | 9 | 8,100 | 23 | 17,800 | 50 | 6,150 | 17 | 0 | 0 | 250 | 1 | 35,450 |
| 11/18/91 | 5,050 | 26 | 4,550 | 23 | 7,600 | 38 | 2,300 | 12 | 0 | 0 | 300 | 2 | 19,800 |
| 11/25/91 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 12/02/91 | 1,900 | 7 | 7,200 | 27 | 14,400 | 53 | 3,300 | 12 | 300 | 1 | 0 | 0 | 27,100 |
| 12/09/91 | 2,600 | 11 | 7,100 | 30 | 11,700 | 50 | 2,100 | 9 | 0 | 0 | 0 | 0 | 23,500 |
| 12/16/91 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 12/23/91 | 3,300 | 17 | 5,800 | 30 | 9,950 | 51 | 300 | 2 | 0 | 0 | 0 | 0 | 19,350 |
| 12/30/91 | 12,700 | 29 | 9,150 | 21 | 19,550 | 45 | 2,350 | 5 | 0 | 0 | 0 | 0 | 43,750 |
| 1/06/92 | 2,600 | 8 | 8,700 | 27 | 17,000 | 54 | 2,800 | 9 | 400 | 1 | 175 | 1 | 31,675 |
| 1/13/92 | 4,100 | 19 | 7,675 | 36 | 5,575 | 26 | 3,200 | 15 | 500 | 2 | 0 | 0 | 21,050 |
| 1/20/92 | 7,300 | 31 | 8,100 | 35 | 6,650 | 29 | 0 | 0 | 1,000 | 4 | 150 | 1 | 23,200 |
| 1/27/92 | 4,300 | 11 | 6,700 | 18 | 16,850 | 44 | 8,700 | 23 | 700 | 2 | 1,000 | 3 | 38,250 |
| 2/03/92 | 8,300 | 23 | 8,300 | 23 | 12,200 | 34 | 4,400 | 12 | 800 | 2 | 1,750 | 5 | 36,050 |
| 2/10/92 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 2/17/92 | 3,600 | 8 | 17,950 | 38 | 18,700 | 40 | 4,100 | 9 | 1,900 | 4 | 650 | 1 | 46,900 |
| TOTAL % | 17 | | 26 | | 41 | | 12 | | 1 | | 1 | | 100 |

Table 8. Results from the workout analysis showing actual yardage and percentages of energy systems trained. The weeks with no values are during breaks from school. The description of each energy system is given in the introduction. EN1 is AT1,2, mVO2 is mVO2 and AOV, AN1 is LP1, and AN2 is ALP2.

Bibliography

- Cook, Bill and Paul Brynteson. "Effect of a Season of Collegiate Swimming Competition and Training on Selected Responses." The Research Quarterly. Vol. 44, pp.63-70. 1973.
- Costill, David, M.G. Flynn, J.P. Kirwin, J.A. Houmard, J.B. Mitchell, K. Thomas, and S.H. Park. "Effects of Repeated Days of Intensified Training on Muscle Glycogen and Swimming Performance." Medicine and Science in Sports and Exercise. Vol. 20, pp.249-254. 1988.
- Counsilman, James. Competitive Swimming Manual. Bloomington, Indiana: Counsilman Company Inc. 1977.
- Darnell, James and Harvey Lodish. Molecular Cell Biology. New York: W.H. Freeman and Company. 1990.
- Donovan, Casey and George Brooks. "Endurance Training Affects Lactate Clearance, Not Lactate Production." American Journal of Physiology. Vol. 244, pp.83-92. 1983.
- Hickson, Robert, William Heusner, and Wayne Van Huss. "Skeletal Muscle Enzyme Alterations After Sprint and Endurance Training." Journal of Applied Physiology. Vol. 40, pp. 868-872. 1976.
- Jacobs, Ira, M. Esbjornsson, C. Sylven, I. Holm, and E. Jansson. "Sprint Training Effects on Muscle Myoglobin, Enzymes, Fiber Types, and Blood Lactate." Medicine and Science in Sports and Exercise. Vol. 19, pp. 368-374. 1987.
- Kindermann, W., G. Simon, and J. Keul. "The Significance of the Aerobic-anaerobic Transition for the Determination of Work Load Intensities During Endurance Training." European Journal of Physiology. Vol. 42, p.25-34. 1979.
- Kirwin, J.P., D. Costill, M.G. Flynn, J.B. Mitchell, W.J. Fink, P.D. Neuffer, J.A. Houmard. "Physiological Responses to Successive Days of Intense Training in Competitive Swimmers." Medicine and Science in Sports and Training. Vol. 20, pp.255-259. 1988.
- Maglischo, Ernest. Swimming Faster: A Comprehensive Guide to the Science of Swimming. California: Mayfield Publishing Company. 1982.

- Medbo, John, A. Mohn, I. Tabata, R. Bahr, O. Vaage, and O. Sejersted. "Anaerobic Capacity Determined by Maximal Accumulated O₂ Deficit." Journal of Applied Physiology. Vol. 64, pp.50-60. 1988.
- NCAA News. Policy and Procedure Manual: Department of Intercollegiate Athletics and Physical Education. Washington and Lee University. 1978.
- Nye, Steve. "Monitoring Workouts With Heart Rate." Swimming Technique. Vol. 24, pp. 25-29. 1987.
- Paxton, M.V.W. Endocrinology: Biological and Medical Perspectives. Iowa: Wm.C. Brown Publishers. p. 251. 1986.
- Sharkey, Brian. Physiology of Fitness. Illinois: Human Kinetics Books. 1990.
- Stainsby, W.N. "Biochemical and Physiological Bases for Lactate Production." Medicine and Science in Sports and Exercise. Vol. 18, pp. 341-343. 1986.
- Stryer, Lubert. Biochemistry. New York: W.H. Freeman and Company. 1988.
- Strauss, Richard, Editor. Sports Medicine. Philadelphia: W.B. Saunders Company. 1984.
- Troup, John, David Costill, and Ernest Maglischo. Level 3: The Physiology School. Florida: The American Swimming Coaches Association. 1990.