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Topic Report: Lactate Metabolism

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In light of the increasing interest in using “Blood Lactate” measurements as a means of monitoring the training of swimmers, it would be useful to have a basic understanding of the role of Lactic Acid in muscle metabolism, its production and use. A recently published text titled “EXERCISE PHYSIOLOGY—HUMAN BIOENERGETICS AND ITS APPLICATIONS” by George Brooks and Thomas Fahey includes a chapter on the subject of Lactic Acid which is both timely and useful. In this brief summary we will highlight some of the major points made by the authors and try to make some observations on the material presented.

The chapter begins by drawing our attention to some of the more popular misconceptions on the subject of Lactate Metabolism. The first misconception is that Lactic Acid is a “metabolic dead-end”, the implication being that once produced in the muscle, it needs to be removed via the blood to the liver in order to be reconverted to a “useful substrate”, namely Glycogen. The section titled “Metabolic fate of Lactic Acid after exercise” outlines a more current belief as to what occurs to Lactic Acid after it is manufactured. By tracing the pathways using radioactively labeled Lactate the diverse paths of the substance are described. The authors state,

“Lactate, in effect, sits close to metabolic crossroads. Because of the proximity of lactate to the TCA cycle, it’s entry into that cycle and subsequent oxidation constitutes a major pathway of metabolism.”

In addition to this pathway, we see that “... the effects of prolonged exercise leading to exhaustion—such as glycogen depletion, and hypoglycemia (low blood sugar)—may favor lesser oxidation and greater conversion of lactate to glucose (gluconeogenesis).” Lactate in this case acts as a precursor or forerunner, to the formation of glycogen. Finally, these radioactive tracings indicate that lactate can be incorporated into Amino Acids and eventually form segments of proteins. All these avenues indicate that lactic acid plays a significantly greater role in the metabolism of the muscle than was previously thought.

Indications are that as lactate levels begin to rise there is a change in the cellular environment of the working muscles. The cells become more acidic as the intensity of the work increases. As a consequence, the contractile properties of the muscle are affected and the outcome is a decrease in the capacity of the particular muscle group to continue at that particular work intensity. We have come to expect that training somehow delays the negative effects of a build up in lactic acid. The authors clearly show that the assumption that a trained subject produces less lactate for a given work intensity, when compared to a less trained subject, may not necessarily be true.

They state “Detailed studies utilizing both radioactive and nonradioactive tracers have shown that lactic acid is a dynamic metabolite both at rest and during exercise. At rest and during easy exercise, lactic acid is produced and removed at equal rates. This balance of production and removal is called turnover. Even though a metabolite such as lactic acid turns over very rapidly, its concentration in the blood may not change so long as the removal (from the blood) keeps pace with the production (entry into the blood). Tracer studies have clearly shown that for a given blood lactate level, the turnover of lactic acid during exercise is several times greater than at rest. Therefore, if the situation is that blood lactic acid levels remain at resting levels during exercise, then it is erroneous to conclude that there is no lactic acid production occurring.”

The discussion continues to point out that the rate of removal of lactic acid is “concentration dependent”. That is, that as the concentration of lactic acid increases this in turn will elicit a response that will force its removal at a faster rate. Therefore, when blood lactate levels fall off during exercise, we must infer that the means available to the muscle for removal of this substance exceeds the process by which it is produced. A rise in the blood lactate level would therefore indicate that the production rate exceeds the rate of removal.
Under the section titled “Effect of endurance training on lactate metabolism during exercise” Brooks and Fahey describe a study on the effects of endurance training on lactate turnover during rest and exercise. They state “. . . trained animals had lower lactate levels during both easy and hard exercises than did untrained animals. However, lactate turnover rates in trained animals during exercise were the same as in untrained animals. Unchanged blood lactate levels in trained animals concealed the fact that lactate production was the same in trained as in untrained animals. The difference due to training was the greater lactate clearance rate from the blood in trained animals.”

The implication then for our purposes is that if we see low values on a blood lactate test, with either a moderate or hard effort, we must not assume that less lactate is being produced but rather that long-term training effects have resulted in a more efficient means of lactate removal. This indeed seems to be the case when distance swimmers have been tested. The values for their consecutive “200 swims” for developing a lactate profile, were comparatively low, while still swimming reasonably fast times. This group, from what is now known, may very well be producing just as much lactic acid, but removing it at a faster rate.

The last portion of the chapter is devoted to a discussion of the “Anaerobic Threshold” and the significance of the “lactate inflection point”. The term “Anaerobic Threshold” as pointed out, is a “misnomer”. The level above which blood lactate begins to increase non-linearly, that is, show a sudden surge, is observed to be at about 60% of the VO2 max. However, this point of inflection for the sudden lactic acid increase in the blood as suggested by the authors “. . . gives no indication about anaerobic metabolism; rather it reflects a balance between lactate entry into and removal from the blood.”

In the context of terminology, the introduction of the term “Onset of Blood Lactate Accumulation” (OBLA) which has been introduced, is recognized as being a more accurate description of the rapid changes in blood lactate above certain work intensities.

As to the causes of the inflection point, the authors suggest that there are several reasons for “the muscle being deprived of oxygen” rather than a singular factor. Since lactate is constantly produced by the muscle, even at rest, “. . . any factor that affects entry and/or removal changes in the blood concentration.” Some of the factors suggested are, the increasing recruitment of fast-contracting muscle fibers, the release of hormones which have a vasoconstricting effect in the working muscles, and the redistribution of blood flow from lactate removing (gluconeogenic) tissues such as the liver and kidney to the lactate producing glycolytic tissues.

We recommend this chapter to readers interested in blood lactate measurements. The material contains new information on the subject and the graphs and diagrams are well chosen to illustrate the points made by the authors.

For this reviewer, the chapter did bring up some unanswered questions. As coaches, what then are we to deduce from the results of blood lactate measurements? It is apparent that fluctuations of lactate levels in the blood, do signify changes in the manner in which the body is adapting to workloads of varying intensities. However, since the process appears to be more complicated than was first thought, it would not be unreasonable to postpone detailed interpretations of blood lactate measurements on swimmers until a substantial volume of data is gathered. The best approach to obtaining useful information from blood lactate measurements on our swimmers would be to begin at the beginning of a season and take these samples at regular intervals throughout the entire training period. Ideally, coaches would be able to plan their weekly workouts based on the results of the lactate tests. Since the absolute values will vary among the individual swimmers, the only useful information would be the values from each person plotted over the course of a season or two. This is a long-term project and must be allowed to stand the test of time. We are told that the Soviet bloc countries have been accumulating this type of data since the early seventies. We have everything to gain from exercising patience and caution rather than expecting immediate results from a concept which is still relatively new to U.S. swimming.
Review: The Cardiovascular System

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Introduction

In the following pages you will find a review of the Cardiovascular system and how it is affected by exercise and physical training. The intent of this article is to offer to the reader a basic understanding of the physiology of the heart and blood vessels, and will not review the multitude of factors that can affect our circulatory system. Indeed, a concerted effort has been made to eliminate those aspects of physiology which might be considered of academic interest only. A more comprehensive review of the cardiovascular system in health and disease can be found in many excellent textbooks (2,12,13).

The Cardiovascular (CV) system is composed of a series of vessels through which blood flows, and a pump, the heart, which produces this flow. As shown in the diagram of figure 1 there are actually two circuits within the CV system, both originating and terminating in the heart. The Pulmonary circulation connects the right half of the heart to the lungs and back to the left half of the heart. This oxygenated blood returning from the lungs is then pumped out of the left heart via the second circuit, the systemic circulation. Here blood is carried out to all tissues of the body, including the heart itself. End-waste products (carbon dioxide and lactic acid) are then transported back through the veins of the circulatory system to the heart and lungs. Carbon dioxide is removed from the system by exhalation, and lactic acid is metabolized in the liver.

In both circuits, those vessels carrying blood away from the heart are called arteries and those returning blood back to the heart are the veins. A further distinction of arteries and veins as well as the function and anatomy of the heart are discussed in the following subdivisions.

The Heart

The heart is a muscular organ with characteristics and functional abilities that are very similar to the skeletal muscles surrounding our bones. The heart, or cardiac muscle, can develop strength as a result of training, as well as respond immediately to the demands of exercise (12). Anatomically the heart is composed of four chambers: The right and left atrium and ventricle. The salient anatomical feature of the heart may be reviewed by considering figure 2 in the forthcoming discussion.

As mentioned previously, blood is pumped out of the right heart via the pulmonary circulation. This blood leaves the right ventricle and returns, rich with oxygen, to the left atrium. Blood entering the left atrium from the lungs flows passively into the left ventricle through a valve. Valves permit blood to flow in one direction only, and in this case, from atrium to ventricle (AV valve). The period of time in which the ventricle receives blood is called diastole, and is considered the resting phase for the heart. The end of diastole is determined by the heart rate which will be discussed in a later section.

With each heart beat the ventricle contracts. This period of time is called systole, and is the working phase for the heart. As the ventricle contracts, the blood contained within is squeezed tightly together, resulting in

Figure 1. Schematic view of the cardiovascular system consisting of the heart and the pulmonary and systemic vascular circuits. The dark shading indicates the oxygen-rich arteriolar blood, whereas the deoxygenated venous blood is somewhat paler. In the pulmonary circuit, the situation is reversed, and oxygenated blood returns to the heart in the right and left pulmonary veins. (Taken from McArdle, Exercise Physiology, 1981).
a large increase in pressure. For a brief period of time the heart is then in a phase of "isometric" contraction where the blood volume contained within the ventricle remains constant and the heart muscle fibers increase their tension without a shortening in muscle length. When the pressure within the ventricle exceeds the pressure of the arteries, a second valve opens, the aortic valve, and blood is ejected out of the ventricle and into the systemic circulation (Figure 1). The ventricles does not empty completely, and the amount remaining after each ejection is called the end-systolic volume.

Following the ejection of blood into the systemic circulation, the pressure within the ventricle starts to recede. When it falls below a critical point, the aortic valve closes again, and the AV valve opens. This is called the period of early diastole.

![Figure 2. The heart.](image)

When the AV valve opens, blood enters the ventricle rapidly at first, and then declines as the atrial volume and pressure decrease. The importance of this rapid filling during the early phase of diastole is most apparent with high heart rates. If not for this rapid filling the increase in heart rate would cause a considerable fall in the quantity of blood entering the ventricle during the resting phase of the heart cycle. The quantity of blood pumped out into the circulatory system would consequently become seriously impaired.

The Heart and Exercise

Control of the heart rate

Resting Heart rate (HR) is determined by an individual's age, body position, level of cardiovascular fitness, and environment conditions (11). Heart rate is controlled by impulses which are generated from the sinoatrial node, or pacemaker cell, located in the right atrium. The activity of the pacemaker is spontaneous, but it is under the constant supervision of the autonomic nervous system (ANS) and circulatory hormones.

The ANS is an involuntary neural system, which means it is not under conscious control. Depending on which neural pathway is stimulated, the ANS can cause a marked increase in HR with exercise, stress, excitement, etc., or it can slow HR down to very low levels (8,11,12).

In addition to the ANS, blood hormones are also very important in HR control. A release of Adrenalin (Epinephrine) into the blood stream will cause an almost immediate increase in the HR. Adrenalin is also released into the blood stream in response to stress, excitement, exercise, etc. (11).

With training, the resting HR can decrease substantially, and in a healthy individual, can represent a measure of cardiovascular fitness. Resting HR is normally between 60-70 beats per minute (b/m), but following intense endurance training has been reported as low as 35 b/m, with a mean rate of between 40-50 b/m (2,12). During a submaximal exercise at the same workload, HR will decrease with training. An individual's maximal HR will either remain unchanged, or will decrease slightly.

There is a linear relationship between the amount of work the athlete is doing, and their achieved HR. From this relationship, it is possible to predict the effort put forth in doing the work based on the individual's HR. An individual's maximal HR and varying percentages of the maximal HR has been used to determine the intensity in which an athlete should be trained. Karvonen's formula (13), developed for Cardiac Rehabilitation, has been particularly useful in estimating the work capacity of an athlete, and a means in which to determine a training effect upon the heart. Karvonen's formula is used in the following way:

1. 220 - Age = maximal HR (HRmax)
2. HRmax - HR resting = HR range
3. HR range x % effort + HR resting = training HR

As an example in how to use this formula, we will use an 18 year old swimmer who wants to train at about 85% of his/her ability in a given interval set. The resting HR is 50:

1. 220 - 18 = 202 (HRmax)
2. 202 - 50 = 152
3. \((152 \times .85) + 50 = 179\)

This particular swimmer would need to maintain a HR of approximately 180 to know that he/she is training at 85% of their maximal ability.

After using this formula for a variety of different athletes, the following results will become obvious:

1. As an individual's fitness increases, the quality of work must also increase to maintain the training HR.
2. Training HR will not be markedly affected by a decrease in resting HR.
3. The age of the individual can affect the HRmax and what an 85% effort training HR should be.
As an example of this last point, we will use a 60 year old male, with a resting HR of 65, who wants to train at 85% effort:

1. $220 - 60 = 160$
2. $160 - 65 = 95$
3. $(95 \times .85) + 65 = 145$

A HR of 145 for this man would then represent the same or similar effort as that of the 18 year old who trained at 180 b/m. The examples given here are assuming a healthy individual. Medications and cardiovascular disease can vastly change the validity of this formula.

**Stroke Volume**

The amount of blood pumped out with each beat of the heart is called the *stroke volume* (SV) and it is normally reported in units of milliliters of blood per heart beat (ml/b). In a healthy adult at rest, the SV will be between 70-100 ml, and for highly trained individuals, a maximal SV can reach as high as 200ml (2).

Resting SV is dependent upon hydrostatic pressures, and will therefore be greater when lying down. When sitting or standing, without the benefit of motion, SV will decrease due to a pooling of blood in the lower extremities. During maximal exercise, however, the same maximal SV can be achieved regardless of the body position, since the pumping action of the muscles against the veins help with the return of blood back to the heart (*venous return*).

Stroke volume increases linearly with increasing HR’s up until approximately 50% of a person’s working capacity (approximated at 120 b/m). At this point, there is a substantial enough decrease in the diastolic filling time to inhibit any further increase in SV (2,8).

Following a training program, resting SV will increase significantly, and SV during maximal exercise may increase up to twice its initial value, depending upon the preconditioning fitness of the individual. The increase in SV with training is due to a greater filling of the ventricle during diastole (i.e., more blood), and a greater force in the contraction of the ventricles during systole (2). The end result would be a larger amount of blood pumped out into the periphery and a decrease in the amount of blood left in the heart.

**Cardiac Output**

When SV is multiplied by the number of beats/min, we have the Cardiac Output (CO):

$$CO \text{ (liters/min)} = HR \text{ (b/m)} \times SV \text{ (ml/beat)}$$

Cardiac Output is dependent upon a person’s body size, fitness, sex, and age. Males will have a larger resting and maximal CO than females, and CO will decrease with age for both.

A resting CO in a healthy individual will be between 4-6 liters per minute(L/m). Resting CO is unchanged by training, but is maintained by an increase in SV and a decreased HR, as was discussed earlier. Since changes in SV and HR are opposite in direction and equal in magnitude, CO remains fairly constant.

For example, we will compare two people of the same age, size, and sex, but where one is highly trained, the other is sedentary. Both have CO’s of 5 L/m (5000ml), but the trained individual has a resting HR of 50, and the untrained has a resting HR of 70:

$5000\text{ml/min} = 70 \text{ b/m} \times SV$  
$SV = 71 \text{ ml/beat for the untrained}$

$5000\text{ml/min} = 50 \text{ b/m} \times SV$  
$SV = 100 \text{ ml/beat for the trained}$

A increase in the SV to maintain a given resting CO indicates a more efficient heart, and allows the heart to stay longer in its resting phase.

During periods of intense activity, the CO may reach values up to 20-25 L/min in the untrained, and as high as 40-42 L/min in the highly trained endurance athlete (2,7,8,12). This difference represents a 68% greater blood supply to the tissue of the body of the trained individual over that received by the untrained. You will see in subsequent sections, however, that this represents only one of many ways in which an individual benefits from regular physical activity.

Regardless of training, CO will increase linearly with workload up to one’s maximal capacity. The increase in CO is supported equally by both SV and HR to approximately 120 b/min. Further increases in CO are then due to an increase in HR alone.

How rapidly CO falls upon cessation of exercise is indicative of the state of conditioning of the individual. Upon cessation of exercise, CO falls quickly, in an exponential fashion, towards resting values, often approaching pre-exercising levels within minutes (3,4). A physically fit person should see a drop in HR of approximately 25 beats within the first minute, and should be under 100 beats by the end of the third minute of recovery. Stroke volume too will decrease quickly back towards the normal resting value.

Research has shown that with training the size of the heart may increase with intense endurance training. This *cardiac hypertrophy* consists of an increase in size of the left ventricle cavity without an increase in the thickness of its wall. For non-endurance athletes, however, just the opposite occurs. Cardiac hypertrophy consists of an increase in ventricular wall thickness, while the cavity size remains the same. Cardiac hypertrophy does not always occur with training, and the mechanism that causes hypertrophy in some instances and not in others is unknown (8,13).
Peripheral Vascular System

Despite the preeminent position of the heart as the dominant feature in the CV system, the vascular system plays a critical role in the circulation of the blood. These vessels are not merely inert pipes, but have a highly characteristic and trainable structure and function.

Blood flow depends on the pressure (P) driving blood through the vessels, and the resistance (R) acting to oppose that flow (13). The relationship between these three factors can be seen by the following equation:

\[ \text{Blood Flow} = \frac{P}{R} \]

Hence, if the pressure within the vessel decreases, or the resistance increases, the blood flow will fall. Similarly, an increase in P at the same R would mean an increase in the blood flow. The pressure taken at the brachial artery of the arm will typically average about 120/80 mmHg, (i.e., systolic pressure/diastolic pressure). Exercise will increase the systolic pressure, whereas the diastolic pressure should remain the same or decrease slightly.

Figure 1 shows the interrelationships of both the systemic and pulmonary circulations. The diagram also depicts the fact that both systems are a closed circuit, with their respective vessels aligned in parallel to each other. The rest of this discussion will focus exclusively upon the systematic vascular system.

Arteries

Arteries are smooth muscle vessels with thick elastic walls. Smooth muscle is not under voluntary or conscious control of the brain but, instead, responds to nerve impulses of the autonomic nervous system. Arteries have a large diameter and cause little resistance to blood flow. They serve as low-resistance pipes, conducting blood to the smaller arteries (3,6,13).

The large arteries branch into smaller arteries or arterioles. The arterioles are responsible for most of the blood flow adjustments seen during exercise. Their strong muscular walls can decrease a vessel's diameter to 1/4th of its former size when stimulated by the ANS. Substantial blood flow changes can result from relatively small adjustments in vessel diameter. For this reason, constriction and dilation of the arteries can control to a very large extent the quantity of blood channeled to the muscles and organs (3,6,13).

The arterioles branch into the capillaries, their thin walls of which act as a semipermeable membrane for interchange of various substances between the blood and tissue. Through muscle biopsy techniques, it has been estimated that there are approximately 500 capillaries per square centimeter of muscle tissue, depending upon muscle fiber type and state of cardiovascular fitness (1,2,10). The greater the capillary density within a muscle, the more available is oxygen for periods of greater demand.

Veins

Metabolic waste products such as heat, carbon dioxide, and lactic acid are transported out of the tissue cells by venules. Venules converge into veins which, in turn, converge into the vena cava which enter the right side of the heart (figure 1). Vessels on the venous system of the circulation are of equal size or diameter as the arteries, but have much thinner walls. They are called capacitance vessels since they are able to store a great deal of blood.

The importance of cardiac output to the needs of the body has already been stressed in detail. However, the heart can only pump out what it receives. For this reason, CO is ultimately dependent upon the amount of blood returning to the heart from the systemic system, that is, it is dependent upon its Venous Return (VR).

Venous return can be enhanced by the muscle pump, the respiratory pump, and constriction of the veins. All three of these mechanisms are important to the quantity of blood returning to the heart, and so will be discussed in more detail.

Muscle Pump: As mentioned briefly in the section on stroke volume, muscular contraction aids in returning blood to the heart. When the muscle contracts, their veins are compressed, and the blood within is forced toward the heart (figure 3). Similar to heart action, reverse blood flow direction is prevented by valves that are located within the veins. The muscle pump is important when standing, walking, running and other similar activities during which the muscles alternatively contract and relax (12,13).

Respiratory Pump: With this pump, the veins of the chest and abdomen are emptied toward the heart during inspiration and filled during expiration. Thus VR is enhanced merely by breathing. This pump plays an increasingly greater role during exercise when frequency and depth of breathing increase (2,13). It is also for this reason that breath-holding during strenuous movements such as weight lifting can be potentially dangerous, since VR and, therefore CO, is impaired.

Venocostriiction. The large veins are used as storage sites of blood during periods of inactivity. When blood flow demands increase, constriction of the veins will reduce the volume capacity of the venous system, and, as a result, blood is forced out toward the heart. Venous constriction is initiated and controlled by the autonomic nervous system (2,11,13).

Distribution of Blood Flow During Exercise

In general, the increase in blood flow and oxygen delivery to working muscles, regardless of training, is
caused by an increase and redistribution of cardiac output. Increased neural activity is responsible for the redistribution of blood flow away from the less active tissues (i.e., GI tract, liver, spleen, kidneys, and resting skeletal muscles) and to the working muscles. The adjustments are proportional to the severity of the workload (5,14).

With large increases in blood flow to the active muscles, reductions must occur in other areas to insure a constant arterial blood pressure throughout the circulatory system, as well as to allow the greater fraction of CO to reach the working muscle. Well-perfused regions which normally extract only small amounts of oxygen are the liver, kidneys, and resting muscles. The blood flow to these regions can be reduced to approximately 20-30% of their resting value during maximal exercise (6,14,15) (figure 4). The quantity of oxygen they will receive will be the same, however, since more is extracted from the blood that is available.

That muscle blood flow decreases at submaximal workloads following training has been well substantiated in the literature. A decrease in blood flow during submaximal exercise implies that a greater amount of oxygen is extracted from the capillaries. Experimental findings support the conclusion that trained muscles extract more oxygen from the blood at a lower blood flow (2,6). These altered metabolic and circulatory responses, however, occur only within the trained muscle and are not transferable to other untrained muscle groups. This points out once again the need for specificity in training.

Training will not alter the blood distribution during maximal exercise (6).

In summary, the distribution of blood flow during exercise in working and inactive tissues can be generalized as follows: Regardless of training, exercise will cause an increase and redistribution of CO away from the inactive tissues to the active muscles. A decrease in blood flow to the inactive tissue is compensated by a simultaneous increase in the amount of oxygen extracted. The muscle blood flow increases linearly with increasing intensity up to maximal effort, and may represent up to 85% of the CO.

![Figure 4. Distribution of blood flow to the various organs and tissues of the body at rest and during strenuous exercise. The numbers show blood flow in milliliters per minute. (Taken from Vanders, Human Physiology, 1975).](image)

**Heart and Local Adaptations to Training**

The blood flow to skeletal muscles depends upon the pressure and resistance in the arterioles. During muscular exercise the arterioles dilate (resistance decreases) in active muscles, and constrict (resistance goes up) in the inactive tissue beds. The cooperative action of this dilatation and constriction helps maintain the blood pressure (6,15). Dilatation of the arteries supplying the working muscles is metabolically controlled by the end waste products of heat, carbon dioxide and lactic acid, i.e., the presence of these waste products override any neural constriction by the ANS.

Studies have shown that training will increase the amount of blood supplying the working muscles through a decrease in arterial resistance. For both heart and muscles to adapt, however, training must include large muscle groups. Many studies have indicated that when either arms only or one-leg training is involved, the cardiovascular system is not sufficiently challenged to induce an increase in the cardiac output (2,3,4,5). In addition, an improvement in endurances as a result of this training would be apparent in the trained limb only.

Some studies have suggested that an increase in the amount of blood supplying the trained muscles could
be due to an increase in the capillary density (1,9,13). An increase in the number of capillaries would decrease resistance, and allow a larger muscle blood flow to be accommodated with little change in capillary transit time. An increase in the number of capillaries can also facilitate the amount of oxygen extracted from the capillary by decreasing the diffusion distance from the vessel to the muscle cells. In addition, a decrease in resistance would also result in a subsequent rise in venous return and therefore the amount of blood entering the heart. Many of these studies that have shown an increase in capillary density with training have been done on laboratory animals. The issue of whether or not training can cause an increase in capillaries within the human muscle remains unclear.

Summary

The cardiovascular system is composed of the heart and blood vessels. Their main purpose is to supply oxygen from the lungs to all tissues of the body.

With exercise, stroke volume, heart rate, and consequently, cardiac output will all rise linearly with the intensity of the activity. An increase in the quantity of blood pumped out of the heart (cardiac output) results in an increase in the amount of oxygen delivered to the exercising muscles. The increase in cardiac output is accompanied by a redistribution of blood from less active tissues, such as the digestive organs and kidneys, to those in greater need, the heart and skeletal muscles, and eventually the skin for heat dissipation. These mechanisms combine to allow an increase in blood flow of up to 20 times over their resting values.

Endurance or “aerobic” training will increase athletic performance in the following ways: (a) increase the strength of the heart to eject more blood out to the body; (b) increase the quantity of blood delivered to the working muscles; and (c) increase the amount of oxygen extracted from the capillaries and utilized by the muscle cells. The adaptations that occur within the cardiovascular system to elicit these changes were discussed in the above review.

References

Review: Energy Systems and Training Considerations

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Introduction

The physiological requirements of swim training are very demanding. Because of this, only a few swimmers can easily achieve outstanding success based on natural talent alone. Success, however, is made difficult by a number of complex physiological interactions and is a result of a finely tuned system with functional diversity. This diversity includes control by the nervous system, fueling and cooling by the circulatory system as well as regulation of many of these properties at rest and activity by the hormonal system (1). When considering all of the systems that must be coordinated to produce peak performance and the wide diversity of activity patterns required in swimming, it is unrealistic to assign to any one system the role of predominant importance for success.

Although we do know factors important to swimming performance include muscle metabolism, strength/power, aerobic and anaerobic capacity (1, 13), each component encompasses disciplines of study with unknowns as to the relative contribution to superior performance. Furthermore, it is possible that major fluctuations occur within the same individual with regard to some of these components (14). These fluctuations, therefore, may account for the variations in performance that are seen in the elite swimmer.

Due to the complex interrelationship of these factors to performance each must be examined separately. In this and future articles we will discuss some of the specific factors important to performance and review work previously found in the literature. Specifically, in this review we will deal with an over view of the energy systems in muscle and discuss how they may be trained. Future reviews will deal with other subjects in a more specific way.

Energy Systems

The way in which energy is supplied during swimming depends on the time of the event and is comprised of two major systems. The relative importance of 1) Aerobic (endurance) and 2) Anaerobic (sprint) metabolism for the total energy yield is fairly well understood (11, 12, 13, 14). Figure one illustrates differences in utilization of the two systems. It should be clear that in a 50 second maximal exercise bout (100 meter freestyle) that the anaerobic processes may account for 80% of the total energy yield. During work periods exceeding 4 minutes, the aerobic processes account for 80% of the energy yield (13, 14). Obviously, during different events energy will be supplied from different systems. This has important implications for training and so deserves closer examination.

![Figure One. Percentage contributions to total energy requirement by aerobic and anaerobic metabolism (In "physiology of swimming man" 1. Holmer, Exercise & Sport Science Reviews Vol7 1979)](image)

Muscle Metabolism

The immediate form of muscular energy is Adenine Triphosphate (ATP). To maintain ATP levels close to resting levels during intense work involves a number of carefully coordinated, quickly responding reactions (9, 16). ATP is supplied in three ways. Two of these function for short periods without oxygen (anaerobically), 1) alactacid—through stored phosphagens, ATP, creatine phosphate (CP), 2) lactacid—through the breakdown of...
fuels, primarily carbohydrates. The third system through which ATP is generated is in the presence of oxygen, or aerobically (16).

The fast-twitch muscle fibers have the greatest anaerobic component, while the slow-twitch fibers are highly aerobic having many mitochondria (1, 2, 5, 7). As ATP is utilized, anaerobic or aerobic processes must function in order for continued fuel mobilization and ATP synthesis. This then allows the muscles to continue working (15, 17). Since the high energy phosphagen stores are rapidly utilized early in exercise, some other fuel source must exist. Intramuscular energy sources are composed of phosphagens, glycogen (carbohydrate) and triglycerides (fats) and when broken down, all of these contribute to the energy requirements (i.e. supply of ATP) of the working musculature during the course of the exercise bout (11, 16, 17). Table one summarizes the characteristics of each energy system and Figure two illustrates biochemical scheme of fuel utilization.

Table 1: Summary of Energy System Characteristics

<table>
<thead>
<tr>
<th>ATP-PC System</th>
<th>Lactic Acid System</th>
<th>Oxygen System</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaerobic (Lactacid)</td>
<td>Anaerobic (Lactacid)</td>
<td>Aerobic</td>
</tr>
<tr>
<td>Utilized in Bouts 30 Sec. (Sprints)</td>
<td>Utilized in Bouts 30 Sec. to 1 Min. (Spring-Mid. Distance)</td>
<td>Utilized in Bouts 3 Min. (Distance)</td>
</tr>
<tr>
<td>Limited ATP Production</td>
<td>Limited ATP Production</td>
<td>Unlimited ATP Production</td>
</tr>
<tr>
<td>Limited ATP Stores</td>
<td>Lactic-Acid By-Product Causes Muscular Fatigue</td>
<td>No Fatigue By-Products</td>
</tr>
</tbody>
</table>

Transformation of Bound Energy to Mechanical Energy

Figure Two. Biochemical pathway, describes the transformation of bound energy to mechanical energy (ATP).

The relative contribution of these energy sources depends on 1) intensity of the exercise, 2) duration of work, 3) amount of fuel available, 4) continuous or intermittent work, 5) size of working muscles, 6) duration of work and rest periods. Depending on these factors one of the major energy systems will be used (1).

**Anaerobic System.** In the presence of oxygen (work of 3 min), the complete breakdown of carbohydrate stores (glycogen) yields enough energy for the production of 36 ATP. Like the anaerobic processes, this takes place in muscle but is confined in the subcellular organelles (mitochondria (1, 8, 16). Since enough ATP can be produced during aerobic metabolism, no fatigue by-products are formed. The trained individual has more mitochondria so more ATP can be produced by a larger muscle mass more efficiently (17). Furthermore, these individuals have higher levels of enzymes which control aerobic metabolism (2, 12).

In swimming, perhaps the most important source of energy for muscular work is glycogen (13, 14) since at workloads greater than 80% of maximal Oxygen Consumption of VO2max (most swimming events) carbohydrates become the exclusive fuel for working muscles. The rate at which muscle carbohydrate stores are used increases with an increase in workload (2, 8, 11). Studies have shown that during submaximal exercise slow-twitch fibers lose their glycogen first and fast-twitch fibers were recruited when slow-twitch fibers were depleted (5, 7, 17). This research determined that work was related to muscle glycogen stores.

**Anaerobic system.** Previous studies have shown that for maximal swimming at different distances, there is an exponential increase in the total energy required (11). Estimates have indicated a major reliance on anaerobic processes as swimming speed is increased. This suggests an importance of phosphagens and carbohydrates as fuel sources.

The primary source of fuel in exercise under 30 seconds are the high energy phosphagens stored in muscle cells, ATP, CP. When the phosphate group is removed a large amount of energy is released. Total muscle stores of ATP and CP are small and are reduced rapidly during the first 30 seconds of swimming (13, 15). However, the usefulness of this system lies in the fact that ATP and CP stores are rapidly replenished as each CP broken down, one ATP is resynthesized (1, 18). This system has been referred to as the alactacid component of the anaerobic system.

The other component of the anaerobic system has been called lactacid and is referred to as anaerobic glycolysis. Glycolysis refers to the breakdown of carbohydrates from which ATP is made. When the carbohydrates are only partially broken down, one of the end products is lactic acid (3, 4, 15). The formation of lactic acid is associated with high intensity work between 30 seconds and 4 minutes (1, 13, 19). The increase in lactate is associated with a proportionate increase in the production of hydrogen ions (2, 4, 5, 18). This results in temporary muscle fatigue as the increased hydrogen ion concentration has been shown to limit the force-generating capacity of the muscle and decrease the rate of ATP production (18, 19).

The production of lactate can indirectly be considered
a limiting factor of performance. Training can reduce the production of lactic acid by making the rate of glycolysis more efficient (8, 11, 12, 18, 17). This increased efficiency is due to adaptations in the metabolic and enzymatic reactions.

Lactate production is influenced by the glycolytic enzyme, lactate dehydrogenase (LDH). Several forms of LDH exist (1, 16), and each differs in the rate at which it affects lactate production. An H form predominates in slow-twitch fibers resulting in a slower rate of lactate production. Another, M form of LDH, predominates in fast-twitch fibers and results in lactate production at high rates (1). Various studies have shown that lactate levels in fast-twitch fibers were higher following intense exercise (11), while slow-twitch fibers tend to have a greater removal of lactate following exercise (1, 11). This suggests that the fast-twitch fibers produce lactate, while the slow-twitch fibers remove lactate from the blood as well as from the fast-twitch fibers during exercise. It would therefore appear that lactate levels are influenced by fiber type as well as the type of activity.

Energy Transition
The capacity of each of the above systems to supply the necessary ATP for a given event is determined by the specific type of activity performed (5, 3, 11, 12, 17). For example, during sprint swimming (i.e., 50 meters) most of the ATP is supplied by the readily available phosphagen system (anaerobic-lactacid). On the other hand, during distance events (500 meters) requiring lower intensity activity, most of the energy is derived from the aerobic system. In contrast, middle-range events (100 to 200 meters) depend on the lactacid system for ATP. Within these ranges of activity exist events that require a combination of both anaerobic and aerobic metabolism (200 to 500 meters). During these activities both anaerobic systems supply the majority of ATP during the sprint at both the start and finish of the race while the aerobic system will supply most of the energy during the middle part of the race. It should be clear that the three stages of energy metabolism are at work in each competitive event (13, 14).

Training Specificity
Though it may seem fairly easy to pick the energy system to train for the sprint or distance swimmer, the decision is complex when designing workouts for middle distance swimmers. Guidelines for estimating the emphasis that should be placed on improving the appropriate energy system for various swimming events is shown in Table two.

Houston (14) first illustrated links between metabolic parameters of each energy systems with the desired adaptation and type of training condition. Table three presents this information. The glycolytic rate refers to the rate of ATP generation through the sequence of reactions first shown in Figure two. The glycolytic (anaerobic) capacity refers to the ability of these reactions to continue to work even while lactate levels and hydrogen ions accumulate. As mentioned earlier, if the accumulation of these substances can be tolerated, performance can continue for longer periods (12, 14, 17). It suggests that limitations imposed by key biochemical reactions preclude improvements in the rate and capacity of anaerobic processes in muscle (14). Therefore, short term sprint training and longer duration high intensity anaerobic training (i.e. 200 to 400 meters) can produce gains in aerobic events (12, 15, 17).

### Table 2: Predominant Energy Systems For Competitive Events

<table>
<thead>
<tr>
<th>Event Distance (All Strokes)</th>
<th>% Emphasis Per System</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ATP/CP (Lactacid)</td>
</tr>
<tr>
<td></td>
<td>LA (Lactacid)</td>
</tr>
<tr>
<td></td>
<td>O$_2$ (Aerobic)</td>
</tr>
<tr>
<td>50 yds/meters</td>
<td>98</td>
</tr>
<tr>
<td>100 yds/meters</td>
<td>80</td>
</tr>
<tr>
<td>200 yds/meters</td>
<td>30</td>
</tr>
<tr>
<td>500 yds/meters</td>
<td>20</td>
</tr>
<tr>
<td>1650 yds/1500 meters</td>
<td>10</td>
</tr>
</tbody>
</table>

The rate of anaerobic metabolism should be maximally stressed when swimming is longer than 5 to 10 seconds. This type of speed swimming lasting for up to 30 seconds will severely tax the ATP synthesis reactions, resulting in increased ATP-CP stores in the muscle (15). Further, the levels of enzymes controlling these anaerobic reactions are increased which may lead to an improved rate of ATP resynthesis during the exercise bout (1, 3, 15).

In order to stress the anaerobic capacity, near maximal efforts should be swum for a period long enough to allow accumulation of anaerobic end-products (i.e., lactate, hydrogen ions) in the muscle. Repeated bouts with short rest intervals could be performed; this would result in continued increase of the waste products. This type of training is very demanding and should gradually be introduced reaching a peak by mid-season. The result of this type of training is excess lactate levels reducing the anaerobic capacity and rate, causing a painful sensation in the arms (14). Continued use of this type of training leads to an eventual reduction in the rate of acid accumulation either due to improved aerobic or by conversion of the lactate precursor pyruvate to alanine instead of lactate (3). More recently, Donovan (9) suggested that with training, lactate production may not be changed but rather, its rate of removal may be improved. This may be the result of 1) improved blood flow removing waste products (1), 2) by converting lactate into a useable substrate (glucagon) 5, 7), 3) an increased enzymatic activity, or 4) improved buffering capacity (6, 18).

### Overreaching vs Overtraining

In this discussion we have emphasized the importance of high intensity swimming. Working hard is of course important if the desired physiological adaptations are to be met. Excessive high intensity training, however,
Table 3: Energy Systems & Training Summarizes

<table>
<thead>
<tr>
<th>Metabolic parameter</th>
<th>Desired physiological response</th>
<th>Training conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxidative metabolism (aerobic)</td>
<td>To increase oxygen delivery to muscle; to improve muscle capillarization; to increase the number and size of muscle mitochondria and increase oxidative enzyme activities</td>
<td>Continuous and intermittent swimming at submaximal intensity; longer duration swimming intervals with short rest periods</td>
</tr>
<tr>
<td>Glycolytic rate (primarily Anaerobic) ATP, synthesis</td>
<td>To increase the flux of substrates through the glycolytic reactions and thus the rate of lactic ATP resynthesis</td>
<td>Short duration (10-25 sec) maximal speed swimming intervals with or without added resistance, high speed, high repetition (20-30 repetitions) resistance training</td>
</tr>
<tr>
<td>Glycolytic capacity (primarily Lactacid system)</td>
<td>To maintain optimal glycolytic flux despite rising end product accumulation and falling muscle pH; to enhance muscle buffering capacity</td>
<td>Repeated intervals of 1-3 min duration at fast pace, with less than full recovery rest pauses</td>
</tr>
</tbody>
</table>

(Adapted from Houston, Swimming Medicine IV; 1978. Univ. Park Press)

may result in diminished performance levels. This “syndrome” has been referred to as overtraining and has been defined as the incomplete recovery from a work bout that causes a decline in work capacity (1). The onset of this condition may be attributed to the fact that an adaptation threshold is exceeded which results in a decline of the physiological systems. It seems that there is an optimum amount of training intensity that will lead to the necessary physiological adaptations while just above this level, adaptations will not result. Once the swimmer is in this state, it is hard to bring them out of it and becomes even harder the further away from that optimal threshold they get.

Characteristics of the overtrained swimmer have been described and can be useful in determining if the swimmer suffers from this syndrome. Some signs of overtraining include an increase in the resting heart rate and recovery heart rate following a particular event. Weight loss, insomnia and depression are seen. Following these occurrences, injuries can easily occur. All of these lead to a low psychological state where the swimmer feels run down, is depressed and loses motivation. Additional symptoms may also include high enzyme levels, elevated CPK, high blood lactate levels and chronic muscle soreness.

Summary

The role of science in swimming has grown over the years as more and more swimmers search for a competitive edge based on something more than on tradition or guesswork. The information provided by scientific research helps us to understand what training modalities might be effective and which are not. It is for this reason that an understanding of some of the more important factors effecting swimming performances be reviewed. This has been the purpose of this article and will remain the focus of future review articles.

References

Comparison Between Blood Lactate and Heart Rate Profiles During a Season of Competitive Swim Training

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Abstract

This study was undertaken to determine if blood lactate/velocity and heart rate/velocity profiles would sense the changes in endurance capacity associated with a season for training in competitive swimmers. Since heart rate and exercise intensity are known to be closely related, it was hypothesized that a heart rate/velocity profile could provide the same information concerning the success of training programs as the lactate/velocity profiles proposed by Mader in 1976. Twelve male collegiate swimmers were asked to perform two 200 yard freestyle swims at three times during a season for training; once at the beginning of the season T1, after 2 months of training (T2) and after 4 months of training (T3). At each testing session, the swimmers would perform a 200 freestyle at approximately 90% effort followed by 20 minutes of rest and another 200 freestyle at 100% effort. Blood samples and heart rates were taken after each swim and values for each were plotted opposite mean velocity for each swim. The results of the study showed that both profiles showed a significant rightward shift during the first 2 months of training and a small further shift during the final 2 months of training. The degree of shift was similar between the two profiles and both were noted to increase in slope by approximately the same magnitude. Performance times predicted from the lactate profiles were, judging from group means, not significantly different from actual competition performances. The correlation between the actual and predicted performance was, however, low (r = 0.61). It is concluded that both monitoring techniques are sensitive to adaptations made to training and can thus provide useful information to the coach and athlete concerning the relative effectiveness of a training program. In light of the possible expense and difficulty of blood lactate measurements, it was concluded that the heart rate profile can provide a useful and non-invasive alternative to blood lactate testing.

It is well known that blood lactate concentration is proportional to exercise intensity. The increase in blood lactate due to incremental increases in workload is characterized by a breakpoint from resting levels at an intensity between 50-80% $V_{O2max}$. This breakpoint is followed by an exponential increase in blood lactate concentration until exhaustion is reached (2). This breakpoint has been studied by several investigators who have found that the endurance trained individual has a lower blood lactate concentration than the untrained person both at a given workload and at a given percentage of $V_{O2max}$ (10,13).

Based on this information, Mader et al. have proposed a technique which can be used to characterize the training status of track and swimming athletes (12). The model consists of two time trials over a known distance followed by blood lactate (BL) measurement. Performance time is then plotted against BL and a straight line is drawn through the resulting data points.

It is also well known that heart rate increases in proportion to exercise intensity (1). The heart rate response of the endurance trained individual, like the lactate response, is lower at both a given workload and at a given percentage of maximum work capacity than the untrained person. In addition, the recovery time of heart rate is faster in the endurance trained person both after submaximal and after maximal exercise intensities (1).

Because of the similarities in the response of blood lactate and post-exercise heart rate, the present authors felt that a heart rate/velocity profile could be used in much the same way as the lactate/velocity profiles suggested by Mader for the purpose of monitoring the training state of swimmers. Thus, the purpose of this study was to compare the lactate/velocity profile with a heart
rate/velocity over the course of a collegiate swimming season. The intent was to characterize the training status of 12 male swimmers before training (T1), following 2 months of training (T2), and following 4 months of training (T3) using both techniques and to determine the extent to which the models parallel each other. In addition, performance times in 200 yard freestyle events were predicted from the lactate profiles (12) and compared to performance in actual competitive events.

**Methods**

Twelve male competitive swimmers from the university swimming team volunteered as subjects for this investigation. Each subject was fully informed of all the risks and benefits of the study before giving his written consent to participate. All of the subjects were freestyle specialists.

Lactate/velocity profiles were constructed as described by Mader (12). Two 200 yard freestyle swims were performed at 90% effort on the first swim and 100% effort on the second. Twenty minutes of rest were allowed between the two trials to allow lactate levels to return to near resting levels between the trials. In no case did any of the pre-exercise blood lactate levels exceed 2 mM. Following each of the swims, 0.02 ml of arterialized blood was drawn from a heated fingertip, protoplastized in cold 2N perchloric acid and analyzed for lactate concentration (11). Blood samples were taken at 1.3,5.7 and 9 minutes post-exercise. Performance times were converted to mean velocity (meters/sec) and plotted on x,y coordinate graphs with lactate concentration on the ordinate and velocity on the abscissa. A straight line was then drawn through the two data points and used to represent the training state of the subject. The equations presented by Mader were then used to predict performance capacity in a 200 yard freestyle competitive event.

The heart rate/velocity profiles were constructed by counting heart beats after each of the same swims used for the lactate profiles. Heart beats were counted by independent observers who palpated the radial artery of the subjects. Heart beats were counted for 1.5 sec at three separate times post-exercise: 15 sec post, 45 sec post and at 90 sec post. The sum of the three counts was subsequently plotted against velocity in the same manner as the lactate profiles.

It was decided to use the sum of these counts since the sum would be influenced by both the intensity of the exercise bout and the recovery rate following the swims. Both of these parameters are known to improve with training and are proportional to the physiological intensity of the exercise.

In order to characterize the changes in these profiles with training, standard values were established and the velocity predicted from these standard values was read from the graphs. Standard values used for the lactate profiles were 6mM and 12 mM and on the heart rate profiles were 88 and 105 beats. These values were used because they represent the group mean on the submaximal and maximal swim, respectively (Fig. 1).

In addition to these standardized values, predicted lactate and heart rate responses to a standard velocity of 1.50 m/sec were used. Group means (+ SE) were determined for each of the testing periods and differences were analyzed for significance with a two-way ANOVA followed by a Newman-Keul multiple range test. The level of probability considered sufficient to reject the null hypothesis was set at p<0.05.

**Table 1.** Mean ± SE velocities, heart rate sums and blood lactate concentration at the three testing dates during a season of swim training.

<table>
<thead>
<tr>
<th></th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_1$ (m/sec)</td>
<td>1.37 ± 0.02*</td>
<td>1.47 ± 0.01</td>
<td>1.48 ± 0.01</td>
</tr>
<tr>
<td>$V_2$ (m/sec)</td>
<td>1.53 ± 0.02*</td>
<td>1.58 ± 0.02</td>
<td>1.58 ± 0.02</td>
</tr>
<tr>
<td>HR1</td>
<td>88.58 ± 3.01</td>
<td>89.00 ± 2.30</td>
<td>86.92 ± 3.45</td>
</tr>
<tr>
<td>HR2</td>
<td>107.00 ± 2.62</td>
<td>104.83 ± 3.06</td>
<td>105.75 ± 3.23</td>
</tr>
<tr>
<td>LAC1 (mM)</td>
<td>5.73 ± 0.53</td>
<td>5.57 ± 0.59</td>
<td>5.22 ± 0.51</td>
</tr>
<tr>
<td>LAC2 (mM)</td>
<td>11.53 ± 0.48</td>
<td>11.76 ± 0.87</td>
<td>11.14 ± 0.99</td>
</tr>
</tbody>
</table>

*p < 0.05

$V_1$ velocity of submaximum effort
$V_2$ velocity of maximum effort
HR1 heart rate sum of submaximum effort
HR2 heart rate sum of maximum effort
LAC1 blood lactate concentration after submaximum effort
LAC2 blood lactate concentration after maximum effort

**Results**

The velocities of the submaximal swims were 89.90, 93.18 and 93.51% of the velocities on the maximal swims at T1, T2 and T3, respectively. The actual mean (+ SE) velocities of each of these swims are presented in Table 1. These data indicate that the velocities for the two 200 swims were significantly lower at T1 than at the other two testing dates. In spite of the increased actual speed of the submaximal efforts during the training period, the sum of heart beats and the blood lactate were not significantly different at the three testing dates on either the submaximal or the maximal efforts. These data are also presented in Table 2. This finding indicates that the subjects swam at the same relative physiological intensity at each of the testing dates but the velocity required to reach this intensity increased.

The predicted response to a standard velocity of 1.50 m/sec showed similar changes with training between the two profiles (Fig. 2). This figure shows that the estimated lactate concentration at 1.50 m/sec decreased from 10.70 ± 0.57 mM at T1 to 7.40 ± 0.94 mM at T2 and 6.20 ± 1.07 mM at T3. The same pattern was shown by the heart rate sum at 1.50 m/sec, decreasing from 104.17 ± 2.01 beats at T1 to 94.25 ± 2.73 beats at T2 and 91.50 ± 3.33 beats at T3. This would suggest that a training response did occur since the physiological response, measured with blood lactate and heart rate sum, to a given speed decreased.

By using two standard lactate and heart rate sum
predicted and actual velocities were quite close, the relationship between the two was low ($r = 0.61$) with a standard error of the estimate at 0.05 m/sec which translates to approximately $\pm 3.5$ sec over this distance.

Figure 1. Example of procedures used for plotting relationship between velocity and either blood lactate or heart rate sum. Also shows how responses to standard values were predicted.

Figure 2. Predicted blood lactate and heart rate sum response to a standard velocity of 1.50 m/sec at beginning of season (T1), after 2 months of training (T2) and after 4 months of training (T3).

values, the degree of change in the profiles at submaximal and maximal intensity was characterized (Fig. 3). The velocity required to reach a blood lactate of 6 mM changed from $1.37 \pm 0.02$ m/sec at T1 to $1.48 \pm 0.02$ m/sec at T2 and $1.50 \pm 0.02$ m/sec at T3 while that required to reach blood lactate of 12 mM changed from $1.54 \pm 0.02$ at T1 to $1.58 \pm 0.02$ m/sec at T2 to $1.61 \pm 0.01$ m/sec at T3. The same pattern was shown by the heart rate sum. The velocity required to reach a heart rate sum changed from $1.37 \pm 0.02$ m/sec at T1 to $1.46 \pm 0.02$ m/sec at T2 to $1.47 \pm 0.02$ m/sec at T3 while that required to reach a heart rate sum of 105 beats changed from $1.51 \pm 0.02$ m/sec at T1 to $1.59 \pm 0.02$ m/sec at T2 to $1.58 \pm 0.02$ m/sec at T3.

Using Mader's equations for the prediction of performance from the lactate profiles, predictions of performance capacity were made at each of the testing sessions. These predictions were then compared to actual performance times at competitive events that were in all cases held within two days of the profiles. These data are shown in Figure 4. There was no significant difference between the mean predicted velocity and the mean actual velocity when the data from all three test sessions were pooled. The mean predicted and actual velocities converted to time for the 200 freestyle were 1:49.84 and 1:50.77, respectively. Although the means for the

Figure 3. Mean ± SE lactate and heart rate profiles for the group at beginning of season (T1), after 2 months of training (T2) and after 4 months of training (T3).

Discussion

The major finding of this study was that both the heart rate and lactate profiles were capable of sensing changes in training status of competitive swimmers over the course of one season of training and competition. In addition, the profiles showed the same relative shift during the season with the greatest degree of change occurring during the first 2 months of the season. This finding is in agreement with studies that have shown that the greatest increase in maximum aerobic capacity occurs during the first few weeks of training (4,7,8).

Closer inspection of the change in these two profiles reveals that during the training period, the steepness of the slopes of both profiles increased. This finding indicates that the adaptations to training were manifest more during submaximal exercise intensity than during maximal intensity. It should be noted that this effect was not observed in all of the subjects and may have been due to the specific training programs of the individuals. For instance, if a relatively greater amount of emphasis were placed on endurance training than on sprint training, one might expect such a response. It is not possible from these data, however, to determine the exact cause of this response or whether it is a desirable one for improving performance. Consequently, further studies are necessary to examine the effects of different forms of training on the slope of these profiles.

The change in the predicted response to a mean 200 yard velocity of 1.50 m/sec (121.9 sec per 200 yards) was similar in direction and magnitude between the two profiles. The decrease in blood lactate concentration to
this velocity agrees with numerous studies that have shown a progressive decline in blood lactate at an absolute exercise intensity with training (1). The decrease in the heart rate sum at this velocity also agrees with published data showing that heart rate taken during exercise decreases at absolute workloads with endurance training (1,3,5,13).

The prediction of 200 performance time from the lactate profiles was very weak. The degree of relationship between the actual and predicted times was not high enough to give a very reliable indication of performance ability. A likely reason for why the prediction is unreliable is that the prediction equation predicts time based on an assumed maximum blood lactate concentration of 16 mM (12). This level may not be appropriate for all individuals since it is well known that maximum blood lactate shows both intra- and interindividual variability depending on factors such as training status (9,13), muscle fiber type (6,15) and nutritional status (14).

Consequently, for these predictions to be of any value in assessing performance capacity, a maximum blood lactate for each individual would need to be used in the equation. This determination of the maximum lactate for each individual would be difficult, requiring repeated sampling of blood following maximum competitive performances. During pilot studies currently underway, we have found that although the maximum lactate levels of swimmers do indeed vary with training, the range in variability is rather small. Thus, if this maximum lactate is known to be within a fairly narrow range, the prediction of performance times may be significantly more reliable than when a maximum lactate level is assumed for all individuals.

Although the heart rate profiles of the group during the season appear to respond similar to the lactate profiles to training, it was noted that the heart rate profiles of some individuals tended to be more variable than the lactate profiles thereby resulting in occasional erratic shifts in the profile. This is perhaps due to the fact that heart rate response to exercise can be quite variable, depending on extraneous factors such as the anxiety of the individual. Because of this possibility of contamination from variables other than improved endurance performance capacity, the heart rate profiles would not be as reliable as the lactate profiles in sensing improvements in training status. The present data indicate, however, that in the absence of blood sampling supplies, personnel and analytical equipment necessary for lactate measurement, the heart rate profiles offer the coach and athlete a reasonably objective and non-invasive alternative to lactic acid measurements in monitoring their training programs both within a season and across several seasons.

References


Relationship of Body Composition to Swimming Performance in Female Swimmers

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Abstract

The purpose of this study was to determine the relationship of body fatness to swim performance in competitive, female swimmers (n = 284). Subjects' best performance in their best event and in the 100 yard freestyle were determined by questionnaire. Body composition was estimated by densitometry, with residual lung volume measured by oxygen dilution. Best time in either event was not significantly related to body density, fat percentage, fat body mass or years of competition except in the 13-year olds. Performance was, however, found to be related to lean body mass. Since lean body mass was significantly intercorrelated to age, the effect of age was controlled for by using partial correlation procedures. Independent of age, lean body mass was shown to be unrelated to performance in the 100 yard freestyle. However, when swimmers were compared on the basis of their performance in their best events, the better performers were found to have a greater lean body mass, but were similar in body fatness when compared to the poorer performers. Thus, we conclude that for female competitive swimmers ages 12 to 17, while lean body mass appears to influence swimming performance, body fatness is relatively unimportant.

Key Words: BODY COMPOSITION, FEMALE SWIMMERS, PERFORMANCE, BODY FATNESS

Introduction

Women in today's society are acutely aware of body fatness. This is particularly so for women athletes due to the almost universal assumption that low fatness equates with enhanced athletic performance. Continual attempts are made by women athletes and their coaches to minimize body fatness even though the relationship between fatness and performance has only been defined for a few athletic activities. Studies of running events have shown body fat to have a significant negative effect upon endurance performance (5, 7, 11, 12, 14, 16) as well as upon sprint performance (1, 8, 16). In addition, the belief that athletic performance may be improved by reducing body fat may be accounted for by several other observations. A low relative fatness and a high lean body mass have been suggested as two of the few morphological characteristics common to outstanding male and female performers, irrespective of the sport (18). The difference in performance between males and females has been reported to be at least partially accounted for by the lower relative fatness of males (7).

One aerobic sport in which body fatness may not be detrimental to performance is competitive swimming. Although the evidence supporting a relationship between fatness and performance in swimming is largely anecdotal (22), it has been suggested that relatively lean individuals might be at a disadvantage in that fatness and bouyancy are known to be related (2). Individuals with a high body density float lower in the water and expend more energy while treading water than those with a low body density (21). Furthermore, low body density may increase the drag coefficient and thus affect energy expenditure during swimming. This, coupled with the
observation that swimmers have more fat when compared to athletes competing in other sports (8), suggests that either fatness is indeed a positive factor in swimming performance or it could indicate, as Counsilman suggests (6), that swimming performance is relatively independent of body composition.

Because of the recent suggestion that low body fatness in women may have an adverse effect upon reproductive function and development (9), the relationship between fatness and performance needs to be better defined. Thus, the purpose of the present study was to determine if body composition and swim performance in women swimmers are related.

Methods

In order to assess the relationship of body composition to swim performance, competitive female swimmers (n = 280) were hydrostatically weighed and then asked to complete a questionnaire concerning health, current and past training intensity and competitive performance. The subjects ranged in age from 12 to 17 years old and were solicited from United States Swimming Clubs and high school swim teams in California, Colorado, Hawaii, Indiana, Michigan, Texas and other Midwestern states. All subjects were informed as to the intent of the study and supplied written consent.

Body composition was determined by the hydrostatic technique (4) with residual lung volume (RV) determined prior to or after immersion using a closed circuit, oxygen dilution method (26). Body fat percentages were calculated from body density by use of the Brozek equation (2). Lean body mass (LBM) and fat body mass (FBM) were subsequently calculated by subtracting absolute body fat from total body weight.

Best time in the 100 yard freestyle (American crawl) was recorded and used as a performance measure. This specific swim stroke and distance were chosen for several reasons. First, 160 of the 280 women (57%) had participated in this event within the preceding six months. Secondly, of the four competitive strokes (butterfly, backstroke, breaststroke and American crawl), crawl is the stroke primarily used during training sessions and is thus the stroke in which the greatest number of women are proficient. The 100 yard distance was chosen because it represents an intermediate distance in the swimming age group studied. When compared to the other event distances, nearly twice the number of women routinely participated at this event distance and could therefore supply recent, accurate times.

In addition, to facilitate comparisons between various swimming events, best performance in each subject's best event was ranked and assigned a code number according to qualifying standards defined by United States Swimming (20). Seven performance levels within each age category were identified. The performance levels (qualifying standards) ranged from "B" standards to those which would allow competing in the National championships. Better performers and poorer performers in all events were pooled and compared using Student's T test.

A correlation matrix was generated to evaluate the interrelationships of all the variables for the 100 yard freestyle. In addition, partial correlation coefficients were calculated for selected relationships so as to cancel out the confounding influence of certain variables. Coefficients of variance were determined for all of the independent variables. Because of the effect of large sample sizes upon the criteria required for statistical significance, correlational relationships were considered significant when P < 0.001.

Finally, in order to examine the relationship between performance and body composition within a given age group, the population was divided into five age categories and the relationships between body composition and swim performance were determined.

Table 1. Subject characteristics.

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>Mean</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>280</td>
<td>14.2</td>
<td>1.7</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>280</td>
<td>164.0</td>
<td>7.1</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>280</td>
<td>53.8</td>
<td>9.1</td>
</tr>
<tr>
<td>Density (g/cm^3)</td>
<td>280</td>
<td>1.038</td>
<td>0.0108</td>
</tr>
<tr>
<td>% fat</td>
<td>280</td>
<td>16.8</td>
<td>4.4</td>
</tr>
<tr>
<td>FBM (kg)</td>
<td>280</td>
<td>9.2</td>
<td>3.4</td>
</tr>
<tr>
<td>LBM (kg)</td>
<td>280</td>
<td>44.5</td>
<td>6.4</td>
</tr>
<tr>
<td>RV (l)</td>
<td>280</td>
<td>1.052</td>
<td>0.26</td>
</tr>
</tbody>
</table>

FBM = fat body mass; LBM = lean body mass; RV = residual volume

Results

Subject characteristics are presented in Table 1. The correlation matrix for these variables is shown in Table 2. Height, weight, age, LBM and RV were significantly (P < 0.001) inversely related to the time for the 100 yard swim. However, density, relative fat (% of total body weight) and FBM were not significantly related to the time for the 100 yard swim. Since height, weight, LBM and RV were also significantly related to age (Table 2), partial correlation coefficients were calculated to control for the confounding effects of age on these various relationships (Table 3). After age was controlled, no significant relationship existed between time for the 100 yard swim and LBM, weight, height or RV. The coefficient of variance (r^2 x 100) calculated for the various relationships of time for the 100 yard swim to the independent variable are shown in Table 4. Of the variables inspected, age was the only one which accounted for any appreciable variance in swimming performance.

When the population was divided into age groups and
the relationship of the 100 yard swim to fat percentage, FBM and LBM were calculated (Table 5), the only significant relationship noted was between relative fatness and performance in the 13-year-old age group. Fat percentage was positively related ($r = .57, P = .001$) to the swim time indicating that in this age group higher fat percentages were associated with poorer swim performances. Table 6 illustrates the results of comparing the best performers with the poorest performers. As noted earlier, performance in each swimmer’s best event was assigned a number according to which performance standard (B to Sr. nationals) this time qualified. Swimmers making Sr. and Jr national or AAAA standards were compared to those meeting B or A time standards. Results indicated the better performers were older and had a significantly greater LBM than the poorer performers. However, no differences were found between the groups in either relative fatness or fat body mass.

Table 3. Partial correlation coefficients (controlling for age; n = 162).

<table>
<thead>
<tr>
<th></th>
<th>Height</th>
<th>Weight</th>
<th>LBM</th>
<th>RV</th>
</tr>
</thead>
<tbody>
<tr>
<td>100 yard time</td>
<td>.17</td>
<td>.16</td>
<td>.23</td>
<td>-.10</td>
</tr>
</tbody>
</table>

$r = .24$ required $P \leq .001$

Discussion

Body Fat

Body fat expressed as a percentage of total body weight previously has been reported to be inversely related to running and/or jumping performance (1, 5, 7, 11, 12). Indeed, it is generally believed that low levels of fat are desirable for outstanding performance in almost all sports. In contrast, the results of the present study suggest that for young women swimmers, performance is relatively independent of body fatness. Higher levels of body fatness in the population observed were not necessarily detrimental to performance in their best event or in the 100 yard freestyle. As fatness is not a criteria necessary for outstanding performance, this may in part explain the findings that when compared to athletes competing in other sports, swimmers have a greater relative level of fatness (8).

From biomechanical considerations, the reasons for the minor influence of body fat upon swimming efficiency can be proposed. It is clear that body fatness and body density are related. This relationship forms the basis of estimating body composition by hydrostatic weight (4). In any event, individuals with a low body density (high body fat) are more buoyant than those with a greater density (less fat) (15, 21). This tendency to sink must therefore be counteracted by muscular effort and is thus a component of the overall energy output during swimming. The lean individual will thus require a greater energy component to counteract his/her sinking tendency than a fatter individual. Individuals at the upper extremes of body density may be at a relative disadvantage.

On the other hand, the rate of a swimmer’s forward movement is a function of two forces; resistive forces and propulsive forces. The resistive forces (components commonly referred to as drag) are at least partially a function of the size and shape of the object moving through the water. Thus, for a given body shape the larger the body size, the larger is the resistive force. A swimmer with a large body fat mass, although less dense, will present a larger surface area to the water. The drag component will be greater when compared to the smaller
(less fat) individual. The individuals at the lower extremities of body density may be at a disadvantage in this regard.

When considered together, it is clear that these two components counteract each other. As fatness increases, drag increases and the tendency to sink decreases. As fatness decreases, drag decreases although the energy required to stay on top of the water thus increases. The overall effect of body fat upon performance is, therefore, relatively insignificant. It is possible, however, that the influence of body fat may be greater in events other than the 100 yard freestyle. For instance, the distance swimmer might be more affected by body composition than the sprinter. Continued research in this area is necessary before generalized conclusions can be drawn.

Table 5. The relationship between body composition and performance within each age group. All values are means ± S.D. Numbers within parentheses are the correlation coefficients for that variable and time.

<table>
<thead>
<tr>
<th>Age</th>
<th>n</th>
<th>100 yd free (sec)</th>
<th>% fat</th>
<th>Fat (kg)</th>
<th>LBM (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>12.0 to 12.9 yrs</td>
<td>38</td>
<td>64.5 ± 9.0</td>
<td>16.5 ± 0.04 (.29)</td>
<td>8.1 ± 2.5 (.25)</td>
<td>40.4 ± 4.7 (.03)</td>
</tr>
<tr>
<td>13.0 to 13.9 yrs</td>
<td>43</td>
<td>62.9 ± 4.3</td>
<td>17.5 ± 0.04 (.57)*</td>
<td>9.0 ± 2.6 (.05)</td>
<td>41.8 ± 5.0 (.04)</td>
</tr>
<tr>
<td>14.0 to 14.9 yrs</td>
<td>37</td>
<td>60.5 ± 3.5</td>
<td>17.3 ± 0.05 (.06)</td>
<td>9.7 ± 3.9 (.08)</td>
<td>45.2 ± 5.4 (.07)</td>
</tr>
<tr>
<td>15.0 to 15.9 yrs</td>
<td>24</td>
<td>59.0 ± 2.2</td>
<td>18.0 ± 0.03 (.15)</td>
<td>10.2 ± 2.3 (.02)</td>
<td>46.4 ± 3.8 (.39)</td>
</tr>
<tr>
<td>16.0 to 16.9 yrs</td>
<td>12</td>
<td>59.5 ± 1.9</td>
<td>18.4 ± 0.03 (.46)</td>
<td>11.7 ± 2.4 (.31)</td>
<td>51.6 ± 5.6 (.11)</td>
</tr>
<tr>
<td>17.0 to 17.9 yrs</td>
<td>8</td>
<td>58.9 ± 2.2</td>
<td>18.6 ± 0.05 (.01)</td>
<td>11.3 ± 4.8 (.08)</td>
<td>47.9 ± 7.0 (.07)</td>
</tr>
</tbody>
</table>

* P ≤0.001

Lean Body Mass

The most common model of body composition identifies two compartments; lean body mass and fat mass (23). Adipose tissue makes up the fat mass while the rest of the body, i.e. muscle, bone organs, etc., comprise the lean body mass. It is somewhat misleading to state for this population of swimmers that performance and lean body mass are unrelated. Tables 2 and 6 suggest that these two variables are related but as primarily a function of age. The most significant factor influencing performance in our population is age with lean body mass being a distant second. However, it is likely that age is not in and of itself the significant variable. It most likely represents an index of the maturation process.

When swimmers were compared on the basis of their performance in their best events (Table 6), the better performers were found to have a greater lean body mass but were similar in body fatness when compared to the poorer performers. These results are more consistent with findings in other sports (18). It has been shown that the greater the proportion of lean body mass, the greater is the maximal oxygen consumption, both absolutely and/or per kilogram of body weight basis (24). It is difficult to identify how lean body mass influences swimming performance from the current study. More research is needed in order to define the potential relationships which may exist between lean body mass, metabolic capability and strength in competitive women swimmers.

Although there are many questions which remain unanswered concerning the relationship between body composition and performance in women swimmers, the current findings are important for several reasons. Since low body fatness is almost universally equated with outstanding performance, many coaches may unnecessarily encourage their athletes to lose weight with the...
Body Composition and Swimming Performance

intent to improve performance. Our results suggest that
lean body mass is more important than is the relative
proportion of body fat. It is not usually recognized that
lean tissue as well as fat tissue is decreased by dieting
(’24). Nevertheless, it is well accepted that nutrition and
performance are coupled (17). Again, our results would
seem to indicate that dieting or undue concern over body
fatness may be more significant in terms of limiting per-
formance than is excess body fat.

There is evidence that reproductive function and men-
struation may be disrupted in competitive female athletes
who tend to have lower levels of body fat than their
respective non-athletic peers (9). Although the relation-
ship between fatness and reproductive function is being
debated, it is clear that the current preoccupation with
low fatness during adolescents is resulting in a greater
prevalence of eating disorders, such as anorexia and/or
bulimia (10). These disorders not only threaten perform-
ance but can threaten life itself.

Before formulating generalized conclusions, it should
be recognized that several weaknesses exist in a cross-
sectional research design such as that employed in the
present study. First, the variability of a characteristic or
criteria which may enhance performance tends to become
smaller as the population becomes more elite (3). Because
of the smaller variance, correlational relationships are
difficult to identify. With this in mind, we included in
the present study swimmers at all performance levels,
rather than only national qualifiers. Nevertheless, we
recognize that any population of athletes is non-random
and some selection factors must already be affecting the
specific characteristics of the group. This is particularly
true as the age of the athletic population studied is in-
creased. As indicated by our data, age is the most signifi-
cant criteria associated with performance. This should
not be interpreted to mean that the younger swimmers
cannot be good swimmers. It is more likely evidence
that the population becomes more select as it grows older.

Clearly, although no relationship between fatness and
performance was identified in our swimmers, there will
be an optimum range outside of which fatness will
adversely affect performance. That individuals with these
characteristics are present in the athletic population
studied is doubtful. Again, the obese individual will, for
many reasons, be likely to discontinue participation at
an early age.

Secondly, it cannot be resolved from a cross-sectional
study what the effect of lowering body fatness will be
upon any given individual’s performance. The answer
to this question can conceivably be obtained through a
longitudinal study. However, the ability to isolate all
other variables important in swimming performance, i.e.
training, motivation, psychological factors, stroke
mechanics, health, genetics, etc., reduce the effectiveness
of longitudinal designed studies.

Finally, an additional difficulty in interpreting the find-
ings of the present study is that estimations of body com-
position were performed at a point in time independent
of when the individual’s best performances were achieved.
However, prior studies have shown little (.6%) (22) or
no change in composition throughout a training session
in competitive women swimmers (13). In addition, many
of our swimmers compete and train year around.
Therefore, the changes which might occur over the course
of a several-month interval between a swimmer’s best
performance and her body composition estimate would
be less than what may occur if the untrained state were
compared to the trained state (25).

In conclusion, the results of the present study failed
to indicate the existence of a relationship between body
fatness and performance in adolescent competitive
women swimmers. No index of body composition inde-
pendent of the influence of age was shown to relate to
performance in the 100 yard freestyle. The absolute
amount of lean body mass, but neither absolute fat mass
nor the relative proportion of body fatness, was shown
was related to performance when comparisons were
made using the swimmer’s best events. Thus, we would
propose that coaches spend more time on activities which
would promote conditioning or increase strength (and
thereby increase LBM) and less time on activities related
to reducing body fat.

Acknowledgments. The authors would like to express their appreciation to
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mers and coaches who took part in this study but are too numerous to mention
individually.

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Nutritional Status of Elite Swimmers

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Abstract

Observations were made to determine whether any nutritional deficiencies exist in the diet of competitive swimmers. Highly trained, elite female (F) and male (M) athletes participated. Survey of their diets over a three day period was conducted following the 1984 Short Course National Championships. A computerized comparison of records to recommended daily allowances (RDA) was made and was based upon an extensive data base of foods provided by the Anjon Nutritional Analysis System. Data are reported as group means (plus/minus standard deviation and range). While mean total caloric intake was representative of other age-matched athletic populations for men and appeared to be adequate, females may not be consuming enough calories. Also the distribution of calories between carbohydrate and fat was deemed not conducive to optimal performance. As an example, both sexes obtained a relatively low proportion of their total calories from carbohydrate (75% and 81% of recommended). On the other hand, with the exception of iron status of the women, vitamin, mineral and electrolyte intake was adequate if not even excessive in some cases. Overall, these observations suggest that on an average basis, while non-caloric nutrients may be obtained in adequate amounts, these athletes may not be providing optimal levels of the "energy" producing foods. The data also indicate, however, that individual athletes may have extremely poor dietary habits and as a result, severe nutrition problems. Nutrition education of the athlete and coach is warranted.

Nutrition is one of a wide variety of physiological, biomechanical and psychological factors which combine to ensure optimal athletic performance. Like all Americans, athletes require a well-balanced diet, containing nutrients to sustain not only normal daily activities, but also those associated with training and competition (1, 6). Caloric requirements vary and are dependent, in part, upon sex, body size, age and level and intensity of training. It is not unusual for athletes to consume 3000 to 6000 Calories (Kcal) per day (6) while still maintaining body weight.

Assuming well-balanced selections from the four food groups (meat/poultry/fish, dairy products, grains/cereals, fruits/vegetables), athletes should be able to achieve nutrient intakes adequate to sustain training and competition (4). That coaches and athletes have sound dietary habits and understand the importance of good nutrition is questionable however (7).

The observations described below were made to (A) evaluate the diets of competitive swimmers, and (B) to form the basis for recommendations on meal planning during training and competition.

Methods

Fourteen women and 13 men who had swum at the 1984 Short Course National Championships and who were preparing for the Olympic Trials participated. Six subsequently made the Olympic Team. Each athlete recorded food intake on special forms (Anjon, Inc.) over a three-day period which was typical of the overall diet. Instructions on the importance of including all fluids suggested, substances used in the preparation of meals, and the need for accuracy in describing amounts and serving sizes were given to each swimmer. Reports also included all snacks.

The dietary records were analyzed for total caloric intake (food energy), amounts of protein, carbohydrate and fat (here expressed as percent of the total food

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energy), minerals and electrolytes (sodium, potassium, calcium, iron) and vitamins (A, C, niacin, thiamin-B1, riboflavin-B2). The above are known as "leader" nutrients, i.e. if adequate amounts of these are obtained from a balanced diet, the other 40 or so known nutrients will also probably be obtained at optimal levels. Note that the four "food groups" were originally set up using these leader nutrients. Analysis was completed by computer (Anjon Nutritional Analysis System, South Bend, IN). The total database is extensive (3, 10), containing some 1300 food items including all popular snack and fast food items by trade name.

Estimates of body fatness were made by skinfold measurement. Because of problems associated with conversion of these to percent fat by standard equations, absolute values of thickness are reported here.

The mean and standard deviation are used to describe each sample population. A Student’s Test was applied to detect significant differences between the sexes. Significance was determined to be at the 0.05 level of confidence.

Results

Subject characteristics are presented in Table 1; results of the dietary analysis in Tables 2-4. The males were older, weighed more but were leaner than the females. Note the general competitive level of these athletes. AR and WR stand for American and World Records, respectively.

Table 1: Subject Characteristics. Generally speaking, the men were older, heavier and leaner than the females. Note the overall competitive level of these athletes. AR and WR stand for World and American records, respectively.

<table>
<thead>
<tr>
<th>SUBJECT CHARACTERISTICS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (Yrs.)</td>
</tr>
<tr>
<td>Males</td>
</tr>
<tr>
<td>Females</td>
</tr>
</tbody>
</table>

+ denotes significant sex difference (p < 0.05) | *Tricep, Subscapula, Midaxilla, Suprailliac, Abdomen, Front Thigh

Table 2 presents information on caloric (energy) intake. The females consumed significantly less total calories than the males (2300 vs 4339 Kcal, respectively).

While this would be expected due to body size differences, the relationship still exists when caloric intake is expressed independent of body weight, i.e. as Kcal per pound. Notice that the men consume 50% more Calories per lb than do the women (24.8 vs 16.8, respectively).

Table 2 also presents the relative distribution of calories from the three basic energy providing nutrients, protein (PRO), carbohydrate (CHO) and fat (FAT). There were no significant sex differences in this distribution pattern and the amounts coming from CHO (49% and 53%) and FAT (34% and 30%) are close to Dietetic Association (1) recommended proportions (55% and 30% for CHO and FAT respectively).

CHO, FAT and PRO intake can also be expressed as percent of recommended based upon laboratory studies of athletes. Note that these proportions are different than those expressed by the Dietetic Association (more CHO and less FAT). As would be expected (see right hand panel of Table 2), both men and women were getting far more fat and less carbohydrate than recommended by sports scientists. For example, while CHO provided 49% of the total calories for the men, this is actually only 75% of the recommended based on athlete studies. "Ideal" levels would be 100% for each nutrient.

Tables 3 and 4 present information on the mineral, electrolyte and vitamin intake. With the exception of iron intake by the females, the diets met (or exceeded) the RDA for all items.

Discussion

A nutritionally balanced diet based upon the four food groups (1, 5, 6) with total calories sufficient to meet the body's energy demands is the goal for all individuals. Energy "cost" and nutritional balance studies have described the basic caloric and nutrient requirements. These amounts or levels are described by the Recommended Daily Allowance (RDA) (3). Allowances provide for individual variations among most normal persons under usual environmental stresses. Of total calories consumed, recommendations for the average American are that roughly 30% come from fat and 55% from complex carbohydrates (starches, breads, potatoes, pasta, etc.). This is in contrast to the observed distribution of calories in the average diet which contains nearly 40% fat and only 45% carbohydrate. Individuals should consume less fat and more carbohydrate.

The simple concept of providing the RDA for nutrients and shifting caloric distribution is difficult to put into practice, however, especially for the athletic population. Numerous myths, misconceptions and even dangerous practices are found when dealing with athletes. Two of the more prevalent are that (A) "more" is better "because I'm training" and (B) that weight (fat) loss can occur with dietary (caloric) restriction while still
Table 2: Expressions of Calorie Intake. The men consumed significantly greater calories, even expressed per pound body weight. Carbohydrate intake (% of total calories) could be greater for both men and women. Individual athletes have dietary problems with calorie distribution.

<table>
<thead>
<tr>
<th>CALORIE INTAKE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total Daily</strong></td>
</tr>
<tr>
<td><strong>Calorie Intake</strong></td>
</tr>
<tr>
<td><strong>(Kcal)</strong></td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td><strong>Males</strong></td>
</tr>
<tr>
<td>n = 13</td>
</tr>
<tr>
<td>range</td>
</tr>
<tr>
<td><strong>Females</strong></td>
</tr>
<tr>
<td>n = 14</td>
</tr>
<tr>
<td>range</td>
</tr>
</tbody>
</table>

*Recommended in Anion Nutritional Analysis System. Levels are PRO = 15%, CHO = 65%, FAT = 20% of total calories. See text for discussion.+
+ denotes significant sex difference (P < 0.05).

maintaining the caloric requirements for energy production during training and competition.

Training places extraordinary demands upon the body systems including those responsible for energy production. "Calories" provide the energy, the remaining nutrients used in various ways to help provide that energy. Thus the nutritional status of the athlete and the dietary habits on a day-to-day basis are important factors for success in both training and competition.

Energy "cost" studies would suggest that swimmers require approximately 3000 Calories for workouts (9) and another 1200-1500 for maintenance of body functions during the rest of the day. Generally speaking, if caloric "balance" is achieved (calories IN from food = calories OUT for metabolism), body weight remains relatively stable. Upsetting the balance one way or the other results in either weight gain or weight loss.

From the present data, it would appear that the men have adequate caloric intake in terms of total calories (4339 Kcal/day; Table 1) assuming they compare generally with the above "costs". The females are considerably below optimum levels (2300 Kcal/day). This assumes however, that training levels are similar in terms of total caloric demand and that body weight was remaining stable. As we have no information on these factors, no definite conclusion can be made regarding the adequacy of caloric intake for the women. It is interesting to note in this regard, that ancillary information from both coaches and athletes would suggest an obsession with fatness and a view that these women must lose weight.

The large sex differences in caloric intake are also attributable, in part, to differences in size (height, weight). Note however, that significant differences still exist when caloric intake is expressed independent of weight, i.e. as Kcal/pound. Comparison to information from other sports also suggests that the females may have a low caloric intake.

Energy "cost" studies have not only described the caloric requirement for activity, but also the source (CHO, PRO or FAT) of energy at various levels of work intensity (5, 11). These clearly point out the importance of carbohydrate as a limiting energy source for both training and competition. On the basis of this research, it can be recommended that approximately 65% of the total calories come from carbohydrate sources (and 20% from fat, assuming protein remains at 15%). This recommendation is based upon studies of muscle and blood during exercise and the effects of altering diet upon nutrient stores.

Compared to the typical American diet (45% Calories from CHO) or that recommended by the Dietary Association (55%), the swimmers consumed fairly adequate amounts of carbohydrate (49% and 53% total Calories; Table 2). When compared to that recommended by athlete studies, however, both men and women are substantially low (75% and 81% of optimum, men and women respectively). Protein intake is near optimum, while fat consumption is extremely high. It should be noted that the fat intake of these athletes (34% and 30% of total Calories; Table 2) is below that seen in the typical American diet (40%) and that the 20% of total calories level on which the calculations in the right hand panel of Table 2 are based, is probably unrealistically low and from a practical point of view, impossible to achieve for a training athlete.

In contrast to the calorie distribution data, these athletes were able to supply from their diets, the RDA for all electrolytes, minerals and vitamins listed except iron for the women (Tables 3 & 4). In fact some concern may be expressed regarding the high sodium (250% RDA), iron (300% RDA) and vitamin A (225% RDA) intake by the men.

Among other functions, these nutrients are used in various chemical reactions to help release the "energy" available in the calorie containing foods. There is no conclusive evidence that intake of these nutrients above that available from a balanced diet improves performance, nor is there any indication that training athletes have a greater requirement than does the average population.
(1, 6), assuming caloric intake meets requirements for activity. For example, there is evidence that exercise training increases the needs for the water soluble vitamins niacin, thiamin and riboflavin. The increased need is related to the total calorie need and can be met by appropriately increasing caloric intake (3). The RDA is set to account for differences in activity level. Thus the idea that “more is better” is not only inappropriate but can actually be dangerous when high levels of fat soluble vitamins or other minerals and electrolytes are ingested as supplements.

The one exception cited earlier was for iron intake by the females (only 80% of RDA). This observation is consistent with the female population in general and other athletic groups (2, 8). There is also some evidence that training can induce a greater than normal “draw” on the iron stores of the body so that a transient “sports” or “iron deficiency" anemia can be present (8). The significance of this for performance, however, is unclear.

Iron is a major component of hemoglobin, serving to hold oxygen in the red blood cell. It also serves in several chemical reactions which release “energy” in the muscle cell. Thus some potential exists for the training athlete with inadequate iron intake (especially females) to have a reduced capacity for oxygen transport by the blood and/or energy production in the muscle.

This does not mean that mega doses of iron must be supplied the training athlete. Indeed, iron intake is generally related to caloric intake so that more iron could be obtained just by providing more total calories (balanced from the four food groups). This other possibility is to provide an iron supplement. Indeed it can be stated that any woman, athlete or not, consuming less than about 2000 Calories per day is a likely candidate for iron and possibly other deficiencies. In this case we would recommend a standard multi-vitamin/mineral supplement with iron (CHECK the RDA levels on the bottle. Percent RDA should be about 50-150% daily for every item).

Table 3: Mineral and Electrolyte Intake. While average values appear normal, individual athletes are not meeting daily requirements or are getting too much of these. Concern can be expressed for calcium and iron intake for the women.

<table>
<thead>
<tr>
<th>MINERAL OR ELECTROLYTE</th>
<th>Sodium</th>
<th>Potassium</th>
<th>Calcium</th>
<th>Iron</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>% RDA</td>
<td>± SD</td>
<td>± SD</td>
<td>± SD</td>
</tr>
<tr>
<td>n = 13</td>
<td>250</td>
<td>100</td>
<td>144</td>
<td>270</td>
</tr>
<tr>
<td>range</td>
<td>156-442</td>
<td>65-227</td>
<td>70-599</td>
<td>124-554</td>
</tr>
<tr>
<td>Females</td>
<td>% RDA</td>
<td>± SD</td>
<td>± SD</td>
<td>± SD</td>
</tr>
<tr>
<td>n = 14</td>
<td>142+</td>
<td>50</td>
<td>105</td>
<td>60</td>
</tr>
<tr>
<td>range</td>
<td>67-247</td>
<td>40-224</td>
<td>46-208</td>
<td>40-166</td>
</tr>
</tbody>
</table>

RDA Food and Nutrition Board. Recommended Dietary Allowances, 9th Ed., National Academy of Sciences, Washington, D.C., 1980. There is no RDA for several vitamins and minerals including sodium (Na+) and potassium (K+). These are listed as “safe and adequate” daily intakes.

+ denotes significant sex difference (P 0.05)

Special Concerns

Any well-controlled laboratory or field study bases its conclusions (and recommendations) upon appropriate statistical analysis of the data and also makes the assumption that the sample studied is representative of the whole population of athletes described. At this point we cannot determine whether or not the present observations on these 27 swimmers are representative of the total swim community. In this regard we are currently gathering more information on the dietary habits of these athletes.

From a practical point of view this concern may be irrelevant however. For the coach, the individual response is important, not the group average. We would suggest then, that the trends demonstrated by the mean (average) data and, more importantly, the individual responses should be considered.

Table 4: Vitamin Intake. Similar to mineral/electrolyte intake, averages do not tell the whole story. Both excesses and deficiencies appear to exist.

<table>
<thead>
<tr>
<th>VITAMIN</th>
<th>B₁ (Thiamin)</th>
<th>B₂ (Riboflavin)</th>
<th>Niacin</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>% RDA</td>
<td>± SD</td>
<td>± SD</td>
<td>± SD</td>
</tr>
<tr>
<td>n = 13</td>
<td>225</td>
<td>100</td>
<td>189</td>
<td>144</td>
</tr>
<tr>
<td>range</td>
<td>97-408</td>
<td>90-355</td>
<td>94-353</td>
<td>112-1200</td>
</tr>
<tr>
<td>Females</td>
<td>% RDA</td>
<td>± SD</td>
<td>± SD</td>
<td>± SD</td>
</tr>
<tr>
<td>n = 14</td>
<td>175</td>
<td>100</td>
<td>128</td>
<td>101</td>
</tr>
<tr>
<td>range</td>
<td>92-900</td>
<td>86-355</td>
<td>52-286</td>
<td>112-670</td>
</tr>
</tbody>
</table>


+ denotes significant sex difference (P 0.05)

The latter are expressed in the tables as ranges and clearly show that while on a group average basis nutritional status may be satisfactory or at least marginal, there are individual athletes who should be concerned, in particular those getting only about 35% of total calories from carbohydrate and those with low calcium and iron intakes. Many values are far below recommended. Likewise concern can be expressed over abnormally high values as well, as example vitamin A for both sexes.

Summary

The present group observations suggest that these elite swimmers, while essentially providing all of the basic non-caloric nutrients, may have inadequate total caloric intake and/or non-optimal calorie distribution. The former is likely due to attempts to control weight by reducing food intake. Capacity for provision of energy needed for training is compromised. The latter is due to consumption of too much fat and too little carbohydrate. As carbohydrate is the recommended energy source, attempts should be made to alter this propor-
tion. It should also be noted that per unit weight of food, fat has more than double the amount of calories than does carbohydrate (for 1.0 gram, fat = 9.0 Calories, CHO = 4.1 Calories). Reductions in total caloric intake and yet provision of adequate energy sources can be had by shifting to a diet greater in carbohydrates content at the expense of fat. The importance of a balanced diet from the four food groups cannot be over emphasized as adequate calories and nutrient intake can only be achieved by following good dietary habits.

The data also show, however, that individual athletes may have extremely poor dietary habits. As a result they are not providing adequate nutrients for maintenance of their training programs.

As many athletes and coaches are unaware of not only what they are eating (in terms of nutrients), but also how much they eat and what the requirements are for exercise (7), a good nutrition education program would seem to be warranted.

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References

Determining the Proper Training Speeds for Swimmers

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Abstract

MAGLISCHO, ERNEST W., CHERYL W. MAGLISCHO, RUSSEL E. SMITH, RICHARD A. BISHOP, AND PETER N. HOVLAND. PRESCRIBING TRAINING THROUGH BLOOD ANALYSIS. The purpose of this study was to determine the effect of a season of competitive swim training on the swimming speeds and heart rates required to produce blood lactate concentrations of 2 mM/l, 4 mM/l, 8 mM/l and 12 mM/l. Blood samples were collected from six male intercollegiate swimmers on three different occasions during the course of six weeks of training. These samples were analyzed, in duplicate, for lactic acid content. On every occasion, three blood samples were taken, following each of three 200 yard time trials that varied in intensity from 70 to 98 percent of race speed. Immediate post-sprint heart rates were graphed opposite the blood lactate concentrations they produced and a line of best fit was drawn so that the swimming speeds and heart rates that would produce blood lactate concentrations of 2 mM/l, 4 mM/l, 8 mM/l and 12 mM/l could be predicted. Increases in the speeds and heart rates estimated to produce the blood lactate concentrations under investigation were analyzed statistically. The results were: (1) an average increase in percent effort of 16 percent was required to produce a blood lactate concentration of 2 mM/l from the beginning (65 ± 11.25) to the end (81 ± 3.7) of the training period. At the 4 mM/l level the required increase in effort between test I (74 ± 9) and test III (87 ± 2.5) was 13.3. On test I the athletes required a percent effort of 83 ± 7.5 to reach the 8 mM/l level. This was increased 9% to 92 ± 1.3 for test III. All of the above differences were significant. Increases at 12 mM/l were not significant; (2) an average increase in post-exercise heart rates of 38 bpm's was required to produce a blood lactate concentration of 2 mM/l from the beginning (124 ± 14) to the end (162 ± 11) of the test period. There was an increase of 29 bpm's at the 4 mM/l level from test I (143 ± 9) to test III (172 ± 172 ± 6). At the 8 mM/l level there was an increase of 28 bpm's from the beginning (158 ± 17) to the end (186 ± 11). All of these results were significant (.01). Increases at 12 mM/l did not attain significance. Some suggestions were presented as to the optimum training speeds and heart rates for improving the aerobic and anaerobic capacities of competitive swimmers.

Key words: BLOOD LACTATE, BLOOD TESTING, COMPETITIVE SWIMMING, HEART RATE COUNTING, OPTIMAL TRAINING PACE
Introduction

Recently, a considerable amount of attention has been focused on "blood testing" as a means of determining swimmers' adaptations to training. Details of the blood testing procedure were made available when Mader, one of the architects of the East German swim training program, defected to the West. Mader reported research in which he and his colleagues had collected small samples of blood following a series of time trials ranging from 60% to 100% of race speed. By determining the blood lactic acid concentration required to swim a particular speed they were able to predict adaptations to training. In addition, they were able to predict each athlete's optimal training speeds for various repeat distances (8).

We decided to use similar blood testing procedures to determine if we could find a non-invasive method for predicting optimal training speeds. Before discussing that research, we want to provide some background information on the blood testing procedure and the training concept that has evolved from this procedure.

Blood Testing and Training

Lactic acid is an intermediate waste product of glycogen metabolism (glycolysis). It is produced in muscles during exercise when the supply of oxygen is not sufficient to continue the breakdown of glycogen to its final end products, CO₂ and H₂O. At any time of the season a swimmer will have a finite limit of lactic acid accumulation that he or she can tolerate before performance deteriorates.

If small amounts of blood are withdrawn and analyzed following races or practice repeats, the lactic acid content of that blood can provide an estimate of the quantity of lactic acid that was produced in the swimmer's muscles during the swim. This is because the lactic acid that is produced in muscles will diffuse into the bloodstream during the swim and during the recovery period that follows (8). When repeated blood tests indicate a reduction in the rate of lactic acid accumulation during exercise an athlete should be able to swim further or faster before becoming fatigued.

Blood testing can also be used to predict an individual swimmer's proper training speeds. This is done by determining those speeds that produce certain blood lactic acid concentrations. The resting concentration of lactic acid in the blood is usually between 1 mM/1 and 2mM/1. Aerobic training adaptations are believed to occur when a swimmer's repeat speed is sufficient to increase that concentration beyond 2mM/1 (11). After that, further increases in swimming speed will cause a slow but steady increase in blood lactic acid until a critical speed is reached where the production of lactic acid in muscle fibers greatly exceeds its rate of removal to the bloodstream. When this occurs, further increases in speed will result in a rapid increase of blood lactic acid until the maximum tolerable level is reached.

The critical speed where the balance between muscle lactic acid production and removal is exceeded is considered to be optimal for aerobic training. Those physiological mechanisms involved in aerobic metabolism are believed to be maximally overloaded at that point (8).

Mader and his associates have indicated that, for most athletes, the critical training speed corresponds to a blood lactic acid concentration of 3-5 mM/1 (8). Thus, they have suggested swimming speeds that will produce a blood lactic acid concentration of 4 mM/1 as optimal for aerobic training.

Swimming coaches have traditionally used percent efforts and heart rates to determine the proper training speeds for their athletes. Blood testing is thought to be more accurate than either of these methods (9). However, it is doubtful that coaches will use this method extensively because most do not have the time, facilities nor the expertise to perform such analyses. It would be good if they could predict their athletes' blood lactic acid concentrations from non-invasive methods such as post-swim heart rates and percent efforts.

It is well known that increases in heart rate and swimming intensity have a linear relationship up to the point where a maximum heart rate is attained. The proper heart rates for aerobic training have generally been suggested to be in the range of 85% to 95% of maximum. This would correspond to heart rates of 150-190 for most high school and college age athletes (10).

Exercise physiologists commonly define the optimal training speeds for aerobic training as a percentage of maximum oxygen consumption. This concept has not been useful to coaches who generally do not know the VO₂ max of their athletes. Coaches generally suggest percent efforts between 75% and 90% of a swimmer's maximum speed for the repeat distance as optimal for aerobic training.

Purpose

The purpose of this study was to investigate whether blood lactic acid concentrations could be predicted from repeat times and from post-swim heart rates. We hoped to find a method for predicting optimal training speeds that was more practical than blood testing and more accurate than the noninvasive methods in present use.

Test Procedures

Six members of the men's varsity swimming team at Oakland University were subjects for this study. There were three freestylers, two backstrokers and one butterfly in the group. Samples of blood were taken by fingertip pricks after each of three 200 yard swims. The samples were analyzed for lactic acid content by the
simplified method described in the 1979 edition of Analytical Methods for the Measurement of Human Performance published by the staff of the Human Performance Laboratory at Ball State University (3). The pace of the first swim was to be within 70 to 80 percent of the swimmer's lifetime best. The second was to be between 80 to 90 percent, and the pace of the third was to be in excess of 90 percent. The swimmers rested for 30 minutes between each 200 swim, which was sufficient time to permit nearly complete recovery (2). All athletes swam their specialties.

Heart rates were recorded immediately at the conclusion of each swim by palpating the carotid artery for ten seconds. We selected this procedure because it is the method most often used by swimmers. An "Insta Pulse" heart rate counter was also used to measure immediate postswim heart rates.

The swimmers were tested on three different occasions. The subjects had been in training for four months prior to testing period I. They were just completing the major portion of their precompetition training at this time. The emphasis had been on aerobic endurance training. The second series of swims, testing period II, were completed 4 weeks later during the competition period. The quantity of training had been reduced and the quality of training increased during this period. Testing period III occurred 2 weeks later. The swimmers had completed the first week of a gradual taper at that time.

The subjects were not able to duplicate their time trial speed from one test period to the next, therefore, it was necessary to standardize their performances in order to make comparisons. After each test session, the times for the three 200 yard swims were converted to percent efforts by the following formula: %EFFORT = 100(200 - (time in seconds/100)). The three percent efforts were then plotted opposite the blood lactic acid concentrations they produced. Connecting the plots with a straight line and then determining the point of intersection of this line with any designated blood lactate acid concentration made it possible to estimate the percent effort required to produce that concentration. One such graph is shown in Figure 1. The dotted line extended from the ordinate at a concentration of 4 mM/1 and then dropped downward at a ninety-degree angle to the abscissa indicates that an effort of 89% was required to produce that concentration.

The accuracy of this procedure is based on the assumption of a linear, rather than curvilinear, relationship between swimming speed and blood lactic acid concentrations. According to Mader and colleagues, this assumption is accurate for speeds that produce blood lactic acid concentrations between 4 mM/1, and maximum (8).

The heart rates associated with certain blood lactate concentrations were estimated by a similar procedure.

Heart rates were graphed opposite blood lactic acid concentrations and a line of best fit was drawn (see figure 2).

We selected the percent efforts and heart rates that were estimated to produce blood lactic acid concentrations of 2 mM/1, 4 mM/1, 8 mM/1 and 12 mM/1 for comparison purposes. Our reasons for doing so were as follows:

1. We selected a concentration of 2 mM/1 because it represents the minimal training speed that will improve aerobic endurance (11). An estimate of the percent efforts and heart rates that produce this concentration may help swimmers identify those training speeds below which training becomes ineffective. This training speed is sometimes referred to as the "aerobic Threshold." We cannot be certain of our estimates at this level since, as indicated earlier, the relationship between changes in speed and blood lactic acid concentrations is not linear below 4 mM/1.

2. The 4 mM/1 level was selected because it is considered to most closely approximate the optimal training speed for improving aerobic endurance (8, 11).

3. A concentration of 8 mM/1 was selected because there is some indication that it approximates the intensity where maximal oxygen consumption is reached (5).
Since it is important to train VO₂ max, predicting training speeds and heart rates that produce blood lactic acid concentrations of 8 mM/1 could help coaches monitor training for this purpose.

4. Most athletes are capable of tolerating blood lactic acid concentrations between 12 and 20 mM/1 before becoming exhausted (8). Comparisons at the 12 mM/1 level, because it represents the threshold of maximum intensity, might identify the minimum repeat speeds and heart rates that will improve anaerobic endurance.

The data were analyzed for changes in heart rates and percent efforts estimated to produce blood lactic acid concentrations of 2 mM/1, 4 mM/1, 8 mM/1 and 12 mM/1 during the six weeks of training. An ANOVA and Tukey Test were applied and the results interpreted at the .01 level of probability.

Results

Effects of Swimming Speed on Blood Lactic Acid Concentrations

The effects of six weeks of training on the percent efforts estimated to produce the blood lactic acid concentrations we investigated are shown in Figure 3. Notice that, as the season progressed, faster speeds (indicated by greater percentages at later test periods) were required to produce the same blood lactic acid concentration. The increase in speed estimated to produce blood lactic acid concentrations of 2 mM/1, 4 mM/1 and 8 mM/1 were significant between the first and second and between the first and third test periods.

Figure 3: Effects of Training on the Percent Efforts Estimated to Produce Blood Lactate Concentrations of 2 mM/1, 4 mM/1, 8 mM/1 and 12 mM/1.

Increases in speed were not significant between the second and third test periods, although the improvements at 2 mM/1, 4 mM/1 and 8 mM/1 approached significance. The lack of significance was probably due to the shorter interval, (2 weeks) between the second and third test periods. We believe an additional two weeks of training would have produced significant increases.

Figure 3 shows that, during testing period I, subjects had to swim at speeds that were between 54% and 76% of maximum, (mean 65%), to produce a blood lactic acid concentration of 2 mM/1. The group became more homogeneous later with most requiring speeds between 77% and 84% of maximum, (mean 81%), to achieve the same blood lactic acid concentration during testing period II. There was no change between testing periods II and III.

The majority of the subjects had to swim at speeds between 65% and 83% of maximum, (mean 74%), to produce blood lactic acid concentrations of 4 mM/1 during testing period I, (see Figure 3). Once again, the group became more homogeneous later in the season. Training speeds had to be increased between 82% and 88% of maximum, (mean 85%), by testing period II. The speeds were further increased to within 84% and 90% of maximum, (mean 87%), during testing period III.

Regarding speeds that produced blood lactic acid concentrations of 8 mM/1, the information presented in Figure 3 indicates that speeds between 76% and 91% of maximum, (mean 83%), were required for this purpose during testing period I. During testing period II, speeds between 86% and 92% of maximum (89%) were required. Still later, during testing period III, speeds between 90% and 94% of maximum, (mean 92%), were required to produce blood lactic acid concentrations of 8 mM/1.

Our results indicate that repeat speeds between 88% and 99% of maximum, (mean 94%) produced blood lactic acid concentrations of 12 mM/1 during testing period I, (see Figure 3). The groups appeared to become more homogeneous later in the season with repeat speeds between 92% and 98%, (mean 95%), required for this purpose during testing period II and speeds between 95% and 98%, (mean 96%), needed to produce the same result during testing period III. The differences between mean repeat speeds for these testing periods were not significant, however.

Selecting the Proper Heart Rates for Training

The results depicted in Figure 4 show the effect of six weeks of training on the range of heart rates estimated to produce blood lactic acid concentrations ranging from 2 mM/1 to 12 mM/1. Progressively higher heart rates were required to produce the same blood lactic acid concentrations from the beginning to the end of the six weeks testing period.

Increases in heart rates between testing periods I and II and between periods I and III were significant at concentrations of 2 mM/1, 4 mM/1 and 8 mM/1. The increases at 12 mM/1 were not significant between testing periods. Heart rate increases noted between the testing
periods II and III were not significant for any of the blood lactic acid concentrations under investigation. Once again, we felt that an additional two weeks of training would have produced significant increases at concentrations of 2 mM/L, 4 mM/L, and 8 mM/L.

Heart rates between 110 and 138 bpm (mean 124) indicated a blood lactic acid concentration of 2 mM/L for the majority of subjects during testing period I. Heart rates between 139 and 167 bpm (mean 153) were required for this same purpose during testing period II. During testing period III, blood lactic acid concentrations of 2 mM/L were produced by heart rates ranging between 151 and 173 bpm (mean 162) for the majority of the subjects.

Heart rates between 134 and 152 bpm (mean 143) were needed to produce blood lactic acid concentrations of 4 mM/L for the majority of the subjects during testing period I. Heart rates between 149 and 173 bpm (mean 161) were required during testing period II. During testing period III, after an additional week of training and one week of tapering, it required heart rates between 162 and 182 bpm (mean 172) to reach the same concentration.

There was considerable variation among the subjects as to the heart rates estimated to produce a blood lactic acid concentration of 8 mM/L during testing period I. For the majority of subjects the range was 141 to 175 with a mean of 158 bpm. This range increased to 167-187 bpm with a mean of 177 by the time of testing period II. During testing period III it increased further to a range between 175 and 197 bpm with a mean of 186.

**Discussion**

**Effects of Swimming Speed on Blood Lactic Acid Concentrations**

Our findings are in agreement with the theory that training permits athletes to swim nearer maximum speed without increasing their blood lactic acid concentrations. Among our subjects the average increases in swimming speeds were 16% at 2 mM/L, 13% at 4 mM/L, and 9% at 8 mM/L during the six weeks testing period.

Realizing that our estimates of the optimal pace for aerobic training might only apply to repeats of 200 yards, the test distance, we took blood samples from one swimmer as he completed sets of 100 and 500 yard repeats during mid-season. He was asked to swim these repeats at speeds within the appropriate range of effort, (3-5 mM/L). In one session he swam twenty-four 100 yard repeats on a 1:20 interval. Blood samples were taken following completion of the 8th, 16th, and 24th repeats. The predicted pace was 1:00. However, the swimmer’s actual repeat times ranged from 1:05 to 58. + with the average time at 1:01. +. His results are shown in Figure 5.

On another day he swam four 500 yard repeats on 6 minutes. Blood samples were taken after each repeat. The results are shown in Figure 6. The predicted pace was 5:33.0.

**FREESTYLE**

24 X 100 on 1:20 secs.

**AVG. TIME 1:01 +**

![Figure 5: Blood lactate concentrations during a set of 24 one hundred yard freestyles.](image)

In both sets of repeats, repeat speeds were sufficient to produce blood lactic acid concentrations between 5 and 8 mM/L. Although he swam faster than the predicted paces it seems obvious that those paces would have produced blood lactic acid concentrations that were within the optimal range for improving aerobic endurance, (3-5 mM/L).

There is, however, an indication that shorter repeats should be swum slightly slower than the percent efforts predicted from the 200 swims of our subjects while longer repeats should be swum faster than the predicted percent efforts.

The average speed for the 100 yard repeats was 81%
of the swimmer's maximum 100 yard speed. The 500 yard repeats were swum at an average speed which was 91% of the swimmer's maximum 500 yard speed. At this same point in the season this swimmer required efforts of 84% of his maximum 200 yard speed to predict a blood lactic acid concentration of 4 mM/L. Therefore, if coaches wish to use our results to predict the percent efforts for training, it is suggested that the speed of shorter repeats should be approximately 10% slower than the percent efforts we have recommended for 200 yard repeats. Longer repeats should be swum approximately 5% faster.

According to the results of this study, percent efforts between 54% and 76% of maximum can produce some improvement in aerobic endurance early in the season. This range produced a blood lactic acid concentration of 2 mM/L during the first testing period. Later in the season percent efforts that were between 77% and 84% of maximum were required for this purpose.

Improvements in VO2max may be brought about by training between 75% and 90% of maximum (actually 76% and 91%) early in the season. This approximate range produced a blood lactic acid concentration of 8 mM/L in most of our subjects. By mid-season, during the second testing period, efforts between 86% and 92% were required for this purpose with efforts of approximately 90% to 95% required for this purpose late in the season.

![Blood Lactate Concentration](image)

**Figure 6:** Blood lactate concentration during a set of four 500 yd. freestyles.

Based on these results, it appears that efforts in excess of 95% of maximum may be satisfactory for improvement of anaerobic endurance (see Figure 3).

**Predicting the Proper Speeds for Training**

Our purpose was to determine if blood lactic acid concentrations could be predicted from percent efforts and heart rates. Because of the individual differences among the subjects in our group it is obviously not possible to predict, for each individual swimmer, the exact percent effort that will produce certain blood lactic acid concentrations. However, predictions that are made within ranges encompassed by the standard deviations of the group should allow the majority of swimmers to train near the blood lactic concentrations they choose.

Several textbooks on physical conditioning suggest that exercise intensities of 70% of VO2max (which is less than 70% of maximum speed), are optimal for aerobic endurance training (4, 6, 10). Such speeds would obviously have been below optimum for most of our subjects. Perhaps they would have been adequate in the early months of the season, before we began testing. However, they were clearly too slow by the time of our first testing period.

Our results are in agreement with the suggestions by some coaches that swimmers should do their aerobic training at speeds which are between 75% and 90% of maximum. However, swimmers would be best advised to train in the low end of this range during the early season, in the mid-point during the middle of the season, and at the high end of the range late in the season.

Our results indicate that the optimal intensity for aerobic training was between 65% and 83% of maximum speed during the first testing period. This range produced blood lactic acid concentrations of 4 mM/L for the majority of our subjects. Training speeds between 82% and 88% of maximum were required for this purpose during the second testing period while speeds between 84% and 90% were needed during the final testing period.

**Predicting the Proper Heart Rates for Training**

It was possible to predict certain blood lactic acid concentrations from heart rates. As with percent efforts, however, it was only possible to predict within ranges encompassed by the standard deviation of the group. These ranges were much larger than the ranges listed for percent efforts and the group did not become more homogeneous as the season progressed (see Figure 4) as they had when percent efforts were compared to blood lactic acid concentrations (see Figure 3). Therefore, it should be less accurate to use heart rates, than to use percent efforts, as a guide to aerobic training. Nevertheless, it is still possible to use heart rates for this purpose since during later testing periods, the lower end of the range for each concentration was usually above the mean of the previous testing period. For example, the mean was 143 bpm for a concentration of 4 mM/L during the first testing period while the range of heart rates that would produce this same concentration during testing period II was 149-173 bpm's with a mean of 161, and was 162-182 with a mean of 173 during the third testing period.

Our results are in partial agreement with the belief that the proper range of heart rates for aerobic training is in the range of 150-190 bpm. While the proper heart rates are generally within this range during mid-season and late season, 150 bpm is probably higher than necessary.
for early session training. According to our data the low end of this range is adequate for mid-season aerobic training while the mid-point is adequate for late season training. Where improving aerobic endurance is concerned, it is probably not necessary to train at the high end of the range at any time during the season.

Heart rates of 130-150 bpm (actually 134-152) indicated optimal intensity for aerobic training during the first testing period. We assume that even lower heart rates, in the range of 120-140 bpm might have been optimal for this purpose earlier in the season. During the second testing period, heart rates in the range of 150-170 indicated the proper training intensity, while later, during the third testing period, a range of 160-180 bpm was indicative of the proper training intensity (see Figure 4).

Our results indicate further that heart rates in the range of 110 to 140 bpm can produce some aerobic training effects early in the season (see Figure 4). This is the range of heart rates that produced blood lactic acid concentrations of 2 mM/1 for most of our subjects during the first testing period. Apparently, heart rates of 140-160 bpm and 150-170 bpm will do likewise at later points in the season (see Figure 4).

It seems that athletes cannot swim at the same heart rate all season long and expect to continue improving their aerobic endurance. Instead, they must swim at progressively higher heart rates throughout the season if they wish to maintain the proper intensity for such improvement.

Where improvements in VO2max are concerned, heart rates of 140-170 bpm seem to be adequate early in the season. Heart rates of 170-190 bpm and 175-200 bpm may be required for this purpose at later times.

Conclusions

Based on the results reported here, the following conclusions seem justified:

1. Six weeks of swim training will reduce blood lactic acid accumulation at certain submaximal speeds. This statement is supported by the finding that increases in swimming speed were required to produce blood lactic acid concentrations of 2 mM/1, 4 mM/1, and 8 mM/1 throughout the testing period.

2. Athletes must swim at progressively faster rates of speed throughout the season in order to maintain the proper intensity for improvements in aerobic endurance. Support for this statement comes from the finding that progressively greater percent efforts were required to produce certain blood lactic acid concentrations throughout the testing period.

3. Athletes must swim at progressively higher heart rates throughout the season in order to maintain the proper intensity for improvements in aerobic endurance. The finding that, throughout the testing period, progressively higher heart rates were required to produce blood lactic acid concentrations of 2 mM/1, 4 mM/1, and 8 mM/1 supports this statement.

We have attempted to present a practical method for predicting the proper intensities for aerobic and anaerobic training. Repeat speeds, expressed as a percentage of one’s lifetime best swim (percent efforts) and immediate post-swim heart rates were recommended for this purpose. Due to the varying and changing physiological capacities found among our group of subjects, our recommendations were made in ranges that were based on the standard deviations of the group.

It should be understood that these ranges are not represented as proper for all swimmers. There will be certain athletes who must train at speeds which are faster and heart rates that are higher than those we have recommended. Still others will receive greater benefit from training at slower speeds and lower heart rates. Nevertheless, the suggested guidelines were accurate for the majority of swimmers that we tested. Therefore, we believe they can be used for training most of the swimmers on a team provided allowances are made for the minority who do not fit these data.

References

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The American Swimming Coaches Association suffered the loss of Keith Sutton on September 4, 1984. Keith’s death was both sudden and unexpected.

Keith served as Executive Director of the American Swimming Coaches Association for a period of nine months. During his brief tenure, Keith infused a measure of enthusiasm and vitality into our organization which is difficult to measure. We do know however, that it was Keith’s vision that led to the funding of the Journal of Swimming Research. He worked hard at creating a Journal the ASCA could be proud of. We can pay no better tribute to his memory than to continue working toward this goal.

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