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Lactate Threshold

John M. Kelly D.P.E.
St. Cloud State University

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JOHN M. KELLY, D.P.E.*

ABSTRACT—The purpose of this paper is to review the delicate metabolic balance an endurance athlete must maintain to achieve a desirable performance. The optimum pace is ultimately determined by the athlete's ability to deliver large volumes of oxygen to the working muscles while simultaneously preventing excessive lactate accumulation in the tissues and blood.

Lactic acid has been associated with fatigue for nearly 80 years. This anaerobic metabolic by-product plays an important role in fatigue; however, many of the accusations concerning lactate's role in causing fatigue are unfounded or exaggerated. Its negative reputation is the result of an inadequate understanding of lactate kinetics during exercise.

Lactic acid is a naturally occurring product of anaerobic metabolism. It is not bad or an undesirable substance; in fact, it is useful as an energy source, as a temporary pyruvate reservoir and as a means of preventing the body's pH from falling to dangerously low levels.

The lactate threshold is defined as the highest metabolic rate obtainable while keeping the blood lactate at a steady state. At this level of intensity, the body is clearing lactate as rapidly as it is produced. Should the intensity increase beyond this critical point, lactate production exceeds removal rate causing a rapid increase in lactate accumulation.

For athletes to reach their greatest endurance potential, they must train their bodies to process lactate efficiently. This "fine tuning" allows them to compete at the highest possible intensity while maintaining relatively low concentrations of lactic acid.

Introduction

Highly trained endurance athletes in action are a thing of beauty. Whether running, skiing, cycling, swimming or skating, their muscles contract repetitively in a fashion that permits graceful movement for extended periods, sometimes hours. Because their movements appear to require so little effort, the delicate metabolic balance athletes must maintain to support their muscular efforts is often overlooked. Each has selected the precise pace that permits completion of the race in the fastest possible time.

The energy permitting the working muscle to keep on pace is dependent upon its oxygen supply. The muscles of an athlete who miscalculates and selects a pace requiring too great an oxygen supply will be flooded with the "dreaded" chemical athletes know as lactic acid. This spontaneous release of lactic acid will almost certainly cause a less than desired performance.

The purpose of this paper is to review the delicate metabolic balance an endurance athlete must maintain to achieve a desirable performance. The optimum pace is ultimately determined by the athlete's ability to deliver large volumes of oxygen to the working muscles while simultaneously preventing excessive lactate accumulations in the tissues and blood. This highest metabolic rate obtainable, while keeping the blood lactate at a steady state, is referred to as the lactate threshold (LT).

*John M. Kelly, D.P.E., is a professor in the Department of Physical Education, Recreation, and Sport Science and Director of the Human Performance Laboratory at St. Cloud State University. *This paper is a invited review based on Dr. Kelly's remarks to the Sports Medicine Symposium at the Minnesota Academy of Science annual meeting, April 1988.*

Maximum Oxygen Uptake ($\dot{V}O_2$ max)

Before discussing the role played by the lactate threshold in endurance activities, it is appropriate to consider the oxygen transport capabilities of the athlete, since oxygen remains the bottom line in determining how much energy will be available to support muscle activity.

The physiologist measures oxygen transport ability with the maximal oxygen uptake ($\dot{V}O_2$ max) test. Until recently it has been considered the best single indicator of physical fitness. Simply stated, $\dot{V}O_2$ max is the greatest amount of oxygen consumed during an all-out effort. $\dot{V}O_2$ max is measured with an 8-15 minute test to exhaustion, generally on a treadmill or bicycle ergometer. Test results are expressed in liters$\cdot$min^{-1} (absolute) or in ml$\cdot$kg$^{-1}\cdot$min^{-1} (relative to body mass). Average $\dot{V}O_2$ max values for college females and males are 40 and 47 ml$\cdot$kg$^{-1}\cdot$min^{-1}, respectively. Aging and deconditioning cause an approximate 10 percent decline in $\dot{V}O_2$ max per decade from age twenty-five onward. While apparently an inevitable result of aging, this downward trend can be reduced by 50 percent if modest amounts of physical training are continued throughout one's life (1).

As might be expected, elite endurance athletes have higher than average $\dot{V}O_2$ max values, ranging from 70-94 ml$\cdot$kg$^{-1}\cdot$min^{-1} for males and from 60-75 ml$\cdot$kg$^{-1}\cdot$min^{-1} for females.

Although endurance training can improve an individual's $\dot{V}O_2$ max, these gains are limited to 10-30 percent and are determined by initial fitness levels. The highly trained athlete experiences little or no improvement while the sedentary person stands to benefit substantially following only a few months of exercise.

The cardiorespiratory system dictates the size of the $\dot{V}O_2$ max by its ability to deliver oxygen to the cells. Ultimately, this

is decided by the rate at which the heart can deliver blood to the working muscles; the more blood pumped, the more oxygen transported to the cells. Because this ability is so greatly influenced by the heart's pumping action, $\dot{V}O_2$ max is, to a large extent, determined by heredity.

Recently, however, exercise physiologists have been forced to reevaluate the predictive power of $\dot{V}O_2$ max in endurance events. Since the person with the highest $\dot{V}O_2$ max frequently fails to win the race, other factors must also be important predictors of performance.

Lactate Threshold (LT)

One of the most promising new methods of evaluating endurance potential revolves around the kinetics of lactic acid formation and clearance during exercise. The ability to operate at a high percentage of one's $\dot{V}O_2$ max while maintaining relatively low concentrations of lactic acid within the cells and blood appears to be a prerequisite for success in endurance activities; this is known as the lactate threshold (LT). Highly trained athletes are able to sustain efforts requiring 90 percent of their $\dot{V}O_2$ max while maintaining relatively low concentrations of lactate. Conversely, sedentary individuals experience overwhelming lactate surges at intensities as low as 50 percent of their $\dot{V}O_2$ max (1).

Besides LT there are several other terms in use describing essentially the same phenomenon. Some of these names are: anaerobic threshold, lactate steady state and the onset of blood lactate accumulation (OBLA). Furthermore, several methods are in use to identify this point of rapid lactate accumulation. Two of the more common procedures use pulmonary ventilation and heart rate for this purpose (2,3). However, the actual measurement of blood lactate levels is the most accurate way of determining the LT.

Lactic Acid

Before continuing the LT discussion, a few words about lactic acid are appropriate. It was identified as a possible fatigue-causing agent as early as 1907 (4) and has received the intense interest of coaches and athletes since that time. Lactic acid is a product of glycogenolysis and glycolysis; the anaerobic breakdown of glycogen and glucose, respectively. Figure 1 presents an abbreviated diagram of the 10 coupled reactions involved in glycolysis.

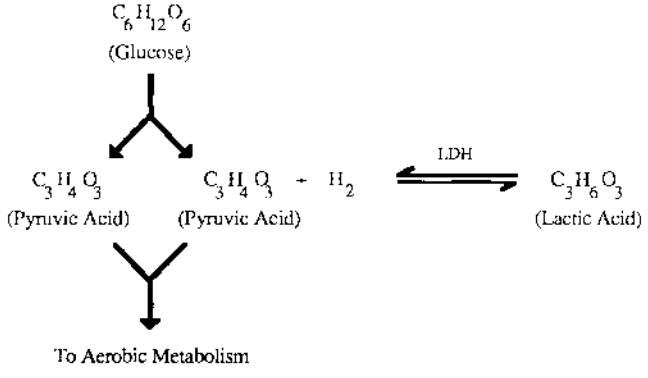

Figure 1. Glycolysis.

Like all acids, lactic acid readily dissociates, forming a positively charged ion (H^+) and a negatively charged lactate ion (lactic acid $\rightarrow H^+$ + lactate -). Under normal physiological conditions, lactic acid dissociates almost completely estab lishing a ratio of 3,000 to 1. For this reason, the terms lactic acid and lactate are usually used interchangeably.

Until recently it was believed that lactic acid was formed only during times of hypoxia. While lack of oxygen will cause more rapid lactic acid formation, we have learned that it can also be produced in a muscle that is fully oxygenated. Skeletal muscle cells, especially the fast twitch fibers, are able to break down glucose to pyruvate far more rapidly than their mitochondria are able to accept pyruvate for aerobic metabolism. In order to allow this very rapid energy transforming system to continue the necessary production of ATP for muscle contraction, the excess pyruvic acid must be removed. With the aid of the lactate dehydrogenous enzyme (LDH), pyruvate is converted to lactic acid (Figure 1). It is estimated that the rate of glycolysis can increase nearly 1,000 fold from rest to maximal effort; a rate far too rapid for the aerobic system to match (5). As the lactate concentration within a muscle cell increases, it diffuses into the plasma where it is transported to tissues that can remove it. Eventually, however, if the high rate of lactate production continues, the body's buffering systems, tissues and enzymes will be overwhelmed, helping to hasten fatigue.

One last point needs to be made. The primary substrates for lactate formation are glycogen and glucose. Consequently, when they are depleted, the body's anaerobic energy system is greatly impaired and little lactate can be generated. For this reason, runners at the end of a marathon race have lactate concentrations close to resting levels and must rely heavily on fatty acids for fuel.

Historical Overview

It is always interesting to discover that something you thought was new really had its foundations laid at a time earlier in history. The LT, a product of the 1970s and 80s, is an example because it is based on the results of several investigations conducted in the 1920s and 30s. We owe much of our present understanding of lactate metabolism to the pioneering work of such legendary investigators as Hill, Jervell, Owles and Bang (6,7,8,9).

W. Harding Owles, one of the first researchers to recognize that blood lactate concentration remained low during low levels of exertion and began to rise in an exponential fashion as exercise intensity increased, wrote: "...it was found that there was no lactate increase as a result of exercise up to a certain critical level... This critical level corresponded to an oxygen utilization of about 1.8 liters per min. (8)." This observation is one of the first descriptions of the LT as we know it today.

Since that time several investigators have confirmed the lactate-exercise intensity relationship. A hypothetical example of this relationship is seen in Figure 2 where blood lactate levels remain unchanged in early exercise and then increase exponentially as the intensity increases. It should also be noted that oxygen uptake is linearly related to exercise intensity until it reaches a maximum level.

Early investigators interpreted this flat response of lactate to low intensity exercise as an indication that little or no lactate was being produced, concluding that the energy demands were being met aerobically. Recently, several investigators have provided evidence to refute this early

belief. Through direct measurement of lactate production in animal muscle and from tracer studies using isotopes in humans, we have learned that lactate production increases, even with low intensity exercise but is cleared as rapidly as it is formed (10, 11). Thus, lactate concentration is determined not only by its production, but also by its rate of removal.

Fast and Slow Twitch Muscle

To help understand this phenomenon, it is necessary to consider the metabolic characteristics of muscle. Human skeletal muscle cells have been classified into two categories according to their contractile characteristics. Type I, or slow twitch fibers, have high concentrations of mitochondria which enable them to efficiently process energy aerobically. Type II, or fast twitch fibers, have limited numbers of mitochondria, but are rich in anaerobic enzymes and therefore possess excellent power.

As exercise begins at low intensity, some lactate is formed as a result of increased rates of glycolysis even though most of the required energy comes from aerobic sources. However, blood lactate concentration remains unchanged because the lactate is cleared as rapidly as it is formed. As exercise intensity is increased (for example, 150-200 watts in Figure 2), lactate production begins to increase at a faster rate and the removal mechanism, reaching its maximum rate of clearance, struggles to keep pace. The increased lactate release is caused by greater fast twitch muscle involvement as a result of increased exercise intensity. Finally, lactate clearance processes become overwhelmed because lactate formation has increased exponentially as more fast twitch fibers are called upon to provide contractile force (Figure 2, 200 to 300 watts).

Fatigue

High lactate levels are indirectly responsible for fatigue during intense exercise bouts lasting 10 minutes or less. Lactate levels from 15-30 mmol·l^{-1} of blood have been reported in humans. The culprit in this case is not lactate but its tendency, along with other acids, to lower muscle pH from 7.1 at rest to 6.2 at exhaustion. Not only does increased acidity interfere with the muscle's contractile mechanisms, but it also inhibits a key glycolytic enzyme (phosphofructokinase) thereby limiting anaerobic energy formation (12). On the positive side, this negative feedback system provides a useful energy source while at the same time preventing us from poisoning ourselves with dangerously low pH levels.

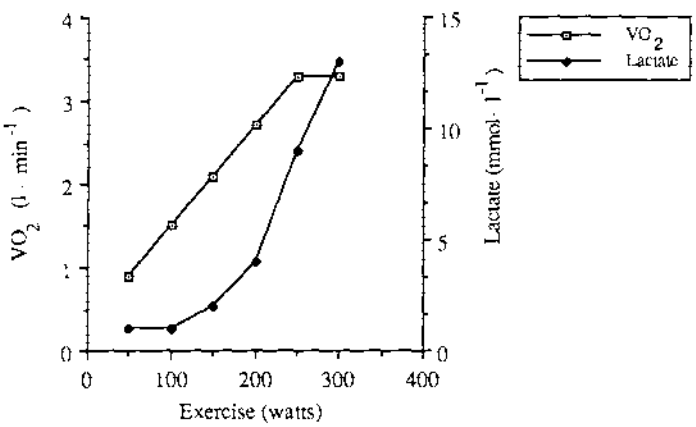

Figure 2. Oxygen uptake and blood lactate responses to a progressive bicycle ergometer test.

Lactate Clearance

As was discussed, blood lactate concentration at any given moment is determined by its rate of production and clearance. Even at rest, lactate is constantly formed in tissues such as the intestines, red blood cells and skeletal muscle. Between 50 and 75 percent of this lactate is oxidized by the heart, skeletal muscle, kidneys and liver. Only 20-25 percent is reconverted to glucose by the liver and kidneys. It is important to recognize that lactate is a valuable energy source and not simply an undesirable waste product (13).

When lactate diffuses from the muscle cell, it may move into another nearby cell that is better equipped for aerobic metabolism, or it may pass directly into the blood. As the lactate is transported, it may be picked up by organs such as the heart, kidneys, or skeletal muscle and burned for energy, or it may be converted back to glucose by the liver.

During resting conditions, blood lactate levels remain at about 1 mmol·l^{-1} of blood. As exercise intensity increases, lactate is formed in greater quantities but clearance mechanisms keep pace and lactate concentrations remain unchanged. Further increases in intensity cause even greater release of lactate into the blood which eventually causes a rise in the blood lactate to about 4 mmol·l^{-1}. Research has suggested that when lactate reaches this level the body is clearing lactate as rapidly as possible (14). Even harder exercise causes the exponential rise in lactate because production is far greater than removal. Decreased intensity will cause lactate levels to fall, indicating a more rapid clearance than production. Interestingly, at any given time lactate concentrations in the blood lag far behind those in the muscle. For this reason, blood lactate often reaches its peak value in recovery, 3 to 5 minutes after the exercise session.

Lactate Threshold Measurement

To evaluate the accuracy of the estimated LT determination, the athlete should complete a 30-minute treadmill run at the threshold pace. In the example presented in Figure 3, it is obvious that the lactate threshold occurred close to the 5:45 minutes per mile pace. When the speed was faster (5:30 pace), lactate accumulated faster than it could be removed, resulting in fatigue. When the treadmill speed was decreased (6:00 min. pace), the lactate rose initially and then gradually returned to a near resting level.

Significance of the Lactate Threshold

It is an obvious advantage for an endurance athlete to compete at the highest possible percentage of his/her $\dot{V}O_2$ max during competition. For example, the data seen in Figure 3 indicate that a 5:45 minute per mile pace could be maintained while keeping steady lactate concentrations. To run faster would be foolish since it would result in fatigue; to run slower would result in a poor competitive effort. An athlete can run at LT for slightly over an hour before fatiguing. Thus, marathon runners must select a pace slightly under lactate steady state since their event requires an effort of over two hours. Ten kilometer runners typically run at paces slightly above lactate threshold.

Jan Ettle, a nationally ranked marathon runner, has been tested in our laboratory on several occasions (Figure 4). Results from two of the tests are depicted in Figure 5.

In both of these tests, she maintained a 5:45 minute per mile pace for 30 minutes. On the most recent test (November 3, 1988), Jan was recovering from an injury sustained while competing in the Olympic trials. It can be observed that during this run she struggled as her lactate was above steady state. From this test her steady state pace was projected to be 6:00 minutes per mile. Two weeks later she participated in the Tokyo marathon and finished in two hours, 41 minutes and 28 seconds, a pace of 6:09 minutes per mile; nine seconds per mile slower than her LT pace. Two additional points of interest should be noted: (1) Jan's lower than average LT (3 $mmol \cdot l^{-1}$) is typical for well-conditioned endurance athletes, indicating excellent lactate removal ability; (2) Her $\dot{V}O_2$ was about the same on each occasion despite the significant difference in blood lactate.

Clearly, Jan's performance in Tokyo was not her best; it was 7.8 minutes slower than her fastest time. Her inability to process lactate as well as she had in the past was an important contributor to her slower time. Consequently, Jan will attempt to improve her ability to process lactate before her next marathon sometime in the spring on 1989. She will not be alone, however, because a major focus of every endurance athlete's training program is directed toward improving their lactate kinetics.

Endurance Training

Three major points, as they relate to lactate formation, must be considered when planning training programs for endurance athletes. First, it is essential that $\dot{V}O_2$ max is at the highest possible level. This increased ability to deliver oxygen to the working muscle permits greater fat metabolism and less carbohydrate use with correspondingly lower lactate production. In essence, more of the load is carried by the mitochondrial-rich slow twitch fibers. At the same time a greater $\dot{V}O_2$ max will permit increased circulation and

Figure 4. Jan Ettle during a lactate threshold test at the St. Cloud State University Human Performance Laboratory

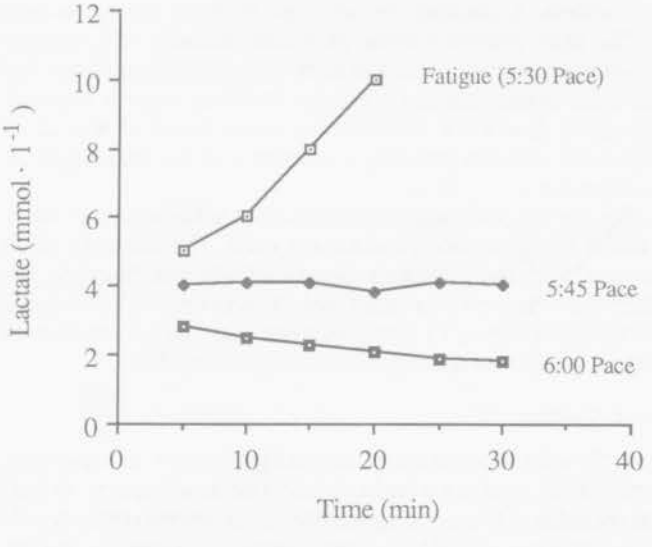

Figure 3. Blood lactate responses to three 30 minute treadmill tests at different speeds.

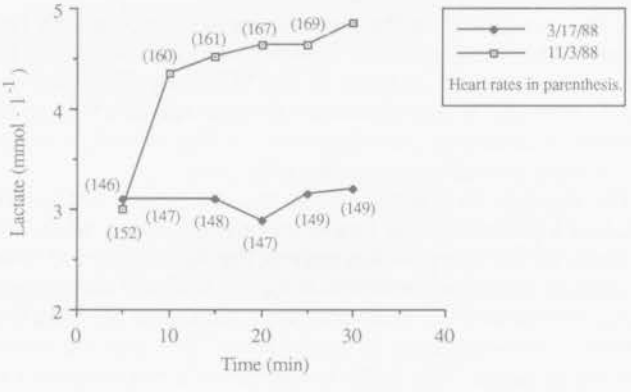

Figure 5. Jan Ettle's response to a 5:45 minute per mile pace on two occasions at different conditioning levels.

clearance of existing lactate. Most authorities agree that $\dot{V}O_2$ max is best improved with high intensity exercise (above lactate threshold) such as interval training.

The second concern is to increase the ability of the muscle cell to utilize oxygen. This includes increasing capillary and mitochondrial density. More capillaries enhance oxygen delivery while increased mitochondria allow for more efficient metabolism; both contributing to increased fat metabolism and less lactate formation. Longer, slower training appears to provide the stimulus for these improvements. Large amounts of training at intensities below the LT are essential. Some researchers are recommending one 30 minute workout every week to 10 days at the LT intensity, reasoning that at this pace lactate clearance is at its very highest level (15). This training is referred to as "tempo training" by most coaches and athletes. The purpose of tempo training is to stress the body's maximum ability to clear lactate during a long workout without causing total fatigue. It is presumed that such training will improve the body's capacity for lactate removal.

The last point to mention is the necessity for athletes to include adequate carbohydrates within their diet. Carbohydrate is the preferred fuel for exercise and the body's reserve must remain at high levels for optimum performance. An athlete with low carbohydrate reserves will experience a much lower exercise capacity in spite of low lactate concentrations. It is almost paradoxical that carbohydrate is the precursor of lactate, but low reserves not only cause low lactate concentrations, but also fatigue.

$\dot{V}O_2$ max vs. Lactate Threshold

By now it is clear that the ultimate goal of endurance training is to elevate $\dot{V}O_2$ max and LT to their highest possible levels. A hypothetical example in Figure 6 is used to illustrate the effects of a six month training program on an average college-aged woman.

Initially, she has an average $\dot{V}O_2$ max (40 ml•kg^{-1}•min^{-1}) and LT (60 percent of $\dot{V}O_2$ max). This means that she would be able to run for approximately one hour at 60 percent of her $\dot{V}O_2$ max (24 ml•kg^{-1}•min^{-1}) before fatiguing. This $\dot{V}O_2$ would provide enough energy for her to sustain a 13.4 minutes per mile pace for about one hour.

At the conclusion of the training period, her $\dot{V}O_2$ max has improved to 50 ml•kg^{-1}•min^{-1} (20 percent gain) while her LT has increased to 80 percent of her $\dot{V}O_2$ max (33.3 percent gain). Now she is able to sustain an hour run at a $\dot{V}O_2$ of 80 percent of maximum (40 ml•kg^{-1}min^{-1}). This would allow her to maintain an eight minute per mile pace.

The example in Figure 6 shows that the subject's $\dot{V}O_2$ max reached a plateau by the fourth month of training while the LT continued to improve throughout the training period. This is a typical response, demonstrating the limits of the oxygen transport system ($\dot{V}O_2$ max) and at the same time showing the gradual improvement in lactate kinetics (LT) over the entire training program. This helps to explain why improvement is possible long after the upper limits of $\dot{V}O_2$ max are reached. Top endurance athletes often reach their peak performance in their late 20s and 30s, long after their $\dot{V}O_2$ max has plateaued. It is not uncommon for these great endurance athletes to sustain $\dot{V}O_2$'s that are 90 percent of the $\dot{V}O_2$ max during long periods of exertion — even up to marathon distance.

$\dot{V}O_2$ max has been called the endurance I.Q., meaning that it is a measurement of one's endurance potential. For this reason it is strongly influenced by genetics and to a great

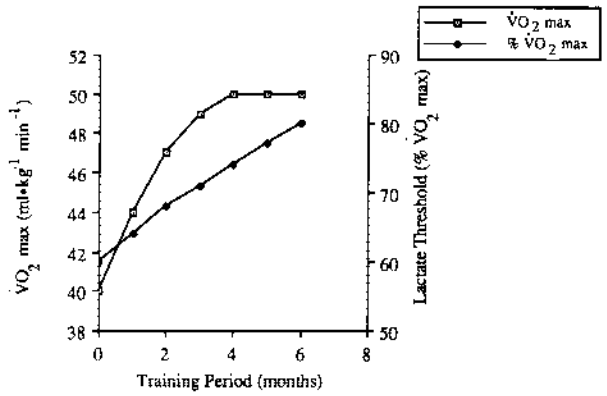

Figure 6. The effects of physical training on $\dot{V}O_2$ max and lactate threshold.

extent unrelated to training. For example, not unlike the I.Q. test, a person can have a high $\dot{V}O_2$ max without training. The reverse can also be true, a person may have a below average $\dot{V}O_2$ max even though he/she trains regularly. Not so with the LT; one must train regularly to achieve above average results. Just as in academic settings where some students have great potential but fail to achieve because of laziness, some athletes have high endurance potential but fail to develop it. For these reasons, we are beginning to recognize the LT is a truer indicator of training status than the $\dot{V}O_2$ max.

Using the Lactate Threshold

The LT has become a valuable marker of endurance fitness and provides an excellent way to evaluate training status. An athlete may periodically (every month or two) have his/her blood lactate monitored during a 30 minute treadmill run at a prescribed pace. Technology has improved so that a drop of blood from the fingertip can be analyzed within three minutes, providing almost immediate feedback to the coach and athlete. A decline, or no improvement in the lactate profile from previous tests, provides valuable information indicating a problem with the training program or athlete. For example, overtraining, a common problem among athletes, will often lower the LT. On the other hand, if the LT is improved, one can feel very confident that the training is on the right track.

Monitoring and establishing training schedules are other ways in which lactate profiles are used. The athlete's LT is matched with the corresponding heart rate. For example, Jan Ettle's LT occurred at a heart rate of between 145-150 beats per minute (Figure 5). Her coach can use this information in planning and monitoring her training schedule.

Conclusion

Lactic acid is a naturally occurring product of anaerobic metabolism. It is not a bad or undesirable substance. In fact, it is useful as: (1) an energy source; (2) a place to temporarily store pyruvate, allowing a continuation of anaerobic energy reactions; and (3) a link in a negative feedback mechanism which prevents the body's pH from falling to dangerously low levels.

For athletes to reach their greatest endurance potential, they must train their bodies to process lactate efficiently. This "fine tuning" allows them to compete at the highest possible intensity while maintaining relatively low concentrations of lactic acid.

Based upon current knowledge, the most important adjustment the body must make to achieve this objective is to increase its ability for rapid lactate clearance. This change requires an improved oxygen transport system as well as an enhancement in the skeletal muscle's capacity to process oxygen, specifically an increase in mitochondria.

Scientists have been searching for clues to the mysteries surrounding lactate metabolism and its effects on fatigue for the past 65 years. Not all of the questions have been answered, but recent findings have given us the basis for a clearer understanding of these phenomena. One thing we can be sure of: the success of an endurance athlete is closely related to his/her ability to process lactic acid.

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