Anaerobic Threshold: The Concept and Methods of Measurement

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Abstract/Resume

The anaerobic threshold (AnT) is defined as the highest sustained intensity of exercise for which measurement of oxygen uptake can account for the entire energy requirement. At the AnT, the rate at which lactate appears in the blood will be equal to the rate of its disappearance. Although inadequate oxygen delivery may facilitate lactic acid production, there is no evidence that lactic acid production above the AnT results from inadequate oxygen delivery. There are many reasons for trying to quantify this intensity of exercise, including assessment of cardiovascular or pulmonary health, evaluation of training programs, and categorization of the intensity of exercise as mild, moderate, or intense. Several tests have been developed to determine the intensity of exercise associated with AnT: maximal lactate steady state, lactate minimum test, lactate threshold, OBLA, individual anaerobic threshold, and ventilatory threshold. Each approach permits an estimate of the intensity of exercise associated with AnT, but also has consistent and predictable error depending on protocol and the criteria used to identify the appropriate intensity of exercise. These tests are valuable, but when used to predict AnT, the term that describes the approach taken should be used to refer to the intensity that has been identified, rather than to refer to this intensity as the AnT.

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Le seuil anaérobie (AnT) correspond au plus haut niveau d'intensité d'effort physique dont l'énergie provient exclusivement de métabolisme aérobie. Au seuil anaérobie, la quantité de lactate diffusant dans le sang est égale à la quantité en sortant. Bien qu'un transport d'oxygène inadéquat puisse accroître la production d'acide lactique, il n'y a pas d'indication solide voulant que la production d'acide lactique au-delà du seuil anaérobie soit due à un transport d'oxygène déficient. Nombreux sont les arguments militant en faveur de la quantification de l'intensité d'exercice au seuil anaérobie, notamment: évaluation de la santé cardiovasculaire ou pulmonaire et des programmes d'entraînement ainsi que la catégorisation de l'intensité de l'effort soit léger, modéré, et intense. Des tests ont été mis au point pour indiquer l'intensité d'exercice au seuil anaérobie: maximum de lactate en régime stable, minimum de lactate, seuil de lactate, SASL (OBLA), seuil anaérobie individuel, et seuil ventilatoire. Chacune de ces approches donne une estimation de l'intensité d'exercice au seuil anaérobie, mais l'erreur associée varie selon le protocole d'évaluation et les critères d'identification de l'intensité d'exercice. Ces tests sont utiles, mais quand ils servent à établir le seuil anaérobie, on devrait préciser le nom de l'approche utilisée pour identifier l'intensité d'effort au seuil anaérobie plutôt que d'associer cette intensité au seuil anaérobie.

Introduction

Few concepts in the field of exercise science have generated such debate as that of anaerobic threshold. Disagreement among researchers stems not only from the absence of methodological standardization but also from a lack of consensus on the theoretical basis of the concept itself. Efforts to accurately describe a threshold intensity have resulted in an immense pool of scientific data. Yet the issue remains an unresolved controversy. One reason for the ongoing controversy is the lack of consensus for the definition of anaerobic threshold and the persistent inappropriate use of the term. It is important to recognize that anaerobic threshold is a concept, and that the definition is a conceptual definition. In contrast, the various ways to detect the intensity of exercise associated with the anaerobic threshold have resulted in a proliferation of terms that are more appropriately given operational definitions. These measurements should not always be equated with anaerobic threshold, since there are clear differences between conceptual and operational definitions. Considering the inconsistency with which these terms are used, readers should interpret a term like anaerobic threshold or lactate threshold from the context of its use.

The purpose of this review is to provide a conceptual definition of anaerobic threshold and related terms, and to discuss the theoretical concept and methods of measurement. An historical perspective on the meaning of anaerobic threshold is presented, with a discussion of likely (and unlikely) mechanisms. This is followed with a brief description of some of the tests that have been proposed as providing an estimate of the anaerobic threshold.

DEFINITIONS

These definitions are generalized and are intended to provide a framework for subsequent discussion. The definitions are elaborated upon later in this paper.

Anaerobic threshold: The term "anaerobic threshold" is defined as an intensity of exercise, involving a large muscle mass, above which measurement of

oxygen uptake cannot account for all of the required energy. Stated in other terms, this is the exercise intensity above which there is a net contribution of energy associated with lactate accumulation.

Maximal lactate steady state: Maximal lactate steady state (MLSS) is defined as "the highest exercise intensity at which blood lactate concentration does not increase beyond the initial transient during constant load exercise" (Tegtbur et al., 1993, p. 620). In other words, the intensity at MLSS represents the highest intensity for which there is an equilibrium between lactate transport into the blood and lactate removal from the blood (Heck et al., 1985).

Lactate minimum speed: The lactate minimum speed is the speed of locomotion at which blood lactate reaches a minimal value during an incremental exercise test (increments in speed of locomotion), which is initiated in the presence of lactic acidosis.

Lactate threshold: Lactate threshold is the exercise intensity that is associated with a substantial increase in blood lactate during an incremental exercise test. Various specific criteria are used to identify this increase, and some of these have their own special name.

Onset of blood lactate accumulation: Onset of blood lactate accumulation, or OBLA, is defined as the intensity of exercise at which blood lactate concentration reaches 4 mM during an incremental exercise test (Sjodin et al., 1981).

Individual anaerobic threshold: The individual anaerobic threshold (IAT) is a special version of a lactate threshold. IAT is defined as the intensity of exercise identified by a line drawn from a recovery lactate concentration, tangent to the lactate concentration observed during an incremental test (Stegmann et al., 1981).

Ventilatory threshold: Ventilatory threshold is defined as the exercise intensity at which the increase in ventilation becomes disproportional to the increase in power output or speed of locomotion during an incremental exercise test.

What Is the Anaerobic Threshold?

The definition of anaerobic threshold relates to exercise involving a large muscle mass. It is recognized that within a single muscle, glycolysis can occur, resulting in net output of lactate even at rest (Gladden, 2000; Stainsby et al., 1984; 1991). Under these circumstances, measurement of oxygen uptake could not account for all the energy use by the muscle. Therefore the concept of anaerobic threshold must apply only to the intact whole body when a substantial portion of the muscle mass is active. To understand the concept of anaerobic threshold, it is important to understand the metabolic systems that provide energy during exercise.

Technically speaking, if "anaerobic metabolism" is defined as replenishment of ATP without the use of oxygen, then substrate level phosphorylation would be considered anaerobic. This would include reactions associated with creatine kinase, glycolysis, and the Krebs cycle. Since measurement of oxygen uptake permits accounting for some of these steps, the presence of glycolytic activity is not necessarily evidence that the exercise intensity has exceeded the anaerobic threshold.

Typically, pyruvic acid resulting from glycolysis is either incorporated into oxidative metabolism via the Krebs cycle or is converted to lactic acid. The con-

version of pyruvic acid to lactic acid is a valuable step in that cytoplasmic NADH is oxidized. This ensures a continued supply of NAD^+ for glycolysis. Therefore, instead of inhibiting glycolysis, lactic acid formation permits continued glycolysis. Furthermore, it is clear that lactate can be oxidized either within the muscle fiber in which it is produced (Brooks, 2000; Brooks et al., 1991) or in an adjacent fiber or another muscle (Donovan and Pagliassotti, 2000). In this case, measurement of oxygen uptake could account for this glycolytic production of ATP.

It is the accumulation of lactate or other glycolytic intermediates, not simply evidence of lactate production, which should be considered to represent the metabolic rate above anaerobic threshold. This accumulation could be in muscle tissue and/or in the blood. Accumulation of lactate represents the situation whereby glycolytic production of pyruvic acid and lactic acid exceeds the rate of incorporation of these molecules into the Krebs cycle. It seems reasonable to assume that if lactate is accumulating in the blood while exercise intensity is constant, then the intensity of exercise exceeds the anaerobic threshold, as defined above.

Is There an Anaerobic Threshold?

To address the question of whether or not there is an anaerobic threshold, it is important to consider the fate of lactate in the body (see Donovan and Pagliassotti, 2000). As mentioned above, a single muscle can have a net lactic acid production even at rest. However, it is known that lactate may be taken up and oxidized in another organ or tissue in the body. In defining anaerobic threshold, the ultimate (short-term) fate of the lactate that is released from a muscle must be considered in order to determine whether that lactate represents accumulation. If the lactate (or pyruvate) that makes its way to the blood is subsequently taken up by another muscle or other organ, and oxidized, then it would not accumulate. If on the other hand the lactate that is released from a muscle results in increasing blood lactate concentration, then the measurement of oxygen uptake could not account for the ATP replenishment associated with that lactate formation. Therefore, by definition, when blood lactate concentration increases over a prolonged duration at a given intensity of exercise (power output or speed of locomotion), the intensity would be considered as being above the anaerobic threshold.

When blood lactate concentration is not increasing, the rate of lactate removal from the blood must equal or exceed the rate at which lactate is moving into the blood. If all of the lactate removed from the blood is oxidized, the intensity of exercise would be considered as being at or below the anaerobic threshold. However, lactate can have several pathways of metabolism. Lactate (or pyruvate) can be taken up by the liver and the kidney, and undergo gluconeogenesis. It is possible that oxidative metabolism in the liver or kidney provides the energy needed to transform the lactate and/or pyruvate back to glucose by oxidative metabolism, so this pathway of disposal represents a means by which oxygen uptake can account for the glycolytic formation of ATP. If we accept this argument, then the anaerobic threshold would occur at the highest intensity of exercise for which a steady state for blood lactate can be sustained. This intensity of exercise has also been referred to as the maximal lactate steady state (MLSS).

The only circumstance when MLSS would not be equal to anaerobic thresh-

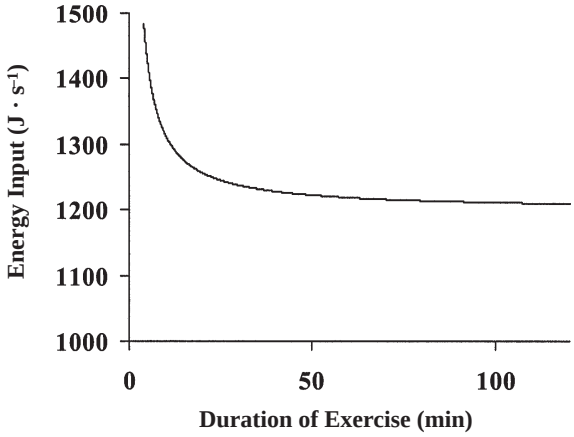

Figure 1. The exercise intensity/duration relationship, for intensities equal to or less than maximal oxygen uptake. As exercise intensity decreases, time to fatigue increases.

old would be if blood lactate concentration could remain constant while lactate accumulates in muscles. Under these circumstances the measurement of oxygen uptake cannot account for glycolytic ATP formation, although the rate of lactate entry into the blood was equal to the rate of lactate removal from the blood. This would probably occur if the volume of active muscle was relatively small. Otherwise it should be considered that MLSS actually represents the intensity of exercise at the anaerobic threshold.

Since the anaerobic threshold refers to an intensity of exercise, it is important to recognize that this intensity is presumably just one point on the intensity/duration relationship. Figure 1 presents a typical intensity/duration relationship. Intensity in this case is expressed as energy input. Alternatively, intensity could be expressed as mechanical power output, or speed of locomotion. This particular depiction of the intensity/duration relationship presents the duration of exercise when intensity is at or below maximal oxygen uptake, and is based on the following: At maximal oxygen uptake, exercise can be sustained for up to about 60 min (Billat et al., 2000); at anaerobic threshold, which typically occurs at 60 to 80% of maximal oxygen uptake, exercise can be sustained for up to about 60 min (Lajoie et al., 2000); at less than the anaerobic threshold, exercise can be sustained for several hours.

Exercise intensity is best quantified by measuring the rate of metabolic energy input while performing a task. This can be done by measuring oxygen uptake when the intensity is below the anaerobic threshold. However, the rate of oxygen uptake is not constant while exercising at an intensity above anaerobic threshold, and by definition, oxygen uptake does not account for all of the energy input above this intensity. When the energy demand for the exercise is near maximal oxygen uptake, there is a steady increase in oxygen uptake while the conditions of the exercise (speed or power) remain constant (Gaesser and Poole, 1996). This steady increase is called the slow component of oxygen uptake.

It is thought that the slow component exists when exercise intensity exceeds the anaerobic threshold (Jones et al., 1999), but the mechanism of this slow increase in oxidative metabolism is not known (MacIntosh et al., 2000). Since oxygen uptake is not constant when a slow component exists, it may be more appropriate to designate the intensity of exercise by power output or speed of locomotion, but this depends on the reason for expressing the intensity of exercise. Heart rate is often used, but this is not the most appropriate or precise means of expressing intensity, due to the occurrence of cardiac drift even below the apparent anaerobic threshold (Lajoie et al., 2000). Furthermore, there is daily variation in heart rate response at a given intensity of exercise (MacIntosh et al., 2002).

A brief history of our understanding of the circumstances of lactate formation and its appearance in the blood will now be presented, with the intent of giving some perspective to the use of the term anaerobic threshold. It is not our purpose here to provide an extensive review of this history but rather to point out some key observations.

Historical Perspective on Lactic Acid Formation

Much of the information presented below on the early recognition of a role for lactic acid formation in skeletal muscle metabolism has been obtained from a very interesting book by Dorothy Needham (1971). The reader is directed to this source for the specific references for this material.

It was recognized as early as 1807 that lactic acid was formed in muscle. Needham (1971) indicates that Berzelius was the first to identify lactate in muscles, and this was in the muscles of hunted stags. Another early scientist who observed lactate in muscle was Claude Bernard, who reported that the amount of lactic acid in muscle was proportional to previous exercise. In the early 1900s, an intensive search to understand the biochemistry of energy metabolism resulted in considerable advances in the understanding of the role of lactic acid and its involvement in providing energy for muscle contraction, but this was not without contradiction and confusion. In 1907, Fletcher and Hopkins made a profound observation which is as true today as it was then: "it is notorious that, quite apart from the question of the oxidative removal of lactic acid—which has not previously we think been examined—there is hardly any important fact concerning the lactic acid formation in muscle which, advanced by one observer, has not been contradicted by some other" (as cited in Needham, 1971, p. 45).

It was reported by Pflüger in 1875 (as cited in Needham, 1971) that muscle contraction could occur in an anaerobic (oxygen-free) environment. The metabolic pathway that can provide energy under these circumstances came to be recognized as glycolysis. Therefore we refer to anaerobic glycolysis now, when lactic acid is formed, whether or not oxygen is present.

This misuse of the term "anaerobic" may be an important factor in the pervasive misunderstanding of the circumstances in which lactic acid formation occurs. The persistent use of the term anaerobic has led to the common belief that the presence of lactic acid in muscle is evidence that oxygen delivery was insufficient to satisfy the demand. A net production of lactic acid has often been interpreted as a symptom of inadequate oxygen delivery (Hill and Lupton, 1923). This is not necessarily the case.

The Cause of Increased Lactic Acid Formation

There is no doubt that when oxygen availability is limited, lactic acid will be formed in muscle, making a net contribution to the provision of energy. However, this is not sufficient rationale to conclude that the presence of lactic acid in muscle means that limited oxygen availability was restricting oxidative metabolism. It is important to consider whether lactic acid can be formed in muscle when adequate oxygen seems to be present. The most striking evidence for this was presented by Jobsis and Stainsby (1968), who 35 years ago showed that the mitochondrial redox state becomes more oxidized when contractions are initiated in the dog gastrocnemius muscle. Graham and Saltin (1989) confirmed that the mitochondrial redox state (NAD^+/NADH) rose in humans during exercise at a time when lactic acid formation was accelerated.

Lactic acid production is known to be accelerated when contractions are initiated (Stainsby et al., 1991). Richardson et al. (1998) and Connett et al. (1984) have shown that oxygen availability is sustained when lactic acid formation is substantial. Recently Hogan (2001) has shown that it is *not* lack of oxygen that stimulates the glycolysis which results in lactic acid formation at the onset of exercise. In Hogan's study it was observed that oxygen content of single skeletal muscle fibers decreases with a time constant similar to the time constant for the increase in oxygen uptake. This observation confirms that the relatively slow increase in oxygen uptake at the start of exercise is not due to limitations in oxygen delivery. Presumably, oxidative metabolism has a high inertia, and phosphocreatine and glycolysis provide the ATP replenishment while oxidative metabolism is accelerated. Glycolysis resulting in the formation of lactic acid should be interpreted as a process occurring without the use of oxygen, not necessarily in the absence of oxygen. It is now recognized that although hypoxia may result in increased formation of lactic acid, absence of oxygen is not a prerequisite for lactic acid formation (see review by Gladden, 1996).

Several factors can promote lactic acid formation in muscle. One of these is accelerated glycogenolysis and glycolysis (Febbraio et al., 1998; Richter et al., 1982; Stainsby, 1986), resulting from increased sympathoadrenal activity. The control mechanism for activation of phosphorylase-b was delineated by Rall et al. (1957). This effect of sympathoadrenal enhancement of lactic acid formation could very well be the primary mechanism for the marked elevation of blood (or plasma) lactate during an incremental test. Mazzeo and Marshall (1989) reported a strong correlation between plasma lactate and epinephrine concentration among runners and cyclists during incremental exercise tests. They also observed no significant difference between an inflection in plasma lactate concentration and an inflection in plasma epinephrine concentration when expressed as a percent of maximal oxygen uptake. This was the case for both cycling and running tests, although the inflections occurred at different relative intensities (i.e., higher for running among runners and higher for cycling among cyclists).

McMorris et al. (2000) also found a significant correlation between power output at lactate threshold and power output at catecholamine thresholds, but they argue that there is not a clear cause and effect (lactate threshold sometimes preceded the catecholamine threshold). In contrast, Dickhuth et al. (1999) found low correlations between catecholamine and lactate thresholds. These discrepancies

between investigators probably relate to the different criteria for identification of the thresholds. This is consistent with the observations of McMorris et al. (2000), who evaluated different criteria for detection of the lactate threshold and found varying correlations with a catecholamine threshold.

Other factors may contribute to accelerated lactic acid formation. Another possible reason for increased lactic acid formation in a muscle is inadequate transfer of reducing equivalents to the mitochondria (Holloszy and Coyle, 1984). Under these circumstances, lactate formation can help to maintain the NAD^+/NADH ratio in the cytoplasm. The lactate-to-pyruvate ratio would be expected to increase under these circumstances, a symptom that Wasserman interprets as indicative of oxygen limitation (Wasserman et al., 1999). See Graham (1991) and Gladden (1996) for further discussion of these metabolic implications.

Blood lactate concentrations can be elevated at rest, even in the presence of adequate oxygen delivery. It is not the presence of lactate in the blood, nor even the presence of a concentration above resting that is important. Rather, the net result of lactate transport into and out of the blood must be considered. At a certain exercise intensity, the rate of lactate production and transport into the blood will exceed the rate of removal from the blood. This could be due to redistribution of blood flow away from lactate removal sites (nonexercising muscle, liver, kidney, heart), or to transformation of some tissue from lactate removal sites to lactate producing sites, as the intensity of exercise increases. This includes recruitment of additional motor units within an active muscle, since some lactate is likely to diffuse between active and inactive muscle cells within a muscle (Karlsson and Jacobs, 1982). As the pool of motor units becomes more active, there are fewer inactive (or only mildly active) muscle fibers available to serve as lactate removal sites. Under these circumstances, lactate will accumulate and measurement of oxygen uptake cannot account for all the energy requirements of the exercise.

Origin of the Concept of a Threshold Intensity

The notion of a "threshold" or intensity of exercise, above which there is accumulation of lactate, also has a long history of scientific investigation. Owles (1930) wanted to quantify lactate in the blood during low intensity exercise and found that when the exercise was mild, the blood lactate concentration did not rise above resting values. However, at intensities well below maximal oxygen uptake, blood lactate was above resting levels. Consistent with the point raised above, Owles interpreted this to indicate that oxygen delivery became insufficient, leading to the formation of lactic acid. This appears to be the first reference to a relevant threshold intensity of exercise. However, it should be noted that Owles measured lactate after 30 min of constant intensity exercise, and when lactate was elevated above the resting level, he inferred that there had been an accumulation of lactate. There was no attempt to determine whether blood lactate concentration was changing at this intensity of exercise, so this accumulation cannot be related to the anaerobic threshold.

There is no question that as exercise intensity increases, there will be a higher concentration of lactate in the blood regardless of the underlying cellular mechanisms. In the 1950s and early 1960s Hollmann and colleagues (see review by Hollmann, 1991) started using the measurement of blood lactate in submaximal

exercise tests to detect a critical intensity of exercise indicative of exercise intolerance in cardiac and pulmonary patients. They assumed that if arterial blood lactate could be maintained at a constant level, then the exercise was "purely aerobic." This could be considered the beginning of a concept of maximal lactate steady state.

The term "anaerobic threshold" was proposed by Wasserman and McIlroy (1964). Similar to Hollmann, they wanted to identify an intensity of exercise that provided a substantial, yet safe, amount of physical stress for patients suffering from cardiovascular disease. Their rationale was that if a submaximal test could reliably detect an objectively determined level of stress, then it would not be necessary to expose these patients to maximal exercise testing. They saw some value in identifying the intensity at which there appeared to be a limitation in the cardiovascular system's ability to deliver oxygen to the working muscles. Wasserman and McIlroy believed that when this occurred there would be a substantial increase in blood lactate concentration, and proposed identifying this intensity of exercise in several ways. They reported that the anaerobic threshold was associated with decreased plasma bicarbonate and pH, as well as increased R (respiratory exchange ratio) and increased ventilatory equivalent for CO_2 ($\dot{V}E/\dot{V}CO_2$). Within the scope of their initial work, little consideration was given to the notion that these three events may not occur in synchrony. This problem was exacerbated by the selection of the term "anaerobic threshold" to designate this intensity of exercise—a term that has instigated much debate and controversy. Wasserman still maintains that accumulation of lactate in the blood is a symptom of inadequate oxygen delivery (Wasserman et al., 1999).

Wasserman and McIlroy used an incremental test to identify the anaerobic threshold. With this type of test, a steady state of lactate transport into and out of the blood would not be established. However, there were several compelling reasons to use an incremental test, and the utility of this approach is not reduced by the disagreements over a potential underlying mechanism for the increased lactic acid formation. Indeed, although some symptoms associated with this threshold intensity of exercise are commonly seen in healthy subjects and even in endurance athletes, it is not certain that cardiac patients are not limited by oxygen delivery at the intensity that is associated with an accelerated accumulation of lactate in the blood. The disagreements over mechanism should not detract from the value of an incremental test to identify an objectively determined intensity of exercise associated with metabolic stress.

Discussion of the cellular mechanisms associated with lactic acid formation and accumulation of lactate in the blood will probably continue to be debated for many years. However, it can be agreed that there is an intensity of exercise above which lactic acid will accumulate in the blood, and several tests have been developed to detect this intensity. It can also be agreed that detection of this intensity is an important predictor of capability for endurance exercise, a fact that was not considered when the concept was first proposed.

It is too late to suggest changing the name of the anaerobic threshold. The use of the term is pervasive, not only in the scientific and clinical literature but also by coaches, athletes, and people who exercise regularly. However, the various ways of detecting an intensity of exercise, above which measurement of oxygen uptake cannot account for all of the energy use, provide disparate results. For this reason, it should be acknowledged that these methods provide only an estimate or

approximation of the anaerobic threshold, and it is strongly recommended that terms with appropriate operational definitions be used in place of the term anaerobic threshold. These alternatives are presented below, but first, the rationale for undertaking this measurement is provided.

Why Quantify the Anaerobic Threshold?

As noted, the initial purpose for estimating the anaerobic threshold was to assess exercise capacity in cardiac patients (Wasserman and McIlroy, 1964). Clinical assessment or approximation of the anaerobic threshold is also useful in respiratory disease (Hollmann, 1991). Tests for the detection of anaerobic threshold have also gained widespread use in athletic populations (Beneke, 1995; Billat, 1996; Jenkins and Quigley, 1990; Rusko, 1992; Sjodin et al., 1982). Focus has shifted away from maximal oxygen uptake ($\dot{V}O_2max$) as a predictor of success in endurance performance, because studies have shown poor correlations between $\dot{V}O_2max$ and performance results when athletes with similar $\dot{V}O_2max$ values are compared (Costill et al., 1973; Hagberg and Coyle, 1983). In addition, endurance performance of trained athletes continues to improve even after $\dot{V}O_2max$ levels have ceased to improve with further training. For example, despite similar $\dot{V}O_2max$ values between junior-age and adult elite runners, the younger athletes were unable to perform at the same level (Murase et al., 1981).

For middle- and long-duration exercise, $\dot{V}O_2max$ may not be the best predictor of endurance capability. It has been realized that athletes who can utilize a larger fraction of their $\dot{V}O_2max$ for the duration of an endurance event will perform better than those who are physiologically limited to completing the event at a lower intensity. It has been demonstrated that various techniques which purport to measure anaerobic threshold provide a good estimate of the fraction of $\dot{V}O_2max$ that can be sustained in endurance exercise (Bassett and Howley, 2000; Coyle et al., 1988; Kindermann et al., 1979). Consequently, submaximal performance indicators, most of which claim to measure some type of "threshold," have gained widespread utility.

A cautionary note is presented here. It is true that the intensity of exercise (oxygen uptake, power output, velocity of locomotion) at some measured threshold (lactate threshold, OBLA, ventilatory threshold, etc.) may provide an accurate prediction of performance in endurance events. However, a high $\dot{V}O_2max$ is still a prerequisite for elite caliber performance in such events.

The concept of anaerobic threshold is also commonly referred to in training programs. Measurement of the anaerobic threshold provides a benchmark intensity around which training programs can be designed. Exercise performed at an intensity around the anaerobic threshold would be considered moderate, while exercise below this intensity would be mild. When the intensity of exercise substantially exceeds the anaerobic threshold (i.e., approaches $\dot{V}O_2max$), it would be considered intense. There are differences in the adaptations that occur due to training at various intensities, and the most appropriate intensity of training depends on the goal of the program. It is beyond the scope of this review to further evaluate the consequences of training at these specific intensities.

It is recognized that there are several reasons for identifying the intensity of exercise associated with the anaerobic threshold, and several methods have been

proposed for these purposes. To be useful, the method must be reproducible and must identify the threshold with some accuracy. The method must also be objective. Evaluation of the advantages of such tests should include practical considerations for the subjects, including time commitment, invasiveness, and cost. Several approaches are described below.

How the Anaerobic Threshold is Detected

An operational definition relates to the manner in which a measurement is obtained. For example, if the intensity of exercise associated with anaerobic threshold is identified by determination of the intensity at which blood lactate remains at a steady state, then the term "maximal lactate steady state" is a more appropriate manner of referring to that intensity than to say it is the anaerobic threshold. Maximal lactate steady state is defined operationally by the method of obtaining this measure. Definitions and explanations for MLSS and several other terms that should be operationally defined are presented below.

MAXIMAL LACTATE STEADY STATE

Maximal lactate steady state (MLSS) is defined as "the highest exercise intensity at which blood lactate concentration does not increase beyond the initial transient during constant load exercise" (Tegtbur et al., 1993, p. 620). In other words, the intensity at MLSS represents a point of equilibrium between lactate transport into the blood and lactate removal from the blood (Heck et al., 1985). Under these circumstances lactate is not accumulating, measurement of oxygen uptake can account for the energy requirement of the exercise, and exercise time to exhaustion is relatively long. As previously noted, MLSS is equivalent to the anaerobic threshold, as long as there is no progressive accumulation of lactate or other glycolytic intermediates in the muscles.

Although it is unclear as to where the term "maximal lactate steady state" originated, the term "maximal steady state" was used by Londeree and Ames (1975). Their work examined the ability of several maximal steady-state criterion measures to predict level of conditioning. Differences were observed in heart rate and oxygen uptake at blood lactate concentrations of 2.2 and 4.4 mM between groups with varying levels of conditioning. No mention was made of a maximal lactate steady state; however, the exercise intensity at which blood lactate increased from 10 to 15 min of a constant-intensity treadmill test was identified. This intensity was considered to be that at which glycolysis, leading to the formation of lactic acid, began to make a net metabolic contribution. The concept and terminology was explored more extensively by Stegmann and colleagues (Stegmann et al., 1981; Stegmann and Kindermann, 1982) and Heck et al. (1985). More recently, the work of Tegtbur et al. (1993) seemingly reintroduced the concept of MLSS as a valid parameter for athletic testing and training.

The only valid method for measuring MLSS involves blood sampling during multiple sessions of constant-intensity exercise over a range of intensities. The constant-intensity tests should last at least 20 min (Aunola and Rusko, 1992), but tests lasting 30 min or longer have been used more commonly (Beneke, 1995; Beneke and von Duvillard, 1996; Jones and Doust, 1998; Swensen et al., 1999). In

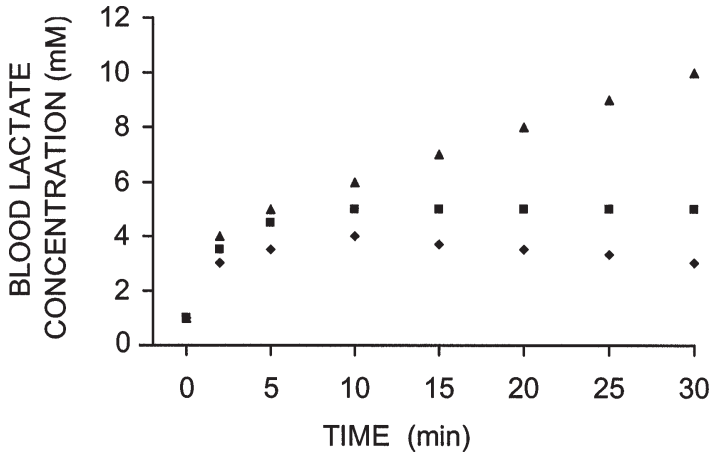

Figure 2. Blood lactate concentration over time for three exercise conditions relative to maximal lactate steady state (MLSS): below MLSS (diamonds), at MLSS (squares), above MLSS (triangles).

theory, the range of selected intensities should include the intensity corresponding to MLSS, in addition to an intensity slightly above it. At MLSS, an initial increase in blood lactate concentration will occur, followed by a steady-state condition for blood lactate. The curve depicting blood lactate over time that is generated at exercise intensities below the MLSS will also show an initial increase, but this will be followed by a gradual decrease in blood lactate concentration. Above the MLSS, blood lactate levels are expected to rise steadily throughout the exercise session (see Figure 2).

The commonly accepted criterion for achieving MLSS is the highest intensity of exercise for which there is a change in blood lactate concentration of no more than 1.0 mM during the final 20 min of constant-intensity exercise lasting at least 30 min (Carter et al., 1999; Heck et al., 1985; Jones and Doust, 1998; Swensen et al., 1999). However, more stringent criteria have been used, such as changes in blood lactate concentration of no more than 0.2 to 0.5 mM (Aunola and Rusko, 1992; Haverty et al., 1988).

The increment in exercise intensity that is needed to accurately reflect MLSS has not been established. Considering the substantial change in endurance that is expected for a small change in intensity of exercise at the anaerobic threshold (see Figure 1), the increments between two constant-intensity exercise bouts should be very small. We have recently observed that an increase in cycling speed of just 0.9 $km \cdot hr^{-1}$, or approximately 2.5%, gives an increase in plasma lactate concentration of 0.7 mM during the last 20 min of a 30-min bout of exercise, while at the lower intensity, plasma lactate was unchanged (MacIntosh et al., 2002). Commonly, researchers involved in the study of MLSS use step increases in intensity of 4 to 5%. The precision of the estimate of MLSS depends on the size of increment in intensity between tests. Essentially a series of tests will yield an intensity that is clearly

above MLSS (blood lactate increased during the final 20 min of a 30-min test) and an intensity just less than this, which will be at or below the MLSS.

Narrowing the intensity range over which trials must be conducted is one of the challenges in devising a strategy to determine MLSS in as few trials as possible. Investigators have come up with various preliminary tests that permit estimation of a starting point that should be close to MLSS. These preliminary tests typically predict an apparent anaerobic threshold, and a series of constant-speed trials would then be conducted to establish the actual intensity at which the blood lactate remains in steady state.

A number of other tests have been designed in order to predict MLSS, rather than using direct measurement. Often these methods are based on the average response to endurance exercise, such as heart rate, velocity, or time trial duration. Foster et al. (1995) designed a protocol to predict MLSS in speed skaters by calculating the relative velocities and heart rates associated with constant blood lactate concentrations. Swensen et al. (1999) expanded upon Foster's work and applied it to cycling, using a windload simulator to determine what percentage of 5-km time-trial velocity corresponded to MLSS. Hoogeveen et al. (1997) had elite cyclists and triathletes complete a 40-km time trial, which they deemed as representing MLSS since heart rate and lap times remained constant throughout the test and a steady-state blood lactate response was observed.

The problem with these approaches is that average physiological responses are not uniformly applicable to all individuals. Predictive tests tend to overlook one of the main conceptual advantages of MLSS, which is the fact that it is an individualized measurement, dependent on individual lactate kinetics rather than absolute blood lactate concentrations or percent of maximal heart rate. It should be appreciated that group statistics can result in false confirmation of the validity of a test. If the results of one test are no different from those of another test, this lack of difference can be due to either true agreement or large variability between subjects for a given test. It is not appropriate to use group statistics to validate a technique for estimating MLSS. Individual results are more relevant. This is true for any test to estimate the intensity of exercise close to the anaerobic threshold.

Direct measurement of MLSS, however, is not an attractive approach for the detection of anaerobic threshold. The procedures are too time consuming and always require multiple laboratory visits for confirmation of the measurement. Although the most common methods of MLSS determination employ somewhat lengthy protocols, efforts to streamline the process are the focus of recent research endeavors. In contrast to some of the aforementioned methods, the lactate minimum test seems to be a valid and reliable method of estimating MLSS.

LACTATE MINIMUM SPEED

The lactate minimum speed (LMS) is the speed at which blood lactate reaches a minimal value during the lactate minimum test (an incremental exercise test with increments in speed of locomotion which is initiated in the presence of lactic acidosis). The lactate minimum speed is theoretically representative of the MLSS (Tegtbur et al., 1993).

Tegtbur et al. (1993) hypothesized that MLSS could be predicted using a protocol consisting of two short-duration, high-intensity efforts, followed by an

active recovery period and several submaximal workloads of progressively increasing intensity. The above sequence produces a "lactate minimum intensity," which is objectively determined by curve fitting of the resultant U-shaped lactate curve. Verification of the relationship between the lactate minimum point and MLSS was done with two constant-intensity tests (8-km runs) undertaken at intensities at and above the LMS. The criterion for MLSS was met if the change in blood lactate concentration during the final 20 min of constant-load exercise was ≤ 1.0 mM. For all of Tegtbur's subjects, this condition was satisfied during the constant-load test equivalent to the LMS (mean change in blood lactate concentration 0.4 ± 0.4 mM). However, 5 of the 25 subjects demonstrated a decrease in blood lactate concentration, indicating that perhaps they were exercising at an intensity below their true MLSS. Running at 0.2 $m \cdot s^{-1}$ above the LMS produced an increase in blood lactate concentration greater than the 1.0-mM criterion, and 11 of the 25 subjects were unable to complete the 8-km test at this intensity.

Tegtbur's work is unique in that it uses an incremental test with previous lactic acidosis, resulting in a clear change in direction of the resulting lactate curve. In comparison, other researchers have attempted to predict MLSS using incremental tests without previous lactic acidosis; therefore the lactate curve shows an exponential increase rather than a definitive turning point.

The reproducibility of the lactate-minimum test is protocol-dependent. Variation in stage duration during the incremental portion of the test has significantly affected test results (Foxdal et al., 1996; Tegtbur et al., 1993). If intervals are not long enough to allow an indication of the steady-state lactate exchange within the whole body distribution space, the LMS may be inaccurately predicted. The LMS is also affected by the initial workload for the incremental test (Carter et al., 1999). The fact that the lactate minimum point is both intensity- and time-dependent highlights the importance of a valid protocol. It is critical that changes in blood lactate values be related to the true metabolic demand of a given exercise intensity, rather than being affected by the lactate kinetics of previous exercise stages.

The validity of using the LMS to estimate MLSS has been investigated by Jones and Doust (1998), who reported that the LMS gave a less accurate estimate of the velocity at MLSS than did the velocity at lactate threshold, measured by an incremental test. We have conducted an evaluation of the lactate minimum test in our laboratory, and have found it to be a reliable and valid predictor of MLSS (MacIntosh et al., 2002). Clearly, more research in this area is warranted.

Advantages of this method include the fact that it is a single test, and some of the variability and subjectivity inherent to other methods are avoided by using a mathematical model. Also, Tegtbur et al.'s (1993) original work demonstrated that altering glycogen stores did not affect the LMS, although absolute blood lactate concentrations were different between normal and low-muscle-glycogen conditions. A disadvantage of the LMS method is the level of effort needed for the initial high-intensity workloads, rendering the test impractical for clinical populations. As noted, caution must be exercised with regard to the test protocol, as protocol manipulations have produced variability in results (Carter et al., 1999; Tegtbur et al., 1993). Furthermore, in our experience (Svedahl and MacIntosh, unpublished), variable results were obtained when subjects engaged in strenuous exercise up to 2 days before the test. This may represent a problem with the test, or it may reflect a desirable sensitivity to altered metabolic and performance capabilities.

LACTATE THRESHOLD

Lactate threshold (LT) is the exercise intensity that is associated with a substantial increase in blood lactate during an incremental exercise test.

Lactate threshold is probably the term most commonly used in the literature in association with estimates of the anaerobic threshold, and in most cases the use of this term is appropriate. The specific criteria used to detect the substantial increase have become important parameters of the definition, and this has led to specific terms according to the criteria for detection of this threshold (i.e., OBLA, or individual anaerobic threshold). For example, the substantial increase may be detected as an increase by a fixed amount above resting blood lactate levels (i.e., +1 mM), or by the first intensity at which a given absolute level of blood lactate is detected (i.e., 2 mM or 4 mM). Figure 3 presents a typical lactate curve, showing an exponential increase in blood lactate as exercise intensity increases. Several objective criteria for LT detection are indicated, including departure from linearity, 1-mM increase above resting, absolute 4 mM, and "individual anaerobic threshold" (see below).

All of these techniques will detect an intensity of exercise that is reasonably close to the anaerobic threshold, but individual variability results in discrepancies when each measurement is compared with the actual anaerobic threshold (or more practically, MLSS). Parameters of the incremental test will affect the outcome, including magnitude of increment, duration of each step, and continuous vs. dis-

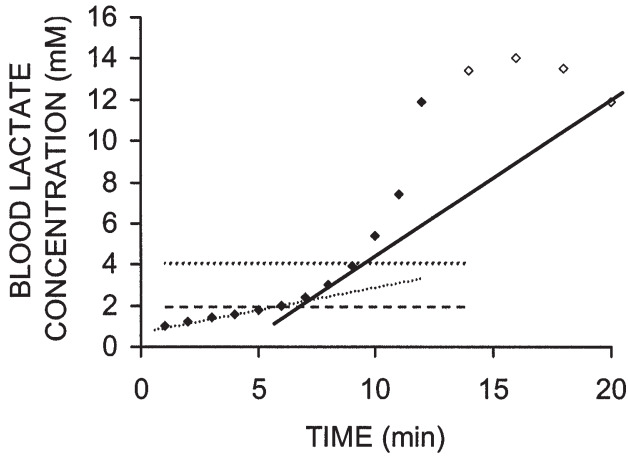

Figure 3. A typical lactate curve, showing an apparently exponential increase in blood lactate as exercise intensity increases. The following objective criteria for lactate threshold detection are shown: departure from linearity (small dotted line); 1-mM increase above resting (thick dashed line); absolute 4 mM (thick dotted line); and indivdual anaerobic threshold (IAT, solid line). The IAT is represented by a line drawn tangent to the blood lactate curve produced during an incremental exercise test, originating at the time that recovery blood lactate falls to the blood lactate value observed at the highest exercise intensity.

continuous test protocols. Lactate kinetics may be quite different between continuous and discontinuous incremental tests, with some discontinuous protocols shifting the lactate curve to the right due to lactate elimination outweighing production during the break in exercise (Heck et al., 1985).

Break durations of 30 s have shown negligible effects (Gullstrand et al., 1994). Workload duration (Foxdal et al., 1996; Wasserman et al., 1973), rate of increase in work rate (Hughson and Green, 1982), blood sampling site (Robergs et al., 1990), and measurement error (Aunola and Rusko, 1992) are all potential sources of variability in measuring the LT. Consequently, it is important to recognize the sources of variability and realize when results are appropriate (due to physiological changes) versus inappropriate (due to error or inconsistency). Under similar testing procedures and similar physiological conditions, the LT is reasonably reproducible (r = 0.90) (Dickhuth et al., 1999).

Table 1 presents a sample of various conditions and parameters of reported tests for LT. It is important to note that there is considerable variability in increment durations and step sizes, as well as criteria for identification of LT. These differences could lead to different estimates of the criterion intensity between tests. However, if a given test provides a reliable estimate of LT, then that test will have utility.

Technology, such as portable lactate analyzers, has made utilization of the LT more practical and convenient. LT tests are simple to administer, can often be combined with a maximal oxygen uptake test, and a single test is sufficient for identifying the intensity of exercise associated with the "substantial change in blood lactate." Furthermore, blood sampling is a minimally invasive technique and does not demand much technical skill. The cost of supplies and equipment is reasonable, results can be obtained quickly, and on-site lactate sampling can be used to monitor athletes in their sport-specific environments.

The relationship between LT and MLSS is variable and largely based on testing protocol. High correlations (r = 0.94) have been noted between running velocity at LT and running velocity at MLSS (Jones and Doust, 1998). When carefully completed, LT tests will yield consistent results, and this is sufficient for evaluating functional fitness in clinical populations or for assessing benefits from a training program in either clinical or athlete populations.

OBLA/4 mM

Onset of blood lactate accumulation, or OBLA, is defined as the intensity of exercise at which blood lactate reaches 4 mM during an incremental exercise test (Sjodin et al., 1981).

This approach to the estimation of anaerobic threshold assumes that the anaerobic threshold is synonymous with an absolute blood lactate concentration of 4 mM, and was originally described by Mader et al. in 1976 (as cited by Heck et al., 1985). One reason for selecting 4 mM as the blood lactate concentration associated with OBLA was the recognition that at 4 mM muscle lactate, muscle and blood lactate are related. This is not the case at higher and lower values (Jacobs and Kaiser, 1982). The transport of lactate out of muscle reaches a peak rate as muscle lactate reaches 4–5 mmol per kg wet weight (Jorfeldt et al., 1978). However, the logic of this rationale is limited since the relevant concentration is muscle and not blood lactate. OBLA is typically measured with an incremental testing

Table 1 Various Lactate Threshold Test Parameters

Protocol	Increment step	Increment duration	LT criterion	Source
Cycle, continuous	30 W	1, 3, 5 min	Breakpoint [a]	McLellan (1985)
	28 W	2 min	Mathematical [b]	McMorris et al. (2000)
	16 W	3 min	Nonlinear increase in [la] vs $\dot{V}O_2$max	Neary et al. (1985)
Cycle, discontinuous	34 W	3 min	Breakpoint [c]	Henritze et al. (1985)
Treadmill, continuous	0.27 m·s^{-1}	2 min	Systematic increase in [la]	Haverty et al. (1988)
Treadmill, discontinuous	0.5 km·h^{-1} [d]	3 min [d]	Breakpoint [a]	Jones and Doust (1998)
	2 km·h^{-1}	3 min	Mathematical [e]	Dickhuth et al. (1999)
	1.0 km·h^{-1}	5 min	Multiple [f]	Nicholson and Sleivert (2001)
Swim, discontinuous	7 × 200 m	5 min	Mathematical [g]	Pyne et al. (2001)

[a] Exercise intensity preceding an increase in [la] for successive workloads.
[b] Algorithmic linear regression, log-log and semi-log transformation methods.
[c] Plot of [la]-work rate; highest work rate not associated with an elevation in [la] above resting levels.
[d] Until 95% HR max or 4 mM [la], then increase 1% grade each minute.
[e] Plot of [la]-running velocity; smoothed with an equalizing spline procedure; lowest value of the ratio of [la] to performance.
[f] Velocity preceeding two consecutive increases in [la] ≥ 1 mM; velocity associated with maximum perpendicular distance between nonlinear regression line and straight line formed by two end-data points of blood lactate profile; velocity corresponding to [la] of 4 mM.
[g] Velocity at LT calculated as a function of the slope and y-intercept from a plot of [la]-swimming velocity.

protocol and subsequent interpolation to determine the intensity of exercise that would be expected to elicit 4 mM blood lactate.

The theoretical basis behind this method was supported by Kinderman et al. (1979), who reported that elite cross-country skiers could sustain a constant running speed corresponding to 4 mM blood lactate for at least 45 to 60 min. However, this support ignores the fact that constant speed with sustained 4 mM blood lactate is not the same intensity of exercise as that at which blood lactate reaches 4 mM during an incremental exercise test. Furthermore, although the average blood lactate concentration was 4 mM, there was some variability between subjects. Clearly, associating a lactate threshold with a fixed blood lactate concentration ignores individual variability. For example, the sustained blood lactate concentration at MLSS ranges from 3 to 9 mM among individuals (MacIntosh et al., 2002).

The advantage with using 4 mM lactate as the criterion estimate of OBLA is that it provides a very objective assessment of lactate threshold. A further advantage is that 4 mM is substantially higher than resting levels, which can be quite variable. This means that 4 mM will represent a rather narrow region of intensity during an incremental exercise test (Karlsson and Jacobs, 1982). The problem with using an absolute blood lactate concentration is the insensitivity to individual physiological differences. As previously noted, many factors affect lactate production and distribution within the exercising body. For example, since blood lactate concentrations are influenced by active muscle mass (Schneider et al., 2000), a fixed blood lactate concentration represents different relative exercise intensities and different relative contributions from glycolysis for different activities. Other important factors to consider while evaluating the usefulness of OBLA are training status and substrate availability, particularly glycogen stores.

Although prediction of anaerobic threshold using OBLA is very objective in that it always occurs at 4 mM, performance at the level of OBLA (i.e., workload, heart rate, oxygen uptake) is not as consistent. In some cases endurance-trained subjects have been unable to sustain workloads at OBLA (Foxdal et al., 1996). Conversely, non-endurance-trained subjects have demonstrated the ability to complete 50-min runs at the velocity corresponding to OBLA, but with blood lactate levels consistently above 4 mM (Foxdal et al., 1996). These results may be attributed to physiological differences between trained and untrained individuals with respect to the intensity (relatively higher for trained) at which 4 mM lactate was reached. For example, the total blood volume of endurance-trained individuals may be at least 10% greater than that of untrained individuals (Green et al., 1991). Although it has not been directly investigated, the additional blood volume would dilute the blood lactate concentration, resulting in a different intensity corresponding to OBLA which may or may not represent a maximal lactate steady state. Dehydration may have the opposite confounding effect.

Studies have also shown that OBLA is protocol-dependent (Foxdal et al., 1996; Heck et al., 1985). There is evidence both supporting and refuting the use of OBLA to predict a maximal steady-state blood lactate response. In some cases no significant relationship has been reported between OBLA and MLSS ($r = 0.57$) (Aunola and Rusko, 1992). In rowing ergometry, high correlations ($r = 0.80$) have been shown between the intensity at the individual anaerobic threshold (see below) and OBLA, while both were significantly higher ($p < 0.01$) than the workload at MLSS (Beneke, 1995).

INDIVIDUAL ANAEROBIC THRESHOLD

The individual anaerobic threshold (IAT) is defined as the exercise intensity identified by a line drawn tangent to the blood lactate curve produced during an incremental exercise test, originating at the time that recovery blood lactate falls to the blood lactate value observed at the highest exercise intensity (see Figure 3). Like OBLA, this is simply a special case of a lactate threshold.

This concept was introduced by Stegman et al. (1981) and was one of the first attempts at providing a single test to identify the intensity at which MLSS should occur. Theoretically, this intensity is representative of the metabolic rate whereby the elimination of blood lactate during exercise is both maximal and equal to the rate of lactate diffusion into the blood (Stegmann et al., 1981). The IAT is measured by an incremental exercise test followed by a passive recovery period, with monitoring of blood lactate levels throughout both phases of the test. Blood lactate concentration is then plotted versus time, and a line tangent to the rising blood lactate curve is drawn from the recovery blood lactate value that equals the final exercise blood lactate concentration. The point of intersection of this line with the blood lactate curve is referred to as the IAT (see Figure 3).

Essentially, the IAT represents a diffusion/elimination model derived from blood lactate kinetics during incremental exercise and recovery (Stegmann et al., 1981). The model presumes to take into account diffusion through biologic membranes, a progressive increase in blood lactate concentration with increasing exercise intensity, the existence of a lactate gradient between working muscle and blood, and the fact that the rate of elimination approaches maximum at higher workloads. It is assumed that the rate of diffusion and the lactate gradient are maximal at the incremental-test end point, and that both decrease during the recovery period (Stegmann et al., 1981). This model also assumes that the rate of decline in blood lactate concentration during passive recovery represents the ability to dispose of lactate. For subjects with a faster decline in blood lactate, the tangent intersects at a higher blood lactate concentration and represents a higher intensity of exercise.

A recent study has shown that determination of the IAT is insensitive to small changes in testing protocol (Coen et al., 2001). The protocol manipulations included previous warm-up, variation in step duration, and test ending point (maximal or submaximal). However, changing the incremental test starting point produced significantly different results. Other researchers have reported varying results due to changes in duration of increments (McLellan, 1985) and test ending point (McLellan et al., 1991; Urhausen et al., 1993). Under identical testing conditions, the reliability for IAT determination is high ($r = 0.98$) (Coen et al., 2001; McLellan and Jacobs, 1993). Endurance trained athletes have been able to sustain exercise at the IAT for 30 min of cycle ergometry and 45 min of treadmill running (Urhausen et al., 1993). Subjects in other studies have not been able to maintain a steady-state lactate response while exercising at the intensity corresponding to IAT (McLellan and Jacobs, 1993).

This method is advantageous in that it is a single test protocol and permits individualized measurement, thereby avoiding many of the shortcomings inherent to OBLA. It is likely not necessary for subjects to put forth maximal effort, although a peak blood lactate concentration of at least 6 mM is recommended (Urhausen et al., 1993).

The relationship between IAT and MLSS is somewhat variable. One report indicates that in rowing, IAT occurs at a higher workload than does MLSS (Beneke, 1995). The relationship is perhaps best summarized by Urhausen et al. (1993), who report that IAT is a reliable estimation of the range of MLSS, although the two are not identical in all subjects.

VENTILATORY THRESHOLD

Ventilatory threshold (VT) is defined as the exercise intensity at which the increase in ventilation becomes disproportional to the increase in power output or speed of locomotion during an incremental exercise test.

Several scientists have noted a nonlinear increase in ventilation when the exercise intensity associated with anaerobic threshold is exceeded. This observation has led to the attempt to use ventilation to detect anaerobic threshold, and various specific techniques have been reported. These include nonlinear increases in ventilation and carbon dioxide output, and an increase in the respiratory gas exchange ratio (R). However, it may be difficult to discern a clear breakpoint using these criteria, and interpretation of the data is not completely objective, with a number of studies reporting variability between reviewers (Powers et al., 1984; Yeh et al., 1983).

Additional criteria have been established based on the occurrence of increased buffering when a net production of lactic acid occurs. In order to minimize the magnitude of change in blood pH, various buffer systems are involved, including the bicarbonate system. The reaction of H^+ with bicarbonate results in the formation of carbonic acid, which dissociates to H_2O and CO_2. This excess CO_2 and the slight fall in pH stimulate ventilation, and the extra ventilation results in excretion of the extra CO_2. An increase in the ratio of ventilation to oxygen uptake, in conjunction with no change in the ratio of ventilation to CO_2 output, represents isocapnic buffering and is considered to be a more specific method of threshold determination from gas exchange parameters (Chicharro et al., 2000; Wasserman, 1987). However, it should be noted that the ability to observe the isocapnic buffering region is dependent on the increment duration of the exercise protocol (Davis, 1985; Hughson and Green, 1982; Wasserman et al., 1973).

The drawback to this approach (and most of these other single test methods) is that it does not necessarily detect the exercise intensity that can be called anaerobic threshold. This may be because several physiological parameters contribute to increased ventilation during exercise. These mechanisms have been reviewed by Walsh and Banister (1988) and include CO_2/H^+ stimulation of the carotid bodies, respiratory mechanics, temperature effects, and skeletal muscle neurogenic stimulation. Consequently, the detected increase in ventilation cannot necessarily be exclusively attributed to buffering of lactic acid. Studies of patients with McArdle's disease, a metabolic disorder in which affected individuals do not produce substantial amounts of lactic acid, have shown ventilatory breakpoints at higher exercise intensities during incremental tests (Hagberg et al., 1982). In healthy individuals it has been shown that the lactate and ventilatory thresholds do not always occur together, nor does the LT cause the VT (Neary et al., 1985). This leads one to question the reliability of predicting anaerobic threshold from noninvasive gas exchange measurements (Powers et al., 1984).

One advantage of using gas exchange measurements to predict anaerobic threshold is that it is a noninvasive technique. Strong test-retest relationships in work rate and $\dot{V}O_2$max at VT have been reported (Yamamoto et al., 1991), and reliability of the method is enhanced if test conditions and personnel are kept constant. In terms of practicality, this method has clinical value, particularly when maximal exercise is contraindicated and invasive blood sampling is not appropriate or desired.

Yamamoto et al. (1991) reported that the VT measured during a stepwise incremental test was equivalent to MLSS, and subjects were able to exercise for 30 minutes at constant intensities corresponding to the VT as well as at 4.9% above. However, during the trial at 4.9% above VT, only 1 of 13 subjects exhibited a rise in blood lactate concentration greater than 1 mM, while 3 subjects had a slight decline in blood lactate concentration. Therefore, in the study by Yamamoto et al., it is likely that VT underestimated MLSS. This is another case of inappropriate use of group statistics for validating such a technique.

Conclusions

It has been the purpose of this review to provide insight and clarification as to the concept and methods of measurement and prediction of anaerobic threshold. The anaerobic threshold, which is the highest intensity of exercise for which measurement of oxygen uptake can account for the energy requirement of the exercise, clearly does exist. Years of research have shown that it is a difficult concept to define and measure. Yet despite the lack of theoretical and methodological consensus, there undoubtedly is value in having a test to estimate the intensity of exercise associated with the anaerobic threshold. The ideal test should consistently yield an intensity of exercise that is close to maximal lactate steady state, which is considered to be the best predictor of anaerobic threshold. It is important for exercise science practitioners to be aware of the different definitions that are commonly applied to anaerobic threshold. Interpretation of the literature with different definitions of these terms relies on taking the meaning of each term from the context in which it is derived. Use of the appropriate operational term when referring to the estimate of anaerobic threshold is encouraged.

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