

VCAA Bulletin VCE, VCAL and VET Supplement 1

VCE Physical Education Units 3 and 4: 2007

Clarification of content: Lactate inflection point (anaerobic threshold)

Introduction

The anaerobic threshold (AT) is one of the most important but controversial concepts in exercise physiology. This supplement provides advice on the preferred approach when teaching this concept in VCE Physical Education Units 3 and 4. Section A contains a series of recommendations for teaching the concept of AT in VCE Physical Education Units 3 and 4. Section B contains teacher professional reading outlining the concepts of AT and lactate inflection point (LIP). Section B is for teacher reference only and is not intended to be used with students.

The VCAA requires that the seven points contained in Section A be incorporated into teaching VCE Physical Education Units 3 and 4 in 2007.

The information provided specifically relates to VCE Physical Education Unit 3: Physiological and participatory perspectives of physical activity, Area of Study 2. This includes key knowledge of:

- characteristics and interplay of energy systems for physical activity and recovery in relation to duration, intensity and type of activity;
- muscular fatigue mechanisms, specifically fuel depletion, metabolic by-products, and dehydration.

Unit 4: Enhancing physical performance, Area of Study 2 Strategies for enhancing sports performance. This includes key knowledge of:

- chronic adaptations of the cardiovascular, respiratory and muscular systems to training.

The understanding outlined in Section A will be examinable from 2007.

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The information has been reviewed by a panel of VCE Physical Education teachers, Dr David Bishop from The University of Western Australia and Dr Carl Paton from Waikato Institute of Technology, Hamilton, New Zealand.



Section A

VCE Physical Education Units 3 and 4: 2007

The VCAA recommends the following points be emphasised when teaching the concept of anaerobic threshold (AT) as part of VCE Physical Education Units 3 and 4:

1. Traditionally the term anaerobic threshold has been used to describe changes in anaerobic metabolism in general and blood lactate metabolism specifically. However, it is now widely recognised that the term is a misnomer. The extent to which anaerobic metabolism is involved in providing energy for exercise cannot be accurately identified by changes in blood lactate concentration or pulmonary ventilation and thus the term should be avoided.
2. Although the relationship between blood lactate accumulation and anaerobic metabolism is unclear, exercise physiologists describe blood lactate accumulation and its relationship to exercise intensity using a variety of terms. Collectively, these terms often refer to a range of blood lactate inflection points. Therefore, it is more appropriate to use the term lactate inflection point (LIP) to represent this group of terms. The specific terms should not, however, be used interchangeably.
3. The LIP does not provide specific information regarding anaerobic metabolism, rather it is thought to reflect the balance between, lactate entry into and removal from the blood.
4. At exercise intensities beyond the LIP blood lactic acid concentration increases.
5. Exercise intensities beyond the LIP are associated with fatigue; the greater the exercise intensity above the inflection point, the more rapid the fatigue. This fatigue is generally considered to be a consequence of a greater reliance on anaerobic metabolism to supply the adenosine triphosphate (ATP) demands of the exercise task and the resultant accumulation of the by-products of anaerobic metabolism. The LIP is often used to predict the speed or power an individual is able to sustain over a prolonged period.
6. Several methods have been developed to estimate LIP. The estimation of LIP involves complex testing that is often conducted in a laboratory and involves analysis of blood or ventilation.
7. LIP tests may provide guidance as to the training intensity required to improve endurance performance and predict the speed or power output an athlete is able to sustain for a prolonged period of time. In fact, where maximal oxygen uptake VO_2 max) is equivalent between athletes, LIP is more likely to distinguish performances of middle and long distance athletes than a VO_2 max test.

Section B

Teacher professional reading

Anaerobic threshold: Is it a misnomer?

Introduction

The AT is one of the most important but controversial concepts in exercise physiology. It was originally thought to represent the exercise intensity beyond which aerobic metabolism was unable to exclusively meet the ATP demand and anaerobic metabolism was switched on in attempt to sustain the exercise intensity (Wasserman and McIlroy, 1964).

It was proposed that this abrupt increase in anaerobic metabolism could be detected as a non-linear increase in blood lactate concentration with increasing exercise intensity (Wasserman and McIlroy, 1964) (Figure 1). However, the metabolic basis of the AT has been debated extensively and the term is now considered to be a misnomer (Brooks et al., 2000). The misnomer arises from the fact that the point of non-linear increase in blood lactate concentration (the 'Lactate Inflection Point') gives no information about anaerobic metabolism and only reflects the balance between blood lactate entry and removal from the blood (Brooks et al., 2000). There has even been a debate as to whether a true inflection point in the exercise intensity/blood lactate concentration relationship exists at all (Campbell et al., 1989). Furthermore, the term anaerobic threshold can lead to confusion, as anaerobic metabolism occurs during mild exercise intensities, and insufficient oxygen supply does not necessarily result in lactic acid production (Stainsby, Brechue and O'Drobinak, 1991).

Despite the fact that AT cannot be precisely identified, the term has crept into the mainstream terminology of the athletic and sporting realm. It is, however, more appropriate to use the term lactate inflection point to describe the point beyond which a given power output cannot be maintained, as it is in fact the variable that has been measured. Exercise intensities above the LIP have been associated with a decreased time to exhaustion, which in turn, are associated with the accumulation of the by-products of an increase in anaerobic metabolism. Specifically, the increase in anaerobic metabolism is accompanied by an increase in lactic acid accumulation; which has historically been implicated in initiating fatigue (Allen and Westerblad, 2005).

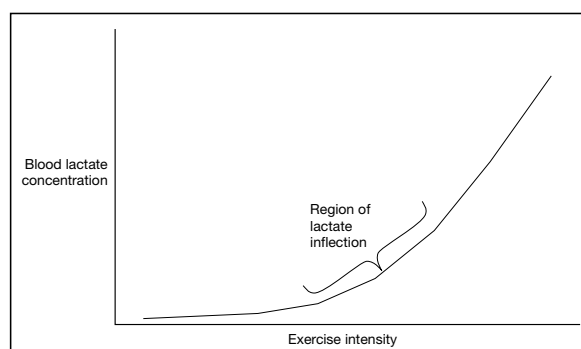


Figure 1: General representation of the relationship between exercise intensity and blood lactate concentration and Lactate Inflection.

It must be noted, however, that recent research reveals lactic acid accumulation may actually help to maintain muscle force output rather than depress muscle force as originally proposed (Allen and Westerblad, 2005). Irrespective of the uncertainty of the mechanisms of fatigue, the effect of exceeding the LIP on fatigue is evident from studies that reveal exercise time to exhaustion is inversely related to the extent to which the work-rate exceeds the LIP (Stegmann and Kindermann, 1982).

Endurance athletes, coaches and exercise physiologists have embraced and utilised the LIP concept in an attempt to define training intensities able to be sustained in preparation for endurance competition. Consequently, numerous techniques were developed as potential indicators of the LIP including maximal lactate steady state (Tegtbur, Busse and Braumann, 1993), lactate threshold (Wasserman, 1987),

ventilatory threshold (Wasserman et al., 1973), onset of blood lactate accumulation (Heck et al., 1985) and individual anaerobic threshold (IAT) (Urhausen et al., 1993). However, the development of several different methodologies and terminologies proclaiming to measure LIP has contributed to the confusion amongst the sporting fraternity regarding the term itself, and the most appropriate methods used to identify the concept. The varying methodologies often provide disparate results by identifying different exercise intensities able to be sustained by the athlete. Furthermore, the average speed and power output sustained by an athlete decreases as the event duration is extended. Consequently, the speed or power output able to be sustained by athletes over a number of distances cannot be precisely identified by one method alone. In this sense, the LIP has not been precisely identified for sporting events of differing durations and intensities using a single measure, and can therefore be considered to represent a region of the graph representing the relationship between exercise intensity and blood lactate concentration (Figure 1).

In order to alleviate the confusion surrounding the topic, this review endeavours to provide clarity on how LIP should be defined and how the various methodologies should be described when teaching the concept to students enrolled in VCE Physical Education.

How is lactic acid formed?

The conversion of glucose to pyruvic acid or lactic acid is defined as glycolysis. Whether glucose becomes pyruvic acid or lactic acid depends on the capacity of nicotinic adenine dinucleotide (NAD⁺) in the muscle cytoplasm to receive hydrogen ions generated by glycolysis to form NADH. If sufficient oxygen is present in the electron transport chain (located in the mitochondria), NADH can be oxidised back to NAD⁺ by transferring its hydrogen ion to the electron transport chain. This process enables a continual supply of NAD⁺ that is able to receive the hydrogen ions continually generated by glycolysis. The constant ferrying of cytoplasmic hydrogen ions in the form of NADH to the electron transport chain prevents hydrogen ions accumulating in the cell cytoplasm and its acidity to rise (pH to fall). However, if there is insufficient oxygen available in the electron transport chain, NAD⁺ is not regenerated at a rate sufficient to ensure hydrogen ion clearance from the cytoplasm. In a reaction that minimises the disturbance in cytoplasm pH, excess hydrogen ions combine with pyruvic acid resulting in the formation of lactic acid. Hence, lactic acid formation is associated with insufficient oxygen supply. The disassociation of a hydrogen ion from lactic acid results in the formation of the salt, lactate, and a decrease in muscle pH and generally blood pH (Brooks et al., 2000). It is the concentration of lactate which is normally measured in the blood by exercise physiologists.

Pyruvic acid is able to enter the mitochondria. Once in the mitochondria, it undergoes a series of reactions involving the Krebs cycle and is converted to oxaloacetic acid. During this process, mitochondrial NAD⁺ becomes reduced to NADH. The mitochondrial NADH transfers its hydrogen ions to the electron transport chain, where the formation of H₂O from the interaction of hydrogen and oxygen results in aerobic ATP formation.

Insufficient oxygen supply (hypoxia) is not the only explanation for the appearance of lactic acid. Lactic acid may be produced despite adequate oxygen supply to the muscles (Richardson et al., 1998). Accelerated glycogenolysis and glycolysis resulting from increased sympathetic stimulation and/or catecholamine concentration may increase lactic acid production (Febbraio et al., 1998). Furthermore, blood lactate levels may not represent increased muscle lactic acid production. Blood lactate concentration is representative of the net transport into and out of the blood. The net accumulation of blood lactate depends on the ratio of lactic acid producing muscle fibres relative to muscle fibres able to oxidise lactic acid, and the blood flow distribution to lactate removal sites (liver, heart, kidney and non-exercising muscle) (Donovan & Pagliassotti, 2000).

What causes the LIP?

The LIP is not the exercise intensity beyond which anaerobic metabolism becomes active. Anaerobic metabolism occurs constantly in the body, even at rest (Stainsby, Brechue and O'Drobinak, 1991). Anaerobic production of ATP via glycolysis precedes aerobic production of ATP via the electron transport chain, irrespective of whether oxygen is present. There is a net gain of two ATP for every single glucose molecule oxidised to lactic acid. Consequently, anaerobic metabolism constantly contributes to the body's ATP yield. Furthermore, lactic acid formation from glycolysis occurs in single muscle fibres even at rest (Stainsby, Brechue and O'Drobinak, 1991). Pyruvic acid resulting from glycolysis is incorporated into the Krebs cycle (for aerobic ATP production) or is converted into lactic acid. This lactic acid may be oxidised in the muscle fibre in which it was produced, or by adjacent muscle fibres (Donovan and Pagliassotti, 2000). Consequently, the presence of muscle lactic acid or blood lactate does not necessarily provide evidence that LIP has been exceeded.

An incremental increase in exercise intensity accelerates glycolytic rate in order to meet the metabolic ATP demand. This increase in glycolytic rate in turn increases lactic acid production. However, while the exercise intensity is below the LIP, blood lactate concentration does not increase, as the rate of its oxidation is equivalent to its production. Thereby, at intensities below the LIP, the end-products of glycolysis, pyruvic acid and lactic acid, are able to be oxidised and accounted for by oxygen uptake. At exercise intensities above the LIP the glycolytic rate is accelerated even further to meet the ATP demand. However, the rate at which the aerobic metabolic pathways can oxidise lactic acid and pyruvic acid is not sufficient and results in an accumulation of blood lactate. Subsequently, it is the accumulation of blood lactate and not simply the presence of it that indicates LIP has been achieved. In this scenario, the required rate of ATP supply cannot be achieved by oxidation of pyruvic acid and lactic acid and in turn, oxygen uptake.

Why quantify the LIP?

The initial purpose for calculating at the LIP was to prescribe safe exercise training intensities for cardiac disease patients (Wasserman and McIlroy, 1964). The LIP may also provide invaluable information on the severity of respiratory disease (Svedahl and MacIntosh, 2003). Additionally the concept has gained immense interest in sporting circles for its potential

ability to predict endurance performance, monitor training outcomes and serve as a basis for setting the intensity of training programs.

It has long been recognised that it is important for an endurance athlete to possess a high maximal oxygen uptake ($\text{VO}_2 \text{ max}$). However, when $\text{VO}_2 \text{ max}$ values are similar between athletes, the fractional percentage of $\text{VO}_2 \text{ max}$ which can be sustained for a prolonged period of time ultimately determines success (Costill et al., 1973). It is widely reported that the intensity associated with the LIP is more highly correlated with middle and long distance performance than $\text{VO}_2 \text{ max}$ (Coyle et al., 1988) and the correlation between $\text{VO}_2 \text{ max}$ and endurance performance is poor when athletes have similar $\text{VO}_2 \text{ max}$ values (Coyle et al., 1973, Hagberg and Coyle, 1983).

Furthermore, using $\text{VO}_2 \text{ max}$ to monitor the effectiveness of a training program may not be relevant for endurance-trained individuals. Endurance performance can improve in athletes without an increase in $\text{VO}_2 \text{ max}$ (Denis et al., 1982; Murase et al., 1981; Sjodin et al., 1982). Denis et al. (1982) demonstrated that 40 weeks of endurance training significantly increased estimated LIP from 72 to 79% of $\text{VO}_2 \text{ max}$, with no change in $\text{VO}_2 \text{ max}$. Finally, eliciting an improvement in LIP is related to the intensity of training. Henritze, Weltman, Schurrer & Balow (1985) demonstrated that training at intensities above LIP resulted in a 42% increase in LIP when expressed as a percent of $\text{VO}_2 \text{ max}$, compared to 16% in a group that trained at LIP. In general, it has been demonstrated that the LIP can vary from approximately 60–90% of an individual's $\text{VO}_2 \text{ max}$ depending upon factors such as the individual's level of endurance training and cardiorespiratory fitness (Hurley et al., 1984). Further, this intensity of exercise has been estimated to correspond to a heart rate of between 65–90% of maximal heart rate depending on the type of LIP test used (Hurley et al., 1984; Foster et al., 1999). It should be noted, however, that the percentage of maximum heart rate at a given LIP is generally unaffected by endurance training (Hurley et al., 1984; Foster et al., 1999, Lucia et al., 2000).

Consequently, estimating the LIP is important for endurance athletes as it provides a measure of the fractional percentage of $\text{VO}_2 \text{ max}$ that could be emphasised during training that is aimed at increasing the ability of an athlete to sustain high levels of power output. In order to estimate the exercise intensity able to be sustained in endurance competition, several tests estimating the LIP have been developed by exercise physiologists. The next section will identify the major tests developed.

How is the LIP detected?

The LIP can be measured using a large range of techniques. Each of these techniques involves determining an intensity of exercise that can be sustained without a pronounced rise in blood lactate concentration. However, as mentioned previously, it has been recognised for some time that these inflection points do not provide information regarding anaerobic metabolism, rather they are considered to reflect the balance between, 'lactate entry into and removal from the blood' (Brooks, Fahey, White and Baldwin, 2000).

Maximal lactate steady state

Maximal lactate steady state (MLSS) is defined as the highest exercise intensity at which blood lactate does not increase beyond the initial transient rise during constant rate exercise

(Svedahl and MacIntosh, 2003). In other words, MLSS represents the exercise intensity at which equilibrium is observed between lactate transport into and out of the blood (Heck, 1985). The only valid measure of MLSS requires the athlete to perform a number of constant rate exercise intensities during which blood lactate concentration is measured. The constant rate exercise intensities need to be of at least 20 and preferably 30 minutes in duration (Bencke and von Duvillard, 1996). The exercise intensities vary by 4–5%, and are performed in an incremental fashion (Svedahl and MacIntosh, 2003). Increases in blood lactate of greater than 1.0 mmol.L^{-1} during the final 20 minutes of exercise (some protocols use $0.2\text{--}0.5 \text{ mmol.L}^{-1}$) are used to define the exercise intensity above MLSS (Svedahl and MacIntosh, 2003). The intensity of exercise below which lactate does not continuously accumulate is defined as the MLSS. Precision of measurement depends on the size of increment in intensity between tests. Enhancing the precision increases the amount of constant rate tests required to be performed by the athlete. Therefore, assessment of MLSS is time consuming, as it involves multiple assessments in the laboratory and consequently more time efficient single tests are generally conducted.

Lactate threshold

Lactate threshold is the exercise intensity that is associated with a substantial increase in blood lactate during an incremental test. Specific criteria have been developed to detect the lactate threshold. These include departure from linearity, an increase of 1 mmol.L^{-1} above resting concentration, when a fixed absolute concentration is achieved (2 mmol.L^{-1} and 4 mmol.L^{-1}) and the Dmax method (Cheng et al., 1992). These methods allow approximation of the anaerobic threshold, yet frequently report variable results when compared to the MLSS (Svedahl and MacIntosh, 2003). Numerous factors affect the estimated exercise intensity corresponding to lactate threshold including; the particular mathematical approach to detecting changes in linearity, duration of each step, the magnitude of the increment in exercise intensity, the nature of the test (continuous or discontinuous), the duration of the break between steps in discontinuous protocols, the blood sampling site and measurement error (Svedahl and MacIntosh, 2003). However, under controlled testing situations the lactate threshold measurement is reproducible for a number of measures (Cheng et al., 1992, Dickhuth et al., 1999) and highly correlated with the intensity calculated from MLSS (Jones and Doust, 1998).

Onset of blood lactate accumulation

Onset of blood lactate accumulation (OBLA) is specifically defined as the intensity of exercise at which blood lactate concentration reaches 4 mmol.L^{-1} during an incremental test (Heck, 1985). While the test criteria of determining anaerobic threshold (4 mmol.L^{-1}) is very objective, the method does not account for individual variation in ability to tolerate lactic acid. For example, the blood lactate concentration at MLSS varies from $3\text{--}9 \text{ mmol.L}^{-1}$ (MacIntosh, 2002). Additionally, endurance trained runners have been unable to sustain work-rates at OBLA, while sedentary people have been reported to sustain exercise intensities above 4 mmol.L^{-1} for 50 minutes (Foxdal et al., 1996). Research has shown OBLA has a low correlation to MLSS ($r = 0.57$) (Aunola and Rusko, 1992).

Individual anaerobic threshold

The individual anaerobic threshold (IAT) is determined from the changes in blood lactate both during and after an incremental test, and in theory represents a metabolic rate where the elimination of lactate during exercise is equal to the rate of diffusion into the blood (Stegman et al., 1981). The anaerobic threshold estimated may vary depending on the initial starting and ending intensity and the increment duration (McLellan and Jacobs, 1993). Researchers have shown IAT can be maintained for 45 minutes of running and 30 minutes of cycling (Urhausen et al., 1993), while subjects in other studies have not been able to maintain a steady state at IAT (McLellan and Jacobs, 1993). The reproducibility of IAT is high ($r = 0.98$) if the test conditions are controlled (McLellan and Jacobs, 1993). The correlation between IAT with MLSS is variable with some studies reporting strong correlations and others low correlation (McLellan and Jacobs, 1993).

Ventilatory threshold

Ventilatory threshold can be defined as the exercise intensity at which the increase in ventilation become disproportionate to the increase in oxygen uptake observed at a power output or speed during an incremental exercise test (Svedahl and MacIntosh, 2003). Wasserman and McIlroy (1964) proposed at this exercise intensity, accumulation of hydrogen ions (from lactic acid dissociation) and the resultant buffering by bicarbonate ions to form carbonic acid produces non-metabolic carbon dioxide stimulating a disproportionate increase in ventilation. The disproportionate increase in ventilation is identified by a breakpoint in ventilation relative to oxygen uptake. There are several concerns correlating breakpoints in ventilation relative to oxygen uptake to lactate threshold. Several factors are responsible for changes in ventilation including hydrogen ion/carbon dioxide stimulation of the carotid bodies, an elevation in temperature and skeletal and muscle afferent nerve stimulation. Therefore, a breakpoint in ventilation is not exclusively attributable to an increase in carbon dioxide. Furthermore, in patients suffering McArdle's disease, a condition that limits lactic acid production, breakpoints in ventilation are still observed during incremental intensity exercise (Hagberg et al., 1983), which is contrary to the model proposed by Wasserman and McIlroy. Breakpoints in ventilation are not always observed during incremental tests and if a breakpoint in ventilation is observed, often it occurs at a different intensity to the lactate breakpoint, suggesting an absence of a cause and effect relationship (Neary et al., 1985). Bicarbonate ions are not the sole hydrogen ion buffers in the body, and bicarbonate number does not decrease (and carbon dioxide production increase) in a 1:1 fashion as hydrogen ion number increases (Peronnet and Aguilaniu, 2006). Therefore, an elevated glycolytic rate and hydrogen ion production is not necessarily mirrored by changes in blood carbon dioxide levels. Finally, respiratory analysis of carbon dioxide exhaled by the lungs is not a measure of carbon dioxide within the blood (Peronnet and Aguilaniu, 2006). The greater volume of carbon dioxide expired at the lungs may be a result of hyperventilation, rather than a cause of it.

LIP testing and training methods summary

Several tests have been developed to potentially detect LIP. None of these tests are able to precisely measure the extent of anaerobic metabolism. They merely estimate a point above

which exercise performance is unlikely to be sustained. Consequently, in defining the exercise intensity an athlete is able to sustain, the specific term describing the method used to estimate the LIP should be used, in preference to stating that the intensity estimated is at the LIP.

The maximal lactate steady state (MLSS) test most precisely determines inflection points in lactic acid during sustained exercise, although due to the length of time required in the laboratory to conduct this test, other tests have been developed. Lactate threshold, OBLA, IAT and Ventilatory threshold are several tests that have been developed to estimate LIP. These tests have a predictable error in estimating the MLSS but in controlled environments it appears tests of lactate threshold and IAT in particular correlate well with MLSS.

Training at an intensity that is above the LIP has been shown to result in an improved performance in endurance sports compared to training at the LIP.

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