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The notion of anaerobic, lactic and ventilatory threshold: Decades of contradiction.

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Abstract

For decades, many studies have addressed the notion of threshold in sports training given its importance in optimizing of the effort. However, the terminology varies depending on the method of measurement and the method of determining this threshold. There are several notions such as the aerobic and anaerobic threshold, the lactate threshold 1 and 2, the ventilatory threshold 1 and 2, and in this respect, different authors have reported many contradictions, and many received ideas, often erroneous, were conveyed to sports actors.

In this article, we present an analysis of the contradictory results of several previous studies on the notion of threshold.

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Introduction

The concept of "threshold" was proposed in 1930 by Owles W.H of Oxford. This physiologist established a close relationship between the evolution of VCO2, ventilation (VE) and the increase in [La] during exercise. (Jacques R, 2003; Le Meur Y, 2010)

Over the past few decades, physiologists have developed numerous methods to determine a threshold that corresponds to a limit for aerobic metabolism. The term "anaerobic threshold" is the first term that was used to describe a breakpoint between aerobic and anaerobic metabolism (Wasserman K and McIlroy M.B, 1964; Le Meur Y, 2010; Mastouri M, 2016). But the terminology varies depending on the method of measurement and the method of determining this threshold.

Some names are purely descriptive: lactate threshold (LT), onset of blood lactate accumulation (OBLA), lactate turn point, etc. Other names refer explicitly to hypothetical metabolic events (aerobic threshold, anaerobic threshold, etc.), or to real ventilatory events such as ventilatory threshold 1 (VTS1) or ventilatory threshold 2 (VTS2). Other names refer explicitly to hypothetical metabolic events (aerobic threshold, anaerobic threshold, etc.), or to real ventilatory events such as ventilatory threshold 1 (VTS1) or ventilatory threshold 2 (VTS2), or even both, such as the anaerobic gas exchange threshold. Finally, some are arbitrary, such as the lactatemia threshold of 2 mmol/L, 4 mmol/L, etc. (see for example, for a non-exhaustive list of terms designating the transition zone between aerobic and aerobic-anaerobic metabolism) (Péronnet F, Aguilanniu B, 2014).

The methodological choices of threshold determination (choice of test protocol, ergometer, blood sampling site, graphical or numerical determination technique, etc.) can modify the value of thresholds. These choices are made by the experimenter. They must be individualized and adapted to the subject being experimented on up to the specificity of the physical activity. The technique for determining thresholds can be visual or computerized (Vallier J.M and al., 2000).

Threshold determination plays an important role in the assessment of an incremental cardiopulmonary exercise test and describes significant changes in blood lactate accumulation with increasing workload. (Binder R.K and al., 2008), and on the other hand, the knowledge of this threshold, particularly interesting to optimize the training of endurance athletes, is approached most of the time during tests with increasing power, (known as "triangular") (Chatard J.C, 2003).

If this theory were true, the trainer would then have the objective reference points necessary to individually manage aerobic or anaerobic training intensities (Cazorla G and al., 2001).

1. The anaerobic threshold (A.T.)

In the first minutes of muscle exercise at constant power, energy expenditure is largely covered by anaerobic lactic metabolism, which causes an increase in blood lactate concentration ("early lactate", Ceretelli P and al., 1979; Bakli A and Medjili S, 2018). After a delay, variable according to the power, lactatemia [La] may decrease if the exercise power (W) is low, or continue to increase for high powers until the event is stopped. Between these two situations, it is reasonable to assume that there is an exercise power that leads to a stabilization of [La] after the initial period (Kiniffo F and al., 1990). This stability would then be due to an equality between the rate of appearance (which we know is never zero during exercise) and the rate of disappearance of lactate (muscular and myocardial consumption, hepatic metabolization and renal excretion) (Hermansen L and Stensvold I, 1972; Brooks G.A, 1986; Svedahl K and MacIntosh B.R, 2003). This power boundary, or "critical lactate power" (WCL) (Chassain A.P, 1986) is therefore the maximum power of exercise allowing the body to maintain itself in aerobic conditions: this is also the definition of the anaerobic threshold (Kiniffo F and al., 1990).

Conversely, there are theoretical data that define this threshold as the physiological point during physical effort, during which the production of muscle lactate is greater than its elimination (Mastouri M, 2016). And also for Wasserman K and McIlroy M.B (1964), this anaerobic threshold refers to the exercise intensity above which the oxygen supply to the active muscles would be insufficient compared to the demand of the latter. The latter explained this accumulation of lactate in the blood by the occurrence of local hypoxia in the active muscles (Le Meur Y, 2010).

As seen in the definition given in the MeSH®, the S.A is the power beyond which the production of anaerobic glycolytic energy comes to support that from aerobic metabolism whose contribution would be insufficient (Péronn F, Aguilanniu B, 2014; Hakkoumi A, 2018). What Péronnet called here an aerobic insufficiency. According to the classical model, the involvement of anaerobic glycolytic metabolism to compensate for this insufficiency would be responsible for the increase in [La] which signs (according to the theory) the presence of anaerobiosis. It would also be responsible, via the drop in pH and the increase in CO2 production (which

result from anaerobiosis), for triggering hyperventilation and the development of fatigue.

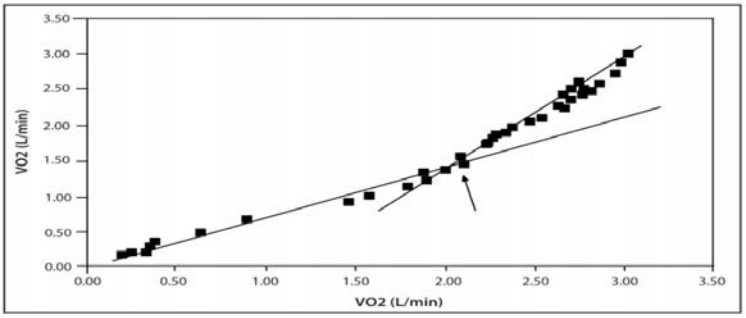
The early onset of A.S. in the untrained subject or in the patient would indicate an aggravation of this aerobic insufficiency. It would be responsible for dyspnea and limitation of work capacity and would reflect an insufficiency of oxygen supply by respiration and circulation to the working muscles or to an inability of the working muscles to use it (Péronnet F, Aguilanniu B, 2014).

Articles and reviews related to the concept of anaerobic threshold are numerous. These publications have given rise to many controversies between proponents and opponents of what G.Brooks calls "the misnomer" (Jacques R, 2003).

1.1 Is the S.A. a threshold and is it anaerobic?

The concept of threshold was first proposed half a century ago by Wasserman and McIlroy based on the observation that when the power of the exercise increases, there is a zone of rapid increase in the RER (ratio VCO2/VO2). (fig.1) Without measuring changes in lactate concentration or any other variable that might indicate the presence of anaerobiosis, Wasserman and McIlroy assumed that the sudden increase in RER reflected ventilatory compensation for the acidosis due to lactate accumulation, indicating the onset of anaerobic metabolism.

Figure 1**:** VCO2/VO2 ratio (Ghosh A.K, 2004).



They therefore proposed that this zone defines a threshold, which reflects a lack of aerobic energy and consequently, that anaerobic energy must be provided (hence the name anaerobic threshold that they gave to this zone). (Péronnet F, Aguilanniu B, 2014; Le Meur Y, 2010)

1.2 The rejection of the threshold nomenclature

The choice of the name "threshold" is not a happy one: it gives the impression of a phenomenon responding to the law of all or nothing and

occurring abruptly, like the depolarization of the nerve fiber when the "depolarization threshold" is reached, which is obviously not the case (Péronnet F, Aguilanniu B, 2014). The work of Green H.J and al, (1983) show that at a power corresponding to 50% of VO2max, which is lower than the power where the S.A. is usually located, the concentration of muscle lactate reaches 4.5 mmol.l-1, while the [La] does not increase above its resting values: 1.3 to 1.5 mmol.l-1. Furthermore, the work of Connett R.J al, (1984), Chirtel S.J and al, (1984) and Fukuba Y and al, (1989) in Cazorla G and al, (2001) show that in a progressive intensity exercise, the muscle produces lactate from the first powers of work and then the intramuscular concentration increases linearly with the successive powers.

Consequently, it cannot be concluded that there is no lactate production by the muscle from the absence of changes in [La] at the beginning of triangular exercise. Therefore, there is no threshold power below which the muscle does not produce lactate and above which it does (Cazorla G and al. 2001).

The lack of change in [La] at the start of triangular exercise probably results from the interaction of three phenomena:

- the too low concentration gradient of intramuscular lactate (Karlsson J, 1971)

- the membrane transporters of lactate are insufficiently active when the lactate gradient is low, recently discovered proteins (Roth R.A and Brooks G.A, 1990, Bonen A, 2000) the MCT (Monocarboxylate Transporter) allow the transport of lactate through the sarcolemma, hence their speed, depends on the passage of muscle lactate to the extracellular medium and to the blood. This speed depends on the level of stimulation of the transporters and the number of transporters involved (Roth D.A, 1991).

- the dilution of small amounts of lactate produced in a large extracellular space (Zouloumian P and Freund H, 1981) so that compared to the five liters of circulating blood, the concentrations of lactate reaching it are negligible.

1.3 The rejection of anaerobic nomenclature

The qualifier "anaerobic" is no happier because it is an assumption. Indeed, no measurements are made in this 1964 article to ensure that beyond the S.A so defined, there is indeed aerobic insufficiency and that energy metabolism is both aerobic and anaerobic (Péronnet F and Aguilanniu B, 2014).

Glycolysis and glycogenolysis do not use oxygen directly, hence their name of anaerobic processes and result in the formation of lactic acid hence the concept of anaerobic lactic often referred to (Cazorla G and al., 2001).

Indeed, Brooks G.A (1985) report relatively high PO2 values during exercise in the mitochondria of active muscles. The increase in [La] is therefore not due to local muscle hypoxia but to the increase in energy demand (Le Meur Y, 2010).

Previously Pirnay F and al (1972) have already highlighted the PO2 of the muscles used during maximum exercise (i.e. at VO2 max), the work shows that the PO2 of the venous effluent blood did not fall below 20 mm Hg, while the [La] increased sharply. Locally in the muscle cell, the work of Chance B and Quirstorff B (1978) using microspectrophotometric techniques showed that the minimum PO2 necessary for maximum oxidative phosphorylation activity was less than 0.5 or even 0.1 mm Hg (Cazorla G and al, 2001), while a few years later, Connett R.J and al, (1984) extended this result by showing that at VO2max there is no muscle area where PO2 is less than 2mmHg, a value 4 to 20 times higher than the PO2 value (Le Meur Y, 2010).

As a result: despite its production and especially its accumulation of lactate, skeletal muscle working even at high power (≥VO2max) is never in hypoxia, either globally or locally. Contrary to what is often said, it is not the absence of oxygen that causes the accumulation of lactate, because there is always more oxygen than the maximum amount that can be used by the muscle. Thus, the assumption underlying the "anaerobic threshold" theory according to which the muscle produces lactate because it is in hypoxia beyond a certain "threshold" power is not tenable (Cazorla G and al., 2001), which a recent consensus of the French Society of Sports Medicine establishes both the definition and the practical determination of lactate and ventilatory thresholds (Vallier J.M and al., 2000). This consensus proposes to abandon all terminology referring to a physiological explanation of these "aerobic and anaerobic thresholds" and to adopt a new one that refers to the technique of determining lactate and ventilatory thresholds (Jacques R, 2003).

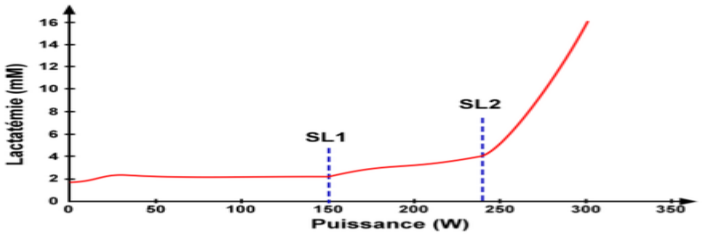
2. The lactic threshold (LT)

In the first few minutes of muscle exercise at constant power, energy expenditure is largely covered by anaerobic lactic metabolism, which causes an increase in blood lactate concentration [La] ("early lactate") (Cerretelli P and al., 1979; Kiniffo F and al., 1990).

There are many controversies regarding the different methods that can be used to determine the lactic threshold. Some authors use the evolution of [La] to determine this threshold (Le Meur Y, 2010). And other researchers propose to determine it from the break in slope in the curve representing [La] as a function of exercise intensity (speed or power) (Cheng B and al. 1992; Lundberg M.A and al., 1986). Although the physiological determinants of lactic threshold are relatively complex (Le Meur Y, 2010).

The first break in the [La] curve (Fig. 2) is also defined as the first turning point of lactate production or the first lactic threshold (LT1), which is at approximately ± 2 mmol.l-1 (Kindermann W and al., 1979; Skinner J.S and Mclellan T.H, 1980; Jacques R, 2003), in other words, this threshold represents the level of intensity at which the amount of lactic acid produced is equivalent to the amount eliminated, and from this level of exercise intensity, there will be the beginning of an increase in [La] (Coulmy N and al., 2002) up to the average value of blood [La] which is very close to 4 mmol/L.

Figure 2**.** The first (LT1) and second (LT2) lactic threshold.



According to some authors (Jones A.M and Doust J.H, 1998), the second break is referred to as the second threshold of blood lactate accumulation (LT2) (fig.2), which is also called OBLA-onset of blood lactate accumulation (Sjodin B and al., 1982; Jacques R, 2003).

With increasing workload above the second break in the [La] curve, muscle lactate production exceeds the rate of removal. This leads to an exponential increase in [La] during incremental exercise, and the energy source is derived largely from anaerobic metabolism. (Aunola S, Rusko H, 1998)

According to Joyner M.J, Coyle E.F. (2008) the very significant increase in muscle oxidative capacity is one of the main factors explaining the high lactic accumulation threshold (OBLA) values measured in high level endurance athletes.

Elite endurance athletes such as triathletes demonstrate VO2max values 50-100% higher than sedentary subjects and their lactic accumulation (OBLA) is associated with higher percentages of their VO2max (Jones A.M and al., 2000). Because lactic thresholds are good indices of performance, Lactate threshold tests can be used to characterize training effects, assess physical fitness, and provide relative training intensity in sports where aerobic metabolism is important (Morton R.H and al., 2012).

2.1 The rejection of lactic threshold nomenclature

It has been shown that there are controversies with LT1 and LT2 thus, these thresholds vary with diet, can severely underestimate or overestimate the actual intensity of LT1 (LT1 can be as low as 2 mmol/l and as high as 7 mmol/l). Thus LT2 is physiologically invalid, varying between individuals (Aunola S and Rusko H, 1992; Aunola S and Rusko H, 1984). However, it has been demonstrated through a metabolic disease that there is more than one correspondence between ventilatory threshold 1 and lactic threshold 1 on the one hand, and ventilatory threshold 2 and lactic threshold 2 on the other hand.

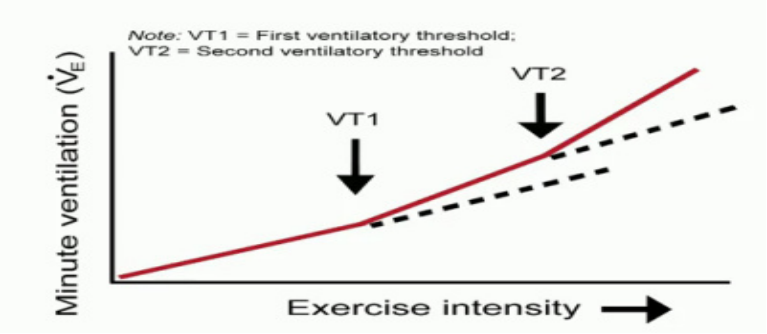
McArdle's disease is a metabolic myopathy, also called glycogenosis type 5, characterized by the congenital absence of the muscle glycogen phosphorylase enzyme due to autosomal recessive mutations of the myophosphorylase gene. It is characterized by severe muscle exercise intolerance from an inability to degrade muscle glycogen (Kuntzer T, 2011).

Exercise intolerance is presented with myalgias, contractures, swelling of muscle masses and motor deficit. In these patients, due to this missing enzyme myophosphorylase, they cannot produce lactate because myophosphorylase is part of the glycogen-lactate chain, so they do not have acidosis, but they have a ventilatory threshold, so their [La] remains stable around a resting value of 1mmol.L-1 (Le Meur Y, 2010). From this disease, it could be clearly demonstrated that there is no relationship between ventilatory thresholds and lactic thresholds, then there are diseases in which there is one and not the other (Neary P.J and al., 1985). In addition, it has been shown that the reproducibility of the curve of lactate production in the blood is low in the same subject from one incremental test to another (Morton R.H and al., 2012), and that the concept of the lactic threshold is unclear since it depends on several factors such as the degree of training of individuals, which can change the values in important ways (Svedahl K and MacIntosh B.R, 2003). Therefore, today it is difficult to rely on lactic thresholds since this nomenclature is highly criticized.

3. Ventilatory threshold (VT)

The method using ventilation is a reference method for determining work intensities useful for planning a specific training program for athletes (Vallier J.M and al., 2000). Current methods allow the determination of two ventilatory thresholds (Daussin F, 2007). From a practical and not theoretical observation by relying on the evolutions of some respiratory parameters that are measured and calculated in real time during the effort (Binder R.K and al., 2008; Beaver and al., 1986; Wasserman and al., 1973; Mastouri M, 2016).

Figure 3. The first (VT1) and second (VT2) Ventilatory Threshold (Wasserman L and al., 1973).



Thus for some studies, the first ventilatory threshold VT1 (fig.3) coincides with the anaerobic threshold (McLellan T.M, 1985; Reybrouck T and al., 1983), which is located at 2 mmol/ml lactate (Orr C.W and al., 1982; Simonton C.A and al., 1988; Wasserman K and al, 1973; Wasserman K and McIlroy M.B, 1964), while for several other authors, the anaerobic threshold is located at 4 mmol/ml lactate (Meyer T and al., 2004) and it represents the second ventilatory threshold VT2 (fig.2) (Ahmaid S and al., 1993).

This notion of threshold was defined as an essential index for the optimization of aerobic capacity or in other words maximum aerobic endurance (Beaver W.L and al., 1986; Nabetani T and al. , 2002; Reybrouk T and al., 1983). It is more relevant to endurance athletes or sports that require a high aerobic capacity such as long distance runners (Jones A.M and Doust J.H, 1998).

For this reason, VT has been considered a good indicator of performance during submaximal exercise (Simões R.P and al., 2013).

The progressive increase in exercise intensity is accompanied by a linear increase in VT up to a level that varies among individuals (Gunepin M and al., 2017). Beyond this level, a sudden and linear increase in EV as a

function of exercise intensity, faster than that of VO2 (Pérronet and Aguilaniu, 2012), a dropout point for which EV increases faster than VO2 is called "ventilatory threshold 1"(Gunepin M and al., 2017). This threshold is between 55% and 70% of VO2max. It is the intensity of exercise from which the energy demand can not be met by oxidative processes. The muscle then uses, in part, glycolysis which leads to the production of lactic acid (David L and al., 2009). The determination of VT1 is based on the assumption that H+ ions, associated with intracellular lactic acid production, are buffered by bicarbonate ions (HCO3-). This induces an excess of CO2 that stimulates ventilation in response to variations in blood CO2 concentration detected by central and peripheral chemoreceptors (Wasserman K and al., 1973; Wasserman K and Whipp B.J, 1975; Daussin F, 2007).

From the first threshold, according to the theory of Wasserman K and al, (1973), ERV and VCO2 increase more than VO2 because an additional amount of CO2 is produced by the buffering of hydrogen ions via the buffer-bicarbonate system. At this intensity, there is therefore an increase in EqO2 without an increase in EqCO2 (the latter remaining constant or decreasing), which defines the first ventilatory threshold (VT1) (Le Meur Y, 2010). This threshold is also highlighted by the break in slope observed in the VCO2/ VO2 relationship (Beaver W.L and al., 1986 in Daussin F, 2007). The power associated with this threshold (pVT1), expressed as an absolute value of VO2 or as a percentage of VO2max, is on average ± 55% of VO2max in a non-trained subject (Jacques R, 2003). While in professional road cyclists, is 70% to 75% VO2max (Lucia A and al., 2001 in Daussin F, 2007).

A second "break" in this evolution appears later for higher intensities (± 220 W), it is the second ventilatory threshold (VT2) (Jacques R, 2003). From VT2 onwards, ERV increases proportionally more than VCO2, in order to compensate for the metabolic acidosis. There is an increase in EqO2 accompanied by an increase in EqCO2 which corresponds to the beginning of the hypocapnic hyperventilation phase (Skinner J.S and Mclellan T.H, 1980; Le Meur Y, 2010). This hyperventilation is explained, at the cellular level, by a decrease in HCO3 stocks, in the cytoplasm and in the blood compartment, which induces an increase in the quantity of H+ ions. These ions gradually diffuse from the muscle compartment to the blood compartment. This increase stimulates the peripheral chemoreceptors located in the aortic arch and induces a second hyperventilation (Meyer T

and al., 2005). VT2 is determined by a second drop in the ventilation curve and an increase in the respiratory equivalent of CO2 (Daussin F, 2007).

Conclusion

Numerous studies have addressed the notion of threshold which has been defined as an essential index for the optimization of aerobic capacity or in other words maximum aerobic endurance. However, there are several ways to estimate this famous threshold. However, depending on the way it is measured, the vocabulary used is not necessarily the same. There is a lot of controversy about the different methods that can be used to determine it.

The validity and reliability of the threshold (whatever it is called), remains an area that needs to be explored further, and that more research should be done.

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