REVIEW

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Historical Remarks on the Development of the Aerobic-Anaerobic Threshold up to 1966

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Abstract

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During the years 1957 to 1963, we introduced the concept of the onset of anaerobic metabolism to measure cardiopulmonary and peripheral aerobic performance capacity. On the basis of bicycle and crank ergometer work with load increments of 3 min duration, we described a point at which the pulmonary ventilation ($\dot{V}_E$) increases at a greater rate than O_2 uptake ($\dot{V}O_2$). Because the changes of the arterial blood lactate (La_a) and $\dot{V}_E$ coincide we defined this point as the "point of the optimal ventilatory efficiency," identical with the "O_2 endurance performance limit," later called "anaerobic threshold" by Wasserman et al.

Introduction

At present the aerobic-anaerobic threshold and lactate evaluation play an important role both in sports and in clinical medicine. Between 1957 and 1963, we introduced the concept of onset of anaerobic metabolism to measure the cardiopulmonary and peripheral aerobic performance capacity. During graded bicycle and crank ergometer work with load increments of 3 min duration, a point is reached at which the pulmonary ventilation ($\dot{V}_E$) increases at a greater rate than O_2 uptake ($\dot{V}O_2$). Because the changes of the arterial blood lactate (La_a) and $\dot{V}_E$ coincide, we defined this point as the "point of optimal ventilatory efficiency," identical with the "O_2 endurance performance limit" (60–62). A few years later, Wasserman et al. (124, 125) called it the anaerobic threshold (AT).

The popularity of AT measurement has increased dramatically during the past 10 years in Germany and during the past 5 to 7 years in many countries. At present, a large number of cardiology, pulmonary, and exercise laboratories include the measurement of AT as a part of their standard exercise protocol; only the techniques used for its detection differ. AT was defined as the point at which there was (1) a nonlinear increase in ventilation (60–62, 124); (2) a nonlinear increase of the arterial blood lactate level (60–62); (3) a nonlinear increase in CO_2 production (125); (4) an increase in end-tidal O_2 (126); (5) an increase in RQ (125); (6) an arterial lactate level of 4 mmol/l (88, 89); (7) an abrupt increase of FEO_2 (average expired O_2 fraction) (24a); (8) an increase of end-tidal O_2 partial pressure (PET_{O_2}) without a decrease of end-tidal CO_2 partial pressure ($PETCO_2$) (24a); (9) a nonlinear increase in the integrated electromyogram (IEMG) (102a) and (10) at which running speed vs heart rate departs from linearity (23a).

The following treatise attempts to outline some of the main stages in the historical development of the AT up to 1966.

Anaerobic Work and Lactic Acid

As early 1871 Hermann observed that muscle can work for quite a long time without oxygen supply (48). In 1906 Fletcher and Hopkins (40) showed that the muscle is capable of producing a specific work performance without any supply of oxygen. At almost the same time, Meyerhof (97, 98) and Hill (53–56) distinguished on the basis of these experiments between an aerobic and an anaerobic phase in muscle contraction. Both researchers believed that lactic acid was the substance that triggered the contraction. Embden (36–38), however, deduced that the formation of lactic acid was merely an energy-supplying process, an "endothermic recharging of a direct source of energy, the exothermic discharging of which would have resulted in muscle contraction."

Hill et al. (57) determined the concentration of lactic acid in the blood and the associated oxygen consumption before and after a given muscular work. They described a close correspondence between the level of lactate acid in the blood and oxygen consumption. This apparent parallelism between lactic acid in the blood and oxygen consumption was questioned by Martin et al. (96) and by Gollwitzer-Meier and Simonson (41, 42). Hewlett et al. (52), Wilson et al. (126, 127), and Liljestrand et al. (79) discovered large quantities of lactic acid and phosphoric acid in the urine.

In 1925 Embden (35) provided evidence for an increase in anorganic phosphate in muscle during a series of muscular contractions. Then, in 1927 and 1928, Eggleton and Eggleton (33) and Nachmanson (107) identified creatine phosphate and demonstrated that its concentration in muscle decreased during contraction. In 1930 Lundsgaard (86, 87) showed that a muscle contaminated with iodoacetic acid can perform several contractions without forming lactic acid. In 1933, Margaria et al. (95) described a fraction of the oxygen debt which showed no relationship to the eliminated lactic acid during the recovery phase and was thus referred to as an alactacid oxygen debt (93, 94).

Further major advances were the discovery of ATP by Lohmann in 1928 and his assumption that this was an important energy supplier for muscular contraction (1934)

(81–84). Finally, the discovery of the ATPase activity by Engelhardt and Ljubimova in 1939 enabled to relate structure and function in muscle (38a).

As early as 1909 Douglas and Haldane (30) assumed that the formation of lactic acid is important for respiratory stimulation during intensive physical work. The previous year Boycott and Haldane (18) described an oxygen deficit during strenuous work, as a result of which lactic acid was supposed to be formed in the exercising muscles. In 1925 Schenk determined a venous lactate concentration of 150 mg% after a 400-m race (119). A year later Hewlett et al. observed a drop in the lactic acid concentration of the blood during oxygen respiration (52). In 1927 Douglas (29) ascertained that the degree to which lactic acid is formed in the working muscles is decisively influenced by the availability of oxygen in the same muscles. As mentioned earlier, at that time the function of ATP and creatine phosphate was still unkown. Consequently, Douglas in the United States and Herbst in Germany believed that the lactic acid formed at the beginning of work was an expression of the energy being supplied for contraction (46). This is why Douglas, in the Oliver-Shaperg lecture he presented to the Royal College of Physicians in London in 1927, had difficulties explaining the working capacity (29). Schenk (1925) (119, 120) and Jervell (1928) (65a) showed correlations between the rise in the lactate concentration and the drop in the alkali reserve in the blood during sporting performance.

This is how Herbst described the importance of the blood lactate concentration in 1929: "The greater the amount of lactic acid that can accumulate in the blood during exercise before exhaustion, the higher the endurance level. Because the lactic acid that is formed has to be bound by the body's alkali, man's performance capacity is dependent on the alkali reserve in the blood and the tissue" (46).

The alkali reserve and the maximum oxygen uptake capability were specified as performance-limiting factors for long-distance races. In 1933 Herxheimer described correlations between lactate levels in the blood and ventilation (50).

The increase in lactic acid even during the first few minutes of recovery after a maximum work load was observed by a large number of workers: Margaria et al. (91, 95); Dill et al. (25); Crescitelli and Taylor (24): Åstrand (4); Huckabee (65); Carlson and Pernow (20); Wolkow (128); Hartung et al. (43).

A rise in the level of lactic acid even during a submaximal constant work load was described by a number of authors: Bock et al. (14, 15); Margaria et al. (95); Hollmann (60–62); Åstrand et al. (40); Hartung et al. (43). The same applies to the constancy or the slow reduction of the lactic acid level in the blood during a given moderate work load: Bang (9); Hartung et al. (43).

Several authors also demonstrated at a relatively early date that, in the case of identical work loads, trained subjects display a lower lactate level than untrained subjects: Bang (9); Robinson and Harmon (114); Crescitelli and Taylor (24); Holmgren and Ström (64); Hollmann (62); Keul et al. (70). The notable difference between the lactate concentration of the arterial blood (e.g., A. brachialis) and the venous blood (e.g., V. cubitalis) during a submaximal bicycle ergometer

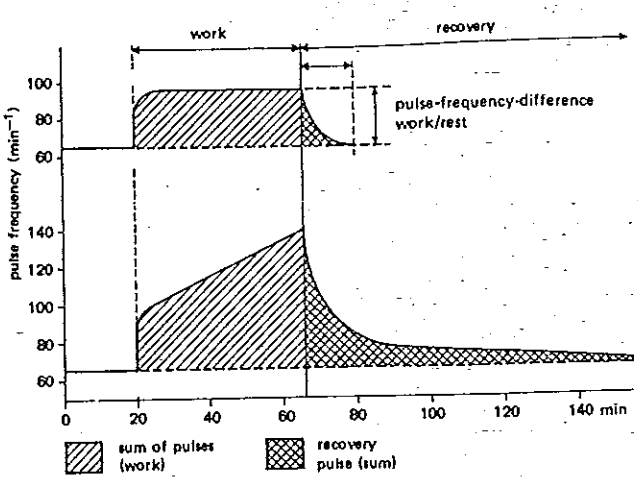

Fig. 1 The recovery pulse frequency sum after two work loads of different intensities (103)

work load was recognised to be an expression of the metabolisation of the lactate in the skeletal muscle at rest (in animal experiments: Barr and Himweck (16), in human tests: Hollmann (60–62).

Endurance Performance Limit and Aerobic-Anaerobic Threshold

The first tests on the endurance performance capacity of man were carried out by Loewy (80) in 1891 and by Zuntz and Schumburg (132) in 1901. They discovered that for a given work load oxygen uptake per minute rose with increasing fatigue. In 1927 Herbst and Nebuloni (47) found that the increase in energy consumption commences earlier the more strenuous a constant work load is. At the Congress of German Sports Physicians in 1929 Herbst stated that in sport the intensity can be increased to a level at which the resultant oxygen requirement can just about be supplied for the duration of the exercise through maximum cardiac and respiratory activity (46).

The first particularly careful investigations to assess endurance work capacity in man were carried out in the 1940s. Müller performed his research in this field on large and small groups of muscles engaging in dynamic work (103, 104). He assumed that, to prepare an individual physiological exercise programme it is essential to know the range of physical working capacity in which endurance performance, i.e., work over a period of 8 h, is possible. The criterion he used in his tests was recovery pulse sum. This is the sum of all the heart beats after completion of the work that exceeds the pulse rate at rest. In the case of work that lasts for 20–30 min and lies below the endurance work limit, the recovery pulse sum does not rise above the empirical value of 50–75 beats above the initial resting value measured in the working position (Fig. 1). This forecast of the limit can be verified in an 8 h experiment. Consequently, Müller demonstrated in his investigations that maximum endurance performance capacity on bicycle ergometers involves a cardiac frequency of about 33 beats above the cardiac frequency at rest. This corresponds to about 30%–33% of

the maximum oxygen uptake (105, 106). This finding was confirmed by Åstrand (1960) who showed an endurance performance limit at about 30% of the oxygen uptake when there was no break during the work. If breaks were included, it was possible to work up to a maximum of 60% of maximal oxygen uptake (3).

These values refer exclusively to bicycle ergometer work carried out in a sitting position. For this reason they can not automatically be applied to job work since among other things values differ, depending on the proportion of static and dynamic work and on the size of the muscle groups involved. Nevertheless, if we assume an average maximum oxygen uptake of approx. 3 l/min and define the endurance performance limit as 30% corresponding to an oxygen uptake level of 1 l/min, this represents an energy expenditure of 21 kJ (5 kcal)/min. Applied to a shift lasting 480 min this gives approx. 10,080 kJ (2,400 kcal) per shift, a figure which lies at the upper limit of empirical values for strenuous physical work in industry.

In clinical applications the assessment of endurance working capacity via the measurement of the pulse rate met little interest since the number of heart beats is considerably dependent on surrounding factors, such as the ambient temperature, and since patients with cardiac rhythm disturbances cannot be subjected to an assessment of their exercise performance capacity by means of the pulse rate. Consequently, in 1955 we wondered whether it was possible to determine the endurance working capacity of man more accurately via a metabolic variable than via the pulse rate.

In connection with the spiroergometric continuous determination of the gas metabolism and respiration, the determination of the lactate and pyruvate concentrations seemed particularly suitable to achieve this. We based our investigations on the two possible states of the muscle cell metabolism:

1. The state of balance of the oxidative metabolism (steady state) in which the continuous ATP consumption for maintaining all cell functions including contraction is covered by an ATP resynthesis of the same magnitude by the respiratory system. In this case, the inner physicochemical milieu, the pH value of the muscle cell, remains unchanged because the carbon dioxide is continuously discharged via the lungs.
2. The state in which the energy requirement of muscle contraction exceeds the actual energy supply from respiration so that the residual energy requirement has to be met through an increase in ATP synthesis via glycolysis. Because of the necessary accumulation of lactic acid which takes place primarily in the muscle and later in the entire body, a "non-steady state" exists which can only be tolerated up to a certain level of lactate concentration.

In 1956 (16) we determined the pyruvate and lactate concentrations in the venous cubital blood of healthy subjects at various work loads and varying intervals during work performed in a sitting position on a bicycle ergometer. We found that lactate, as an end product of the anaerobic breakdown of carbohydrates, is a far more suitable variable

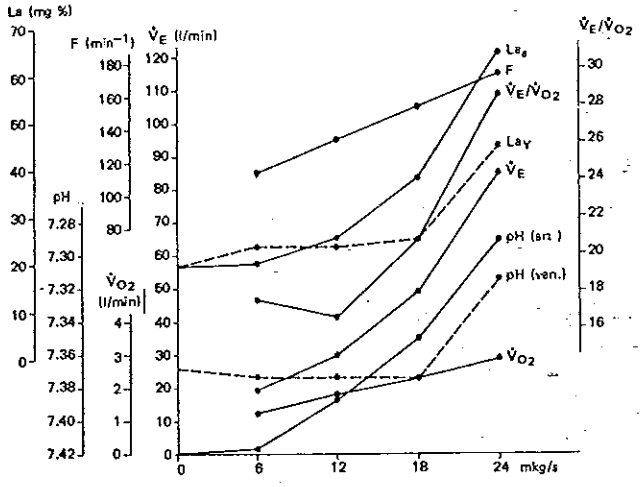

Fig. 2 The changes of the lactic acid concentrations in the arterial (La_a) and venous blood (La_v), of the pH value in the arterial (pH_a) and the venous blood (pH_v), the ventilatory equivalent ($\dot{V}_E/\dot{V}_{O_2}$), the ventilation ($\dot{V}_E$), the pulse frequency (F), and the O_2 uptake ($\dot{V}O_2$) during a stepwise increasing work load (every 3rd min) on a bicycle ergometer (60–62)

for assessing the exercise intensity than pyruvate. However, the lactic acid in the venous blood proved to be rather insensitive to changes of exercise intensity at submaximal work loads even though it suddenly rose steeply at high work loads. In comparative investigations of the lactate concentrations in the venous and arterial blood of the same arm, the arterial values were clearly higher. The difference between the arterial and the venous blood was explained by the fact that the venous blood had also flowed through the muscles of the forearm which were not involved in the exercise. The lactate must have been removed in the resting arm muscles. It was understandable, therefore, that the arterial blood lactate reacted more sensitively even at submaximal work loads than the venous blood. The logical conclusion from this was that only the measurement of the lactic acid level in the arterial blood is sensitive enough to provide adequate information on the work load of the exercising subject (60–62).

In the course of these tests we also measured the pH in arterial and venous blood and assessed the oxygen uptake, the ventilation, the respiratory equivalent, and the pulse rate. We found largely parallel changes between the arterial lactate level, the ventilation and the respiratory equivalent, and – in the opposite direction – the arterial pH value (Fig. 2).

By determining the lactate in the arterial blood it was possible to determine the highest work load intensity that could be mastered purely aerobically. This was the work load that could be tolerated even over a long period without the additional formation of lactic acid. On the other hand, we observed that, for example, during a work period of 30 min, the lactate concentration which had increased during the first 1 or 2 min of work returned almost to the initial resting value if only light or moderate exercise was performed (Figs. 3–4).

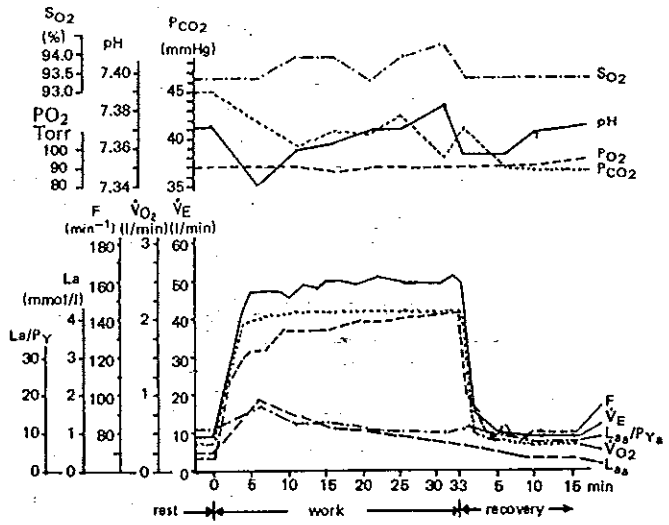

Fig. 3 The changes of oxygen uptake ($\dot{V}O_2$), ventilation ($\dot{V}_E$), arterial lactate (La_a), lactate/pyruvate, pulse frequency (F), arterial pH value, arterial blood gases, and O_2 saturation during a constant work load at 50 % $\dot{V}_{O_2max}$ on a bicycle ergometer (43)

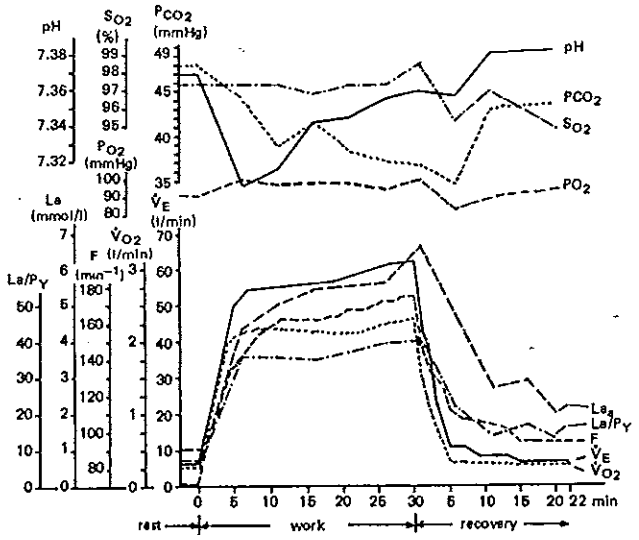

Fig. 4 The criteria as in Fig. 3 during a load intensity of 65 % $\dot{V}_{O_2max}$

However, this lactate-related endurance performance limit did not coincide with the endurance performance limit that had been determined by Müller on the basis of the pulse rate. The limit he determined was higher. As a result we coined the terms "*pulse endurance performance limit*" and "*oxygen endurance performance limit.*" Our explanation for the difference in the level of the two endurance performance limits was that above the pulse endurance performance limit but still within endurance performance limit determined by means of the arterial lactate concentration, nonoxidised substances accumulate and are oxidised or resynthesised in the resting muscles, which do not participate in the exercise, and in the liver among other organs.

Our next aim was to find the simplest possible method of determining this oxygen endurance performance limit. At that time, there as still no micromethod available for determining the lactate level and naturally a routine puncture of the A. brachialis was out of the question. When, during increasing physical work, the point is reached at which the work intensity increased so much that the muscle cells performing the work can no longer be supplied with an adequate amount of oxygen, the ventilation curve increases more steeply than the oxygen uptake. In view of the linear increase in oxygen uptake, a point can be expected at which a maximum amount of oxygen can be taken up with a minimum of ventilation. Therefore, at this point we should look for the aerobic endurance performance limit, i.e. the work load which can still be managed without additional lactic acid formation and thus without an increase of the lactic acid in the arterial blood.

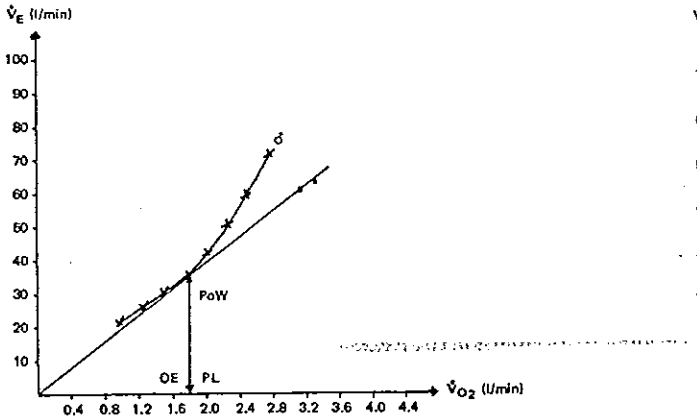

Fig. 5 Point of optimal ventilatory efficiency (POW) and the oxygen endurance performance limit (OEPL) during a stepwise increasing work load (every 3rd min) in a standing position at the crank ergometer (n = 20 male students) 60–62)

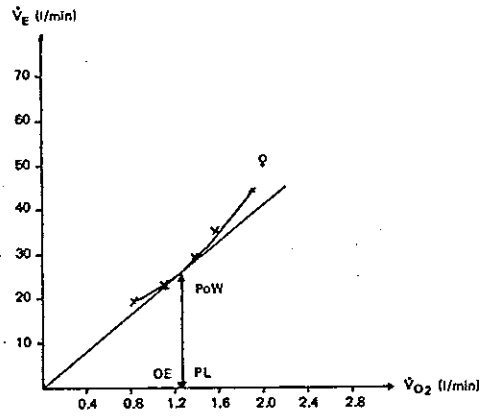

Fig. 6 Same variables as in Fig. 5, but values for female students (n = 32) (61, 62)

To this end we entered the ventilation values obtained every 3rd min of stepwise incrementing exercise tests (steps of 3 min) and which was carried out either in a sitting position on a bicycle ergometer or in a standing position with a crank ergometer, into a system of coordinates. The x axis indicated the oxygen uptake, the y axis the ventilation. We then drew a tangent from the zero point to the curve of the ventilation and called the point of contact the "*point of optimum ventilatory efficiency*" (POW). A perpendicular line dropped from this point onto the x-axis indicated the oxygen uptake corresponding to the highest work load which could still be covered totally by an aerobic energy supply. This was the logical conclusion from the parallel increase of the ventilation and the arterial lactate curves (Figs. 5–6).

Between 1957 and 1963 we examined by this method several hundred healthy subjects and patients of all ages. In the case of sports students who were not endurance trained, the oxygen endurance performance limit for bicycle ergometer work carried out in a sitting position averaged about 60% of their maximum oxygen uptake. This corresponded to pulse rates of around 130–140 min. In the case of females aged between 20 and 30, the corresponding value was reached at a work load which was about 30 % lower, the corresponding pulse rate about 140/min (Fig. 6). On the other hand, when the exercise was performed with a crank ergometer in a standing position, the average oxygen endurance performance limit was about 40 % lower than in the case of the bicycle ergometer work performed in a sitting position. The reason for this is that for a given work load, crank exercises depend on a smaller muscle mass.

Some Later Developments

Wasserman et al. (124, 125) were the first to use the term "aerobic-anaerobic threshold." A criterion of its definition was the start of the nonlinear increase in ventilation in relation to the oxygen uptake. From 1973 onwards this English term was also used internationally. The method and the results are identical with our oxygen endurance performance limit and with the point of optimum ventilatory efficiency (POW).

In 1976 Mader from our laboratory demonstrated that the highest work load of nonaccumulating lactate production is approximately equivalent to an arterial lactate concentration of 4.0 ± 1 mmol/l (88, 89). On an average this is about 2 mmol/l higher than our oxygen endurance performance limit and the aerobic-anaerobic threshold as defined by Wasserman, Keul et al. (71) and Kindermann et al. (72) also obtained similar results. The determination of this threshold value for the evaluation of the endurance performance capacity and as a routine method in physiological and clinical ergometry laboratories, in fitness tests, and for controlling training in sports depended on the possibility of determining lactate from micro blood samples. These are taken from the hyperaemised earlobe. 20–50 µl blood are taken in a capillary tube. Following a minimal incision, more than 20 samples of blood can be taken over a period of about 1 h. These possibilities, which were developed in the 1970s, thus represented a considerable progress in comparison to the potentially dangerous puncture of the A. brachialis which, in addition, was only possible in the laboratory and would never have been suitable for examining athletes during field exercises, for instance, with the possibility of regulating the exercise intensity to obtain an optimal training effect.

At present this method, which is carried out in many countries, is of a great practical importance for the determination of the 4 mmol lactate threshold to evaluate the physical working capacity of top-class athletes, amateur sportsmen, and patients in laboratory as well as in field tests (the latter by means of the Mader "two-speed test").

This development has resulted in the elaboration of a large number of tests. It has now become a popular pastime of many researchers to create in their scientific publications a "new" or "better" definition of the aerobic threshold without the existence of a scientific or a practical necessity. The reliability of the aerobic-anaerobic threshold determination is increased when the intensity of the work load is gradually increased, the exercise prolonged at one intensity step and the nutritional state kept constant. A longer lactic acid accumulation is also necessary to be able to detect small changes in the rate of lactate formation. This is because the final lactate concentration that is attained increases in a linear manner as far as the rate of formation remains constant. However, since the anaerobic threshold represents a range of intensities in which the rate of lactate formation changes considerably and not in a linear manner, a relatively rough graduation of the work load is sufficient for this range to be determined accurately enough.

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