

Cardiorespiratory Testing: Anaerobic Threshold/Respiratory Threshold

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Key words: Exercise — Cardiorespiratory testing — Ventilatory anaerobic threshold — Children

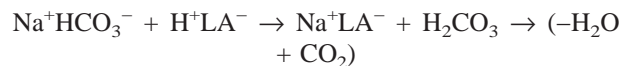
Complete exercise testing is more than simply running on a treadmill or riding on a cycle ergometer. These simple measures may be used to evaluate endurance, peak heart rate, blood pressure, and the presence or absence of dysrhythmias during exercise. With the use of rapid gas analyzers and microcomputers, information about ventilation, oxygen supply and use, and the metabolism at the muscle level may be obtained noninvasively. The concept of the ventilatory anaerobic threshold and the transition from aerobic to anaerobic metabolism during a graded exercise test are discussed. Tests that evaluate only anaerobic performance (i.e., the Wingate test) will not be discussed.

Gas Exchange During Exercise

The amount of oxygen required to perform a given amount of work exclusively aerobically is known and appears to be independent of the degree of fitness, age, or gender of the subject [8, 22]. Thus, the failure of the circulation to deliver oxygen at the required rate to sustain exercise will slow the rate of aerobic regeneration of ATP by the muscle. The oxygen supply to the exercising muscles depends on the blood flow, hemoglobin concentration, the partial pressure of oxygen in the patient's arterial blood, and the iron content of the patient's blood. Failure of the circulation to deliver oxygen at the rate needed to sustain exercise will limit the rate of aerobic regeneration of ATP and the oxygen consumption by the muscles (VO_2).

At very low exercise intensities, the concentration of the blood lactate is nearly identical to levels recorded at rest. At some particular level of exercise intensity, which varies among subjects, blood lactate concentration begins to increase. As the oxygen demand of the exercising muscle exceeds the oxygen supply, anaerobic metabolism is used to supply the energy required to continue

work. Lactic acid is a by-product of this anaerobic metabolism. Once formed, lactic acid will be almost completely dissociated in the serum, and it is buffered predominantly by the bicarbonate system.



Consequent to the buffering of lactic acid, the partial pressure of CO_2 in the venous capillary blood increases. The ventilatory control mechanisms try to maintain homeostasis of PCO_2 resulting in an increase in ventilation; thus, as lactic acid increases during exercise, ventilation responds to two different CO_2 sources: (1) the metabolic CO_2 generated from aerobic metabolism and (2) the excess CO_2 resulting from buffered lactic acid [19].

Arterial lactate measurement is one of the most sensitive ways of determining the fitness of a subject to perform aerobic work. The more fit the subject, the higher the VO_2 before the arterial lactate starts to increase. Once the arterial lactate starts to increase, its slope does so more steeply than the slope for VO_2 [21]. Patients with congenital heart disease causing exercise limitation may have an increase in arterial lactate concentrations at extremely low VO_2 levels. This reflects their inability to supply the active myocytes with the oxygen flow necessary to sustain aerobic metabolism. This same phenomenon can also be observed in sedentary subjects who are deconditioned.

Onset of Blood Lactate Accumulation

Blood lactate concentrations can be measured during exercise. There is a point at which the blood lactate concentration sharply rises, and this point has been termed the onset of blood lactate accumulation (OBLA) [19]. Green et al. [7] investigated the interrelationship between the acute increase in ventilation during exercise and the OBLA. They found that the oxygen consumption at the point of increased ventilation expressed in terms of power output occurred at a higher value than the OBLA,

also expressed in terms of power output. They concluded that the hyperventilation that occurs during exercise and the anaerobic threshold (AT), as determined by blood lactate accumulation, do not represent identical biochemical events. This observation suggested that an increase in anaerobic metabolism by exercising muscle precedes both the onset of hyperventilation during exercise and the OBLA. Similar observations have been reported by others [2, 11].

It is difficult to clearly identify the threshold point on a lactate intensity curve because there are usually very few discrete points obtained during an exercise study. However, most investigators agree that the threshold usually occurs around 4 mmol/L. Therefore, the anaerobic threshold is commonly defined as the intensity corresponding to a lactate value of 4 mmol/L [24].

This value of 4 mmol/L may be valid for adults, but it is also close to the peak VO_2 in children between 11 and 16 years of age [19, 24].

Although fixed lactate concentrations of between 2.5 and 4.0 mmol/L increased the objectivity of the anaerobic threshold determination, such criteria do not respect the individuality of the inflexion point that occurs at different lactate values. This is particularly true for children because the peak percentage VO_2 corresponding to 2.5 mmol/L decreases from about 90% to 80% from 12 to 15 years of age [24], suggesting lower lactate values at the inflexion point for younger children. Using any value for all ages during growth makes it difficult to compare AT values that have been obtained and used in adult studies.

Also data [6] suggest that the increase of serum lactate level begins immediately after the onset of graded exercise and continues throughout a graded exercise study. This observation suggests that the acute increase in blood lactate levels actually represents a point at which the clearance of lactate from the body is exceeded by the production of lactate at the muscular level, and it implies that hypoxia and anaerobic metabolism begin at the cellular level long before the accumulation of lactate in the serum.

Blood lactate levels therefore reflect not only lactate production but also clearance. Indeed, lactate levels may be best interpreted as an index of the balance between these two processes. Other factors may significantly influence the lactate level as well. These include, but are not limited to, the rate of release of the lactate from the muscle cell, the rate of lactate utilization by other tissues (liver, heart, and other muscle), and the volume of distribution within the body fluids.

There are other methodological factors that are too numerous to mention here in any detail, but such factors as type of exercise, site of blood sampling, timing of blood sampling, and the method of assay are important in evaluating lactate levels. Caution should be used when comparing different studies. Lactate levels also appear to

change during growth. A progressive increase in values has been observed in children between 6 and 19 years of age, more than doubling during that age span [7]. Several cross-sectional studies have confirmed the pattern of progressively increasing peak blood lactate levels as children grow [1, 3, 4]. Some investigators have found that peak blood lactate levels are significantly higher in girls than in boys, whereas others have failed to confirm these results [3, 24].

It may be concluded that several factors are responsible for the change in lactate concentrations observed during exercise. The monitoring of lactate levels requires invasive blood sampling; therefore, they are not used widely in clinical testing. The interpretation of lactate levels deserves further research before practical clinical applications are possible.

Ventilatory Anaerobic Threshold

Recall that during exercise there is a point at which the oxygen supply to the exercising muscle is inadequate to replenish all the ATP, and anaerobic mechanisms are called on to provide energy. This process results in an increase in lactate production, and this lactate is rapidly buffered, resulting in an increased production of bicarbonate and subsequently carbon dioxide that must be eliminated via the lungs. This results in an abrupt increase in ventilation to “rid the body” of this excess CO_2 . The hyperventilation also results in an increase in oxygen consumption but not at the same rate as CO_2 production. In other words, the CO_2 production increases out of proportion to the oxygen consumption. This phenomenon has been termed the AT as first described by Wasserman and McIlroy [20].

Several methods have been proposed for determining the AT. The definition of the AT is not at all clear, and comparison of various methods becomes cumbersome. Some researchers believe that the AT marks the point at which exercising muscle begins to use predominantly anaerobic metabolism for its energy supply. Others define the AT as the point at which the concentration of lactate in the serum abruptly increases. Still others define the AT as the point at which the production of lactate exceeds the lactate clearance from the serum. Methods have been described that determine the AT using any of the previous definitions; therefore, caution must be used when comparing one method to another.

Wasserman and McIlroy [20] observed that ventilation increases out of proportion to oxygen consumption at work rates above the AT. The increase in ventilation reflects an increase in respiratory frequency without an increase in tidal volume. The AT described by Wasserman and McIlroy is now referred to as the ventilatory anaerobic threshold (VAT). Work performed at levels above the VAT is associated with an increase in oxygen

debt. The VAT also demarcates the work rate above which VO_2 kinetics are slowed, resulting in a delay of steady state after changes in work rate during an incremental test.

The VAT probably occurs at a time slightly later than the abrupt increase of lactic acid in the serum during exercise. It is therefore not accurate to compare a technique that attempts to determine the VAT to one that measures lactate levels.

The ventilatory anaerobic threshold has been a useful clinical tool despite the shortcomings outlined previously. The VAT may be used to establish a target heart rate for aerobic training and rehabilitation programs [5, 17]. It also has been used to estimate aerobic fitness in subjects who otherwise would not tolerate maximal exercise testing [14], and it has been used to provide training guidelines for athletes [9, 18]. Finally, it has been used to measure an individual's exercise capacity that may be safely used without triggering anaerobic metabolism [19].

There are several studies that have provided normal values for the VAT. It is important to realize that the VAT changes with growth and appears to be somewhat gender dependent. The VAT decreases with age [13]. Reybrouck et al. [13] investigated the influence of age on the VAT in a cross-sectional study of 257 children between the ages of 5 and 18 years. The VATs at age 5 or 6 were 74% and 69% of $\text{VO}_{2\text{max}}$ in the boys and girls, respectively, whereas values at age 15 or 16 years were 61% and 54%. A total of 52 children, ages 6, 11, and 14 years, were studied by Weymans et al. [23], who performed a graded progressive walking treadmill test. Overall VAT mean was 66% of $\text{VO}_{2\text{max}}$ for both the boys and the girls, but the VAT did decline progressively with increasing age in both genders.

The VAT also appears to be useful when evaluating the effects of training. Several studies have indicated a higher VAT value in child athletes. Nudel et al. [12] found that the percentage of $\text{VO}_{2\text{max}}$ at the VAT averaged 81% for runners and 58% for controls. Wolfe et al. [25] found that VAT was useful in ranking the performance of female cross-country runners. However, studies of changes of VAT after physical training in nonathletic children produced mixed results and are nonconclusive [10, 16].

Effects of Cardiac Malformations on the Ventilatory Anaerobic Threshold

Reybrouck et al. [14] evaluated 50 children with congenital heart disease and showed that the VAT can easily be determined in most children over the age of 5 years. Children with ventricular septal defect or atrial septal defect had no correlation between the VAT and the size of the left-to-right shunt. However, the VAT correlated

significantly with the morphologic variables and the habitual level of physical activity in these subjects. This study also suggested that the VAT is a more sensitive indicator of physical performance than $\text{VO}_{2\text{max}}$ in this population of patients.

The VAT is perhaps most useful in pediatric patients who are unable to exercise to maximum levels required for the use of $\text{VO}_{2\text{max}}$ in determining fitness. It is also true that very often chronotropic mechanisms are altered following congenital heart disease surgery. The VAT is useful in this classification of patients in whom peak heart rate may not be used to evaluate fitness.

On the other hand, patients who have significant pulmonary disease or any type of airway obstruction may not develop a true VAT, and this technique is not useful in this group of patients.

Conclusions

Whereas the biochemical interpretation of the AT is uncertain, the VAT is useful in many clinical situations. Most important, the VAT may serve as an effective sub-maximal marker of aerobic fitness and may be used in setting training guidelines in patients with congenital heart disease. The use of the VAT as an aerobic training threshold for improving cardiovascular fitness is conceptually attractive. It would appear to be more appropriate than the traditionally used target heart rate for patients with congenital heart disease. Future research is needed before the full clinical applications of the VAT are realized.

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