REVIEW ARTICLE

ANAEROBIC THRESHOLD: ITS CONCEPT AND ROLE IN ENDURANCE SPORT

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aerobic to anaerobic transition intensity is one of the most significant physiological variable in endurance sports. Scientists have explained the term in various ways, like, Lactate Threshold, Ventilatory Anaerobic Threshold, Onset of Blood Lactate Accumulation, Onset of Plasma Lactate Accumulation, Heart Rate Deflection Point and Maximum Lactate Steady State. But all of these have great role both in monitoring training schedule and in determining sports performance. Individuals endowed with the possibility to obtain a high oxygen uptake need to complement with rigorous training program in order to achieve maximal performance. If they engage in endurance events, they must also develop the ability to sustain a high fractional utilization of their maximal oxygen uptake (%VO, max) and become physiologically efficient in performing their activity. Anaerobic threshold is highly correlated to the distance running performance as compared to maximum aerobic capacity or VO,max, because sustaining a high fractional utilization of the VO,max for a long time delays the metabolic acidosis. Training at or little above the anaerobic threshold intensity improves both the aerobic capacity and anaerobic threshold level. Anaerobic Threshold can also be determined from the speed-heart rate relationship in the field situation, without undergoing sophisticated laboratory techniques. However, controversies also exist among scientists regarding its role in high performance sports.

Key words : Anaerobic, Lactate, Heart Rate, Deflection Point, Ventilatory, Threshold, Acumulation.

Introduction

Recent decades have witnessed a remarkable expansion of the application of scientific principles to sports and exercise. Application of science to sports are especially evident in the field of physiology; indeed sports practitioners are quick to realize the importance of acquiring basic physiological knowledge that can produce better effect. Exercise physiology has for many years been a respected field in its own right. Exercise has conventionally been used as a medium for perturbing physiological systems to ascertain how they behaved under stress. Exercise physiologists have further established the ceilings in human physiological responses and the factors that limit performances in various conditions.

Physical performance in various competitive sports events depends largely on the integrated status of the different physiological mechanisms of the individual i.e. the state of health and capacity for physiological responses to meet the challenges of the competitive situation, apart from the technique, tactics and skill. Optimum level of performance depends on the development of these responses through training. Therefore, the main purpose of physiological research is to evaluate and monitor the training schedule effectively. The most important physiological factor for high performance in marathon is to possess a high aerobic capacity or VO_2 max. An elite marathoner exhibits a high VO_2 max (more than 80 ml/kg/min), similarly the VO_2 max is an important parameter of middle and long distance runners, road cyclists, long distance swimmers (70-80 ml/kg/min) and team game players (60-70 ml/kg/min), whereas, the sprinters exhibits a comparatively lower VO₂ max (45-55 ml/kg/min) [1]. In long term endurance events, oxidative phosphorylation plays the dominant role and thus the maximum oxygen uptake (VO₂ max) or aerobic capacity becomes one of the major determining factors in high performance sports. VO₂ max is restricted to only the individuals' cardiorespiratory capacity relating the O₂ uptake, transport and utilization. Not only in marathon but also in all other sports events of long duration activity, whether it is a continuous type or non-continuous type, VO₂ max plays the key role. Maximal oxygen intake may be the most physiologically significant and most commonly measured parameter in the physiological assessment of well-trained athletes. For this reason a linear correlation has been observed by various exercise physiologist between VO₂ max and distance running performance, where the correlation coefficient varies from 0.52 to 0.98 (2-11). A high aerobic power (VO, max) is an advantage in endurance sports, but to sustain a high fraction of VO_2 max for a long time is more important (6-8).

Anaerobic Threshold Concept

Individuals endowed with the possibility to

obtain a high oxygen uptake need to complement with rigorous training program in order to achieve maximal performance. If they engage in endurance events they must also develop the ability to sustain a high fractional utilization of their maximal oxygen uptake (%VO₂ max) and become physiologically efficient in performing their activity (6, 8, 12). This hypothesis is the concept of anaerobic threshold or lactate threshold or aerobic to anaerobic transition point. During exercise of increasing intensity there is a rise of blood lactate concentration and this response was first reported half a century ago (13-14).

Concept of anaerobic threshold/lactate threshold was introduced in order to define the point when metabolic acidosis and also the associated changes in gas exchange in the lungs, occur during exercise (15). To explain it in another way, during incremental exercise, at a certain intensity, there is nonlinear steep increase in ventilation, known as ventilatory anaerobic threshold (16), a non linear increase in blood lactate concentration, known as lactate threshold (16), a non linear increase in CO₂ production, an increase in end tidal oxygen, an increase in CO₂ production (15), an arterial lactate level of 4 mM/L, known as onset of blood lactate accumulation (OBLA) [17], and an abrupt increase of FEO₂ (expired O₂ fraction) [18]. All these points

Figure 1 : Curvilinear increase in blood lactate in relation to running speed. (OBLA running speed is 24 km/hr). Data of an Indian middle distance runner (from Ghosh and Mukhopadhaya (Ref. No. 102).



are collectively labeled as Anaerobic Threshold (AT). It is evidenced that ventilatory anaerobic threshold is directly related to and also caused by blood lactate threshold (15-19).

It has been observed that individuals with similar VO₂ max have variability in endurance capacity and that highly trained athletes perform at a high percentage of their VO, max with minimum lactate accumulation (20-21). Furthermore, trained athletes accumulate less lactate than untrained athletes at a given submaximal workload. This concept has prompted the consideration of anaerobic threshold as a determinant of physiological fitness. While the appearance of lactate in blood during exercise is the result of an increased glycogenolysis, it is important to recognize that its concentration is, at any time, the result of a balance between the rate of production and removal (22). Nevertheless, during exercise of increasing intensity, the rise in blood lactate concentration is an indication of increase in glycogen metabolism. This increase in blood lactate has been interpreted as a reflection of the onset of hypoxia in skeletal muscles and the exercise intensity at which anaerobic metabolism complements the regeneration of ATP by aerobic metabolism has been called the Anaerobic Threshold (22).

Lactate Threshold Concept

Different scientists with the same objective to determine the aerobic and anaerobic transition point, of an exercising individual, adopt different methodologies. Production of lactate in the muscle increase curvilinearly with increasing work load (23-24) or with percentage utilization of VO_2 max. The level at which abrupt increase in blood lactate is observed has been described as individual's lactate threshold (Fig 1). Numerous scientists emphasized that blood lactate concentration is the net result of lactate production or appearance in the muscle and its removal from the muscle (25-28). Thus the rise in blood lactate may not necessarily indicate the abrupt increase in lactate production by the exercising muscle, due to simultaneous removal process. Increased lactate production could have occurred much earlier but may not have increased the blood lactate concentration because of increased removal. It is well known that non-exercising muscle, the liver, the kidney and the heart can mobilize lactate (29-32), from the main stream for further biochemical degradation. Some reports (33, 34) however indicate that during the work rate below 50-60% of maximum oxygen uptake, muscle lactate does not increase, since the appearance and disappearance rate of lactate becomes almost equal.

Figure 2 : Ventilatory anaerobic threshold of a long distance runner (from Ghosh and Mukhopadhaya (Ref. No. 102)



This supports the fact that initial increase in blood lactate concentration reflects that appearance rate of lactate in the blood is higher than the disappearance rate. Many investigators have suggested that together with lactate, hydrogen ions are generated (15, 35, 36). Due to its low pK (3.9) lactic acid is presumed to dissociate after its formation, thereby generating hydrogen ion (15, 36). The hydrogen ion thus produced may elevate CO_2 through carbonic acid bicarbonate buffer system and alter homeostasis (15, 22, 35, 38).

Ventilatory Anaerobic Threshold

Wassermann and Mcllory (37) and Reybrouck et al (38) postulated that alveolar ventilation is coupled to CO₂ flow to the lungs, thus leading to maintain arterial isocapnia during moderate exercise. However, as the intensity of exercise increases hydrogen ion released from lactic acid can no longer be buffered by blood bicarbonate stores and thus metabolic acidosis results. This increase in hydrogen ion further stimulate ventilation by stimulating central as well as peripheral chemoreceptors resulting in further increase in ventilation. Thus the linearity of ventilation with changes of work load and oxygen utilization deviates at certain points which is named as ventilatory anaerobic threshold (Fig 2). In other words, during incremental, nonsteady state exercise, a point is reached at which a subject's ventilation shows a non-linear increase and this is termed as ventilatory threshold.

Ventilatory threshold is coincident with its hypothesized causative factor, a non-linear increase in blood lactate concentration termed as the lactate threshold (39). Several ventilatory parameters have since been utilised in assessing ventilatory threshold, among which are oxygen consumption (VO_2) [40-42], pulmonary ventilation (V_E) [18, 42-44], respiratory exchange ratio (RER) [18, 45], excretion of CO₂ (VCO₂) [19, 43], and the ratio of ventilation to oxygen consumption (V_F / VO_2) [46, 47] have been devised to evaluate this critical intensity. Most techniques rely on visual inspection of one or two specific parameters over time and / or velocity to identify the breakaway threshold. Methods have now been devised over tine and/or velocity to identify the breakaway threshold. Beaver et al (19) utilised a computerised regression analysis of the VCO₂ versus VO₂ slope (Fig 3) collected during progressive intensity exercise (V-slope method). Although no significant difference existed between the VO₂ at ventilatory threshold computed by the Vslope method and visual inspection. The V-slope method could more reliably determine ventilatory threshold. Orr et al (48) performed a computerised 3-segment regression analysis to locate the intersection point of the segments in the $V_{\rm F}$ vs. VO₂ plot. Their computerised method also correlated highly (r = 0.95) with visual inspection methods with a difference of 0.05 L / min between VO, at AT determined by computer and by visual inspection.

The ability of gas exchange variables to detect

Figure 3 : The v-slope method for determining anaerobic threshold. VO2 is plotted against VCO2. See the deviation (Data of a female long distance runner from Ghosh and Mukhopadhaya



the onset of lactic acidosis was investigated by Hollman (16) and Wassermann and Mcllory (37). Since this time, numerous studies have investigated the ability of specific respiratory measures in assessing the degree of anaerobiosis. Davis et al (18) investigated the validity of AT detection through nonlinear increase in V_E and VCO₂ and abrupt increase in the fractional concentration of O_2 (FEO₂). No significant difference was observed between the estimation of AT from these gas exchange variables and blood lactate concentration. On average, AT occurred at $59.8 \pm 7.4\%$ VO₂ max and $59.7 \pm 7.1\%$ VO₂ max for respiratory gas exchange and blood lactate methods, respectively. Furthermore, a correlation of 0.95 was observed after plotting %VO₂ max scores for gas exchange AT versus blood lactate AT methods. Caiozzo et al (49) also examined the correlation between gas exchange and blood lactate methods for determination of AT. Comparisons were made for using nonlinear increase in V_E or VCO₂, an abrupt increase in respiratory exchange ratio (RER), an increase in $V_{\rm E}$ / VO₂ without a concomitant increase in V_E / VCO_2 and the systematic increase in blood lactate concentration. All gas exchange methods except RER significantly correlated with blood lactate method in ability to detect the AT. Similar methods have been produced by Reinhard et al (50) who observed a significant correlation between the VO₂ at lactate threshold and the VO₂ at ventilatory threshold, as determined through the ventilatory equivalent for oxygen. In describing the differences of muscle fiber type and the occurrence of both lactate and ventilatory threshold, Aunola and Rusko (51) found no significant difference in the occurrence between lactate threshold and ventilatory threshold when expressed in terms of %VO₂ max.

Several lines of evidence have recently been presented which refute the theory that ventilatory and lactate threshold are casually linked. Most studies examining this theory compare the ventilatory response during exercise in individuals with normal and elevated blood lactate concentrations. These studies suggest that the elevated blood lactate concentrations had no significant effect on ventilation during the progressive intensity exercise. Neary et al. (52) examined lactate and ventilatory thresholds under normal conditions and under glycogen depleted and / or previously exercised states. No significant changes in ventilatory threshold resulted under experimental conditions, therefore suggesting that plasma lactate accumulation was not responsible for the threshold-like responses in ventilation. Cecca et al (53) performed a similar investigation where subjects performed incremental exercise under normal and acidic conditions. Although subjects began the experimental progressive intensity exercise with a mean blood lactate concentration of 9.8 mM/L, ventilation did not significantly differ at each power output from normal conditions. Again, the elevated blood lactate concentration did not alter the pattern of ventilation during the progressive intensity test.

Figure 4 : Anaerbic threshold determination from speed heart rate deflection point. Data of an Indian middle distance runner (from Ghosh and Mukhopadhaya (Ref. No. 102))..



References	Subjects	Change in VO2 max (%)	Change in Lactate variable (%)	Training duration (weeks)
Sjodin et al (77)	Elite swimmers	NS	5	14
Denis et al (77)	Students	NS	18	40
Yoshida et al (86)	Students	14	37	8
Hoffor et al (93)	Children	NS	22	5
Burke et al (94)	Women	5	20	7
Belman and Gaeser (101)	Old persons	7	12	8
Edwards et al (90)	Elite soccer players	NS	5	5
NS = Not significant.				

Table 1 :Effect of endurance training on the changes in VO_2 max and lactate/anaerobic threshold
level (All the values are significant except mentioned NS).

Heart Rate Deflection Point and Anaerobic Threshold

The heart rate deflection point (HRDP) is a deviation point at the linear relationship between heart rate and work load is evinced during progressive incremental exercise testing (Fig 4). The HRDP is reported to be coincident with the anaerobic threshold. In 1982, Conconi and colleagues suggested that this phenomenon could be used as a noninvasive method to assess the anaerobic threshold (54). These researchers developed a field test to assess the HRDP, which has become popularised as the 'Conconi test'. Concepts used to define and assess the anaerobic threshold as well as methodological procedures used to determine the HRDP are diverse in the literature and have contributed to controversy surrounding the HRDP concept (55). Although the HRDP may be assessed in either field or laboratory settings, the degree of HR deflection is highly dependent upon the type of protocol used. The validity of HRDP to assess the anaerobic threshold is uncertain, although a high degree of relationship exists between HRDP and the second lactate turnpoint (lactate threshold).

Conconi et al. (54) reported a noninvasive field test for anaerobic threshold (AT) based upon an observed deviation from the linear heart rate (HR)—running velocity (RV) relationship at high RV (HR deviation). While the validity of the Conconi test has been debated (56, 57), the reliability of the Conconi test has never been independently assessed in athletes performing the protocol outlined

by Conconi. In a study by Jones and Doust (58), the reliability of the Conconi test in 15 well-trained male distance runners was evaluated twice within a 4-8 day period. The results indicated that 6 subjects demonstrated HR deviation in both Conconi tests, 5 subjects demonstrated HR deviation in only one test, and in 4 subjects' deviation could not be demonstrated in either test. They concluded that failure to determine a reproducible HR deviation by subjective assessment in 9 of 15 subjects makes the Conconi test unsuitable for reliable evaluation of AT. Vachon et al (59) designed 4 test protocols, like, 1) a treadmill test for maximal O₂ uptake, 2) a Conconi test on a 400-m track with speeds increasing approximately 0.5 km/h every 200 m, 3) a continuous treadmill run with speeds increasing 0.5 km/h every minute, and 4) a continuous LT treadmill test in which 3-min stages were used, to see whether the HR deflection point accurately predicts lactate threshold (LT). In this study, all subjects demonstrated HR deflection on the track, but only one-half of the subjects showed HR deflection on the treadmill. The researchers concluded that the HR deflection point was not an accurate predictor of LT.

Petit et al (60) examined the relationship between ventilatory threshold (Tvent) determined from a laboratory test and heart rate deflection (HRd) from the Conconi test, to validate a mathematical model (MM) that evaluates the Conconi test and predicts 10-km race time. These researchers found a significant relationships between velocity at Tvent and HRd (r = .95, p < .01), and predicted times from each method (r = .96, p < .01). Hoffman et al (61) investigated 227 young subjects, using the method of Conconi et al. (54) and observed 85.9% of the subjects showed a "regular" deflection, 6.2% showed no deflection at all, and 7.9% showed even an inverted deflection of the heart rate performance curve (HRPC). Among all the endurance athletes, the Conconi test was observed not to be very relevant for determining anaerobic threshold of rowers (62). Ballazin et al (63) observed that the speed and heart rate of deflection values assigned visually by observers were compared to the values obtained through mathematical analysis of the tests by computer. Straight-line equations, correlation coefficients (r), and technical errors of measurement (TEM) obtained by comparing visually determined data to those determined through mathematical analysis were calculated for each observer. Differences were found between the observerassigned and computer-determined results for both speed and heart rate of deflection. The study highlighted that visual analysis provides information that is very similar to that obtained through computer analysis and the accuracy of the visually obtained information varies according to the observer's experience.

Endurance Performance and Anaerobic Threshold

In the exercising human, maximal oxygen uptake (VO₂max) is limited by the ability of the cardiorespiratory system to deliver oxygen to the exercising muscles. This is shown by three major lines of evidence: 1) when oxygen delivery is altered (by blood doping, hypoxia, or beta-blockade), VO, max changes accordingly; 2) the increase in VO_2 max with training results primarily from an increase in maximal cardiac output (not an increase in the a $v O_2$ difference); and 3) when a small muscle mass is overperfused during exercise, it has an extremely high capacity for consuming oxygen. Thus, O₂ delivery, not skeletal muscle O2 extraction, is viewed as the primary limiting factor for VO₂max in exercising humans. Metabolic adaptations in skeletal muscle are, however, critical for improving submaximal endurance performance. Endurance training causes an increase in mitochondrial enzyme activities, which improves performance by enhancing fat oxidation and decreasing lactic acid accumulation at a given VO₂. VO₂max is an important variable that sets the upper limit for endurance performance (an athlete cannot operate above 100% VO₂max, for extended periods). Running economy and fractional utilization of VO₂max also affect endurance performance. The

speed at lactate threshold (LT) integrates all three of these variables and is the best physiological predictor of distance running performance (64).

Several attempts have been made to predict endurance ability of the athletes, particularly in continuous events like long distance running. Farrell et al (65) reviewed that the anaerobic threshold yielded highest correlation (r = 0.98) with marathon running performance. Kumagai et al (66) observed a higher correlation between 5000 and 10,000 meters running performance and lactate threshold (r = 0.95and 0.84) than between running performances with the VO₂ max (r = 0.65 and 0.67). Sjodin and Jacob (67); Karlsson and Jacob (68) related 4mM/L (OBLA) lactate to endurance performance and reported a high correlation between them (r = 0.96). It is very much evident from the earlier observation that in elite runners, the onset of blood lactate accumulation (OBLA) occurred at a higher running speed (65, 69, 70). Besides continuous events, the importance of anaerobic threshold in non-continuous intermittent games has also been discussed (40, 71, 72).

Yoshida et al (73) observed the relationships between running velocity (v) in a 3000-m race and various physiological parameters. The parameters measured among 57 female distance runners during a treadmill running test were v at the lactate threshold (v-Tlac), oxygen uptake (VO_2) at the lactate threshold (VO₂ at Tlac), v at the onset of blood lactate accumulation (v-OBLA), VO₂ at OBLA, running economy (steady-state VO₂ at a standard v of 4 m s⁻¹), maximal oxygen uptake (VO₂ max) and v at VO₂ max (v-VO₂ max). The v-OBLA revealed highest correlation with v over 3000-m race (r = 0.78, P < 0.001), followed by the same with v-Tlac (r = 0.77, P < 0.001). Although v-VO₂ max was strongly correlated with v over 3000 m (r = 0.75, P < 0.001), further analysis by stepwise multiple regression indicated that a combination of v-OBLA, VO₂ at Tlac and v-Tlac could account for 73.2% of the variability in v over 3000 m, whereas v-OBLA on its own explained only 61.5%. Blood lactate variables can account for a reasonably large part of the variance in v over 3000 m. Even, v-VO₂ max can be used as a non-invasive predictor of distance running performance. LT appears consistent with the ventilatory threshold (V_T) described by the V slope method (19). Hence, LT and V_{T} may be independent of each other, but there is a link between ventilatory changes and cellular events. It may be suggested that any point consistently used from the lactate concentration curve during exercise may be used as

a performance index (56) in long duration continuous events.

Training and Anaerobic Threshold

Training also has different effects on blood lactate concentration and ventilatory threshold. Three weeks of cycling endurance training reduces blood lactate concentration without affecting the ventilatory threshold (74). The type of training programme also affects the dissociation between blood lactate threshold and ventilatory threshold. Interval training can increase the time to both blood lactate threshold and ventilatory threshold, but endurance training can delay the onset of venous blood lactate threshold, showing less effect on ventilatory threshold. In fact, there was not a significant correlation (r = 0.13) in the time of onset of the two variables between any of the above different training protocols (75). Simon et al (76) demonstrated that in sedentary males, ventilatory threshold (51% of VO, max) occurred significantly before blood lactate threshold (62% VO, max), whereas, in trained males the onset of ventilatory threshold (66% VO₂ max) was not different from blood lactate threshold (69% of VO, max). Furthermore, Denis et al, (78) investigated the effect of a 40 week endurance training programme on ventilatory and lactate thresholds and lactate threshold at 4 mM/L. In comparing the changes over the 40 week period, the threshold level changes by 10, 15 and 18% respectively. All correlations between the 3 threshold intensities over the 40 week period show significance with a ventilatory and lactate threshold correlation of r = 0.79 (p<0.001) [Table 1]. This gives further evidence that the threshold prescribed by ventilatory parameters (departure from linearity in $V_{\rm F}$) and lactate measures (onset of and abrupt increase in blood lactate) are associated and causally linked.

In long term high intensity exercise the concentration of muscle glycogen may be a limiting factor (79, 80). Exercise just below the AT will result in a much slower depletion of muscle glycogen reserves than exercise above AT level and would therefore be tolerable for much longer period of time. On the other hand, Boyd et al (81) demonstrated that elevation in blood lactate concentration inhibits lipolysis in exercising man and thus force obligatory carbohydrate utilization. This indicate that the training at AT level is suitable for endurance athletes. Training at or near anaerobic threshold level result in change in muscle ultrastructure (82), capilarization (83), oxidative capacity (84) and

substrate utilization (85). The benefit which results from the physiological adaptations to anaerobic training include decrease in O_2 cost for ventilation, the lactate accumulation and depletion of glycogen at a given power output. The end result is the rise in the intensity of efforts that can be sustained aerobically.

In endurance sports, it has been suggested that AT might be a better indicator of aerobic endurance than VO₂ max, as AT may change without changes in VO, max (87, 88). Several studies have demonstrated that in the general population, aerobic training often improves the exercise intensity corresponding to anaerobic threshold without a concomitant increase in VO₂max (88, 89). In team events, like soccer, training at lactate threshold (LT) and ventilatory anaerobic threshold (V_{T}) improved the anaerobic threshold level, while the VO₂max remained unchanged (90). VO₂ at LT as well as at V_T was also improved. There was no significant difference in VO_2max . VO_2max is a less sensitive indicator to changes in training status in professional soccer players than either LT or V_{T} (90). OBLA has been widely used to identify changes in training state, however, it's use has been criticised due to variability between subjects (91) and also because it may be a result of not only muscle anaerobisis, but also a decreased total lactate clearance or increased lactate production in specific muscles (92). VO₂max may provide a useful indicator of the aerobic capacity of the athlete, but it's use is limited in the ongoing process of monitoring changes in training state. Submaximal LT or V_{T} may identify changes in aerobic conditioning.

The effects of the of five week interval training on metabolic parameters at maximal work and at the anaerobic threshold in 11 year old children have been studied, using interval work at 25 and 50% above the anaerobic threshold (93). Following training, the children increased their anaerobic threshold (expressed as %VO₂max) significantly (P < 0.05), by 22%, while oxygen uptake (VO₂, l/min) was increased by 19%, but the difference was not significant (P greater than 0.05). Burke et al (94) observed that a 7 week high intensity aerobic interval-training programme (at 80%, 90% and 95% VO₂max) can produce significant changes in VO₂max, lactate threshold and ventilatory anaerobic threshold and these changes appear to be independent of the length of the work interval. Training at 80% VO₂max for 9 weeks duration can increase the anaerobic threshold level significantly. Even a similar duration of detraining can deteriorate the same (95). Training at little above anaerobic threshold level is more effective in improving the lactate threshold VO_2 than training at anaerobic threshold level (96).

Interval training involves repeated short to long bouts of high intensity exercise (at or little above lactate threshold intensity) combined with active or passive recovery periods. Interval training was first described by Reindell and Roskamm and was popularised in the 1950s by the Olympic champion, Emil Zatopek (97). The most important aspect is to assess the velocity or running speed at this lactate threshold level, rather than the VO₂ at LT. The middle and long distance runners often use this technique to train at velocities close to their own specific competition velocity. In fact, trainers have used specific velocities from 800 to 5000m to calibrate interval training without taking into account physiological markers (97). However, in off season training camps, it is better to refer to the velocities associated with particular physiological responses in the range from lactate threshold to the absolute maximal velocity. The range of velocities used in a race must be taken into consideration, since even world records are also not run at a constant pace. It is better for a middle or long distance runner to possess a high VO₂max, but the velocity of running at VO₂max (v-VO₂ max) is the first criterion (98, 99), while velocity at lactate threshold can also not be underestimated (100).

Conclusions

The present review has highlighted the importance and role of anaerobic threshold in endurance sports. Physiological terms like, individual anaerobic threshold, lactate threshold, ventilatory anaerobic threshold, heart rate deflection point, onset of plasma lactate accumulation, onset of blood lactate accumulation, maximum lactate steady state, denote the aerobic to anaerobic transition level, where lactate appearance in the blood from muscle is equal to the disappearance rate of lactate from the blood. This intensity is highly correlated with distance running performance and even training at or a little above this intensity is effective in improvement of anaerobic threshold not only in elite athletes, but also in sedentary population. Above this intensity, the appearance rate of lactate from the muscle to the blood exceeds the disappearance rate of lactate from the blood to the muscle. Both the continuous and interval type of physical training at anaerobic threshold intensity

have beneficial effect on improvement of not only VO_2 max but also on the anaerobic threshold level. A field method of assessing the anaerobic threshold is available and can be done from heart rate deflection point in speed-heart rate relationship with accuracy either by visual method or by computer programming.

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