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exercise and the heart

Dangerous Curves*

A Perspective on Exercise, Lactate, and the Anaerobic Threshold

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Abbreviations: EMG=electromyogram; LDH=lactate dehydrogenase; NADH=the reduced form of nicotinamide adenine dinucleotide; RER=respiratory exchange ratio; VCO₂=carbon dioxide output; VO₂=oxyen consumption

“If, then, life is an action of the soul and seems to be greatly aided by respiration, how long are we likely to be ignorant of the way in which respiration is useful?”

Galen of Pergamum
Circa 130-201 AD

Though it is unlikely the Greek physician Galen had the anaerobic threshold per se in mind, he may have been the first to document something of relevance to it. The increase in blood lactate level that occurs in response to progressive exercise, and changes in ventilation that are associated with it, have engendered a great deal of interest from coaches, athletes, clinicians, and educators for most of this century. Few areas in the exercise sciences have generated as many scientific reports, editorials, or debate. Among the issues in dispute include the mechanisms responsible for blood lactate increase, the pattern of the lactate response to exercise, and the ability to choose a “threshold” (including intraobserver and interobserver reliability and reproducibility, methods of choosing a threshold, and the relation of lactate appearance to ventilatory changes during exercise). Recent reports have even questioned the existence of an “anaerobic” threshold, a concept that some consider heretical. The anaerobic threshold continues to be used widely in the clinical setting, but many clinicians remain confused as to how to interpret the lactate response to exercise. A wide gap exists between the physiology and clinical literature in regard to this issue. The following summarizes recent studies related to the lactate response to exercise and provides a perspective in the context of clinical exercise testing.

Historical Perspective

The concept that muscle contraction can occur for extended periods of time without sufficient oxygen supply may have been first suggested by Hermann in 1871. As early as 1909, Douglass and Haldane surmised that lactate stimulated respiration during high-intensity exercise. Between 1910 and 1915, Meyerhof and Hill suggested that lactate was the “signal” that initiated muscle contraction, and the role of oxygen was to remove lactate once it was formed (it is now known, of course, that lactate is the product of, not the cause of, muscle contraction). Shortly thereafter, several other laboratories in the United States and Europe independently reported that exercise intensity, lactate production, metabolic acidosis, and bicarbonate buffering appeared to be linked. The accumulation of lactate in the blood was thought to reflect a point during exercise in which oxygen supply was inadequate to meet the energy requirements of the working muscle. In the early 1960s, Hollmann and colleagues linked lactate production in the blood to endurance performance among German athletes. In 1964, Wasserman and McIlroy explicitly developed the concept that a critical threshold exists in which (1) the metabolic needs for oxygen in the muscle exceed the capacity of the cardiopulmonary system to supply them, (2) there is a sudden increase in anaerobic metabolism, and (3) lactate is formed in the muscle.

In recent years, however, evidence has accumulated that dissociates lactate production from anaerobiosis. Studies performed over the last 15 years using isotopic tracer technology have suggested that

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muscles can release lactate when their oxygen supply is more than adequate. Moreover, evidence that muscles become anaerobic when exercise approaches an intensity in which blood lactate level rises precipitously is in dispute. The formation of lactate appears to depend on several factors, including but not limited to the availability of oxygen. In the following, current perspectives on lactate control, lactate as a substrate, and recent studies on the pattern of the lactate response to exercise are reviewed along with their clinical implications.

**CONTROL OF LACTATE PRODUCTION**

The relationship between the increase in blood lactate and oxygen supply to the muscle has a long history. Studies performed early in this century closely linked exercise intensity, lactate appearance, metabolic acidosis, and bicarbonate buffering. These associations provided a convenient and logical explanation for lactate production during exercise. Numerous studies have supported the link between oxygen supply and the magnitude of the increase in blood lactate, albeit without direct evidence of cause and effect. For example, the extent to which lactate increases in the blood during exercise is reduced as fitness increases or with training and is increased in the presence of reduced cardiac output states or disease of the electron transport chain. When oxygen supply is experimentally altered, blood lactate is profoundly affected. With an increase in inspired oxygen tension, either by increasing the oxygen content of the inspired air or by increasing the barometric pressure, the exercise-induced increase in lactate level is attenuated. Decreasing the fraction of inspired oxygen either experimentally or by exposure to altitude increases blood lactate.

In normal subjects, reducing blood volume causes an increase in blood lactate level during exercise. In patients with chronic heart failure, increasing cardiac output with inotropic therapy attenuates the increase in blood lactate during exercise.

Despite these compelling observations, manipulations in oxygen supply that affect the relationship between oxygen availability and lactate do not prove causation, and the concept that the availability of oxygen is the sole determinant of lactate production has been highly controversial. The idea that lactate production is dependent solely on oxygen supply to the tissue is not consistently supported by the available data. It is now appreciated that lactate can be formed under conditions that are fully aerobic, findings which appear to negate the concept that anaerobiosis per se causes lactate production. An observation that has perplexed many in this debate is that, between normoxic and hypoxic conditions, oxygen consumption (VO2) remains unchanged (suggesting that O2-limited metabolism is not present) but lactate level is increased. The picture is analogous to that before and after athletic training, in which lactate level is generally lower for any given submaximal work rate while VO2 is unchanged (although Katz and Sahlin argue that this does not take into account adaptive changes in the muscle).

Katz and Sahlin summarized experimental data and theoretical reasons supporting the concept that lactate production is O2 dependent. In effect, they argue that when oxygen supply is limited, mitochondrial respiration is stimulated by increases in adenine diphosphate, inorganic phosphate (Pi), and the reduced form of nicotinamide adenine dinucleotide (NADH). This favors the stimulation of glycolysis, which will increase cytosolic NADH formation, which will shift the lactate dehydrogenase (LDH) equilibrium toward increased lactate production. While they acknowledge that lactate production is also dependent on the glycolytic and mitochondrial respiration rates as well as LDH, they propose that oxygen supply has the most pivotal role in lactate production. Connett and colleagues describe an alternative scenario in which O2 “deficiency” (O2-limited cytochrome turnover) is characterized by the following: (1) it can be defined only by specifying adenosine triphosphate demand; (2) it is dependent on aerobic capacity relative to glycolytic capacity in a specific tissue; and (3) a critical Po2 exists for a given tissue, and the specific PO2 depends on the supply of substrate to the mitochondrial and glycolytic subsystems, along with the redox state. These metabolic compensations depend on cell Po2, but the glycolytic rate is not directly coupled to O2 supply. Lactate accumulation may occur both above and below a critical Po2 since it depends not only on the glycolytic rate but also on exchange across the cell membrane and consumption in neighboring cells. These investigators have observed lactate formation at low levels of exercise (<10% VO2 max) in dog gracilis muscle (a purely aerobic, red muscle fiber). They argue that lactate formation cannot be due to an O2 limitation, and that the anaerobic threshold cannot apply to red muscle.

These two sources seem to directly contrast one another. In effect, however, both Katz and Sahlin and Connett et al suggest that with increasing work loads, a situation must arise whereby metabolic change within the cell occurs in order for the cell to maintain full aerobic (mitochondrial) function. This change is an increase in the mitochondrial NADH/NAD ratio (ie, the redox potential increases), which in turn is transmitted to the cytosol.
where it pushes the lactate/pyruvate ratio in the direction of lactate. Whether this is labeled anaerobic metabolism or metabolic change does not alter its presence, which is agreed on by all.

Other Factors Controlling Lactate Production

Brooks, Stainsby and Brooks, and others have suggested that β-adrenergic stimulation of muscle glycogenolysis is an important factor controlling lactate production. This is evidenced by high correlations between catecholamine concentration and blood lactate, by similar threshold responses for lactate and catecholamines, by studies demonstrating that infusion of epinephrine elicits an increase in blood lactate, and by the observation that β-blockade causes a reduction in blood lactate during exercise. Mazzeo and Marshall have shown that the inflection point during incremental exercise for plasma epinephrine shifts in an identical manner and simultaneously with lactate from cycling to treadmill running. Podolin and associates extended these findings by observing simultaneous inflections in lactate, epinephrine, and norepinephrine in both normal muscle glycogen and glycogen-depleted states. Weltman et al recently contrasted these findings by observing similar norepinephrine and epinephrine thresholds between running and rowing, but the lactate threshold occurred at a lower oxygen uptake (7 and 10% lower for running and cycling, respectively). Weltman et al suggest that it may not be an epinephrine threshold per se that underlies the lactate threshold, but rather a critical plasma epinephrine level.

An additional persuasive argument put forth involves the association between the increase in lactate level and the progressive recruitment of ever more aerobically scarce motor units (culminating in the recruitment of extreme IIB fibers which, as a result of their aerobic paucity, steadily release H⁺ ions and lactate irrespective of PO₂). Some evidence also exists correlating the percentage of type I fibers with the lactate threshold. However, it could equally be that the recruitment of IIB fibers is a consequence of the increase in H⁺ rather than a cause. A decrease in intracellular pH interferes with the contraction-coupling mechanism and its ability to maintain force. To compensate, more IIB fibers are recruited resulting in greater glycogenolysis and lactate production. Underlying the role of muscle fiber types is the fact that the isozyme form of LDH, which converts lactate to pyruvate and vice-versa, differs between muscle fibers. Fast-twitch fibers have an LDH isozyme form that favors the formation of lactate, whereas the opposite is true for slow-twitch fibers, and different individuals have different percentages of fiber types.

Lactate as a Substrate

Long considered to have only toxic effects, an appreciation for lactate as a metabolic substrate has emerged in recent years. This is due in large part to the application of isotopic tracer technology to the study of metabolism during exercise. It has become clear from these studies that the skeletal muscles actively produce and consume lactate during exercise. It is now recognized that a significant portion of the lactate produced and released into the blood during exercise is taken up by tissues other than those from which it was formed. In fact, these studies suggest that during exercise, lactate may be the predominant fuel for the heart, and the preferred fuel for slow-twitch fibers. Lactate also appears to be an important intermediate in glycogen formation by the liver, and a precursor for liver gluconeogenesis. Lactate formation occurs even at rest, and during exercise, lactate production and removal are highly correlated to the metabolic rate.

Role of Lactate in Fatigue

A misconception that remains pervasive is the perception that lactate is the major cause of both fatigue during exercise and postexercise muscle soreness. In fact, lactate has little direct effect on either. Accumulation of lactate in the muscle occurs only during short bouts of exercise of relatively high intensity. Even after endurance events lasting several hours, lactate levels in the blood are near their levels at rest. Moreover, when lactate is infused into the blood, it has minimal noticeable effects. Rather than lactate causing fatigue per se, it is the accumulation of H⁺ ions during glycolysis that contributes to fatigue. High concentrations of H⁺ lower the blood’s pH which adversely influences energy production and muscle contraction. Energy production is affected by the inhibition of phosphofructokinase, a key glycolytic enzyme. In fact, at a pH of 6.4, glycolysis can cease completely. Excessive H⁺ ions affect muscle contraction by displacing calcium within the muscle fiber, interfering with actin-myosin cross-bridge formation and reducing contractile force. During short-term maximal exercise, this is the major factor limiting performance. During longer endurance-type exercise, fatigue is primarily related to energy supply, the most important factor being the depletion of muscle glycogen.
Because lactate is so rapidly removed from the muscle and blood after exercise, the assumption that lactic acid causes muscle soreness is also incorrect. Even extremely high blood lactate levels return to normal within an hour after intense exercise. Interestingly, most muscle soreness occurs after endurance exercise performed below an intensity required to illicit the accumulation of lactate in the blood. Electron microscopy and other studies have demonstrated that muscle soreness is almost certainly due to muscle cell microdamage and inflammation.

**Ventilation**

The idea that ventilatory variables may be linked to these metabolic processes also has a long history. Recent studies have made the long-held cause and effect relationship between lactate accumulation and ventilatory changes another source of debate. In 1975, Wasserman and associates appeared to confirm what had been widely held for many years by observing an absence of a ventilatory threshold among subjects whose carotid bodies had been resected. In the absence of peripheral arterial chemoreceptors to detect changes in pH caused by lactic acid production, no ventilatory changes during exercise were observed. In the 1980s, however, a wide variety of experimental manipulations have raised questions about how closely ventilatory processes mirror metabolic ones. For example, numerous studies have demonstrated that the ventilatory threshold can be detected prior to the lactate threshold during progressive exercise. In addition, nonlinear increases in ventilation have been observed among subjects who do not produce lactate (McArdle’s syndrome). Others have demonstrated that relative to control conditions, ventilation either increases or remains constant among normal subjects in a glycogen-depleted state, in which both lactate and carbon dioxide partial pressure are reduced. Together, these studies suggest that stimulation of peripheral chemoreceptors by lactic acid cannot be solely responsible for the ventilatory threshold.

Some compelling evidence has come from recent studies on potassium. Paterson has made a strong case for potassium as an important humoral factor in the regulation of exercise ventilation. It is known, for example, that in the anesthetized cat, hyperkalemia stimulates ventilation by excitation of the carotid body chemoreceptors and that denervation of these receptors abolishes this effect. It appears also that for McArdle’s subjects and in conditions of glycogen loading, the plasma potassium level tracks ventilatory changes better than lactate—not only during exercise but also in the recovery period (when lactate levels continue to rise). Despite indications that the picture is not entirely clear cut (e.g., the effect of β-adrenergic blockade, which should, by enhancing exercise-induced hyperkalemia, increase ventilation but fails to do so and the contradictory findings of McLoughlin et al). It is clear that to ignore the role of humoral factors other than lactate and H⁺ is to seriously misrepresent the true picture. For some, the safest course for those who choose to use a threshold model is to refer to two separate but associated thresholds, one for ventilation and one for lactate.

Evidence also exists that the lactate threshold correlates well with an electromyogram (EMG) “threshold” (abrupt increases in the frequency bandwidth at 70% of the peak frequency) and the integrated EMG, and that a gas exchange threshold correlates well with an EMG “fatigue threshold.” These findings have raised the possibility that an increase in neural activity, originating from higher motor centers or the exercising muscle, may contribute to the stimulation of ventilation. Mateika and Duffin attenuated peripheral chemoreceptor activity with hyperoxic breathing, and observed coincident ventilatory and EMG thresholds during exercise. These investigators have also shown that, during normoxic breathing, EMG and ventilatory thresholds occur at similar exercise intensities, whereas the lactate and ventilatory thresholds are uncoupled. These data suggest that changes in lactate concentration and thus peripheral chemoreceptor drive are not strictly responsible for the ventilatory threshold, but rather, the ventilatory threshold is mediated by alterations in neural activity that occur in conjunction with motor unit recruitment.

There has also been an evolution in recommended techniques for measuring the ventilatory threshold. Over the years, there have been at least a dozen criteria recommended for detecting a breakpoint in ventilation, including the following: (1) an increase in the respiratory exchange ratio (RER); (2) an abrupt increase in the fraction of expired oxygen; (3) a nonlinear increase in ventilation; (4) a nonlinear increase in CO₂ production; (5) an increase in end-tidal O₂ partial pressure; (6) a nonlinear increase in the integrated EMG activity; (7) the point at which the relationship between running speed and heart rate departs from linearity; and (8) the beginning of a systemic increase in the ventilatory equivalent for oxygen (Ve/VO₂) without an increase in the ventilatory equivalent for CO₂ (Ve/VCO₂). Early reports suggested that the RER was useful since, as an expression of the slope of carbon dioxide output (VCO₂) to VO₂, a value greater...
than 1.0 should reflect a nonlinear increase in CO₂ production. Later studies suggested the RER was comparatively insensitive to changes in blood lactate levels.⁸⁰,⁸¹ There have been numerous efforts to address the subjective nature of determining an "abrupt increase." Problems have been raised repeatedly concerning interobserver and intraobserver reliability and reproducibility.⁸⁷,⁹¹,⁹² and there have been many computerized efforts to improve the detection of a ventilatory break point with mixed success.⁹⁷,⁹⁸,⁹³,⁹⁶

Caiozzo and associates⁹¹ compared various ventilatory indexes with lactate changes and recommended an "increase in Ve/VO₂ with no change in Ve/VCO₂." This removed some of the subjectivity because the ventilatory equivalent for O₂ would typically decrease initially in an incremental test before beginning a systematic increase. The "no change in the ventilatory equivalent for CO₂" was included to guard against the somewhat erratic nature of exercise hyperventilation. It was apparent, however, that while this method removed much of the subjectivity, it was not discernible in all subjects and could still be affected by the multitude of other factors influencing exercise ventilation. Thus, in 1986, Beaver et al⁹⁵ came full circle in defining a new criterion named the "V-slope." This was essentially the point where the slope of the VCO₂ and VO₂ curve became >1.0. In theory, such a point would be more reliable since it depends only on the bicarbonate buffering response to lactate, and is independent of respiratory chemoreceptor sensitivity and thus the ventilatory response to exercise. In a stroke, they effectively removed observer bias from the measurement. Even more important, however, the choice of an inflection point was not affected by the choice of model used to fit the data. Despite the assertion that the choice of the model is "fundamental to the understanding of exercise energetics,"⁹⁸ whether one takes a tangent to a continuous curve or a set square to a threshold curve, the result will be the same (more on this below). The measurement is reproducible in a way that none of the others are and, not surprisingly, it is discernible in most subjects. Further, although this is not absolutely the case, the fact that ventilation generally cancels out on both axes means that the measure is relatively independent of the vicissitudes of exercise ventilation.

**Pattern of the Lactate Response**

The concept that a "threshold" exists, in which there is a sudden onset of anaerobic metabolism resulting in the accumulation of lactate in the blood, proposed by Hill and coworkers⁸ in the 1920s and popularized by Wasserman et al.¹⁵,¹⁸,⁷⁹ has also been challenged. Recently, this issue has generated some rather lively debate. Studies by Gladden et al.⁸³ and Yeh and associates⁹² raised questions about the subjective intraobserver and interobserver reliability and reproducibility of ventilatory and lactate thresholds. Concerns over the presence or absence of a discrete threshold point led Beaver et al.⁹⁵ to suggest the use of a log-log transformation (ie, plotting oxygen uptake, lactate, or CO₂ production on logarithmic axes). These investigators reported that lactate exhibited an abrupt transition from a slowly increasing phase to a rapidly accelerating phase, findings consistent with the historic interpretation of the anaerobic threshold. Despite its wide adoption, however, such an idea was unacceptable to many⁹⁴-⁹₈,⁹₀₂ since among other things, a log transformation could "create" a visually apparent break-point where none existed before, by spreading out the early data points over the X-axis relative to the later ones.

Five recent studies have used mathematical modeling to describe the relationship between oxygen uptake and lactate during exercise, and addressed whether the increase in lactate level was better represented as a continuous (smooth) function or the data depicted a true threshold. Yeh et al.⁹² used semilog plots of arterial lactate vs time, and reported that an exponential increase in lactate level occurred during exercise without a threshold. Hughson and coworkers⁹⁹ compared the log-log transformation model of Beaver et al.⁹⁵ with an exponential plus constant model. These investigators observed that changes in blood lactate levels during progressive exercise were more appropriately described mathematically as a continuous function, implying the absence of a threshold. This was suggested by a mean squared error term for the continuous model that was 3.5 times lower than that for the threshold model described by Beaver and associates.⁹⁵ Similar observations were made by this group over a variety of ramp rates.¹₀¹ Dennis et al.¹₀² corroborated the findings of Hughson and coworkers,⁹⁶,¹₀¹ reporting that plots of ventilation, CO₂ production, and blood lactate vs oxygen uptake were more appropriately described by continuous rate laws rather than threshold linear equations. We considered some of the subsequent adversarial criticisms of these studies (ie, insufficient number of subjects, inconsistent or overly demanding ramp rates, subjectivity, frequency of data sampling)⁹₄ and found that a computerized threshold model fit the data slightly better than a continuous model. However, the differences were quite small, leading to the conclusion that (1) a meaningful difference did not exist between the
models, or (2) these models may not be capable of detecting a difference, if one exits.

Morton's has added significantly to this debate, arguing among other things, that (1) researchers have sought to model the data and not the process that produces the data, and (2) these studies have ignored the importance of the distinction between the lactate response and its first derivative. Other arguments concerning modeling of lactate no doubt serve only to confuse the average clinician not at ease with the idiosyncrasies of these mathematics. In retrospect, however, many of the points made in regard to modeling the response may be academic. It matters little, for example, that when investigators used the term “threshold system” they actually meant “a system which produces data possessing a discontinuous first derivative,” when in fact a “threshold system” mathematically defined can result in data with either a continuous or discontinuous first derivative. What Wasserman and associates originally intended (and others undoubtedly understood) was that these variables showed a continuous, kinked response—a clear point of abrupt increase in slope—and it was this interpretation which was disputed.

It is important that cross-disciplinary terminology be both consistent and appropriate, but this does not change the substance of the physiologic debate. Phenomenologic modeling (modeling the data rather than the system) is appealing to physiologists because its meaning is readily understood. The practical constraints of clinical exercise testing call for fast and meaningful answers for physician and patient. This is not to discount the substantial contribution that mathematical modeling has made to the scientific debate, but merely to emphasize that a good understanding of others’ fields of interest is a prerequisite for good cross-disciplinary research. What is clear is that all functions fit the data well, as would be expected for monotonic, smooth data, and one must question the physiologic significance of small differences in the fit of statistical models.

Summary

A number of general observations can be made from these recent studies. Lactate is a ubiquitous substance that is produced and removed from the body at all times, even at rest, both with and without the availability of oxygen. It is now recognized that lactate accumulates in the blood for several reasons, not just the fact that oxygen supply to the muscle is inadequate. Lactate production and removal is a continuous process; it is a change in the rate of one or the other that determines the blood lactate level. Rather than a specific threshold, there is most likely a period of time during which lactate production begins to exceed the body’s capacity to remove it (through buffering or oxidation in other fibers). It may be appropriate to replace the term “anaerobic threshold” to a more functional description, since the muscles are never entirely anaerobic nor is there always a distinct threshold (“oxygen independent glycolysis” among others has been suggested) Lactate plays a major role as a metabolic substrate during exercise, is the preferred fuel for slow-twitch muscle fibers, and is a precursor for liver gluconeogenesis.

The point at which lactate begins to accumulate in the blood, causing an increase in ventilation, is important to document clinically. Irrespective of the underlying mechanism or specific model that describes the process, the physiologic changes associated with lactate accumulation have significant import for cardiopulmonary performance. These include metabolic acidosis, impaired muscle contraction, hyperventilation, and altered oxygen kinetics, all of which contribute to an impaired capacity to perform work. Thus, any delay in the accumulation of blood lactate which can be attributed to an intervention (drug, exercise training, surgical, etc) may add important information concerning the efficacy of the intervention. A substantial body of evidence is available demonstrating that lactate accumulation occurs later (shifting to a higher percentage of VO₂max) after a period of endurance training. In athletes, the level of work that can be sustained prior to lactate accumulation, visually determined, is an accurate predictor of endurance performance. Presumably, these concepts have implications related to vocation/disability among patients with cardiovascular and pulmonary disease, but few such applied studies have been performed outside the laboratory. Blood lactate during exercise and its associated ventilatory changes maintain useful and interesting applications in both the clinical exercise laboratory and the sport sciences. However, the mechanism, interpretation, and application of these changes continue to rely more on tradition and convenience than science.

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