The Transition from Aerobic to Anaerobic Metabolism

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There is little or no doubt about the primary metabolic processes involved in very brief, high-intensity exercise (phosphagen depletion and/or anaerobic glycolysis) or in prolonged, low-intensity exercise (oxidation). There is some controversy, however, about the relative importance of each during the transition from aerobic to anaerobic metabolism and about where this transition occurs. Since these questions have theoretical and practical implications, an attempt will be made here to:

1. Summarize the events occurring during exercise of progressively increasing intensity
2. Construct a hypothetical model to explain what seems to be occurring at various phases
3. Suggest terminology to clarify and possibly reduce the controversy
4. Discuss how different factors can affect measurements taken during progressive exercise
5. Provide insight about the practical application of this knowledge for training and research.

Course of Events During Progressive Exercise

There appear to be three phases during the progressive transition from exercise of low to maximal intensity. Figure 1 is a schematic representation of typical changes occurring in blood lactate (La), heart rate (HR), and various measures of gas exchange during progressive exercise.

Phase I. With increasing levels of low-intensity exercise, a greater amount of oxygen is being extracted by the tissues, resulting in a lower fraction of oxygen in the expired air (F_EO_2). As well, more CO_2 is being produced and expired (F_ECO_2). There is also a linear increase in oxygen intake ($\dot{V}O_2$), ventilation ($\dot{V}_E$), volume of CO_2 expired ($\dot{V}CO_2$), and HR. Since little or no La is formed and values of .7–.8 for the respiratory quotient (R or $\dot{V}CO_2 \cdot \dot{V}O_2^{-1}$) are found during this low-intensity, steady-state exercise, there is little doubt that this first phase primarily involves aerobic metabolism.

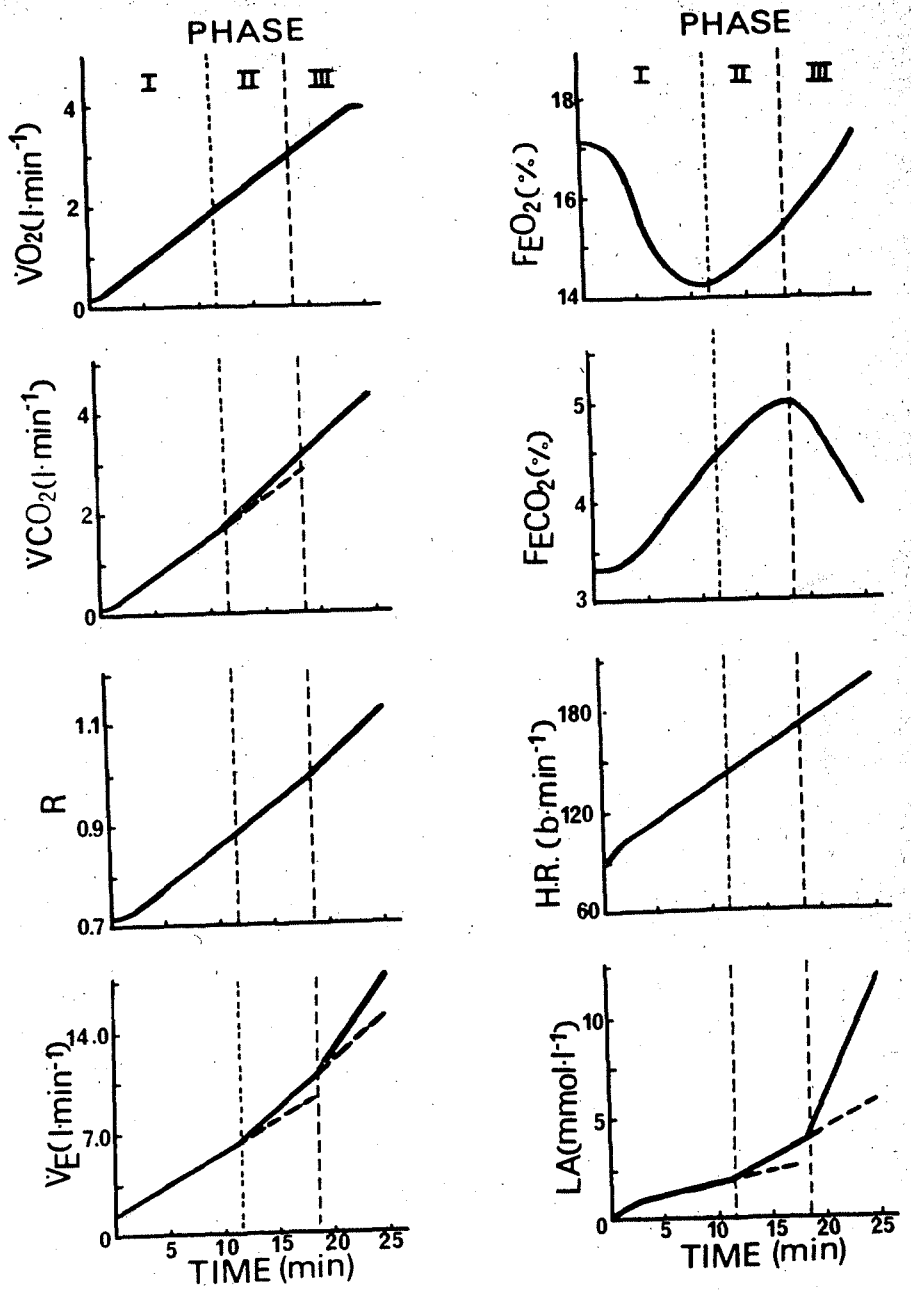

Figure 1—Schematic representation of typical changes in blood lactate, heart rate and selected gas exchange parameters during progressive exercise from rest to maximal oxygen consumption.

Phase II. As the exercise intensity increases and reaches a point between 40% and 60% $\dot{V}O_2$ max, $\dot{V}O_2$ and HR continue to rise linearly and there is an initial rise in La to twice resting values (about 2 mmol·l^{-1}). The acidity (H^+) produced by La is principally buffered by the base bound as bicarbonate (HCO_3^-) (Bouhuys, Pool, Binkhorst, & Van Leeuwen, 1966; Turrel & Robinson, 1942), resulting in an increased production of CO_2 from the dissociation of carbonic acid (H_2CO_3) and a continuous rise in F_ECO_2 (i.e., $H^+ + HCO_3^- \rightleftharpoons H_2CO_3 \rightleftharpoons H_2O + CO_2$). In an attempt to compensate for the impending metabolic acidosis due to higher levels of La and CO_2, the respiratory center is stimulated to increase $\dot{V}_E$; the combined effect of a higher $\dot{V}_E$ and a higher level of CO_2 in the blood produces a higher $\dot{V}CO_2$. Since the La rises to a value lower than approximately 4 mmol·l^{-1} during this second phase, this respiratory compensation appears reasonably effective.

The rise in $\dot{V}_E$ and in $\dot{V}CO_2$ is greater than the rise in $\dot{V}O_2$, producing a disproportionate increase in $\dot{V}_E \cdot \dot{V}O_2^{-1}$ and R. As well, since the body does not consume more O_2 than is needed to replace the ATP utilized, the extra increase in $\dot{V}_E$ results in a lower extraction of O_2 per volume of air ventilated and there is a corresponding rise in F_EO_2. Therefore, the onset of Phase II is characterized by a nonlinear increase in $\dot{V}_E$ and $\dot{V}CO_2$, an increase in F_EO_2 without a corresponding decrease in F_ECO_2, plus a rise in blood La from approximately 2 mmol·l^{-1}. This onset corresponds to the anaerobic threshold described by Wasserman, Whipp, Koyal, & Beaver (1973).

Phase III. With further increases in intensity to about 65–90% $\dot{V}O_2$ max, the linear rise in $\dot{V}O_2$ and the HR continues until near-maximal work loads (WL), at which time they begin to plateau. At the onset of this phase, blood La is around 4 mmol·l^{-1} and then increases more rapidly until the subject attains his $\dot{V}O_2$max. There is also a further increase in $\dot{V}_E$ and a continuous rise in $\dot{V}CO_2$ in an attempt to compensate for the marked rise in La. At this point, however, the hyperventilation cannot compensate adequately and there is a drop-off in F_ECO_2, while F_EO_2 continues to rise. In addition, more and more of the $\dot{V}O_2$ has to go to the respiratory muscles for the increased work of hyperventilation and less is available for the skeletal muscles performing near-maximal work. The onset of Phase III is thus characterized by a sharp rise in La from a level of about 4 mmol·l^{-1}, a decrease in F_ECO_2, and a marked hyperventilation. This onset, with its "break-away" ventilation, appears to correspond to the anaerobic threshold noted by MacDougall (1978) and Green, Daub, Painter, Houston, & Thomson (1979).

Discussion

As noted, the onset of Phases II and III have both been designated the "anaerobic threshold." The controversy thus appears to be related to the choice of criteria and to the definition of the onset of anaerobiosis. Early research by Hill, Long, & Lupton (1924) demonstrated that La was produced when there was an insufficient supply of O_2 to the working muscle. Based on these findings, it was then generally assumed that the presence of La implied hypoxia. This assumption, however, has been questioned by a number of investigators.

Graham (1978) has stated that when blood La concentrations were used to indicate the quantity of anaerobic work performed, the assumption was that blood La concentrations were indicative of muscle La concentrations. This may not always be the case, however, since Mader, Heck, & Hollmann (1978) found a time constant of about 2 minutes from intramuscular La production until La reaches the vascular space (the higher the exercise intensity and the higher the resultant blood La, the later blood La reaches its peak value) and since maximal blood La is a reflection of the rate of production, release, distribution, and elimination (Mader et al., 1978). In this regard, Graham, Sinclair, and Chapler (1976) found that neither muscle La concentration nor the muscle-to-blood gradient for La was related to La release into the blood. Depending on the time of blood sampling, therefore, blood La may or may not be indicative of muscle La.

It has been repeatedly shown that athletes can work at high intensities for prolonged periods with low levels of La. Following the assumption that La implies hypoxia, the lower La at a higher relative WL would have to be due to a removal of hypoxic conditions. If these hypoxic conditions were reduced, then $\dot{V}O_2$ at a given submaximal WL would have to increase; this would suggest an alteration in total body efficiency with training. However, since $\dot{V}O_2$ at a given submaximal WL does not change with training, local hypoxia cannot be the reason for changes in La (Holloszy, 1976). Similarly, Welch et al. (1977) demonstrated that although breathing a mixture of 60–100% O_2 did increase arterial O_2 content and reduce blood La, the $\dot{V}O_2$ of the exercising leg was not increased. From this, they also concluded that muscle hypoxia could not have been present.

The total oxidation of carbohydrate by a muscle fiber requires that electrons be removed from NADH for combination with oxygen in the mitochondria, regenerating NAD in the process (McGilvery, 1975). Although a reduction in mitochondrial NAD has been used to indicate hypoxia (Jöbsis & Stainsby, 1968) it has been shown that the levels of NAD in contracting skeletal muscle during steady-state exercise are high enough to suggest adequate oxygenation, even during the production of La (Jöbsis & Stainsby, 1968; Wahren, 1977). Graham (1978) examined the relationship between NAD and La following exercise at 70% and 100% $\dot{V}O_2$ max. There were reductions in NAD levels after both exercise intensities but there was no relationship between NAD concentration and either muscle or blood La. In addition, increases in skeletal muscle water content due to exercise accounted for 73% of the measured reduction in NAD concentration. Nevertheless, only NAD levels after maximal exercise were significantly lower than levels taken at rest, which suggests that hypoxic conditions were present at maximal WLs.

For an adequate discussion of aerobic and anaerobic metabolism, one should also have more information on the manner in which blood La is influenced by muscle fiber composition and recruitment. For example, the production and removal of La are influenced by the content of lactate dehydrogenase (LDH) in the sacroplasm of muscle fibers. This LDH can be present as heart-specific (H) or muscle-specific (M) isozymes. M-LDH facilitates the reduction of pyruvate to La, whereas H-LDH favors the oxidation of La to pyruvate for subsequent utilization in the Krebs cycle (Sjödin, 1976).

There appears to be a relationship between muscle fiber composition and (1) total LDH activity and (2) LDH isozyme distribution. Type I (also called slow-twitch, oxidative) muscle fibers have a greater relative H-LDH activity (Sjödin, 1976), while Type II (also called fast-twitch, glycolytic) muscle fibers have almost three times as much M-LDH activity (Thorstensson, Sjödin, Tesch, & Karlsson, 1977). Graham (1978) hypothesized that Type II fibers would also be more likely to become hypoxic because they have lower values for (1) capillary-fiber ratio, (2) mitochondrial concentration, and (3) the rate of oxidative metabolism. This is in agreement with the finding that La concentration was higher in Type II fibers following intense,

dynamic exercise (Tesch, 1978; Tesch, Sjödin, & Karlsson, 1978). Similarly, Bonen, Campbell, Kirby, & Belcastro (1978) found a moderate but significant correlation (r = .54) between percent Type I fibers and the rate of La removal after heavy exercise. Jorfeldt (1970) suggested that Type II fibers tend to produce La, while Type I fibers continuously extract and oxidize La from the blood and from Type II fibers. In addition, Graham (1978) found an inverse relationship between the percentage of Type I fibers and the La concentration gradient between muscle and blood. Although the blood La concentrations were similar, the muscle La concentration in Type II fibers was three times as high as that found in Type I fibers. The explanation for this was that Type II fibers had a greater rate of La production and/or a lower rate of La release.

During the various phases of progressive submaximal exercise, there appears to be a preferential recruitment of specific fiber types. Based on studies of glycogen depletion in muscle fibers, Essén (1977, 1978a, and 1978b) found a greater loss of glycogen in Type I fibers at intensities of 30–85% $\dot{V}O_2$ max. As the duration or intensity of work increased, more Type II fibers were recruited. Essén (1977) also found that Type IIa fibers (FOG or fast, oxidative and glycolytic) were recruited before the Type IIb fibers (FG or fast glycolytic). Although patterns of glycogen depletion do yield information about muscle fiber recruitment, they are not necessarily indicative of the extent to which the different fibers have been active, since glycogen is not the only substrate used to produce energy (i.e., fat and glucose can also be used).

Thus, it would appear that blood La levels reflect the production, release, and oxidation of La by muscle and that this, in turn, is influenced by muscle fiber composition and the type of fiber being recruited at any given time.

Looking now at the relationship between La and performance, Costill (1970) reported that well-trained endurance athletes could run on a treadmill at 90% $\dot{V}O_2$ max for 30 minutes with a blood La of slightly over 4 mmol·l^{-1} and a HR of 170–180 beats·min^{-1}. Likewise, Kindermann, Simon, and Keul (1978) studied seven cross-country skiers who ran on a treadmill for 45–60 minutes at 80–85% $\dot{V}O_2$ max and had a blood La of around 4 mmol·l^{-1}. These same skiers were able to do 70 minutes of ski-roller training up a slope with a HR of about 180 and a blood La of 5–6 mmol·l^{-1}. Even untrained people are able to ski cross-country for one hour with a HR of 160–170 and a La of 5.5–7 mmol·l^{-1} (Keul, Huber, & Kindermann, 1975). It is difficult to imagine that such high-intensity exercise could be maintained for more than 30–60 minutes if there really were such high levels of hypoxia or anaerobiosis.

Summarizing these theoretical and applied facts, it would appear that there are discrepancies in what is being defined as anaerobic and the relative importance and meaning of La concentrations in the blood. Therefore, it would appear that a better explanation is needed to understand what is happening in the transition from aerobic to anaerobic metabolism.

Hypothetical Model

Phase I. After the first few minutes of low-intensity exercise, increasing amounts of free fatty acids (FFA) are released into the circulatory system and transported to the working muscles. Since the rate of diffusion of FFA across the cell membrane is proportional to its concentration gradient, high levels of FFA in the blood ensure a constant supply, making FFA the dominant source of fuel for contracting muscle at low WLs (Newsholme, 1977).

This increased availability and utilization of FFA also has a profound inhibiting effect on glycolysis, further increasing the dominant utilization of FFA. It has been demonstrated that FFA metabolism produces citrate, an accumulation of which affects glycolysis by inhibiting pyruvate oxidation (Berger, Hagg, Goodman, & Ruderman, 1976; Denton & Hughes, 1978) and the activity of two glycolytic enzymes, namely, glycerol-3-phosphate dehydrogenase (McLoughlin, Shahied, & MacQuarrie, 1978) and phosphofructokinase (PFK) (Essén, 1978; Newsholme, 1977). Also, the higher the ratio ATP/(ADP + Pi), the greater the inhibition of PFK activity and thus glycolysis (Essén, 1978; Newsholme, 1977). As a result of this glycolytic inhibition by FFA metabolism, there should be a reduction in the amount of La produced. Any La that is produced should be oxidized to pyruvate due to the H-LDH isozyme pattern of the preferentially recruited Type I fibers (Essén, 1977; Sjödin, 1976).

As the exercise intensity increases, more Type I and possibly some Type IIa fibers will be recruited. This produces a greater need for and utilization of ATP, with a corresponding increase in the concentration of ADP, AMP, NH_4^+, and Pi. Newsholme (1977) states that an accumulation of these metabolites reduces the inhibitory effect of citrate on PFK activity, enhancing the rate of degradation of carbohydrate (glycolysis) and increasing the production of pyruvate.

Since FFA oxidation is high at this point, some inhibition of pyruvate oxidation is probably still present. As a result, there will be an imbalance between pyruvate production and pyruvate oxidation, with some of the pyruvate being reduced to La (Jöbsis & Stainsby, 1968; Wahren, 1977). The slight rise in blood La to about 2 mmol·l^{-1} thus appears to be due to excess pyruvate and not to hypoxia, since mitochondrial NAD levels indicate adequate oxygenation.

Phase II. With increasing intensity of exercise, there is a greater recruitment of Type IIa fibers and possibly some Type IIb fibers (Essén, 1977, 1978a, 1978b). The greater utilization of ATP reduces the inhibitory effect of citrate on PFK activity even more, further enhancing the rate of glycolysis. This, together with the predominant M-LDH isozyme pattern of Type II fibers (Sjödin, 1976; Thorstensson, Sjödin, Tesch, & Karlsson, 1977), leads to a greater production of La and a rise in $\dot{V}_E$ to compensate for the metabolic acidosis.

There is another possible explanation for the increased $\dot{V}_E$ seen at this point. Since it has been postulated that the exercising hyperpnea at the onset of exercise involves a neural component (Kao, 1977), it is possible that the hyperventilation is partially due to alterations of this neural component and to the altered recruitment of muscle fibers. If one can assume a constant and graded neural ventilatory component for Type I fibers, which seems reasonable considering the homogeneous efferent neural input and stretch characteristics of muscle fibers within the same motor unit (Eyzaguirre & Fidone, 1975), then it is possible that the recruitment of Type II fibers during the onset of Phase II could produce a different and/or additional neural component.

The degradation of fat (lipolysis) is inhibited by the presence of metabolic acidosis (Boyd, Giamber, Mager, & Lebovitz, 1974; Hjemdahl & Fredholm, 1974, 1976; Issekutz, Shaw, & Issekutz, 1975). Although the exact La concentration necessary for lipolytic inhibition is not known, the accumulation of La during this phase

(2–4 mmol·l^{-1}) probably initiates a reduction in fat utilization and an increase in the utilization of carbohydrate.

Phase III. As the exercise intensity increases, a larger number of Type IIb fibers will be recruited (Essén, 1977). Because of the M-LDH isozyme pattern of these fibers, La levels should continue to rise, resulting in an even greater inhibition of lipolysis. The actual mechanism of this inhibition is not well understood but it has been suggested by Hjemdahl and Fredholm (1974) that arterial La concentrations exceeding 5 mmol·l^{-1} increase the rate of FFA re-esterification, reducing the amount of FFA available as substrate. Others have found La concentrations of 8–9 mmol·l^{-1} (Boyd et al., 1974) or 5–6 mmol·l^{-1} (Issekutz et al., 1975) were necessary for lipolytic inhibition.

Another possibility exists to partially explain the marked rise in La during this phase, namely, anaerobiosis. If hypoxia is present, then PFK activity will be further enhanced (Keul, Doll, & Keppler, 1971) and glycolysis will increase. It is known that muscles are practically inexhaustible as long as the tension developed is less than 20% of the maximal voluntary contraction (MVC) performed statically (Frolkis, Martynenko, & Zamostyan, 1976). Likewise, Durnin and Mikulicic (1956) and Åstrand (1967) report no problem working up to 8 hours per day while performing dynamic, light-to-medium work tasks requiring 40–50% $\dot{V}O_2$ max. At these low intensities, the energy requirements can be easily met by oxidative metabolism and there is no problem of muscle blood flow. As the intensity of exercise increases (i.e., increased levels of force and/or speed of contraction are required), more motor units and muscle fibers are activated, especially the stronger and faster Type II fibers at the higher intensities.

Although Pirnay, Marechal, Radermecker, & Petit (1972) found that the total blood flow to the quadriceps muscles rose during progressive cycling to maximum and concluded that muscular circulation was not the essential limiting factor for $\dot{V}O_2$max, the external mechanical pressure of the contracting fibers will be greater during contraction than the internal arterial pressure at some intensity along the way to $\dot{V}O_2$max. This will cause a reduction or occlusion of blood flowing to the muscle during the time of the actual contraction, increasing the dependence on anaerobic energy supplies within the muscle.

It has been estimated that occlusion of the artery occurs during static contractions of about 60–70% MVC (Start & Holmes, 1963). Unfortunately, the percent $\dot{V}O_2$max at which this occurs during the dynamic, rhythmical contractions seen in running, cycling, etc. is not known. Nevertheless, Katch, McArdle, & Pechar (1974) measured the maximal dynamic leg strength on an isokinetic recording dynamometer while subjects pedalled at 60 rpm on a bicycle ergometer. The average maximal leg force for 50 subjects was 118 foot pounds of torque (16.3 kpm) and their $\dot{V}O_2$max was 3.44 l·min^{-1}. Assuming that the maximal dynamic force at this velocity would be about 80% of the MVC performed statically (Asmussen, 1968), the mean MVC of this muscle group should have been around 20 kpm. Assuming also that the maximal WL on the bicycle was 1200–1500 kpm·min^{-1}, this would be 10–12.5 kpm for each leg per revolution or 50–60% MVC at $\dot{V}O_2$max. Therefore, it may be hypothesized that the rapid increase in La during Phase III is related to partial occlusion during the contractions, resulting in a greater need for anaerobic energy sources.

Putting all these facts together, it would appear that the La response found during

Table 1—Hypothetical Model of Selected Characteristics of the Various Thresholds and Phases During Progressive Exercise from Rest to Maximal Oxygen Consumption

	Rest	Phase I	Aerobic Threshold	Phase II	Anaerobic Threshold	Phase III	$\dot{V}O_2$ max
Predominant Type of Metabolism		Aerobic		→		Anaerobic	
Predominant Substrate		Fat>Carbohydrate		→		Carbohydrate>Fat	
Predominant Muscle Fiber Type		I		I, IIa		I, IIa, IIb	
Relative Intensity (% $\dot{V}O_2$max)			40–60		65–90		
Heart Rate (b·min^{-1})			130–150		160–180		
Blood Lactate (mmol·l^{-1})			~2		~4		

Phase III is due to anaerobiosis since the increased energy requirement occurs at the same time as (1) a possible reduction or occlusion of muscle blood flow, (2) an increased recruitment of Type IIb fibers, which are better adapted for anaerobic glycolysis, (3) a decreased utilization of FFA, which can be metabolized only via aerobic mechanisms, and (4) an increased utilization of carbohydrate, which can be metabolized via anaerobic glycolysis.

Suggested Terminology

Given these facts and assumptions, it seems that some of the controversy could be reduced by a modification of terminology, especially relative to the use of the term "anaerobic threshold" and the interpretation of La present in the blood. Several research groups in Germany (Kindermann, Simon, & Keul, 1979; Mader & Hollmann, 1977) have been studying this area and have suggested different terminology, with which we are in basic agreement.

Since the initial rise in La and the nonlinear increases in $\dot{V}_E$ and $\dot{V}CO_2$ at the onset of Phase II are related more to recruitment of Type I fibers and to an imbalance between the rate of pyruvate production and pyruvate oxidation and are related less to anaerobiosis, it is suggested that this be designated the "aerobic threshold" (AerT). Similarly, since the sharp rise in La and the "breakaway" $\dot{V}_E$ seen at the onset of Phase III are related more to anaerobiosis and to the increasing recruitment of Type II fibers (especially Type IIb fibers) with their predisposition to hypoxia, it is suggested that this be designated the "anaerobic threshold" (AnT). Thus, Phase I appears to be predominantly aerobic and involves Type I fibers, Phase III appears to be predominantly anaerobic and involves Type I and Type II fibers, and Phase II seems to be the transitional phase between these two forms of metabolism. Table 1 summarizes some of the characteristics of the various phases and thresholds.

Factors Affecting the Aerobic and Anaerobic Thresholds

Method of testing. During a progressive work test, AerT determination has usually been based on the association of the initial rise in La with several noninvasive measurements, e.g., nonlinear increase in $\dot{V}_E$ and $\dot{V}CO_2$, an increase in end-tidal O_2 tension without a corresponding decrease in end-tidal CO_2 tension, and an increase in R (Wasserman et al., 1973). Davis, Vodak, Wilmore, Vodak, and Kurtz (1976) found a correlation coefficient of .96 between the AerT determined from these gas exchange parameters and AerT determined from repeated serial venous La samples. At the same time, they also reported a test-retest reliability coefficient of only .75 for AerT determinations from gas exchange alterations, suggesting a large intra-individual variability with this technique. Although it would seem that the best method for determining AerT is the direct measurement of La, Stamford, Weltman, & Fulco (1978) reported difficulty in some subjects and suggested that this was due to differences in (1) rate of La transport from tissue to blood, (2) buffering capacity, and/or (3) rate of La elimination at various sites.

AnT determinations have generally been based on direct La measures (Liesen, Mader, Heck, & Hollmann, 1977; Mader, Liesen, Heck, Philippi, Rost, Schurch, & Hollmann, 1976). AnT is determined at the WL which is followed by an abrupt and continuous rise in La, with a mean La concentration at this point of around 4 mmol·l^{-1}. According to Kindermann et al. (1979), AnT determinations are more precise than those of AerT. With the exception of the "point of breakaway $\dot{V}_E$" reported by MacDougall (1978) and Green et al. (1979), noninvasive gas exchange estimates of AnT have not been extensively reported.

Duration of work loads. Wasserman et al. (1973) and Stamford et al. (1978) suggested that work bouts of at least 3 minutes duration were necessary for the accurate determination of AerT. Similarly, Mader and Hollmann (1977) suggested working for not less than 4 minutes and preferably 5 minutes for AnT determinations. Due to the delay in diffusion of La from muscle to blood, shorter WLs are likely to result in overestimates, that is, the subject will be performing a higher WL before the blood La rises due to conditions produced during the previous WL.

Type of exercise. Davis et al. (1976) measured La and various gas exchange parameters during leg exercise on a bicycle ergometer and treadmill and during arm cranking. There were no individual differences between leg cycling and treadmill walking when the respective AerT values were expressed relative to the $\dot{V}O_2max$ (% $\dot{V}O_2max$) obtained in the same work test. Significantly lower values for $\dot{V}O_2max$ and relative AerT were found for arm cranking. The authors speculated that the lower values for arm work could have been due to (1) smaller muscle mass of the arms, (2) little or no experience with arm cranking, so that arms were less trained, (3) differences in Type I and Type II muscle fiber distribution between arms and legs, or (4) lack of uniform motor unit recruitment in arm work. In this regard, Cerretelli, Shindell, Pendergast, DiPrampero, & Rennie (1977) found that there was a higher isometric component associated with arm exercise; this could have affected blood flow to the muscles. Likewise, Stamford et al. (1978) hypothesized that nonfamiliarity could have produced different patterns of motor fiber recruitment. On the other hand, they did not feel that the size of the total muscle mass involved was important since no difference in relative AerT values was found during cycling with one or two legs.

There is little information in the literature on the influence of the type of exercise on AnT. Kindermann et al. (1978) reported that well-trained cross-country skiers were able to run on the treadmill for 45–60 minutes at 80–85% $\dot{V}O_2max$ at a La of around 4 mmol·l^{-1} but were able to do 70 minutes of ski-roller training up a slope with a HR of 180 and a La of 5–6 mmol·l^{-1}. They attributed this difference to the fact that the muscle mass involved (arms and legs) was greater. There is also the possibility of training specificity since the skiers trained both arms and legs. The AnT of 9 physical education students tested on the bicycle ergometer and treadmill can be found in the research reported by Liesen et al. (1977). Although no direct comparison was made, the AnT was 74% of the $\dot{V}O_2max$ on the bicycle, while it was 82% on the treadmill.

Substrate availability. When high levels of blood glucose were present, Ivy, Costill, Essig, Lower, & Van Handel (1979) found AerT values similar to those found under control conditions. However, when they elevated blood FFA levels, there was a significant rise in relative AerT and a reduction in AerT blood La. Since FFA oxidation inhibits glycolysis (Essén, 1977; Newsholme, 1977), an increase in blood FFA concentrations should produce a greater blood-to-muscle concentration gradient and a greater inhibition of carbohydrate metabolism at the same WL. As a result, La production should be reduced and AerT should occur at a higher WL. The observation that blood La values were lower at AerT during exercise with elevated blood FFA levels (Ivy et al., 1979), suggests that the buffering of other metabolic acids (i.e., ketone bodies) may cause a disproportionate rise in $\dot{V}_E$ and $\dot{V}CO_2$ at AerT. Ivy and his co-workers (1979) also found that elevating La values to 9 mmol·l^{-1} by arm cranking had no effect on the subsequent AerT determination during leg exercise on the ergometer. From these experiments, they hypothesized that (1) the accumulation of La was not entirely due to an O_2 deficiency but depended on substrate availability and (2) the simultaneous changes in $\dot{V}_E$ and La observed at AerT were only coincidental.

The development of the hypothetical model to explain alterations in gas exchange and La production (see previous discussion) included the possibility that the recruitment of Type II fibers produced an additional and distinct neural ventilatory component, resulting in an abrupt rise in $\dot{V}_E$. Under normal conditions, this rise would be associated with an increased La production from the increased Type II fiber recruitment (Sjödin, 1976; Tesch, 1978; Tesch, Sjödin, & Karlsson, 1978). However, during conditions where La values are elevated prior to the exercise test (Ivy et al., 1979), the relationship between an increased $\dot{V}_E$ and La production would become dissociated due to similar neural ventilatory components and fiber recruitment patterns.

Muscle fiber composition and training. Since well-trained endurance athletes tend to have higher percentages of Type I fibers (Bergh, Thorstensson, Sjödin, Holten, Piehl, & Karlsson, 1978; Costill, Daniels, Evans, Fink, Krahenbühl, & Saltin, 1976; Costill, Fink, & Pollock, 1976; Gollnick, Armstrong, Saubert, Piehl, & Saltin, 1972; Saltin, Henriksson, Nygaard, Anderson, & Jonsson, 1977) and higher relative values of AerT (Costill, 1970; Volkov, Shirkovets, & Borilkevich, 1975) and AnT (Kindermann et al., 1979; Mader et al., 1976), it is possible that relationships exist among these physiological parameters. Nevertheless, nonsignificant correlation coefficients have been reported between fiber composition of normal subjects and AerT (McLellan & Skinner, Note 2) or AnT (Green et al., 1979). This suggests

that other factors known to increase with training may be involved, for example, oxidative enzyme levels (Henriksson & Reitman, 1977; Holloszy, 1976; Saltin et al., 1977) or LDH isozyme pattern (Brinkworth & Masters, 1978; Karlsson, Sjödin, Thorstensson, Hulten, & Frith, 1975; Sjödin, 1976; Sjödin, Thorstensson, Frith, & Karlsson, 1976).

Green et al. (1979), on the other hand, reported no relationship between SDH activity and AnT. Therefore, an analysis of LDH isozyme pattern differences may help explain some of the variability of relative AerT and AnT values. Karlsson et al. (1975) found a decrease in total LDH activity with endurance training but a shift toward the H-LDH isozyme. Similarly, Brinkworth and Masters (1978) found a greater decrease in M-LDH activity in Type II fibers, while Sjödin (1976) and Sjödin et al. (1976) reported that the relative activity of the H-LDH isozyme increased in both Type I and Type II fibers. Well-trained endurance athletes should therefore have both a higher percentage of Type I fibers and a preferential H-LDH isozyme distribution. As a result of the possibly slower La production, both AerT and AnT should occur at higher relative WLs compared to untrained persons or to elite nonendurance athletes (sprinters, jumpers, etc.).

There are two other reasons why endurance athletes may have higher values for AerT and AnT. Endurance athletes generally have a lower ventilatory response to similar levels of alveolar CO_2 pressure (Stegemann, 1977). Since many determinations of AerT are based on alterations in $\dot{V}_E$ and $\dot{V}CO_2$, this reduced sensitivity of the respiratory center to CO_2 would delay these changes. Likewise, there are alternative pathways for removal of pyruvate in muscle, other than to lactate or by oxidation. Since one possibility for pyruvate removal is its conversion to alanine and since training produces a major increase in alanine transaminase (Molé, Baldwin, Terjung, & Holloszy, 1973), this may be a reason why the muscles of endurance athletes appear to produce less La, even at comparable rates of glycolysis (Saltin & Karlsson, 1971).

Concluding Remarks

The amount and intensity of training necessary to produce changes in AerT and AnT are not yet known. Williams, Wyndham, Kok, & von Rahden, (1967) reported a 16% increase in AerT following 4–16 weeks of daily training sessions lasting up to 4 hours. This increase was greater than and independent of the mean rise in $\dot{V}O_2$max of 7%. Similarly, Davis, Frank, Whipp, & Wasserman, (1979) found a 44% rise in absolute AerT (15% increase in relative AerT) compared to a 25% increase in $\dot{V}O_2$max following a nine-week program (45 minutes per day, 4 days per week). In contrast, Skinner, Lemieux, & Taylor (1977) and McLellan and Skinner (Note 2) found no change in relative AerT after 8 weeks of endurance training, 3 times per week for 30–45 minutes at around 65% $\dot{V}O_2$max. Mader et al. (1976) report the case of an amateur cyclist who had been training about 18 hours per week for a number of years. After increasing the training intensity for 6 weeks to a level around his AnT, there was little change in $\dot{V}O_2$ max from 60 to 62 $ml \cdot kg^{-1} \cdot min^{-1}$ but he was able to do about 12% more work before reaching his AnT. From this, it was obvious that endurance training at this higher but still tolerable intensity produced a relative increase in both AerT and AnT.

A good empirical example of this type of improvement can also be seen in the dramatic rise over the past decade in the number of marathons run per year under

2 hours and 20 minutes, even though the best performances each year have remained unchanged around 2:09 to 2:12. This is a reflection not only of the increase in the number of people participating in marathons but also of the fact that well-trained athletes are able to work closer and closer to their $\dot{V}O_2$max during prolonged exercise (Saltin et al., 1977), even when there is little improvement in their $\dot{V}O_2$max.

Although some of the literature and the hypothetical model propose values of 2 and 4 $mmol \cdot l^{-1}$ blood La for the AerT and AnT, respectively, these are arbitrary values which probably do not reflect the same degree of excess pyruvate production or anaerobiosis in all people. For example, prepubertal children do not have a high glycolytic capacity due to lower levels of PFK (Eriksson, Gollnick, & Saltin, 1973). As a result, maximal blood La levels of about 2, 2.5, 8.5 and 12.5 $mmol \cdot l^{-1}$ have been reported for children aged 3, 5, 7 and 16 years, respectively (Klimt, 1977). Applying an arbitrary value of 4 $mmol \cdot l^{-1}$, therefore, would make little sense for children before and during adolescence.

Likewise, Mader (Note 1) has tested sprinters who have La levels greater than 4 $mmol \cdot l^{-1}$ at 50–60% $\dot{V}O_2$max, compared with highly-trained endurance runners who have a La of around 1 $mmol \cdot l^{-1}$ at intensities of 80–90% $\dot{V}O_2$max (4 $mmol \cdot l^{-1}$ being near their maximal La levels). In these cases as well, an arbitrary level would not mean the same thing for the two types of athletes who probably also have different distributions of muscle fiber types and/or LDH isozyme patterns. As previously discussed, so many factors might influence blood La concentrations (i.e., La production, release, distribution, and elimination)—and each of these may vary depending on muscle fiber type, LDH isozyme pattern, enzyme activity, capillarization, mitochondrial density, etc.—that much more research is needed before these factors and their effects may be understood.

More research is also needed on different kinds of athletes and on different types of activities to determine their specificity and how each might affect AerT and AnT. Noninvasive field tests to determine AnT would be useful. Finally, the application of these findings to the refinement of training programs for the athlete, as well as for the average person, would be a logical and desirable outcome.

In summary, a hypothetical model has been proposed in an attempt to clarify controversial issues about the transition from aerobic to anaerobic metabolism. Whether it has clarified or contributed to the controversy is for the reader to decide. If it has increased the reader's understanding of this complex but interesting field, then the effort put into the literature review and hypothetical model will be repaid. If it also serves to promote further discussion and acts as a catalyst for more research into this challenging area of study in exercise physiology, then the manuscript will have served its primary objective.

Reference Notes

1. Mader, A. Personal communication, June 1979.
2. McLellan, T. M., & Skinner, J. S. University of Western Ontario, unpublished data, 1979.

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Measurement and Prediction Errors in Body Composition Assessment and the Search for the Perfect Prediction Equation

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The assessment and prediction of body composition has gained widespread application in the various exercise science disciplines. There are applications to physiology of exercise, biomechanics, exercise biochemistry, anatomy, motor integration, and other allied medical fields that consider such topics as nutritional and dietary assessment, the man-machine interface, as well as various environmental concerns. Whatever the application, one major area of interest is the predictive accuracy of body composition assessment, particularly percent body fat and lean body weight.

In the last 30 years, at least 100 prediction methods have been proposed to evaluate the fat and lean components of the body. Most authors usually point out that validity is disappointingly poor when prediction equations and formulae are applied to independent samples (validity is the correlation between predicted and actual measurements, taking into account the standard error of estimate). While the prediction of mean values is relatively high, prediction of body composition for an individual subject is much more variable. Of utmost concern is this question, "Should prediction equations be used, and if yes, what is their accuracy?"

The objectives in the present paper are to discuss (1) error sources in body composition assessment by laboratory methods with emphasis on hydrostatic weighing and anthropometric measurement and (2) several aspects of statistical theory as it relates to the search for the perfect prediction equation.

Error Sources in Body Composition Assessment by Laboratory Methods

Many laboratory methods are available for assessing the fat and lean components of the body. The range in sophistication varies considerably from relatively inex-