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Diagnostics of Anaerobic Power and Capacity

Hermann Heck, Henry Schulz, and Ulrich Bartmus

Testing procedures for the assessment of anaerobic energy metabolism during muscular work have not yet gained the relevance of tests assessing maximal aerobic power. The diagnosis of aerobic power allows one, through the choice of an adequate testing protocol, to design a test that mainly measures the power of aerobic metabolism by means of indicators like VO_{2max} and lactate. With regard to tests for the assessment of anaerobic power and capacity, however, alactic, lactic, and oxidative components of energy expenditure as a whole cannot be differentiated by means of simple parameters (e.g., lactate and time until exhaustion). By means of computer simulations of energy metabolism for supramaximal loads with durations until exhaustion of about 10 s and 60 s as well as the isolated variation of the concentration of muscle phosphocreatine, the maximal rate of lactate production, and the maximal aerobic power (VO_{2max}), the influence of the single components on energy metabolism as a whole is presented in a semi-quantitative way. Subsequent testing procedures for the measurement of alactic and lactic power as well as alactic and lactic capacity are presented. Finally critical-power method and method for the determination of maximal accumulated O_2 deficit are described in greater detail, because both methods are widely discussed in contemporary international literature.

Key Words: energy metabolism, anaerobic capacity, anaerobic power, critical power, lactate

Key Points:

1. In contrast to the determination of maximal aerobic power, the assessment of anaerobic power and anaerobic capacity is a much more complex subject.
2. Computer simulations of energy metabolism can help to better understand the interactions between the different metabolic pathways.
3. When introducing new testing procedures and parameters for the assessment of the different components of energy metabolism, it is important to do so on the basis of a physiological model.
4. Because of the complex interactions between the various components of energy metabolism, a detailed assessment of these components can only be achieved by using a battery of tests.

Introduction

In several sport activities, performance ability is considerably determined by the capacity and power of the energy-generating systems of the muscles involved.

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Short-term activities (e.g., the 100-m run) mainly require the anaerobic energy-transfer system, while long-term activities (e.g., the 10,000-m run) predominantly make use of the aerobic energy-transfer system.

It must be the aim of complex performance diagnostics to determine capacity and power of each component of energy metabolism in order to detect deficiencies and to give concrete advice for the planning of training. Additionally, the efficiency of training devices can be examined in the course of longitudinal studies.

Measurement of maximal aerobic power has a long tradition in exercise physiology. The term *maximal oxygen uptake* was introduced by A.V. Hill (8) in 1924. In the following decades, diverse instruments for the measurement of oxygen uptake during exercise were designed. Today the miniaturization of spirometry systems has reached a state that allows continuous registration of oxygen uptake (and thus a direct measurement of aerobic metabolism) during a broad range of sport activities without impairing the athlete.

The situation is fundamentally different with testing procedures for the determination of lactic and alactic capacity and power, the main reason being the fact that all substrata needed for anaerobic processes are located inside the muscle cell. These substances can only be measured directly by complex methods—for example, muscle biopsy and ^{31}P-MR spectroscopy. The determination of intermediate or end products of energy metabolism such as lactate or pyruvate in the peripheral blood only allows an indirect assessment owing to the complex dynamics of diffusion and elimination processes.

It is an aim of this review to illustrate the static and dynamic behavior of different components of energy metabolism and their interactions by referring to the existing literature and also using computer simulations of metabolic processes. These considerations should allow some advice for the construction of anaerobic performance tests. Additionally, some testing procedures from the national and international fields are discussed.

Capacity and Power of Muscular Energy Metabolism

Capacity of energy metabolism may be defined as the sum of all work that can be gained from energy stored in chemical form. Analogously, power may be defined as the sum of the maximal metabolic rates of the different energy transfer systems.

Table 1 shows data (in ATP-equivalents) for power and capacity of alactic, lactic, and aerobic energy metabolism related to kilograms of wet muscle weight.

An *alactic power* of 6 mmol $\cdot$ kg^{-1} $\cdot$ s^{-1} is measured with maximal exercise of about 0.5 s duration. Values for high jumping with a duration of 0.2 s are even higher (25). Maximal running (sprint) results in values of about 3 mmol $\cdot$ kg^{-1} $\cdot$ s^{-1} (24).

Alactic capacity nearly amounts to 20–25 mmol $\cdot$ kg^{-1}. But only 85% can be used, because a reduction in muscular ATP concentration by 30% (e.g., from 5 to 3.5 mmol/kg muscle) leads to a decline in free energy of the adenylic acid system to a point that results in a contraction insufficiency (25).

Lactic power is limited by the maximal glycolytic rate, which itself is mainly limited by the amount of glycolytic enzymes, especially phosphofructokinase (PFK). Assuming normal glycogen values, maximal PFK activity is about 1 mmol $\cdot$ kg^{-1} $\cdot$ s^{-1} glucose in the musculus quadriceps. Because anaerobic glycolysis converts 3 mol ATP per mol glucose (from glycogen) into lactate, maximal lactic ATP resynthesis rate reaches 3 mmol $\cdot$ kg^{-1} $\cdot$ s^{-1}. This metabolic rate is attributed to the 100-m run.

Table 1 ATP Equivalents for Power and Capacity of Alactic, Lactic, and Aerobic Energy-Generating Processes (Following Greenhaff et al., 2)

Energy-generating system	Substrata	Power (mmol · kg^{-1} · s^{-1} wet muscle)	Capacity (mmol · kg^{-1} wet muscle)
Alactic	ATP, PCr → ADP, Cr	3–6	20–25
Lactic	Glycogen → lactate	1.5–3	50
Aerobic	Glycogen → CO_2, fatty acids → CO_2	0.5–0.75, 0.24–0.4	Limitation by substrata

With longer distances (e.g., the 400-m run), maximal lactate production rate is reduced by the increasing concentration of hydrogen electrons; for this kind of activity, the metabolic rate amounts to 1–1.5 mmol · kg^{-1} · s^{-1} ATP.

Lactic capacity is mainly limited by the maximal acidosis that can be tolerated. Maximal blood lactate values amount to 15–20 mmol · L^{-1}; with specially trained athletes (e.g., 400-m runners), even 25 mmol · L^{-1} are possible. Inside the muscle, lactate values up to 30–35 mmol · kg^{-1} can be measured. These values correspond to a phosphagen equivalent of about 45–50 mmol · kg^{-1} ATP.

Aerobic power is determined by the maximal oxidation rate of hydrogen within the mitochondrial electron-transport chain and is thus identical to maximal oxygen uptake. Maximal oxygen uptake of untrained young men (75 kg weight) is between 3000 and 3600 ml · min^{-1}. Assuming a usable muscle mass of 24 kg, a muscle related power of 125–140 ml · kg^{-1} · min^{-1} results. This corresponds to a maximal aerobic ATP resynthesis rate of about 0.75 mmol · kg^{-1} · s^{-1}, if glycogen is used exclusively (which can be assumed with exercise intensities leading to maximal oxygen uptake). Maximal ATP resynthesis rate using free fatty acids is 0.24 mmol · kg^{-1} · s^{-1} (19). Maximal oxygen uptake almost is reduced to 50% with pure fat oxidation, corresponding to results gained from McArdle patients who show only little glycolysis rates (or even none at all) owing to a shortage of phosphorylase.

The *capacity of aerobic metabolism* is limited by glycogen and fatty acids. For a 71-kg male with a muscle mass of 28 kg, Greenhaff et al. (6) report ATP-equivalents for muscle glycogen of 84 mol, for liver glycogen of 19 mol, and for fatty acids of 4000 mol.

The data in Table 1 refer to a muscle with a mixed fiber spectrum. Fast-twitch fibers (type II) as a rule have a higher anaerobic capacity and power, whereas slow-twitch fibers (type I) show a greater maximal oxidative metabolic rate (28).

Dynamics of ATP-Resynthesis System

Depending on intensity and duration of exercise, ATP-resynthesis pathways are used to a different extent. This shall be illustrated by means of computer simulations of all-out exercise with durations of about 10 s and 60 s. The mathematical equations

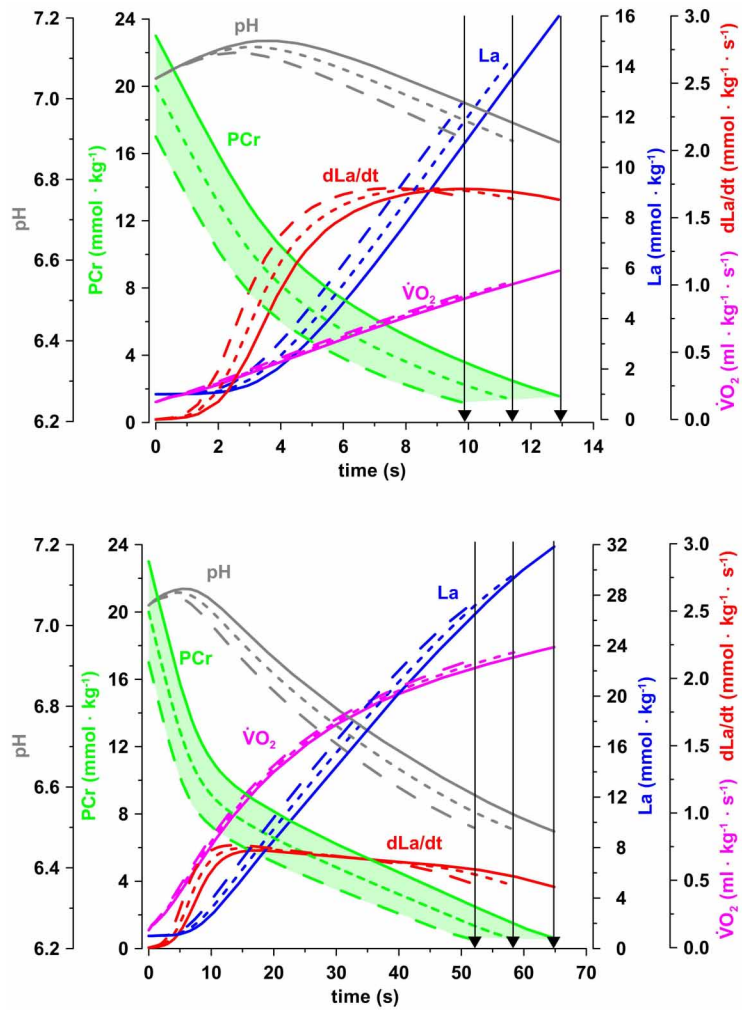


Figure 1 — Simulation of energy metabolism during all-out exercise of about 10-s (top) and 60-s (bottom) duration. Concentration of phosphocreatine is varied by ±15% (shaded area). The arrows indicate the end of exercise. The data refer to wet muscle.

and assumptions on which the computer simulations are based are described in detail in Mader and Heck (17) and Mader (16).

In several performance tests aimed at the assessment of anaerobic power and capacity maximal exercise time, respectively, time to exhaustion and/or submaximal lactate values are used as prediction variables. Because of their interdependence, the exact contribution of the different metabolic processes is difficult or even impossible to determine. In the computer simulations that follow, alactic capacity as well as maximal rates of lactic and aerobic metabolism are varied by ±15% on the basis of a mean value.

As expected, a higher concentration of phosphocreatine results in a longer exercise time until exhaustion for both the 10-s and 60-s ranges (Figure 1). On account of the higher phosphocreatine level, activation of glycolysis is reduced. This leads to lower muscle and blood lactate values (blood values are not included in Figure 1 to avoid confusion) for identical exercise duration.

A variation in maximal aerobic power within the 10-s range has no influence at all on the time to exhaustion and only very little influence on the other parameters. This can be explained by the fact that different levels of aerobic power are of minor significance within this time range because of the delay in increase of the aerobic metabolism (Figure 2, upper part). There is another situation, however, when looking at the 60-s range. In this case, a higher level of maximal oxygen uptake results in

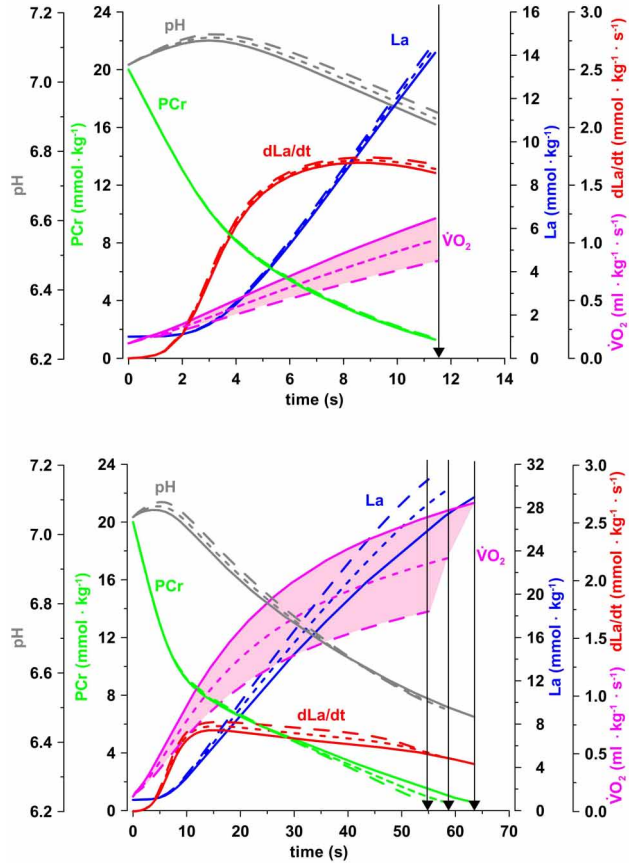


Figure 2 — Simulation of energy metabolism during all-out exercise of about 10 s (top) and 60 s (bottom) duration. Maximal oxygen uptake is varied by ±15% (shaded area). The arrows indicate the end of exercise. In this example, a maximal oxygen uptake of 2.5 ml · s^{-1} · kg^{-1} muscle is assumed. A body mass of 70 kg and a muscle percentage of 35% result in an absolute oxygen uptake of 3825 ml · min^{-1}.

a significant extension of the time to exhaustion. Moreover, lactate concentration for identical exercise duration is reduced (Figure 2, lower part). This means that with performance tests for the assessment of alactic capacity having a duration of 40 s, changes in phosphocreatine as well as maximal aerobic power may influence the prediction variable lactate. Therefore, the exclusive measurement of lactate allows no differentiation between changes induced by an increase of phosphocreatine or maximal oxygen uptake.

Variations in maximal glycolytic rate and thus in the rate of lactate formation have a great influence on the time to exhaustion (Figure 3, upper part). This is not

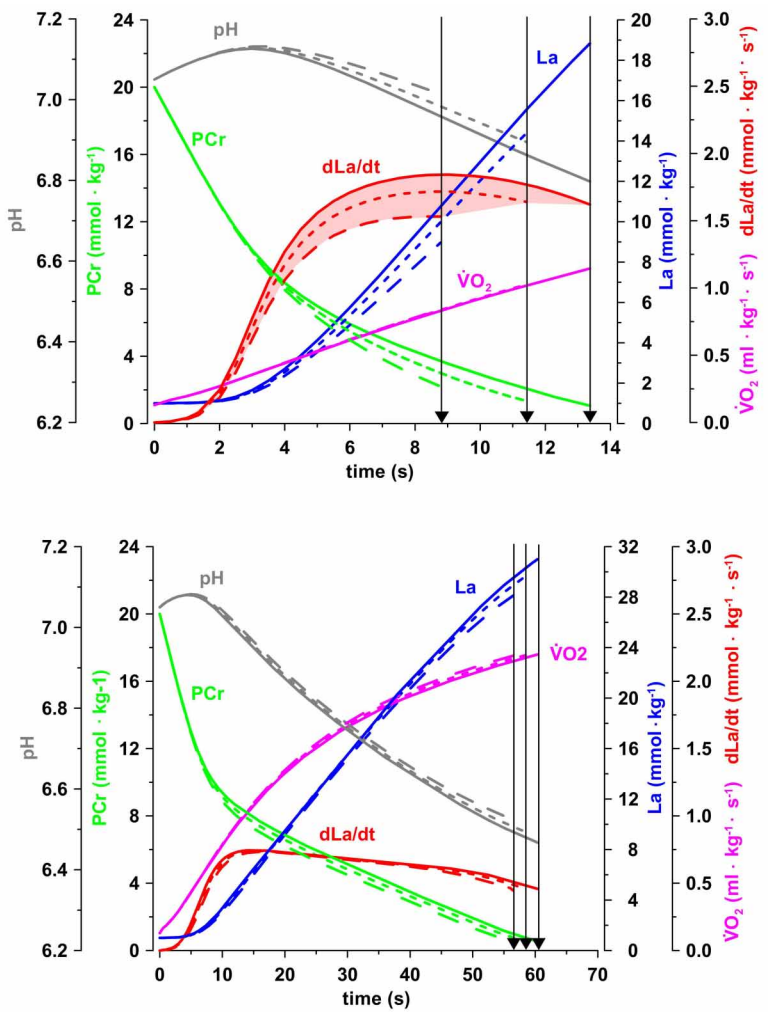


Figure 3 — Simulation of energy metabolism during all-out exercise of about 10-s (top) and 60-s (bottom) duration. Maximal glycolytic rate and lactate production rate, respectively, are varied by ±15% (shaded area). The arrows indicate the end of exercise.

amazing, because even after a few seconds of maximal exercise, glycolytic processes make an important contribution to the total energy output. Looking at the 60-s exercise, the effect of variations in maximal glycolytic rate is less visible (Figure 3, lower part). This can be explained by the reduced activity of phosphofructokinase, along with an increasing concentration of hydrogen electrons. In this situation, even with a massive depletion of the adenylic acid system, high rates of lactate formation are no longer possible (15).

The simulation data illustrate that performance tests for the assessment of alactic power and capacity should have a duration of not more than 5 s to minimize the influence of variations in maximal lactate formation rates.

Anaerobic Performance Tests

The international literature in the field of exercise physiology contains numerous performance tests for the assessment of anaerobic power and capacity. Because of the limitations of space, only few testing procedures can be presented in an exemplary way. A survey can be found in Vandevalle et al. (29) and Green (3).

Diagnostics of Alactic Power

The classic testing procedure for the assessment of anaerobic power is the stair-sprinting test by Margaria et al. (18). After a short distance of acceleration at ground level, the subject runs as fast as possible up a flight of stairs. Velocity is calculated via time measurement with the help of photoelectric beams with a precision of 10 ms. Power (W) then is calculated as a product of vertical velocity and force (dependent on body mass).

The Wingate anaerobic test (9) is another testing procedure for the assessment of alactic power. It is carried out on a bicycle ergometer. After warm-up (5 to10 min), the subject has to pedal as fast as possible against a fixed resistance (0.75–1.05 N/kg body mass); exercise duration is 30 s. Maximal power (i.e., peak power) usually is achieved with the highest pedaling rate after 3–5 s (Figure 4, upper part). Peak power is thought to be identical with maximal alactic power. Methodological studies have shown, however, that peak power is dependent on various factors—for example, the preset resistance. Figure 4 (lower part) shows that with a relative resistance of 1.05 N/kg body mass, maximum power is not yet achieved (9). It seems possible that peak power values are further increased by higher resistance values.

Diagnostics of Alactic Capacity

A direct measurement of ATP and phosphocreatine concentration is only possible by muscle biopsy and to a certain extent by ^{31}P-MR spectroscopy. Some examples for indirect methods are:

Assessment of the Fast Component of the Oxygen Debt That Is Settled During the Recovery Phase Following Exercise. This method is prone to errors, because it is usually not possible to differentiate exactly between fast and slow components of O_2 debt, even with complex mathematical procedures.

Sprint Running Test (3 $\times$ 60 m) According to Hellwig et al. (7). Three sprints are carried out—the first one with maximal velocity, the subsequent ones each with an additional running time of 0.5 s. Maximal lactate values measured during the

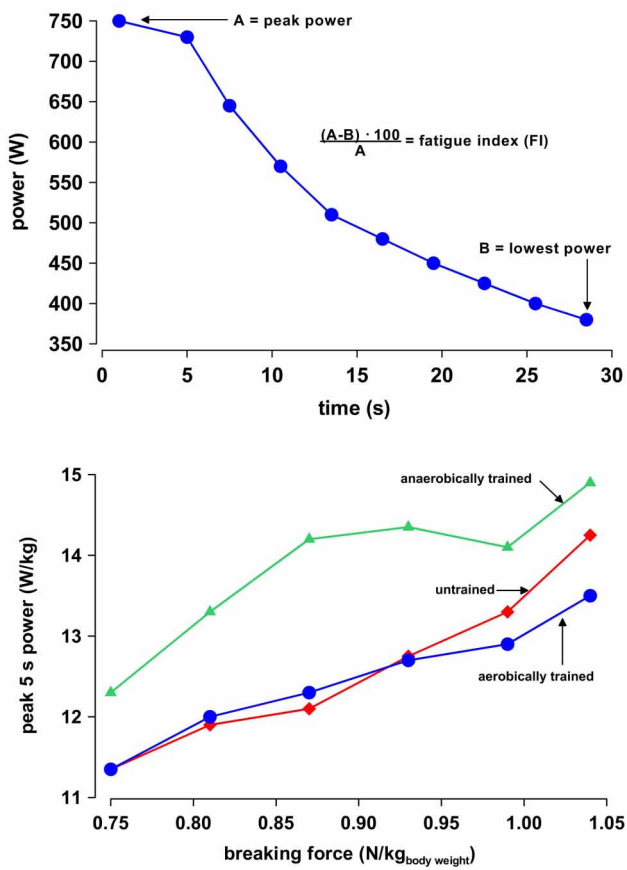


Figure 4 — Power-time function of the Wingate anaerobic test (top); influence of breaking force on peak power (bottom).

recovery phase are entered into a velocity-lactate diagram. Velocity corresponding to a lactate value of 6 mmol/L is thought to be a relative indicator for alactic capacity (Figure 5). This indicator, however, is only valid with identical maximal lactate formation rates. In graded exercise tests, a shift of the lactate-power curve to the right towards higher power values (induced by means of aerobic training) without a corresponding increase in maximal oxygen uptake can be interpreted as a result of a reduced submaximal and/or maximal glycolytic rate. Analogous to this phenomenon, the results from Hellwig et al. show a shift of the lactate-velocity curve to the right without an increase in alactic capacity. Figure 6 can be interpreted in this way. After a 4-week endurance training period within a 10-week preparation period, the mean lactate value after a series of sprint runs (5 × 30 m; Figure 6, middle part) is reduced, although running velocity has remained constant (Figure 6, left part). The significantincrease in running velocity in a graded exercise test at 4 mmol/L lactate points to a higher level of aerobic power. There is no hint in the relevant literature that an increase in alactic capacity can be a result of endurance training.

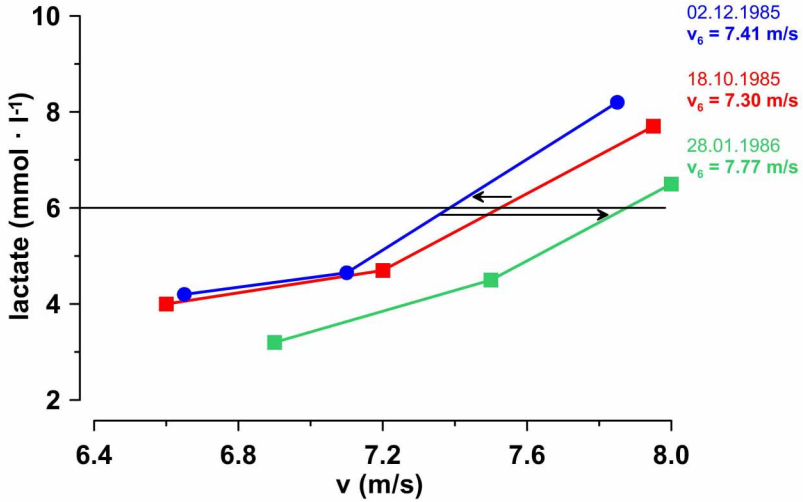


Figure 5 — Lactate-velocity relation in a series of sprint runs (3 × 60 m) according to Hellwig et al. (7); results of a longitudinal study for 1 subject.

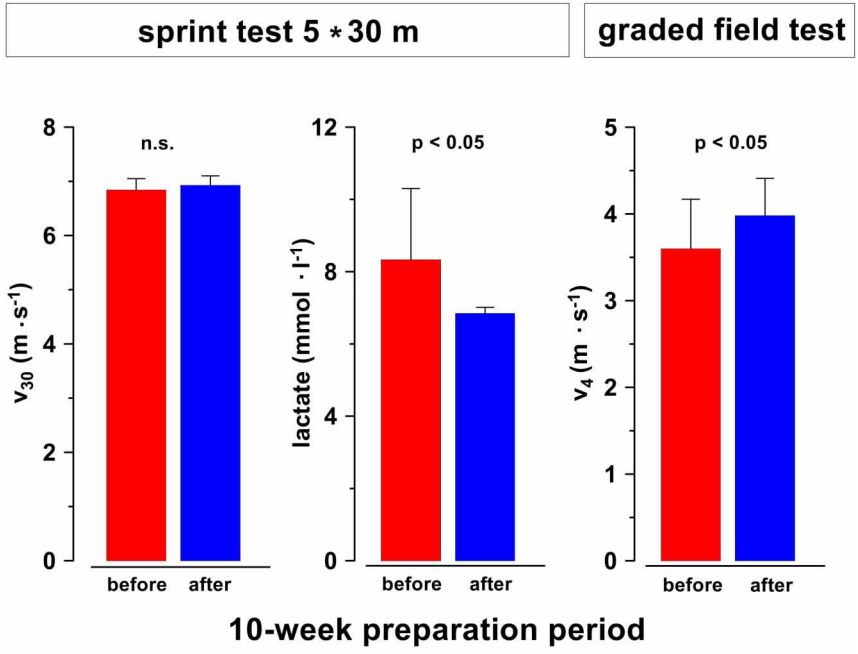


Figure 6 — Effect of endurance training lasting several weeks with soccer players on sprint velocity (v_{30}) and maximal lactate after a series of 5 × 30-m sprint runs and on running velocity at 4 mmol/L (v_4) in a graded exercise field test (increment: 0.5 m/s, stage duration: 3 min; unpublished results by H. Schulz).

Treadmill Test With a Duration of 40 s According to Schnabel and Kindermann (11, 27). The test with a running velocity of 22 km/h and a treadmill grade of 7.5% is broken off after 40 s. It is suggested that the lower the maximal lactate after the end of the exercise, the higher alactic capacity. As mentioned earlier, the test result is not only influenced by phosphocreatine concentration but also by maximal aerobic power. Therefore, a meaningful interpretation of the test result in any case demands the additional measurement of maximal oxygen uptake.

Diagnostics of Lactic Power

Maximal lactate formation rate as an indicator for lactic power cannot be measured directly with simple methods. According to Mader (16), the following formula allows a satisfactory estimation:

$$dLa/dt_{max} = \frac{maxAELa - RLa}{t_{exer} - t_{alac}}$$

where dLa/dt_{max} (mmol · L^{-1} · s^{-1}) = maximal lactate formation rate; maxAELa (mmol · L^{-1}) = maximal lactate concentration after the exercise; RLa (mmol · L^{-1}) = lactate concentration at rest and before exercise, respectively; t_{exer} (s) = duration of exercise; t_{alac} (s) = period at the beginning of exercise for which (fictitiously) no lactate formation is assumed.

If the duration of exercise amounts to 10 s, t_{alac} is about 3 s; the corresponding values for a duration of exercise of 20 s and 50–60 s are, respectively, 4 s and 8 s. A duration of exercise between 10 and 30 s is appropriate, but a period of about 10 s should be preferred, because lactate formation rate is reduced with increasing duration of maximal exercise as a result of a suppression of phosphofructokinase activity owing to acidosis (Figure 7).

Table 2 shows some examples (100-m, 200-m, 400-m run).

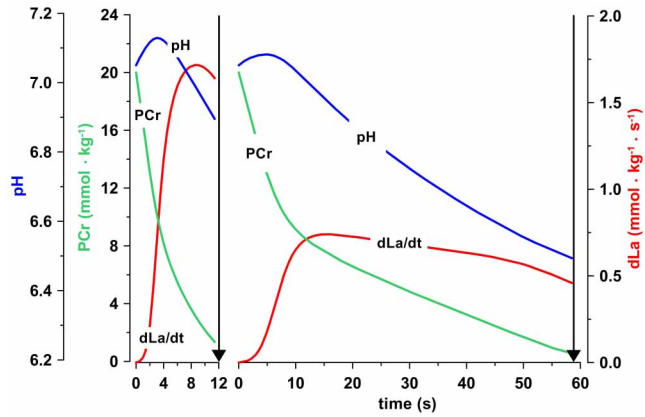


Figure 7 — Simulation of all-out exercise with a duration of about 10 s and 60 s (arrows = end of exercise). Maximal glycolytic rate and lactate production rate, respectively, show a reduction with increasing exercise duration. The data refer to wet muscle.

Table 2 Relation Between Mean Maximal Lactate Production Rate and Running Distance (Exercise Duration)

Variable	Running distance (m)		
	100	200	400
Running time t_{exer} (s)	10.5	21.5	45.0
t_{alac} (s)	3	4	8
maxAELa (mmol · L^{-1})	13	18	22
RLa (mmol · L^{-1})	1.9	1.9	1.9
dLa/dt_{max} (mmol · L^{-1} · s^{-1})	1.48	0.92	0.53

Note. dLa/dt_{max} (mmol · L^{-1} · s^{-1}) = maximal lactate formation rate, maxAELa (mmol · L^{-1}) = maximal lactate concentration after the exercise, RLa (mmol · L^{-1}) = lactate concentration at rest before exercise, t_{exer} (s) = duration of exercise, t_{alac} (s) = period at the beginning of exercise for which (fictitiously) no lactate formation is assumed.

Diagnostics of Lactic Capacity

An isolated assessment of lactic capacity is hardly possible, because lactic and alactic capacities cannot be differentiated with simple methods. If time to exhaustion is used as an indicator for lactic capacity, the lactic component cannot be estimated without knowing the level of maximal aerobic power.

The following are some examples for testing procedures for the assessment of lactic capacity.

Method for the Measurement of Oxygen Debt. As mentioned above, a fast and a slow component of oxygen debt can be registered after supramaximal exercise, with the slow component thought to be identical with lactic capacity. It was also mentioned above that a differentiation between slow and fast components is hardly possible. On the other hand, several studies have shown that oxygen debt often is higher than the sum of lactic and alactic capacity, which is theoretically possible. The reason given for this phenomenon is that glyconeogenesis (from lactate) as well as other metabolic processes taking place during the recovery phase need an additional amount of oxygen, which is measured with the oxygen debt.

Treadmill All-Out Exercise Test According to Schnabel and Kindermann (27). The treadmill all-out exercise test is carried out with a running velocity of 22 km/h and a treadmill grade of 7.5% until exhaustion. Indicators used for the assessment of lactic capacity are duration of exercise and maximal lactate concentration after the exercise. Mean maximal durations of exercise of about 90 s (400-m runners) and 45 s (marathon runners) were measured (11). As mentioned above, maximal duration of exercise in supramaximal tests is also influenced by aerobic power; therefore, duration of exercise is a less useful parameter compared to maximal lactate concentration after the exercise. Maximal lactate concentration after exercise is a direct indicator for muscular lactate taking into account processes of diffusion and elimination.

Diagnostics of Total Anaerobic Capacity

In the following passages, two methods for the assessment of anaerobic capacity are discussed in detail, because they have received broad attention in the international literature related to the subject.

Critical-Power Method. According to Monod and Scherrer (23), there is a hyperbolic relation between workload and time to exhaustion (Figure 8, left part), which can be described by the following formula:

$$t = w'/(P - Pc)$$

where t = duration of exercise (s), w' = anaerobic capacity (J), P = exercise intensity (W), and Pc = aerobic power (critical power; W).

Multiplication of the equation with the term (P-Pc) results in $P \cdot t = w' + Pc \cdot t$. This equation describes a linear relation between exercise duration and work done. The gradient of the straight line (Pc) thus corresponds to the aerobic power, the point of intersection with the ordinate to w' (anaerobic capacity; Figure 8, right part). In the context of this article, w' is of special interest.

The performance test according to Monod and Scherrer is based on the following assumptions:

- Anaerobic capacity is constant and is completely used in every test.
- Mechanical efficiency of muscular work is constant for the whole duration of exercise.
- Maximal aerobic power can already be used completely at the beginning of exercise.

Figure 9 illustrates the concept in a schematic way.

The third assumption is wrong, because there is an O_2 deficit at the beginning of each exercise.

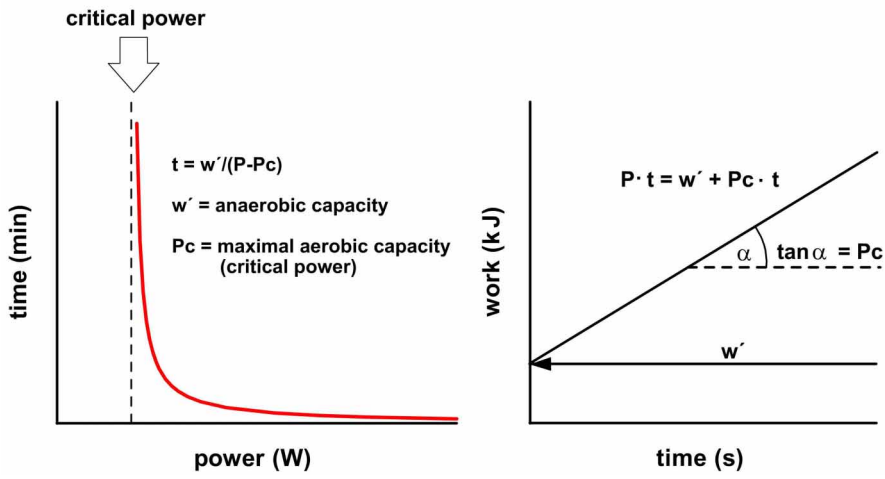


Figure 8 — Relation between exercise duration and exercise intensity (left). A mathematical transformation results in a work-time function (right).

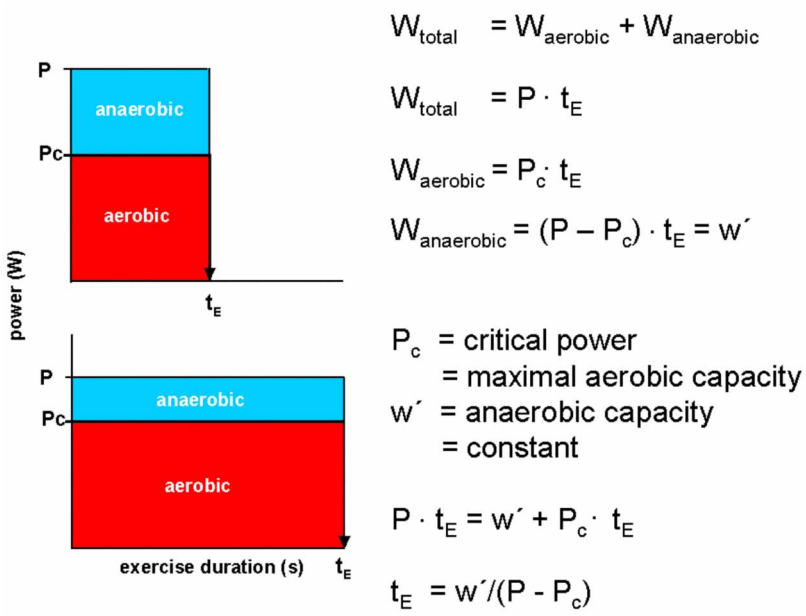


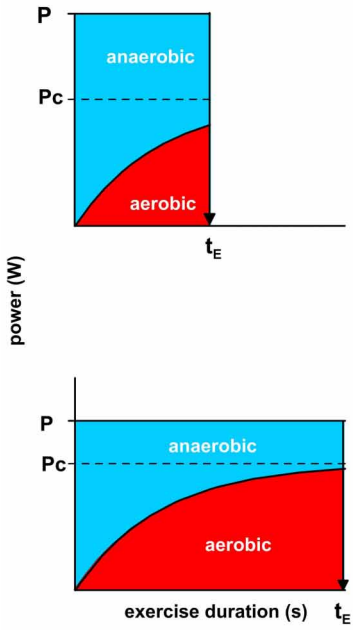
Figure 9 — Model of energy metabolism of the critical-power method. The duration (t_E) until exhaustion is dependent on maximal aerobic power (Pc = critical power) and on maximal anaerobic capacity ([P – Pc] · t_E). Maximal anaerobic capacity is assumed as constant.

On the assumption of a mono-exponential increase in oxygen uptake at the beginning of a constant exercise, the mathematical relations shown in Figure 10 result.

Using the data of Jeschke et al. (10), the function between work and exercise duration was calculated with and without taking account of oxygen deficit (Figure 11).

For the calculation, only the values between 60 s and 140 s were used, because shorter maximal exercise durations are not usual to date. For the time constant τ, a value of 25 s was assumed. The result can be interpreted in the way that, because of the oxygen deficit, a higher anaerobic capacity (24.1 kJ instead of 15 kJ) must be available to be able to tolerate a supramaximal exercise for an identical period of time. For shorter maximal exercise durations (20–40 s), the work values of Jeschke et al. (10) are below the values that have been expected from the calculations with and without oxygen deficit. This result can be explained by the inconstancy of the mechanical efficiency of muscular work dependent on exercise intensity. With increasing exercise intensity, more and more fast-twitch muscle fibers are recruited. The ATP consumption of these muscle fibers is higher for identical work values: for IIB fibers, about three times higher compared to slow-twitch muscle fibers.

Bishop et al. (1) studied the behavior of critical power with longer maximal exercise durations. In addition to usual exercise durations of 60–150 s, they examined periods of 300 s, 400 s, and 540 s. Longer exercise durations resulted in a



$$W_{total} = W_{aerobic} + W_{anaerobic}$$

$$W_{total} = P \cdot t_E$$

$$W_{aerobic} = P_c \int_0^{t_E} (1 - e^{-\frac{t}{\tau}})dt$$

$$= P_c \left[t_E - \tau \left(1 - e^{-\frac{t_E}{\tau}} \right) \right]$$

$$W_{anaerobic} = w' = \text{constant}$$

$$P \cdot t_E = w' + P_c \left[t_E - \tau \left(1 - e^{-\frac{t_E}{\tau}} \right) \right]$$

$$P = w'/t_E + P_c - P_c \cdot \tau \left(1 - e^{-\frac{t_E}{\tau}} \right) / t_E$$

Figure 10 — Like Figure 9 but with the almost exponential increase in oxygen uptake taken into account.

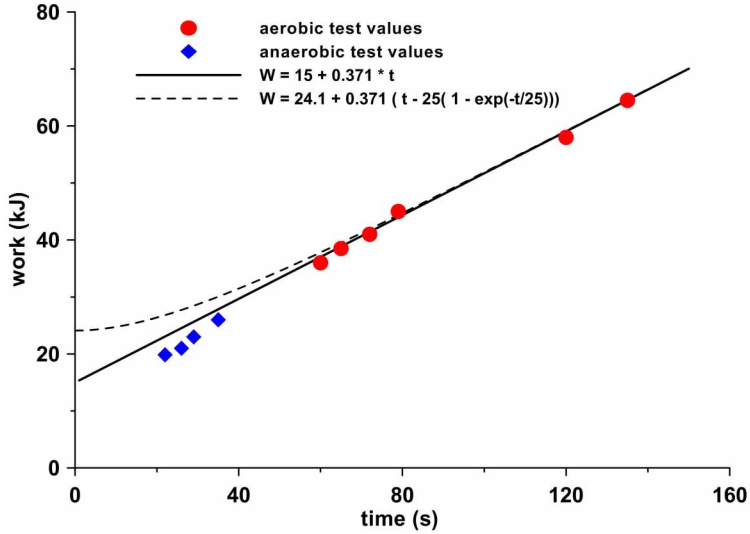


Figure 11 — Work-time diagram with data from Jeschke et al. (10). The dashed line represents the work-time function if the oxygen deficit during the first phase of exercise is taken into account.

reduction of critical power values from 201 W to 164 W, combined with an increase in aerobic capacity from 9.8 kJ to 17.6 kJ. The study was carried out with 10 female students of sport science.

Figure 12 shows the data of Jeschke et al. as well as those of Bishop et al. (1), with the data of Bishop et al. adapted to those of Jeschke et al. by transformation. The calculation of anaerobic capacity (w') and critical power (Pc) on the basis of the data from Figure 12 shows that the values for the two parameters depend strongly on maximal exercise duration and thus on exercise intensity (Figure 13).

The results can be interpreted in the way that high supramaximal workloads (and corresponding short exercise durations) lead to high critical power values and low values of anaerobic capacity. With a reduction in exercise intensity and an increase in maximal exercise duration, anaerobic capacity goes up and aerobic power goes down. Therefore, the critical-power method is neither an adequate testing procedure for the assessment of anaerobic capacity nor maximal aerobic power.

Maximally Accumulated Oxygen Deficit (MAOD)

The term *oxygen deficit* was introduced by Krogh and Lindhard in 1920 (12). *O_2 deficit* is defined as the difference between oxygen steady-state value and actual oxygen uptake at the beginning of muscular work. In 1988, Medbø et al. (20) described a testing procedure that allowed the determination of O_2 deficit as a measure of total anaerobic capacity. They started from the following assumptions (20):

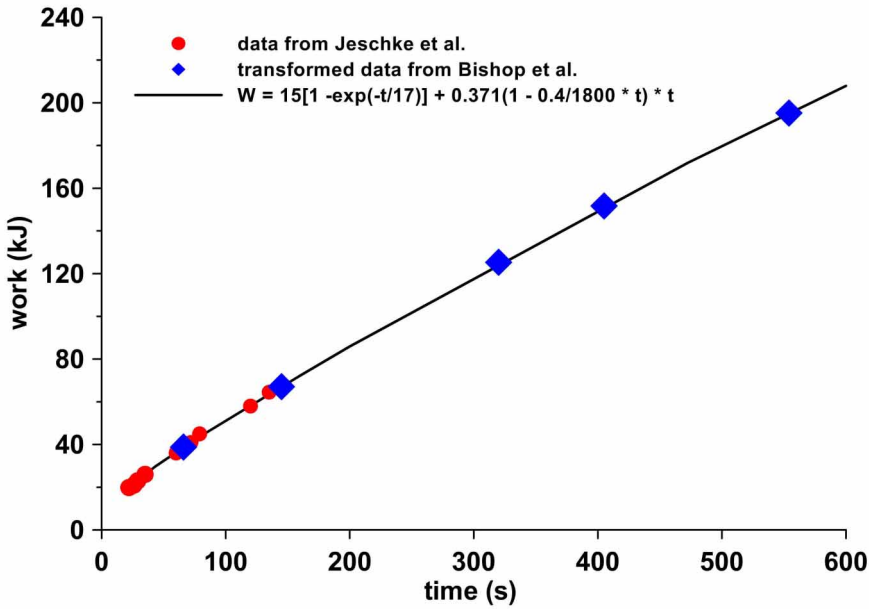


Figure 12 — Relation between work values and maximal exercise duration. The data come from Jeschke et al. (10) and Bishop et al. (1).

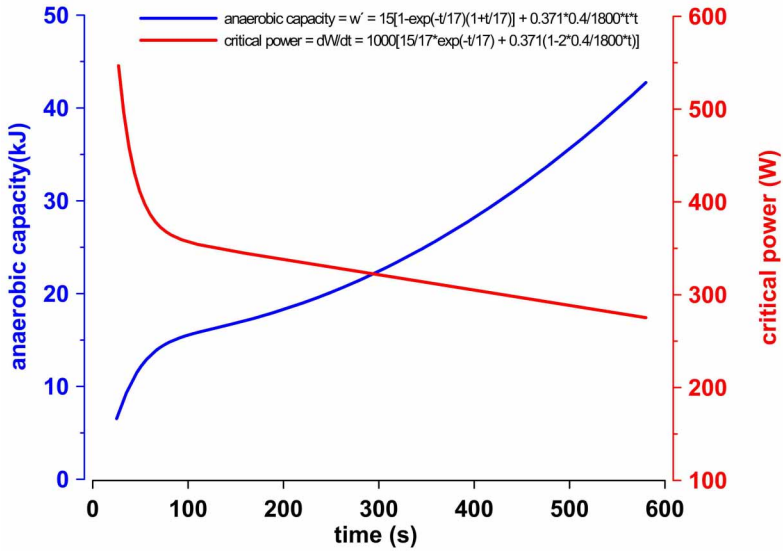


Figure 13 — Anaerobic capacity (w') and critical power (Pc) calculated on the basis of the data presented in Figure 12.

- Anaerobic energy release is the difference between total energy release and the aerobic energy component, which is determined by the accumulated oxygen uptake.
- O_2 demand increases with exercise intensity in a linear way.
- For a given workload, O_2 demand remains constant during the whole exercise duration.

If these assumptions are correct, the knowledge of O_2 demand for a given workload allows, by accumulated measurement of oxygen uptake throughout the whole exercise duration, the determination of accumulated oxygen deficit as the difference between O_2 demand and O_2 uptake. The oxygen deficit is made up of the oxygen stored in the blood and in the muscles, the consumption of phosphocreatine and ATP, and the amount of energy from the glycolysis leading to lactate formation. The contribution of the muscular oxygen stores amounts to only 10% (20).

For the determination of O_2 demand, at least 10 constant exercise tests with a duration of 10 min each and various intensities are carried out (20). Mean oxygen uptake during the last 2 min of exercise corresponds to the oxygen demand for the given workload. By means of a linear regression analysis, the function between power and oxygen uptake is determined. A fictitious example is shown in Figure 14A.

The regression equation $y = 350 + 11x$ allows the calculation of O_2 demand for any exercise intensity. Maximal oxygen uptake was achieved at 300 W, corresponding to 3650 ml · min^{-1}. For the assessment of maximally accumulated O_2 deficit, a supramaximal constant workload should be chosen (e.g., 450 W in Figure 14A–B), which has to be kept up until exhaustion (in this case 2.5 min). A workload of 450 W corresponds to an oxygen demand of 5300 ml · min^{-1}. This results in a total oxygen

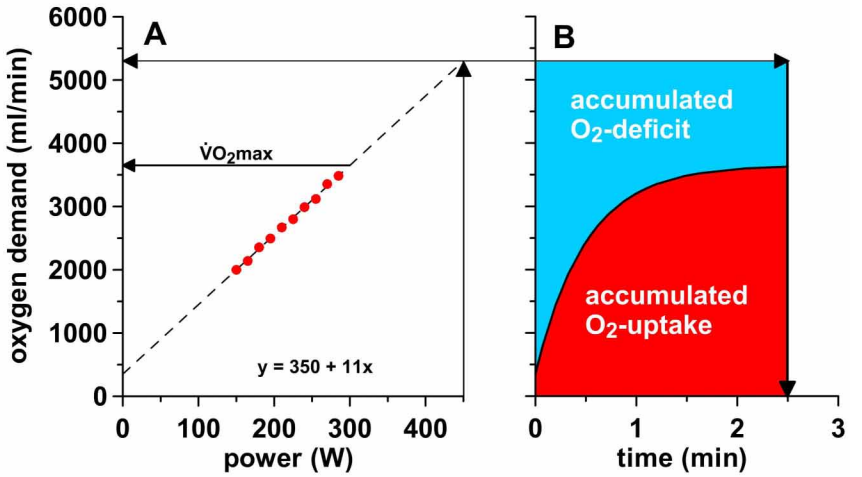


Figure 14 — Example for the method aiming at the assessment of maximally accumulated O_2 deficit (MAOD), modified according to Medbø et al. (20).

demand (for the 2.5-min period) of 13,250 ml. The O_2 uptake measured is 7475 ml. This value is made up of O_2 uptake at rest (350 ml · min^{-1} · 2.5 min = 875 ml) and the exponential component of O_2 uptake, which is calculated according to the formula presented in Figure 10 (τ = 0.5 min):

$$VO_2\,(ml) = (3650 - 350)\left[2.5 - 0.5\left(1 - e^{-\frac{2.5}{0.5}}\right)\right] \approx 6600$$

Thus a value of 5775 ml results for the accumulated oxygen deficit. Assuming a body mass of 75 kg, a relative MAOD of 77 ml · kg^{-1} is calculated. This value fits into the range (52–90 ml · kg^{-1}) of the data reported by Medbø et al. (20). The following problems are discussed:

Test Economy. Ten pretests for the assessment of the individual oxygen-workload function mean a high requirement regarding time and technology of measurement. Medbø et al. (20) suggest assuming a constant value for zero workload (i.e., rest) of 5.0 ml · min^{-1} · kg^{-1}.

For the assessment of the slope of the straight line, two 10-min tests with about 90% of maximal oxygen uptake should be carried out. If the difference between the two lines exceeds 3%, another 10-min test should be added.

Buck and McNaughton (2) examined the influence of a reduction in exercise duration, which means an improvement in test economy. They found an increasing reduction in MAOD parallel to a decline in exercise duration compared to the values of the 10-min test. A similar result was reported by Green and Dawson (4).

Duration of the Supramaximal Test. Medbø et al. found that a test duration of at least 2 min is necessary. A shorter maximal exercise duration leads to a reduction in MAOD, which can be explained by the fact that a complete exhaustion of anaerobic

lactic capacity obviously needs a certain amount of time. This assumption is supported by the observation of higher lactate values after the exercise when exercise duration is extended. Renoux et al. (26) also found an increase in accumulated O_2 deficit corresponding to an increase in exercise duration.

Linearity of the Oxygen-Workload Function. In several studies, Medbø et al. (20, 22) found that oxygen uptake does not always increase linearly with power. Mainly with low workloads, increase is disproportionately low. One important reason for a non-linearity of O_2 uptake in relation to workload with exercise intensities above maximal oxygen uptake is the fact that a nonlinear increase in ventilation demands a considerable amount of oxygen uptake when reaching the limits of performance. It is argued that the increasing O_2 demand needed for ventilation is included in the oxygen-workload function. But this argumentation ignores its disproportionate contribution.

Assuming a reduction in the mechanical efficiency of muscular work with very high workloads (see above), O_2 demand increases even more. If real O_2 demand is higher than the value predicted from the regression function, MAOD is underestimated.

Validity of MAOD. MAOD is determined for about 90% (see above) by alactic and lactic capacity. Therefore, MAOD and results from muscle biopsy should be highly correlated. Green et al. (5) carried out muscle biopsies (vastus lateralis) with 10 racing cyclists. Anaerobic energy contribution was calculated from the differences between resting values and values after exhaustive exercise for ATP, phosphocreatine, and lactate. The correlation coefficient between accumulated O_2 deficit and anaerobic energy contribution (measured in ATP-equivalents) was statistically not significant (r = –0.38). Possibly the group of subjects was too homogeneous regarding MAOD and anaerobic energy contributions.

Medbø and Tabata (21) examined 24 men with muscle biopsy (vastus lateralis) before and after exhaustive exercise of 30 s (n = 7), 1 min (n = 8), and 2–3 min (n = 9). For the calculation of anaerobic energy contribution, they used the same formula as Green et al. In contrast to Green et al., they found a very high correlation coefficient (r = 0.94). The high correlation coefficient can be explained as a group effect. The lowest mean values of MAOD thus are found with a maximal exercise duration of 30 s, the highest ones with a test duration of 2–3 min. The anaerobic energy contribution showed identical results. Additionally, the correlation coefficient was calculated for the mean metabolic rates (energy output per second) thus increasing the group effect even more. It is not quite understandable why Medbø and Tabata (21) did choose this methodological approach, because Medbø et al. (20) had already shown that exercise durations of at least 2 min are necessary to determine MAOD (see above).

This means that other studies using muscle biopsy are needed to answer the question to what extent the MAOD concept is a valid method for the determination of anaerobic capacity.

Isokinetic 90-s All-Out Test According to Lorenz et al. (13, 14)

Modifying the Wingate anaerobic test (see above), Lorenz et al. (13, 14) carried out studies using a bicycle ergometer with an isokinetic mode. Pedaling rates of 90 rpm (13) and 80 rpm (14), respectively, were set. The exercise duration was 96 s.

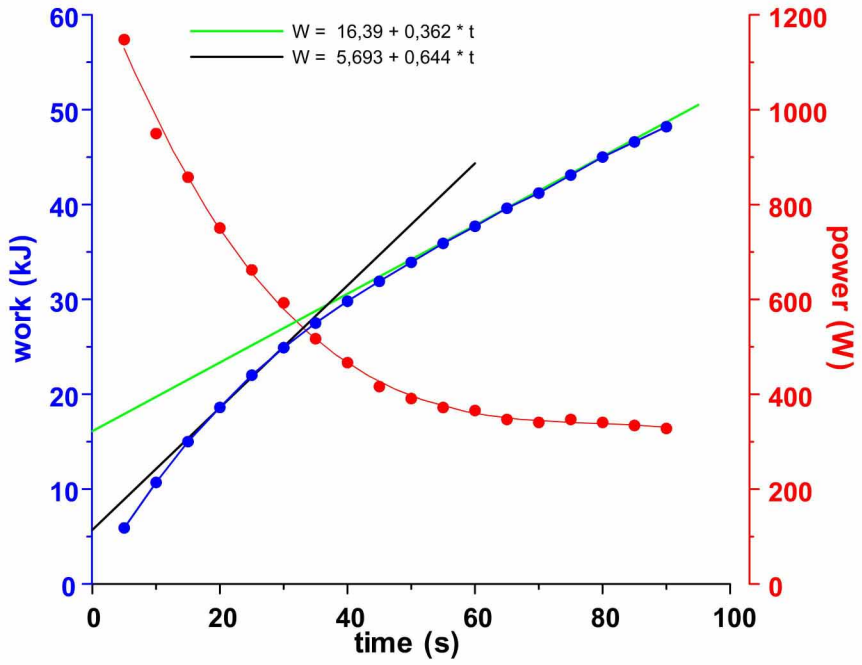


Figure 15 — Example for the isokinetic 90-s all-out test carried out on the bicycle ergometer according to Lorenz et al. (13). The gradient of the upper regression line (green line) is thought to be identical with maximal oxidative metabolic rate; the intersection with the ordinate is assumed to represent total anaerobic capacity. The gradient of the lower regression line (black line) is thought to represent total metabolic rate (sum of maximal glycolytic and maximal oxidative metabolic rate); the intersection with the ordinate is assumed to be identical to alactic capacity (data from Lorenz et al., 13).

Figure 15 shows an example for the relations between work and exercise duration as well as power and exercise duration. The red dots represent the mean power values for every 5-s period; the blue dots stand for the work done (i.e., integral of power over time).

According to the authors, the gradient of the regression line between values of 15 s and 30 s is a measure for the sum of maximal glycolytic and oxidative metabolic rate (W); the intersection of the regression line with the ordinate (work) corresponding to alactic capacity (J).

The authors make analogous claims about the interval between 60 s and 90 s: In this case the gradient of the regression line is to represent maximal oxidative metabolic rate, and the intersection with the ordinate is assumed to correspond to the sum of alactic and lactic capacity (i.e., total anaerobic capacity). For the example given in Figure 15, the following values result:

- anaerobic capacity = 16,388 J (alactic capacity = 5,693 J, lactic capacity = 10,695 J)

- total metabolic rate = 644 W (oxidative metabolic rate = 362 W, glycolytic metabolic rate = 282 W).

A physiological model (analogously to the model behind the critical-power method), however, is not presented by the authors so that the theoretical concept of the method is not discernible.

An analysis of the power-time curve in Figure 15, on the basis of the theoretical explanations, and Figures 1–3, at the beginning of this review, leads to the following statements:

A power value of 644 W (total metabolic rate) is measured for a time value of about 25 s. Assuming a maximal oxidative metabolic rate of 362 W (measured in the interval between 60 s and 90 s), this value cannot be reached after 25 s because of the delayed O_2 kinetics. For the example given in Figure 3, the corresponding value is less than 60% of the maximal value. As the contribution of glycolysis (maximal glycolytic metabolic rate) to total power is calculated as a difference, the resulting value is too low. This also is an explanation for the relatively low glycolytic value of 282 W in contrast to the oxidative contribution (362 W). With untrained persons, lactic power is about twice as high as aerobic power (see Table 1).

A power value of 362 W (maximal oxidative metabolic rate) in Figure 15 is measured between 60 s and 90 s. The method implies that energy metabolism in this period is all oxidative. Keeping up a workload of 362 W would, however, result in a continuous increase in muscle and blood lactate concentration, which are clear indicators for an anaerobic lactic contribution to total energy metabolism. This means, however, that maximal oxidative metabolic rate is determined too high.

Total anaerobic capacity is determined with the help of the regression line representing the relation between work and exercise duration using the interval between 60 s and 90 s. In the example given in Figure 15, the regression line intersects the ordinate at 16.4 kJ. In this case, Lorenz et al. follow the critical-power method which, however, demands a constant workload. Applying this concept to changing workloads (reduction in power with increasing exercise duration) is not allowed. Therefore, a differentiation between alactic and lactic contribution to total power is not possible.

The validation of the method was carried out by calculating several correlation coefficients:

- alactic capacity (J) versus maximal ammonia concentration (36-s test): r = 0.38*
- lactic capacity (J) versus maximal lactate concentration (96-s test): r = 0.62**
- total metabolic rate (W) versus maximal power (W, graded exercise test): r = 0.92***
- oxidative metabolic rate (W) versus power at 4 mmol/L lactate (W, graded exercise test): r = 0.88***
- glycolytic metabolic rate (W) versus lactate in an all-out test after about 25 s: r = 0.59**.

The significant increase in blood ammonia during exercise of high intensity comes from the purine nucleotide cycle. In the myokinase reaction, ATP (and AMP) are formed from 2 ADP. A certain amount of AMP is converted to IMP (inosine monophosphate), this reaction resulting in the formation of ammonia, too. As AMP formation is mainly dependent on the amount of ATP being available and not on the

amount of phosphocreatine, ammonia is no adequate validation parameter for alactic capacity.

This statement is supported by the low correlation coefficient ($r = 0.38$). The correlation coefficient between lactic capacity and maximal lactate concentration ($r = 0.62$) also is too low in the context of validation. This possibly is an indication of the fact that the regression line does not determine what it pretends to. The correlation coefficients between oxidative metabolic rate and power at 4 mmol/L lactate ($r = 0.88$) are within a range that is tolerable for validation, thus fulfilling a necessary condition. But this is not sufficient. Additionally, it should be examined whether the regression line corresponds to the line of identical values. This examination is necessary if the new method is expected to lead to identical results. But this point is not clarified by Lorenz et al.

All points of criticism mentioned above illustrate that the isokinetic 90-s all-out test offers no satisfying solution for the determination of anaerobic capacity or anaerobic power.

Concluding Remarks

In the opinion of the authors, there is at the moment no simple testing procedure to precisely determine anaerobic capacity and anaerobic power subdivided into their alactic and lactic components. Because of the complex interactions between these various components of anaerobic and aerobic energy metabolism, a detailed assessment of these components can only be achieved using a battery of tests.

This test battery should include an all-out 5-s test for the assessment of alactic power, an all-out 10-s test for the determination of lactic power, and an all-out 40–100 s test, with post-test measurement of maximal lactate concentration, for the assessment of lactic capacity. Additionally, the determination of maximal oxygen uptake is recommended, especially when using maximal exercise duration as an indicator of lactic capacity.

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