Anaerobic capacity determined by maximal accumulated O2 deficit

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Anaerobic capacity determined by maximal accumulated O2 deficit. J. Appl. Physiol. 64(1): 50–60, 1988.—We present a method for quantifying the anaerobic capacity based on determination of the maximal accumulated O2 deficit. The accumulated O2 deficit was determined for 11 subjects during 5 exhausting bouts of treadmill running lasting from 15 s to >4 min. The accumulated O2 deficit increased with the duration for exhausting bouts lasting up to 2 min, but a leveling off was found for bouts lasting 2 min or more. Between-subject variation in the maximal accumulated O2 deficit ranged from 52 to 90 ml/kg. During exhausting exercise while subjects inspired air with reduced O2 content (O2 fraction = 13.5%), the maximal O2 uptake was 22% lower, whereas the accumulated O2 deficit remained unchanged.

The precision of the method is 3 ml/kg. The method is based on estimation of the O2 demand by extrapolating the linear relationship between treadmill speed and O2 uptake at submaximal intensities. The slopes, which reflect running economy, varied by 16% between subjects, and the relationships had to be determined individually. This can be done either by measuring the O2 uptake at a minimum of 10 different submaximal intensities or by two measurements close to the maximal O2 uptake and by making use of a common Y intercept of 5 ml·kg\(^{-1}\)·min\(^{-1}\). By using these individual relationships the maximal accumulated O2 deficit, which appears to be a direct quantitative expression of the anaerobic capacity, can be calculated after measuring the O2 uptake during one exhausting bout of exercise lasting 2–3 min.

The concept of O2 deficit was first introduced by Krogh and Lindhard (22) in 1920 as the difference between the curve of the actual O2 uptake at the beginning of exercise and the steady-state level of the O2 uptake. Hermansen reintroduced the principle in 1969 and calculated the accumulated O2 deficit (called "oxygen deficit" in his paper) as the area between the curve of the O2 demand and the curve of the actual O2 uptake (Fig. 2 in Ref. 10). During the last 15 years the accumulated O2 deficit has been determined in several studies to quantify anaerobic ATP formation during exhausting bicycle exercise (12, 18–21, 26, 28, 36). However, most of these studies used one common relationship between the exercise intensity and the estimated O2 demand. In addition they did not show their values to be maximal. Hence it is not

To distinguish between concepts related to energy and to power we define six expressions used in this article.

Three expressions for power expressed in watts or as flow of O2 were used: O2 uptake: the measured flow of O2 taken up; O2 demand: estimate of the rate of total energy release; and O2 deficit: the difference between the estimated O2 demand and the measured O2 uptake.

Three expressions for energy expressed in joules or as volume of O2 were used: accumulated O2 uptake: the measured O2 uptake integrated over time; accumulated O2 demand: the estimated O2 demand integrated over time (this is an estimate of the total energy release); and accumulated O2 deficit: the difference between the accumulated O2 demand and the accumulated O2 uptake.

The relationship between these concepts is as follows: for power, O2 demand − O2 uptake − O2 deficit; for energy, accumulated O2 demand − accumulated O2 uptake = accumulated O2 deficit.

At submaximal intensities (intensities not stressing the ability to take up O2 maximally) the steady-state O2 uptake is assumed to reflect the total rate of energy release during exercise. For supramaximal intensities (intensities with a rate of energy release exceeding the maximal O2 uptake) the rate of energy release or O2 demand can be estimated by extrapolating the linear relationship between exercise intensity and the steady-state O2 uptake at submaximal intensities. The accumulated O2 deficit for a given exercise bout at a constant intensity is then equal to the calculated accumulated O2 demand minus the measured accumulated O2 uptake.

The present study was therefore carried out to show that the accumulated O2 deficit under appropriate exercise conditions seems to provide an accurate estimate of the anaerobic capacity. The concept of O2 deficit was first introduced by Krogh and Lindhard (22) in 1920 as the difference between the curve of the actual O2 uptake at the beginning of exercise and the steady-state level of the O2 uptake. Hermansen reintroduced the principle in 1969 and calculated the accumulated O2 deficit (called "oxygen deficit" in his paper) as the area between the curve of the O2 demand and the curve of the actual O2 uptake (Fig. 2 in Ref. 10). During the last 15 years the accumulated O2 deficit has been determined in several studies to quantify anaerobic ATP formation during exhausting bicycle exercise (12, 18–21, 26, 28, 36). However, most of these studies used one common relationship between the exercise intensity and the estimated O2 demand. In addition they did not show their values to be maximal. Hence it is not

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THE ADENOSINE TRIPHOSPHATE broken down during exercise is continuously resynthesized by aerobic and anaerobic processes. Anaerobic ATP formation during exercise stems from breakdown of phosphocreatine and glycogen, the latter increasing muscle and blood lactate concentrations (18, 26). Anaerobic capacity can be defined as the maximal amount of ATP formed by these anaerobic processes during exercise. Since both the stores of phosphocreatine and the extent to which lactate can accumulate seem to be limited (8, 19, 31–33), it may be hypothesized that anaerobic capacity is a well-defined individual entity.

So far available methods have not allowed precise estimates of the anaerobic capacity. The present study was therefore carried out to show that the accumulated O2 deficit under appropriate exercise conditions seems to provide an accurate estimate of the anaerobic capacity.
known whether these data reflect the anaerobic capacity. For the accumulated $O_2$ deficit to provide a good estimate of the anaerobic capacity the following criteria should be met.

1) **Leveling off with exercise duration.** It has been argued that the amount of ATP formed anaerobically during short-lasting exhaustive exercise is independent of the duration of the bout (10, 25). This is in conflict with the argument that there are probably rate-limiting steps in the anaerobic as well as the aerobic ATP formation. If there are limitations on both the amount of ATP formed by anaerobic processes and the rate of these processes, the accumulated $O_2$ deficit would be expected to increase with the duration of the exhausting exercise until a leveling off is observed. To test this hypothesis treadmill exercise was done at speeds individually selected to cause exhaustion after 15 and 30 s, and 1, 2, and 4 min or more.

2) **Independence from maximal $O_2$ uptake.** The glycolytic enzymes are shared by the aerobic and anaerobic ATP-forming pathways. However, the rate-limiting steps in the former process reside after the formation of pyruvate. The maximal rate of aerobic glycogen oxidation is therefore much lower than the capacity of the glycolytic enzymes. Hence anaerobic capacity and the maximal $O_2$ uptake are expectedly two independent variables. Measurements were therefore repeated in hypoxia to see whether the maximal $O_2$ uptake could be lowered independently of the accumulated $O_2$ deficit.

3) **Agreement with existing methods.** One approach that has been used with some success for estimating the anaerobic capacity is measurements of changes in the metabolites linked to anaerobic ATP formation, that is, changes in the phosphocreatine and lactate concentrations, the latter both in muscle and blood. However, to transform these measurements of concentrations into quantities, assumptions of the distribution volumes must be done. Hence the methods are imprecise but may give a rough picture. In the present study the accumulated $O_2$ deficit was compared with the blood lactate concentration measured after exercise as well as with data in the literature on changes in muscle phosphocreatine and lactate concentrations.

In the APPENDIX we evaluate the precision of the method. Some results have been published in preliminary reports (12, 10).

**SUBJECTS, PROCEDURES, AND METHODS**

**Subjects.** Eleven healthy male volunteers served as subjects; three of them repeated the experiments with reduced $O_2$ fractions in the inspired air. The subjects underwent a medical examination and were fully informed about the experimental procedures before they gave their written consent. Table 1 gives pertinent characteristics of the subjects.

The protocol of the experiments and the procedures involved were approved by the Ethics Committee at the National Institute of Occupational Health.

**Procedures.** All experiments as well as pretests were done on the treadmill at 6° (10.5%) inclination to keep the treadmill speed reasonably low even at the highest exercise intensities. The subjects were trained in treadmill running before any testing started. The maximal $O_2$ uptake was determined by a procedure modified from that of Taylor et al. (11, 39). Over a period of 3 wk before the experiments the steady-state $O_2$ uptake was measured during the last 2 min of ~20 different submaximal intensities (range: 35–100% of maximum $O_2$ uptake) of 10 min duration. In pretests we observed no further increase in the $O_2$ uptake during 30 min of exercise. Hence the $O_2$ uptake during the sampling interval represents a steady state. The subjects were instructed not to do any strenuous exercise the day before these tests to exclude a possible effect on the $O_2$ uptake at submaximal intensity.

For each subject all results relating submaximal treadmill speed to the steady-state $O_2$ uptake were plotted and visually checked for linearity, and deviating values were excluded. A linear relationship was determined for each subject by calculating the regression of the steady-state $O_2$ uptake on exercise intensity (here: treadmill speed, see in Fig. 1, left), thus expressing the $O_2$ demand for all intensities. This time-consuming procedure, which provides a reliable relationship between treadmill speed and $O_2$ demand, can be simplified as described in the APPENDIX (procedure 3).

The subjects exercised on the treadmill at supramaximal intensities (i.e., above the maximal $O_2$ uptake) to exhaustion. At different days the treadmill speed was varied to cause exhaustion within ~15 and 30 s, and 1, 2, and 4 min. Five of the subjects also made an exhausting run lasting >5 min. The exhausting exercises were done on different days in random order. Before each run the subjects warmed up for 15 min at 50% of their maximal $O_2$ uptake followed by 10 min of rest. The treadmill speed used to cause exhaustion within the desired duration was established on pretests.

The individual linear relationship between intensity and $O_2$ uptake was extrapolated to the actual intensity at the different exhausting bouts (see the stippled extrapolation of the regression line in Fig. 1), and the corresponding $O_2$ demand was determined. The accumulated $O_2$ demand was taken as the product of the $O_2$ demand and the duration of the exercise (Fig. 1B), assuming that the $O_2$ demand was constant throughout the whole exercise period. Expired air was continuously collected in Douglas bags during each exhausting run, and the accumulated $O_2$ uptake (Fig. 1, left) was measured.

The blood lactate concentration was determined in capillary blood samples taken from a prewarmed finger before the exercise, immediately after exercise (20–30 s), and 1, 3, 6, 9, and 12 min after exercise. For each blood sample 20 μl of blood was pipetted into 500 μl of 0.4 mol/l perchloric acid and kept on ice for later measurement of the blood lactate concentration.

For the hypoxic experiments the same procedures were followed except for the following. A 600-liter spirometer (chain-compensated gasometer, W. E. Collins, Braintree, MA) was filled with gas containing 13.5 ± 0.2% $O_2$ in
TABLE 1. Physical characteristics of the subjects

<table>
<thead>
<tr>
<th>Subj</th>
<th>Ht, m</th>
<th>Wt, kg</th>
<th>Age, yr</th>
<th>$\bar{V}O_2_{max}$ ml·kg⁻¹·min⁻¹</th>
<th>Slope of Regression Line* ml·kg⁻¹·m⁻¹</th>
<th>Background</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACM</td>
<td>1.86</td>
<td>77</td>
<td>24</td>
<td>64.0</td>
<td>51.1</td>
<td>Middle distance runner</td>
</tr>
<tr>
<td>FH</td>
<td>1.86</td>
<td>69</td>
<td>21</td>
<td>65.3</td>
<td>51.4</td>
<td>Middle distance runner</td>
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<tr>
<td>HCH</td>
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<td>78</td>
<td>19</td>
<td>52.4</td>
<td>43.4</td>
<td>Untrained</td>
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<tr>
<td>IT</td>
<td>1.86</td>
<td>65</td>
<td>27</td>
<td>67.0</td>
<td>53.6</td>
<td>Untrained</td>
</tr>
<tr>
<td>JH</td>
<td>1.85</td>
<td>74</td>
<td>26</td>
<td>52.4</td>
<td>43.4</td>
<td>Middle distance runner</td>
</tr>
<tr>
<td>KTS</td>
<td>1.80</td>
<td>64</td>
<td>19</td>
<td>53.6</td>
<td>43.6</td>
<td>Untrained</td>
</tr>
<tr>
<td>LH</td>
<td>1.77</td>
<td>87</td>
<td>19</td>
<td>48.7</td>
<td>43.9</td>
<td>Untrained</td>
</tr>
<tr>
<td>MKS</td>
<td>1.80</td>
<td>75</td>
<td>23</td>
<td>65.0</td>
<td>43.9</td>
<td>Speed skater (sprint)</td>
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<tr>
<td>PB</td>
<td>1.84</td>
<td>79</td>
<td>34</td>
<td>63.5</td>
<td>43.9</td>
<td>Runner and canoeist</td>
</tr>
<tr>
<td>RB</td>
<td>1.37</td>
<td>94</td>
<td>26</td>
<td>60.9</td>
<td>43.9</td>
<td>Volleyball player</td>
</tr>
<tr>
<td>RN</td>
<td>1.80</td>
<td>79</td>
<td>29</td>
<td>65.9</td>
<td>43.9</td>
<td>Speed skater (all round)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>60.1±8.1</td>
<td>46.1±5.1</td>
<td>0.298±0.018</td>
</tr>
</tbody>
</table>

Means ± SD l&3±0.08 77&B 24+5 60.1±8.1 46.1±5.1† 0.298±0.018

$V_{O_2_{max}}$, maximum $O_2$ uptake. * Slope of the regression of the steady-state oxygen uptake on treadmill speed at 6° inclination. † n = 3.

FIG. 1. Principles for determining $O_2$ deficit. A: relationship between exercise intensity (treadmill speed) and $O_2$ demand. B: accumulated $O_2$ deficit is calculated as difference between accumulated $O_2$ demand and accumulated $O_2$ uptake of exercise. Subject RB ran for 2.45 min at 267 m/min, corresponding to an $O_2$ demand of 86 ml·kg⁻¹·min⁻¹.

The subjects inspired this gas during the exhausting bouts, simulating exercise at ~3,500 m altitude. The exact fraction of $O_2$ was measured for each experiment. The subjects were breathing the hypoxic gas for at least 4 min before the experiments as well as during the exercise. To enable the subjects to do exhausting bouts of approximately the same duration, the treadmill speed had to be reduced by 10–40 m/min (at 6°), corresponding to a reduction in the $O_2$ demand of 3–11 ml·kg⁻¹·min⁻¹ (3–15%). Each subject participating in these experiments did four exhausting bouts lasting 30 s to 4 min.

On separate days the steady-state $O_2$ uptake was measured at the end of at least four different submaximal intensities of 10 min duration while the subjects inspired gas with reduced fractions of $O_2$. For submaximal intensities the $O_2$ uptake was unaltered whether the subjects exercised in normoxia or hypoxia. Therefore the same relationships between intensity and the $O_2$ demand were used for both experimental conditions.

Analytical methods. The expired volume was measured in a wet spirometer. Fractions of $O_2$ and CO₂ were determined on a Scholander gas analyzer (35) or on an automatic system (CO₂: CO₂-analysator, Simrad Optronics, Oslo, Norway; O₂: SSA/I Ametek, Pittsburgh, PA). All gas volumes were expressed as STPD. Blood lactate concentration was measured enzymatically (24).

Statistics and calculations. Individual regression lines were determined by a standard procedure (3, 37). Data are presented as individual values or means ± SD. Statistical tests were done using Student's matched-pair $t$ test (one-sided or two-sided whenever appropriate). The error in determining the accumulated $O_2$ deficit for exhausting bouts of different durations was determined by
summing the variances (SE²) of the error in its components (see the APPENDIX).

To convert the energy released by lactate formation and phosphocreatine breakdown to volume of O₂, 1 mol of lactate formed and 1 mol of phosphocreatine broken down was set equal to 1.5 and 1 mol ATP, respectively, whereas 6.5 mol ATP was considered equal to 1 mol (22.39 liters STPD) of O₂ (as for oxidation of glycogen).

RESULTS

Figure 2 shows the individual values for the accumulated O₂ deficit vs. the duration of the exhausting bouts. For bouts lasting <2 min the accumulated O₂ deficit increased with each increase in the duration (P < 0.001). For durations exceeding 2 min the accumulated O₂ deficit leveled off, and there were no statistically significant differences in the accumulated O₂ deficit between exhausting bouts lasting 2, 4, or >5 min (P > 0.2). It can thus be concluded that the accumulated O₂ deficit reached an easily definable maximum for exhausting exercise lasting 2 min or longer.

There were large differences in the maximal accumulated O₂ deficit between the subjects (range: 52–90 ml/kg). The same relative range of variation was found for the shorter exercise durations. However, when the accumulated O₂ deficit for exhausting bouts lasting 15, 30, and 60 s was expressed as fractions of the maximal accumulated O₂ deficit, the variation was considerably reduced (Fig. 3A). The respective fractions of the maximal accumulated oxygen deficit averaged 34% for 15 s, 50% for 30 s, and 75% for 60 s duration. This means that the subjects with the largest maximal accumulated O₂ deficit were able to accumulate a given O₂ deficit (e.g., 50 ml/kg) faster than the subjects with a smaller maximal value. Figure 3B also illustrates that, since exercise of different durations all were done until exhaustion, the exercise intensity decreased with the duration.

To establish the maximal accumulated O₂ deficit as a separate and distinctive capacity, the same experiments were repeated under conditions that lower the maximal O₂ uptake. By reducing the O₂ fraction in the inspired gas to 13.5 ± 0.2%, the maximal O₂ uptake was reduced by 13 ± 4 ml·kg⁻¹·min⁻¹ or 22% (Table 1, P < 0.001), and the accumulated O₂ deficit increased insignificantly by 0.7 ± 4.5 ml/kg (Fig. 2, P = 0.60). Hence, in our experiments the aerobic power was unrelated to the maximal accumulated O₂ deficit.

Since the treadmill speed was reduced by ∼15% for the 4-min exhausting bout in hypoxia compared with the matched bout in normoxia, the treadmill speed used for these 4-min exhausting hypoxic experiments equaled the highest intensity used for the submaximal bouts in normoxia.

The peak blood lactate concentration, which was found 3–9 min after the end of the exercise, increased with exercise durations <2 min (P < 0.02). For bouts lasting >2 min no further increase in the peak blood lactate concentration was seen (P > 0.3, Table 2). The pooled mean blood lactate concentration 30 s after exercise was 13.4 mmol/l for bouts lasting 2 min or more and less for bouts of shorter duration (P < 0.001).

The peak postexercise blood lactate concentration did not differ significantly after exercise in hypoxia compared with normoxia for all exercise durations (P > 0.10).

The methodological imprecision in determining the accumulated O₂ deficit for bouts of different durations is given in Table 2. The imprecision increases with the duration of the exhausting bouts because several important components of the imprecision are proportional to the duration (see the APPENDIX). Thus, for the exercise durations where the accumulated O₂ deficit reached a maximum, the smallest error in the measurements was observed for 2 min duration. During the last 30 s of the exhausting bouts of 2 min duration, the O₂ deficit was 0.39 ± 0.06 ml·kg⁻¹·s⁻¹. This means that if exercise were terminated 10 s before exhaustion, the accumulated O₂
DETERMINATION OF ANAEROBIC CAPACITY

FIG. 3. A: relative accumulated $O_2$ deficit (in % of maximal accumulated $O_2$ deficit) vs. duration of supramaximal bouts in normoxia. B: $O_2$ demand [relative to maximal $O_2$ uptake ($\dot{V}O_{2\text{max}}$)] vs. duration of bouts in normoxia. All exercise bouts were done to exhaustion, irrespective of duration.

TABLE 2. Lactate concentration immediately after exercise, peak postexercise lactate concentration, and absolute (and relative) precision in determining accumulated $O_2$ deficit for exhausting bouts

<table>
<thead>
<tr>
<th>Duration of Exhausting Bout</th>
<th>15 s</th>
<th>30 s</th>
<th>1 min</th>
<th>2 min</th>
<th>4 min</th>
<th>7 min*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood La concn 30 s postex, mmol/l</td>
<td>5.8±2.0</td>
<td>9.1±2.1</td>
<td>9.6±2.5</td>
<td>12.8±1.7</td>
<td>13.6±2.6</td>
<td>14.2±1.4</td>
</tr>
<tr>
<td>Peak blood La concn, mmol/l</td>
<td>9.4±1.5</td>
<td>12.5±1.7</td>
<td>14.8±1.7</td>
<td>16.6±1.9</td>
<td>17.2±2.5</td>
<td>16.6±1.4</td>
</tr>
<tr>
<td>Statistical error in determining $O_2$ deficit, ml/kg†</td>
<td>0.9 (4%)</td>
<td>1.2 (4%)</td>
<td>1.9 (4%)</td>
<td>3.0 (4%)</td>
<td>4.9 (7%)</td>
<td>7.2 (10%)</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 14. La, lactate; postex, postexercise. * n = 5. † Error as square root of sum of variances in determining accumulated $O_2$ uptake, treadmill speed and inclination, duration of exercise, and accumulated $O_2$ demand for a given treadmill speed.

deficit would be underestimated by 4 ml/kg.

The slopes of the individual regressions of $O_2$ demand on exercise intensity averaged 0.298 ml·kg⁻¹·m⁻¹ and ranged from 0.272 to 0.320 ml·kg⁻¹·m⁻¹ (Table 1). The $Y$-intercepts, on the other hand, averaged 5.1 ml·kg⁻¹·min⁻¹ and ranged from 4.2 to 5.9 ml·kg⁻¹·min⁻¹. Thus the slopes reflects a 16% range of efficiency (running economy), which was six times the mean standard error in the individual slopes. The mean SE in the individual $Y$ intercepts was 1.2 ml·kg⁻¹·min⁻¹, which means that the variations in the $Y$-intercepts was only due to random variation.

DISCUSSION

The accumulated $O_2$ deficit reached a maximum value for exhaustive bouts of running lasting 2 min or more.
We define this maximum as the anaerobic capacity. It has the dimension of energy and can hence be expressed in joules, amount of high-energy phosphates, or volume of O_2. This contrasts with the O_2 uptake having the dimension of flow or power (energy per time). Blood lactate concentration, an indicator of anaerobic catabolism, showed the same trend of leveling off for durations exceeding 2 min, supporting that a maximal value was found by the present method.

There are two basic assumptions for calculating the accumulated O_2 deficit: (1) the O_2 demand is constant during the whole exercise period as originally suggested by Krogh and Lindhard (22), and (2) the O_2 demand can be determined by extrapolating the linear relationship between the steady-state O_2 uptake and treadmill speed at submaximal exercise intensities (10). There are several arguments in support of these assumptions.

First, energy from anaerobic ATP formation is of little significance during submaximal exercise, even when blood and muscle lactate concentrations of several millimolars are seen. A lactate concentration of 4 mmol/l will correspond to roughly 10 ml O_2/kg, which is a small fraction of the total O_2 consumption during a 10-min bout (12). Moreover, the major part of the anaerobic ATP formation occurs during the first few minutes of the exercise, as evidenced by the time required for the O_2 uptake to reach a steady level (2, 7, 40) as well as by the rapid increase in the blood lactate concentration at the beginning of exercise (14). After 5 min of exercise at a constant submaximal intensity, further net lactate accumulation is negligible (14).

Second, running economy probably does not improve at high speeds. Hence, if nonlinearity exists, one would expect the O_2 demand to be underestimated, especially for the highest intensities. If that were the case, the estimate of the accumulated O_2 deficit would be smaller than the true value, in particular for the bouts of the shortest duration (highest intensity). The plateau found for bouts lasting 2 min or more would be less affected.

Third, the accumulated O_2 deficit was not affected by the reduced maximal O_2 uptake in hypoxia. This means that our estimate of the anaerobic capacity was the same for different intensities when normoxia and hypoxia were compared, which is in accordance with the preconditions of the anaerobic capacity concept. Of note is that the intensity leading to exhaustion in 4 min in hypoxia was below the maximal O_2 uptake in normoxia. Since the same relationships between intensity and oxygen demand was found in hypoxia and normoxia, the O_2 demand for this latter bout has been measured directly and was not calculated by extrapolation.

Fourth, the fact that leveling off was easily seen for all subjects and for all bouts lasting 2 min or more is a strong indication that we found a true maximum and not only a random result of two opposing tendencies. We therefore conclude that the linear extrapolation of the O_2 demand is justified even for the highest intensities.

The linear relationship between the steady-state O_2 uptake and the treadmill speed was precisely established for each individual by repeated trials at different submaximal intensities. This proved to be important because the error in the estimate of the oxygen demand is heavily dependent on the slope of the regression line. Previous authors have used a mean exercise efficiency (20, 21, 23, 38). This will clearly introduce individual errors in the determined accumulated O_2 deficit, since the range of slopes varied by as much as 16% in the present study. The importance of an error in the slope as well as other aspects concerning the imprecision is considered in more detail in the Appendix.

The accumulated O_2 demand is the product of the O_2 demand and the duration of the exercise. Besides the slope of the regression of submaximal O_2 uptake on the exercise intensity, the duration of the bout, as limited by the experience of fatigue, is the single most important source of methodological error. It is important that the exercise is an all-out effort, since in this paper we want to compare bouts of different durations where the anaerobic energy sources are used as extensively as possible.

Fatigue is a subjective experience that is influenced by motivation and is therefore difficult to assess objectively. Hence it may seem surprising that the values for the accumulated O_2 deficit were so highly reproducible for all durations. For exhausting bouts lasting 2 min the precision (SD) was 3 ml/kg, corresponding to a relative error of 4% of the maximal accumulated O_2 deficit. Therefore, with motivated subjects well accustomed to the procedures which were strictly standardized, the methodological error was quite small.

An imprecision in measuring the time to fatigue by ±10 s for a bout lasting 2–3 min corresponds to ±4 ml/kg in the accumulated O_2 deficit. This is an error of only 6%. The maximal accumulated oxygen deficit can therefore be measured accurately and with high precision, particularly for an all-out bout of 2–3 min duration (Table 2). The mean absolute difference in the accumulated O_2 deficit between exhausting bouts lasting 2 and 4 min was only 3 ml/kg. Since this difference is the additive effect of a possible systematic difference, the statistical imprecision, and a biological variation (for instance day-to-day variations), the day-to-day variation caused by changes in motivation as well as fitness must be small.

Metabolic components of accumulated O_2 deficit. Energy for ATP formation in excess of what can be accounted for by the measured O_2 uptake is covered by three different means: (1) changes in the O_2 stores in the body, which are comprised of O_2 bound to hemoglobin and myoglobin, O_2 dissolved in the body fluids, and O_2 present in the lungs; (2) breakdown of phosphocreatine and ATP in the exercising muscles; and (3) breakdown of glycogen to lactic acid, which partly distributes in the blood and the extracellular fluid.

Whereas 2 and 3 are true anaerobic energy sources, 1 is per definition aerobic but cannot be measured directly. The accumulated O_2 deficit will provide a poor estimate of the anaerobic capacity if stored O_2 accounts for a large fraction. Information on the relative contribution of stored O_2, phosphocreatine breakdown, and anaerobic glycolysis is available from the literature. During exhausting exercise muscle concentration of high-energy phosphates (phosphocreatine + ATP) has been reported.
to decrease about 18 mmol/kg wet wt muscle (8, 16, 31, 32). Increments in the muscle lactate concentrations of 22–30 mmol/kg wet wt muscle has been found by several investigators (15, 16, 19, 31, 33). Assuming that the exercising muscle mass is 25% of the body weight, these figures indicate that 4.5 mmol high-energy phosphates/kg body wt is broken down, and 6.5 mmol lactate/kg body wt is confined to the exercising muscles immediately after exercise.

In addition, lactate is distributed outside the exercising muscles. The blood lactate concentration was 13.4 mmol/l immediately after the exhausting bouts lasting 2 min or more. Arterialized blood lactate concentration immediately after exercise is assumed to reflect the lactate concentration in a volume corresponding to 15% of the body weight, which suggests that the extracellular lactate concentration was 2.0 mmol/kg body wt. Thus the total lactate production is estimated to be roughly 8.5 mmol/kg body wt. When converted to volume of O₂, the breakdown of phosphocreatine and lactate formation contribute 16 and 44 ml/kg, respectively (Table 3).

The total O₂ stores in the muscles, the blood and other body fluids, and the lungs of an average 77-kg subject were estimated to decrease 450–500 ml or 6 ml/kg body wt (the main component is the reduced O₂ saturation of the mixed venous blood), indicating that body O₂ stores contribute little to the accumulated O₂ deficit; as much as 90% is due to true anaerobic ATP formation.

The experiments in hypoxia substantiate that stored O₂ contributes little to the accumulated O₂ deficit. The reduction in the maximal O₂ uptake of 22% in hypoxia is in close agreement with previous studies (5, 6, 30), and reduced O₂ saturation of the arterial blood is the main cause. The O₂ stores in other body compartments are also reduced compared with normoxia. However, the accumulated O₂ deficit was the same in hypoxia as in normoxia for exhausting bouts of the same duration, as also shown by Linnarsson et al. (23). Hence the reduced O₂ stores did not influence the accumulated O₂ deficit significantly, as would have been the case if the O₂ stores accounted for a large part of the accumulated O₂ deficit.

Calculations of the changes in the O₂ stores, lactate and phosphocreatine concentrations, and the rough estimates of their distribution volumes suggest the maximal accumulated O₂ deficit to be 66 ml/kg (Table 3). This is in fair agreement with our measured values of 72 ± 11 ml/kg (range: 52–90 ml/kg). On the other hand, determination of the O₂ debt after exhausting exercise, another method suggested for assessment of the anaerobic capacity, has given values nearly twice as large (10). Thus, in contrast to the accumulated O₂ deficit, the O₂ debt is unlikely to reflect anaerobic catabolism accurately for exhausting exercise.

About 75% of the accumulated O₂ deficit may be due to breakdown to high-energy phosphates in the exercising muscles and to formation of lactate, which does not leave the muscles during exercise. Thus the anaerobic capacity is presumably highly dependent on the mass of the exercising muscles. This is in contrast to the maximal O₂ uptake, which is primarily a function of the capacity of the circulatory system when a large muscle mass is engaged (1, 11).

**Dependence of accumulated O₂ deficit on duration.** It has been suggested that the amount of energy available from anaerobic sources is almost independent of the duration of the exercise for exhausting bouts lasting 15 s or more (10, 27) or 40 s or more (25). In contrast to this view we found that the accumulated oxygen deficit increased about three times when the duration of the exhausting exercise was increased from 15 s to 2 min. The main process that can account for the increase in the anaerobic ATP formation is the glycolysis. Phosphocreatine can be broken down very rapidly, and it is unlikely that the contribution to the accumulated O₂ deficit from breakdown of phosphocreatine will increase much with increased duration of the exercise (9, 31).

This argument raises the question of what limits performance and causes fatigue. The increase in the relative contribution of lactate formation as the exercise duration approaches 2 min suggests that the maximal rate of glycogen breakdown may be a limiting factor for the rate of anaerobic ATP formation. This is supported by the observation that, independent of the absolute magnitude of the anaerobic capacity, a leveling off of the accumulated O₂ deficit with the duration of the exercise period occurred at 2 min. Hence subjects with a large anaerobic capacity were able to produce ATP anaerobically at a much higher rate than subjects with a low anaerobic capacity (e.g., subjects RB and KTS in Fig. 2). In accordance with this it has been shown that sprint-trained subjects have a higher accumulated O₂ deficit and accumulate more lactate in the blood than endurance-trained subjects during 1 min of exhausting exercise (20).

The present data do not allow any conclusion about the role of lactate in the fatigue process. Even though the extracellular buffer capacity does not differ between sprint-trained and endurance-trained subjects (26), the intracellular buffer capacity may vary considerably (28, 34). Hence we do not know the relationship between the intracellular lactate concentration and pH in different subjects. However, it is interesting that fatigue is experienced during short exercise bouts, even though the blood and muscle lactate accumulations are far from maximal (Ref. 4 and Tabata et al., unpublished observations). Other factors than acidosis may therefore in-

### TABLE 3. Relative contribution of components of maximal accumulated O₂ deficit: calculated data

<table>
<thead>
<tr>
<th>Component of the Accumulated O₂ Deficit</th>
<th>Contribution to Maximal Accumulated O₂ Deficit</th>
<th>% of total</th>
<th>Ref. No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>O₂ stored in blood and muscle</td>
<td>6.0</td>
<td>9</td>
<td>1, 17, unpublished observations</td>
</tr>
<tr>
<td>High-energy phosphate stores in muscle</td>
<td>15.5</td>
<td>24</td>
<td>8, 16, 31, 32</td>
</tr>
<tr>
<td>La in muscle</td>
<td>33.6</td>
<td>51</td>
<td>15, 16, 18–20, 31, 33</td>
</tr>
<tr>
<td>La transferred to blood and ECF</td>
<td>10.4</td>
<td>16</td>
<td>Present study</td>
</tr>
<tr>
<td><strong>Sum</strong></td>
<td>65.5</td>
<td>100</td>
<td></td>
</tr>
</tbody>
</table>

La, lactate; ECF, extracellular fluid.
hibit strenuous exercise at this high intensity.

Conclusions. The accumulated O₂ deficit 1) reaches a maximum when the duration of the exhausting exercise exceeds 2 min; 2) varies independently of the maximal O₂ uptake, as expected for the anaerobic capacity; 3) is in fair agreement with an anaerobic capacity calculated from its metabolic components; 4) to an extent of 90% or more measures true anaerobic energy conversion to ATP; and 5) is heavily dependent on the exercising muscle mass. Hence it seems justified to conclude that the accumulated O₂ deficit provides a good estimate of the anaerobic capacity.

The maximal accumulated O₂ deficit can be determined with a precision of 3 ml/kg or 4%, using individual relationships between the intensity and the O₂ demand. To keep the methodological error as small as possible, the exercise should not last more than 3 min. The method of quantifying anaerobic energy release described in this study may be used for all kinds of exercise satisfying the following two criteria. First, there is a linear relationship between the submaximal exercise intensity and the steady-state O₂ uptake. Second, this linear relationship can be extrapolated to supramaximal intensities requiring large anaerobic release.

APPENDIX

Statistical Evaluation of the Method

Four methodological aspects need further analysis if the method is to be applied as a standard test. First, the visual screening of a graph of the data to disclose outliers and nonlinearities for the control procedure described above must be justified. Second, the most important sources of analytical error should be identified. Third, it can be shown that 10 measurements of the steady-state O₂ uptake at different submaximal intensities is the minimum number needed to get a reliable estimate of the O₂ demand. Fourth, even this is time consuming, and a more convenient way to estimate the O₂ demand may be found.

Visual screening vs. formal elimination criteria. "Wild measurements" of the steady-state O₂ uptake, as determined from plots of the O₂ uptake vs. exercise intensity, were excluded. In addition measurements at low intensities were excluded if they deviated from the linearity. These low-intensity measurements were discarded for the following reasons. First, when all measurements were included, the Y-intercept was far above the preexercise resting O₂ uptake. Second, a value estimated by extrapolation from measured values is more imprecise the more distant the extrapolated value is from the measured ones. Third, the largest deviations from linearity at low intensity were found for the subjects with the largest vertical body movements (as judged by visual inspection).

Because including the measurements at low treadmill speed reduces the calculated accumulated O₂ deficit by ~10%, we used the following iterative procedure to formally eliminate these measurements. Starting with all measurements of submaximal O₂ uptake 1) the linear regression of the steady-state O₂ uptake on treadmill speed was calculated, 2) the accumulated O₂ deficit was calculated, and 3) the measurement at the smallest intensity was eliminated. 4) Steps 1–3 were repeated until a convergence in the accumulated O₂ deficit was found. Thereafter the residuals were calculated, and the regression line was recalculated after measurements with residuals exceeding three times the scatter around the regression line were discarded (i.e., 3 ml·kg⁻¹·min⁻¹ or more).

![Diagram](image)

**FIG. 4.** A: steady state O₂ uptake vs. exercise intensity (treadmill speed). Measurements done below 68 m/min (open symbols) were not included in final calculation (solid regression line, boldface text), whereas all of measurements were used to calculate stippled regression line (lightface text). B: deviation in accumulated O₂ deficit from its asymptotic value vs. lowest exercise intensity used to establish relationship between intensity and O₂ demand by linear regression. Accumulated O₂ deficit was calculated as described in APPENDIX. Data are from subject IT.

The smallest accepted treadmill speed ranged from 55 to 80 m/min (at 6° inclination), corresponding to 35–40% of the maximal O₂ uptake (Fig. 4). The slopes determined by this formal procedure differed in average by 0.0001 ml·kg⁻¹·m⁻¹ from the slopes calculated by the control procedure, the mean Y-intercept differed by <0.1 ml·kg⁻¹·min⁻¹, and the mean scatter around the regression lines was 0.9 ml·kg⁻¹·min⁻¹ for both approaches. The mean accumulated O₂ deficit was 0.4 ml/kg (0.5%) larger than that obtained by the control procedure (P > 0.3). Thus the formal analysis fully confirms that the visual inspection of a plot of O₂ uptake vs. exercise intensity is a reliable way of evaluating the data obtained at submaximal intensities.

Analytical errors: regression lines. The errors in determining the accumulated O₂ deficit were assumed to be independent. Hence the total variance (Σ SE²) was taken as the sum of the variance of the following measured components and statistical parameters: 1) the O₂ uptake, 2) the treadmill speed and...
total variance, speed during exhausting experiment; 2, mean treadmill speed during submaximal lo-min bouts. Error for a 2-min bout means the influence of the methodological error in estimating O2 demand, S, and a are the means of the exercise intensities and the corresponding O2 uptakes used for establishing the relationship, b is the regression coefficient (slope), and A is the Y-intercept (estimated O2 uptake at 0 treadmill speed).

The error in estimating the O2 demand \( \hat{Y} \) was taken as

\[
\hat{\sigma} = \left( \hat{S}_\text{a}^2 + \hat{S}_\text{b}^2 + (X - \bar{X})^2 \cdot \hat{\sigma}_{\text{Y}} \right)^{1/2}
\]

where \( \hat{S}_\text{a} \) is the scatter around the regression line (the estimated SE of the residual variation not accounted for by the regression of \( Y \) on \( X \)), and \( \hat{S}_\text{a} \text{ and } \hat{S}_\text{b} \) are the estimated SE values of \( a \) and \( b \), respectively (3,36,37).

Together with the abovementioned errors the scatter around the regression line \( \hat{S}_{\text{a}} = 36\% \) of the methodological variance, the error in the term \( \hat{S}_\text{a} \) (2%), and a term related to the error of the slope of the regression line \( \hat{S}_\text{b} \) (55%) gives a total methodological error in determining the maximal accumulated O2 deficit of 3 ml/kg or 4%.

Since the imprecision in the accumulated O2 deficit is mainly due to the statistical error in estimating the O2 demand, it is important to reduce this error as much as possible. The scatter around the regression line \( \hat{S}_{\text{a}} \) reflects variations in the \( O_2 \) uptake not accounted for by the exercise intensity. In particular, random errors of variable size were found in 5-10% of all measurements at submaximal intensities. These outliers can easily be excluded by visual inspection of a plot of the measurements.

Two of the components in the statistical imprecision of the estimated O2 demand, \( \hat{S}_\text{a} \) and \( \hat{S}_\text{b} \), are proportional to the scatter around the regression line (see Refs. 3, 36, and 37 for details). Reductions in the scatter around the regression line will therefore also reduce these terms. Provided a linear relationship exists, additional measurements at the lowest and highest intensities will reduce the error of the slope considerably. Since the linearity may not be present for low intensities, measurements of the \( O_2 \) uptake at intermediate intensities should also be included as a check of linearity.

By these means each of the terms \( \hat{S}_\text{a} \) and \( \hat{S}_\text{b} \) \( (X - \bar{X}) \) may be reduced to \( \sim 0.7 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \). This gives a total statistical imprecision of \( \sim 2 \text{ ml/kg} \) in determining the accumulated O2 deficit for a bout lasting 2 min.

Required minimum number of measurements of steady-state \( O_2 \) uptake. To find this minimum number, we used the following iterative procedure. Starting with the first two measurements of the submaximal \( O_2 \) uptake done 1) the linear regression of the steady-state \( O_2 \) uptake on treadmill speed was calculated, 2) the accumulated O2 deficit was calculated, and 3) the next measurement of submaximal \( O_2 \) uptake done was included. 4) Steps 1-3 were repeated until a convergence in the accumulated oxygen deficit was found. A minimum number of 10-15 meas-

<table>
<thead>
<tr>
<th>Component</th>
<th>Methodological Error</th>
<th>Error for 2-min Bout</th>
<th>Fraction of Total Variance, %</th>
<th>Relationship of Error to Variation</th>
</tr>
</thead>
<tbody>
<tr>
<td>( O_2 ) uptake</td>
<td>0.35 ml-kg(^{-1})-min(^{-1})</td>
<td>0.43 0.18</td>
<td>2.0 Independent</td>
<td>Increases</td>
</tr>
<tr>
<td>External work</td>
<td>0.005 min</td>
<td>0.46 0.20</td>
<td>2.2 Proportional</td>
<td>Independent</td>
</tr>
<tr>
<td>Speed</td>
<td>0.7 m/min</td>
<td>0.42 0.18</td>
<td>2.0 Proportional</td>
<td>Proportional</td>
</tr>
<tr>
<td>Inclination</td>
<td>0.02(^{*})</td>
<td>0.32 0.10</td>
<td>1.1 Proportional</td>
<td>Proportional</td>
</tr>
<tr>
<td>Regression line</td>
<td>( \hat{S}_\text{a} )</td>
<td>0.9 ml-kg(^{-1})-min(^{-1})</td>
<td>1.8 Independent</td>
<td>Proportional</td>
</tr>
<tr>
<td>( \hat{S}_\text{b} )</td>
<td>0.2 ml-kg(^{-1})-min(^{-1})</td>
<td>0.4 0.16</td>
<td>1.8 Independent</td>
<td>Proportional</td>
</tr>
<tr>
<td>( \hat{S}_\text{Y} )</td>
<td>0.0074 ml-kg(^{-1})-min(^{-1})</td>
<td>2.2 4.93</td>
<td>54.9 Increases</td>
<td></td>
</tr>
<tr>
<td>( X-\bar{X} )</td>
<td>150 m/min</td>
<td>3.0(^{*}) 8.99</td>
<td>100.0 Proportional</td>
<td></td>
</tr>
</tbody>
</table>

Values indicate means of 11 subjects. \( \hat{S}_\text{a} \), \( \hat{S}_\text{b} \), scatter around regression line; \( \hat{S}_\text{a} \), \( \hat{S}_\text{b} \) estimated SE values of regression parameters. \( X \), treadmill speed during exhausting experiment; \( \bar{X} \), mean treadmill speed during submaximal 10-min bouts. Error for a 2-min bout means the influence of the methodological error in each component on the statistical error in the accumulated oxygen deficit for a bout lasting 2 min. * Square root of total variance.

**TABLE 4. Methodological errors and their influence on precision in determining accumulated \( O_2 \) deficit for an exhausting bout of 2 min duration for the control procedure**

**FIG. 5. Abscissa: number of measurements used to establish the relationship between exercise intensity and steady-state \( O_2 \) uptake; ordinate: deviations in accumulated \( O_2 \) deficit from asymptotic value calculated by each relationship. Data are from subjects ACM, MKS, PB, RB, and RN.**
measurements were required, because at that point the estimates of the accumulated O\textsubscript{2} deficit converged (Fig. 5).

**Evaluation of Simpler Procedures**

Since as many as 10 measurements or more were needed for establishing a reliable relationship between the exercise intensity and the oxygen uptake, three alternative simpler procedures were evaluated.

**Procedure 1.** All the 240 accepted measurements of the steady-state O\textsubscript{2} uptake at submaximal exercise intensities were used to calculate one common regression line between the running speed and the O\textsubscript{2} demand for all of the subjects. This procedure expressed the O\textsubscript{2} demand (Y) as \( Y = 7.0 + 0.277 \times \text{speed} \), with a scatter around the regression line of 1.7 ml \cdot kg\(^{-1}\) \cdot min\(^{-1}\), where the O\textsubscript{2} demand is expressed in milliliters per kilogram per minute and the speed in meters per minute at 6° inclination.

**Procedure 2.** The means of the individual slopes and Y-intercepts of the control procedure were calculated, and a line with these two parameters expressed the O\textsubscript{2} demand as \( Y = 5.1 + 0.298 \times \text{speed} \) (units as in procedure 1).

These two procedures, used by several previous investigators (20, 25, 35), take for granted that interindividual variations in the running economy are negligible. The advantage is that no pretests are necessary before the accumulated O\textsubscript{2} deficit is determined.

**Procedure 3.** The mean (±SE) Y-intercept of the control procedure was 5.1 ± 0.2 ml \cdot kg\(^{-1}\) \cdot min\(^{-1}\). The small variation means that there were insignificant differences in the Y-intercept between subjects, and procedure 3 takes advantage of a common Y-intercept. The O\textsubscript{2} demand was assumed to increase linearly for each subject from this common Y-intercept through the O\textsubscript{2} uptake measured at 85–100% of the maximal O\textsubscript{2} uptake. This procedure gave individual relationships with one common reliable Y-intercept.

The accumulated O\textsubscript{2} deficit determined by procedures 1 and 2 produced large individual deviations compared with the control. The mean absolute differences was 9 and 8 ml/kg (Table 5). These two procedures do not separate differences in running economy from differences in the anaerobic capacity, and since the running economy differed by as much as 16% between subjects, the error introduced was large. In addition, procedure 1 was biased and cannot be recommended, since determination of the accumulation O\textsubscript{2} deficit by this procedure gave significantly smaller values compared with the control procedure (\( P < 0.05 \)). However, procedure 2 can be acceptable for determining the accumulated O\textsubscript{2} deficit on groups of subjects.

**Procedure 3**, which takes advantage of a common Y-intercept, did not give significantly different results from the control procedure (Table 5). The mean absolute difference from the control was 2 ml/kg, and the individual differences never exceeded 3 ml/kg, which was within the statistical precision of the control procedure. As a check of robustness the calculations were repeated for each of the four next-highest steady-state O\textsubscript{2} uptakes (70–95% of the maximal O\textsubscript{2} uptake) for each subject. Out of these 55 determinations four differed between 5 and 11 ml/kg from the control. This shows that "wild values" may sometimes arise by procedure 3. The procedure should therefore be based on two rather than only one measurement to increase its robustness.

**Recommendations**

**Procedure 3** is the best of the three simplified procedures and is a good alternative to the comprehensive control procedure, which requires a minimum of 10 measurements of the O\textsubscript{2} uptake at different submaximal intensities. The properties of procedure 3 can be summarized as follows.

1) The relationship between the exercise intensity and the O\textsubscript{2} demand must be established individually. This is important, since there was a considerable variation in the individual slopes calculated by the control procedure. However, a common reliable Y-intercept of 5 ml \cdot kg\(^{-1}\) \cdot min\(^{-1}\) can be used.

2) Individual relationships can be established by measuring a submaximal steady-state O\textsubscript{2} uptake near the maximal O\textsubscript{2} uptake. The extrapolated estimate of the O\textsubscript{2} demand should be as close to the measured one as possible. Selecting the bout at the largest submaximal intensity improved the results of **procedure 3** significantly.

3) Since the steady-state O\textsubscript{2} uptake may be erroneously determined in 5–10% of all cases, basing the relationship between the oxygen demand and the intensity on only one measurement may occasionally give large errors. To increase the robustness a second measurement of the steady-state O\textsubscript{2} uptake at ~90% of the maximal O\textsubscript{2} uptake should be done. If the two slopes calculated from each measurement differ by no more than 3%, large errors are unlikely. If larger differences in the slopes are found, a third measurement can be done to exclude the outlier.

**Exhausting bout.** Since the accumulated O\textsubscript{2} deficit is defined as the difference between the accumulated O\textsubscript{2} demand and the accumulated O\textsubscript{2} uptake, an error in the measured O\textsubscript{2} uptake will also affect the calculation of the accumulated O\textsubscript{2} deficit. When the exhausting bouts were preceded by sufficient warming up, the O\textsubscript{2} uptake was always found to be more than 90% of the maximal O\textsubscript{2} uptake apart for the first 30–60 s of the bout. Comparisons of the O\textsubscript{2} uptake during supramaximal exercise to the maximal O\textsubscript{2} uptake provide another simple control.

We appreciate the skilful technical assistance of Kari Heldal, Anne Kari Henanger, Ada Ingvaldsen, Bjørn I. Selberg, and Jord T. Stuenæs. We acknowledge the advice and the support from all colleagues at the Department of Physiology at the National Institute of Occupational Health. We thank the subjects who participated in the experiments. The idea of defining the accumulated O\textsubscript{2} deficit as the area between the curves of the O\textsubscript{2} demand and the O\textsubscript{2} uptake was first presented by Lars Hermansen (10). It was his idea to use the maximal accumulated O\textsubscript{2} deficit to determine the anaerobic capacity and to control the method by repeating the experiments in hypoxia. He was the leader of this project until his death. We want to express our gratitude for his always encouraging leadership.

R. Bahr and A.-C. Mohn were supported by grants from the Norwegian Research Council for Science and the Humanities (NAVF). J. I. Medbø received grants from the Norwegian Ministry for Local and Occupational Affairs and from NAVF.

<table>
<thead>
<tr>
<th>Subj</th>
<th>P(_1)</th>
<th>P(_2)</th>
<th>P(_3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACM</td>
<td>-6.0</td>
<td>3.1</td>
<td>2.0</td>
</tr>
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<td>2.0</td>
<td>2.0</td>
</tr>
<tr>
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<td>-15.1</td>
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</tr>
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</tr>
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<td>3.6</td>
</tr>
<tr>
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</tr>
<tr>
<td>RN</td>
<td>6.2</td>
<td>15.4</td>
<td>3.1</td>
</tr>
</tbody>
</table>

| Means ± SE | 8.7 ±1.8 | 7.8 ±1.5 | 2.0 ±0.3 |

| Means ± SE (absolute deviations) | 5.5 ±2.6 | 1.5 ±3.2 | 0.8 ±0.7 |

Values are expressed in ml/kg. P\(_1\), P\(_2\), procedures 1, 2, and 3.
REFERENCES


