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Lactate accumulation in response to supramaximal exercise in rowers

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The aim of this study was to test (a) three methods to estimate the quantity of lactate accumulated (Q_{LaA}) in response to supramaximal exercise and (b) correlations between Q_{LaA} and the nonoxidative energy supply assessed by the accumulated oxygen deficit (AOD). Nine rowers performed a 3-min all-out test on a rowing ergometer to estimate AOD and lactate accumulation in response to exercise. Peak blood lactate concentration [$(La)_{peak}$] during recovery was assessed, allowing $Q_{LaA(m1)}$ to be estimated by the method of Margaria et al. Application of a bicompartmental model of lactate distribution space to the blood lactate recovery curves allowed estimation of (a) the net amount of lactate released during recovery from the active muscles ($NALR_{max}$), and (b) Q_{LaA} according to two methods ($Q_{LaA(m2)}$ and $Q_{LaA(m3)}$). $(La)_{peak}$ did not correlate with AOD. $Q_{LaA(m1)}$, $Q_{LaA(m2)}$ and $Q_{LaA(m3)}$ correlated with AOD ($r = 0.70$, $r = 0.85$ and $r = 0.92$, respectively). These results confirm that $(La)_{peak}$ does not provide reliable information on nonoxidative energy supply during supramaximal exercise. The correlations between AOD and $Q_{LaA(m2)}$ and $Q_{LaA(m3)}$ support the concept of studying blood lactate recovery curves to estimate lactate accumulation and thus the contribution of nonoxidative pathway to energy supply during supramaximal exercise.

During high-intensity exercise, muscle activity depends on a high rate of adenosine triphosphate (ATP) resynthesis via oxidative and nonoxidative pathways. The aerobic contribution to energy supply during exercise can easily be quantified from oxygen (O_2) consumption. The nonoxidative energy contribution is much more difficult to determine.

Previously, Margaria et al. (1963, 1971) had proposed a method to estimate the quantity of lactate accumulated (Q_{LaA}) during supramaximal exercise, taking account of $(La)_{peak}$ during recovery and the volume of lactate distribution. Later, Medbø et al. (1988) proposed a method to assess the nonoxidative contribution by measuring accumulated oxygen deficit (AOD, in L O_2 Eq.). Although this method has limitations (Bangsbo, 1998), it represents a means of assessing nonoxidative energy production during high-intensity exercise (Medbø, 1993). Bangsbo et al. (1990) demonstrated that, during exercise with a small muscle group, AOD and energy yield (based on metabolic alterations in the active muscle) give similar values for nonoxidative energy release. Currently, AOD is the sole noninvasive integrative method for estimating the nonoxidative energy contribution during exercise (Saltin, 1990; Gastin et al., 1995). The main metabolic pathway contributing to AOD during 2–3 min supramaximal exercise up to exhaustion is glycolysis. Oxygen stores (bound to myoglobin and hemoglobin) and ATP and phosphocreatine (PC) breakdown also contribute, but to a minor extent (Bangsbo et al., 1990; Bangsbo, 1998). It can therefore be hypothesized that muscle lactate production during exercise is the main determining factor in AOD.

Several authors proposed post-exercise blood lactate concentration [$(La)_b$] as an index of nonoxidative glycolytic energy contribution during exercise. However, most studies, including those performed on rowing, failed to find significant correlations between $(La)_b$ and AOD (Medbø et al., 1988; Scott et al., 1991; Medbø, 1993; Pripstein et al., 1999; Bishop et al., 2002). Those results are not surprising because a single end-of-exercise or post-exercise blood lactate concentration reflects not only muscle lactate production during exercise but also lactate exchange between muscle cells and other tissues (including the blood) and its removal from those tissues during exercise and early recovery. Indeed, it is important to keep in mind that (a) only a part of the lactate produced is released from active and previously active muscles during exercise and recovery (the remainder being accumulated

and/or consumed in situ), and (b) during exercise and recovery, the blood distributes lactate to the same or other tissues (e.g., liver, heart, brain, and the same or other active and inactive muscles), which consume it by oxidation or gluconeogenesis (Ahlborg et al., 1975; Åstrand et al., 1986; Brooks, 1986; Freund et al., 1986; Stanley et al., 1986; van Hall, 2010).

Several groups have used a bicompartmental model of lactate distribution space which provides information on the mechanisms of lactate exchange and removal during recovery (Freund & Zouloumian, 1981a, b; Zouloumian & Freund, 1981a, b; Freund et al., 1984, 1986; Bret et al., 2003). The present study proposes further applications of this model to estimate the Q_{LaA} in response to supramaximal exercise.

The aim of the present study was to compare the method of Margaria et al. with two new methods of estimating the amount of lactate accumulated (Q_{LaA}) in response to supramaximal exercises. Correlations between AOD and the three methods of estimating Q_{LaA} were investigated.

Materials and methods

Subjects

Nine highly trained lightweight rowers volunteered to participate in the study. Their mean [±standard deviation (SD)] age, height, and weight were 22.4 ± 2.8 years, 1.83 ± 0.03 m, and 72.1 ± 3.0 kg, respectively. The experiment was conducted in accordance with the Declaration of Helsinki regarding the use of human subjects and was approved by the Human Research Ethics Committee of the University Hospital Centre of Saint-Etienne, France. Before giving written consent, subjects were informed of the objectives and of all risks, possible discomfort, and potential benefits of the experiment.

Experimental design and equipment

Trials involved two exercise sessions, carried out at least 3 days apart. Subjects were instructed not to undertake any strenuous activity during the 24-h preceding each exercise session. All tests were performed on a wind-resistance-braked rowing ergometer (Model D; Concept2, Morrisville, Vermont, USA). Rowers were fully familiar with the ergometer that provided continuous feedback on power output and stroke rate.

Session 1: Incremental exercise up to exhaustion

Graded exercise started at 150 W, with power output incremented by 50 W steps. Each step consisted of 3 min rowing and 0.5-min rest. To determine blood lactate concentration [$(La)_b$ in mmol/L], blood samples were taken from the earlobe at rest and during the rest interval in each step. To measure oxygen uptake ($\dot{V}O_2$, in L/min), expired gases were sampled during the last 30 s of each step. Heart rate (HR, in beats/min) was measured continuously. Maximal oxygen uptake ($\dot{V}O_{2max}$, in L/min and mL/kg/min) and the mechanical power output corresponding to $\dot{V}O_{2max}$ (Pa_{max}, in W) were determined. As well, $\dot{V}O_2$ corresponding to 4 mmol/L of $(La)_b$ was determined and expressed in absolute values ($\dot{V}O_2La4$, in L/min) and relative to $\dot{V}O_{2max}$ ($\dot{V}O_2La4\%$). This session was also carried out to estimate the linear relationship between $\dot{V}O_2$ and power output.

Session 2: 3-min all-out exercise

Hyperemic cold cream (Dolpyc®, Pfizer, New York, USA) was applied to the earlobe, and subjects performed a 10-min warm-up at 130 beats/min (~50% of Pa_{max}). After a 2-min rest, the subjects performed a 3-min all-out test (no constant work rate), followed by 90 min passive recovery. Subjects remained seated during the rest and recovery periods. Throughout the 3-min all-out exercise, work rate and oxygen uptake were measured continuously. Gas samples were collected every 30 s in 100-L Douglas bags. Blood samples were collected from the earlobe at rest [$(La)_{rest}$, in mmol/L], at end of warm-up [$(La)_{warm\text{-}up}$, in mmol/L] and of exercise [(La)(0), in mmol/L], and thereafter at 0.5-, 1-, 1.5-, 2-, 2.5-, 3-, 3.5-, 4-, 4.5-, 5-, 6-, 8-, 10-, 12-, 15-, 20-, 25-, 30-, 40-, 50-, 60-, 70-, 80-, and 90-min recovery. Peak $(La)_b$ values (in mmol/L) and the corresponding time (in min) were obtained experimentally [$(La)_{peakMeas}$ and $t(La)_{peakMeas}$, respectively] and by modeling [$(La)_{peakMod}$ and $t(La)_{peakMod}$, respectively: *vide infra*]. Mean power output was expressed in absolute values (P_{AOD}, in W) and relative to Pa_{max} ($\%P_{AOD}$). This session provided individual AOD (in L O_2 Eq.) and blood lactate recovery curves.

Measurements and calculations

Ventilatory and metabolic measurements

After passing through a three-way low-resistance mouthpiece (Hans Rudolph 2700, Hans Rudolph Inc., Kansas City, Missouri, USA) and a low dead-space mixing chamber, the expired gases were collected in a balanced Tissot spirometer for flow measurement (session 1). For session 2, expired gases were collected in Douglas bags. The O_2 and CO_2 fractions were measured in ambient air and in the mixing chamber (session 1) or in Douglas bags (session 2), by D-Fend Datex (Helsinki, Finland) and S3A/I Ametek (Pittsburgh, Pennsylvania, USA) analyzers, respectively. Analyzers were pre-calibrated on precision-analyzed gas mixtures. The expired air volumes and O_2 and CO_2 fractions were recorded to calculate $\dot{V}O_2$. $\dot{V}O_{2max}$ was reached when $\dot{V}O_2$ displayed a plateau as work rate continued to increase. To establish that $\dot{V}O_2$ had been reached during the last step when a plateau was not observed, the following criteria were used: respiratory exchange ratio greater than 1.1, end-of-exercise lactate concentration higher than 9 mmol/L, and cardiac frequency approximating the theoretical maximum (220 – age ± 10 beats/min).

HR

HR was recorded by electrocardiogram (Cardimax FX-121 Electrocardiograph, Fukuda Denshi, Tokyo, Japan).

Blood lactate concentration

Arterialized capillary blood (20 µL) was sampled by micropuncture at the earlobe, then diluted in a hemolyzing solution and stored at 4°C until analysis. Lactate concentration was determined enzymatically in whole blood, using a YSI 2300 lactate analyzer (YSI Inc., Yellow Springs, Ohio, USA).

Work rate and oxygen uptake

$\dot{V}O_2La4$ was determined by linear interpolation between the two closest measured values. Pa_{max} was determined by linear interpolation from the $\dot{V}O_2$ vs work-rate curve. During the 3-min exhaustive exercise, power output was recorded every 5 s.

AOD

AOD was obtained by subtracting O_2 consumption from O_2 demand. In accordance with Green and Dawson (1996), oxygen

demand was calculated by linear regression to extrapolate the $\dot{V}O_2$-power-output relationship equation obtained in session 1. Because the present study concerned all-out exercise, O_2 demand was calculated from instantaneous power output (recorded stroke by stroke) rather than mean power output sustained during exercise (i.e., P_{AOD}) as initially proposed by Medbø et al. (1988).

Bicompartmental model

Individual blood lactate recovery curves were fitted to the biexponential time function:

$$La(t) = (La)(0) + A_1\left(1 - e^{-y1t}\right) + A_2\left(1 - e^{-y2t}\right) \quad [1]$$

where: (La)(0) and La(t) (mmol/L) are lactate concentrations in arterialized capillary blood measured at recovery onset and at a given recovery time, respectively; concentration parameters A_1 and A_2 (mmol/L) are the amplitudes of the exponential functions; and γ_1 and γ_2 (per minute) are the velocity constants that describe lactate exchange and removal capacity, respectively (Freund & Zouloumian, 1981a, b; Freund et al., 1986). The blood lactate recovery curves were fitted to Eqn. [1] by iterative nonlinear regression on KaleidaGraph 4.0 software (Synergy Software, Reading, Pennsylvania, USA) to determine the values of A_1, A_2, γ_1, and γ_2, (La)(0) being an experimental measurement.

The bicompartmental model of lactate distribution space during recovery (Zouloumian & Freund, 1981b) predicted the evolution of the net lactate release rate (NLRR in mmol/min) during the recovery period according to the following equation:

$$NLRR(t) = (\gamma_1 - d_2)\cdot V_s \cdot A_1 \cdot e^{-\gamma_1 \cdot t} + (\gamma_2 - d_2)\cdot V_s \cdot A_2 \cdot e^{-\gamma_2 \cdot t} + \mu \quad [2]$$

where μ is the net muscular release rate of lactate into the blood at complete recovery: i.e., 0.12 mmol/min, in accordance with Freund and Zouloumian (1981b), Zouloumian and Freund (1981b) and more recent studies (Bangsbo et al., 1991; Bergman et al., 1999). In line with previous studies, d_2 was set at γ_2–0.005, to approximate NLRR (Freund & Zouloumian, 1981b; Zouloumian & Freund, 1981a; Bret et al., 2003; Messonnier et al., 2006). V_S (in L) is the remaining lactate space, which represents the difference between the total lactate distribution space (V_{TLS}, 600 mL/kg of body mass, in L) and the lactate distribution volume of muscles previously involved in exercise (V_M in L) (Di Prampero, 1981; Freund & Zouloumian, 1981b; Zouloumian & Freund, 1981a). The total skeletal muscle mass (MM in kg) of the rowers was estimated according to the following equation proposed and validated by Martin et al. (1990):

$$MM = STAT \cdot (0.0553 \cdot CTG^2 + 0.0987 \cdot FG^2 + 0.0331 \cdot CCG^2) - 2445 \quad [3]$$

where STAT is stature (in cm), CTG is thigh circumference corrected for front thigh skinfold thickness (in cm), FG is the uncorrected forearm circumference (in cm), and CCG is calf circumference corrected for medial calf skinfold thickness (in cm). It has been previously estimated that 70% of MM is involved in rowing (Steinacker, 1993). Besides, muscle lactate diffusion volume corresponds to the volume of water in the considered muscles. According to previous studies, mean muscle water content represents 78% of MM after exhaustive exercise (Sahlin et al., 1978; Bangsbo et al., 1990). Therefore, V_M can be approximated by the following equation:

$$V_M = 0.78 \cdot 0.70 \cdot MM \quad [4]$$

The integral of Eqn. [2] estimates the NALR to the blood (NALR, in mmol) from the previously active muscle during recovery. The maximal value of NALR ($NALR_{max}$, in mmol) was then recorded as previously (for further details of the model and its application, see Freund & Zouloumian, 1981a, b; Zouloumian & Freund, 1981a, b; Freund et al., 1984, 1986; Bret et al., 2003; Messonnier et al., 2006) and taken into account.

Estimated Q_{LaA} at the end of exercise

Total lactate production during exercise equals Q_{LaA} at the end of exercise plus the quantity of lactate removed from the body during exercise. The present study exclusively focused on Q_{LaA}. This choice was made for the following reasons: estimation of lactate removal during exercise is highly speculative, because the time courses during exercise of (a) muscle and blood lactate concentrations, and (b) lactate removal capacity in the active muscle and the remainder of the lactate space, are all impossible to assess (even using invasive methods). Moreover, lactate removal is in any case expected to be small during short high-intensity exercise, because oxidation does not mainly concern lactate during high-intensity exercise (Bangsbo et al., 1990; Bergman et al., 2000). Finally and most importantly, the main fate of lactate during exercise being oxidation (Brooks, 1986; Bergman et al., 2000), its disappearance from the active muscles and the remainder of the lactate space during exercise must be attributed to oxidative metabolism and should not be taken into account in assessing nonoxidative glycolytic energy supply. For all these reasons, the present study focused on lactate accumulation at end of exercise, rather than on total lactate production.

Method 1

Peak blood lactate concentration obtained during recovery [$(La)_{peak}$] was used previously by Margaria and coworkers to estimate lactate accumulation during supramaximal exercise (for further details, see Margaria et al., 1963, 1971; Di Prampero & Ferreti, 1999). Assuming that the water fraction was 0.6 for the whole body and 0.8 for the blood, the Q_{LaA} at the end of exercise ($Q_{LaA(m1)}$, in mmol) can be written as follows:

$$Q_{LaA(m1)} = (La)_{peakMeas} \cdot 0.6/0.8 \cdot \text{body mass} \quad [5]$$

The following two methods are based on applications of the bicompartmental model of lactate distribution space during recovery proposed by Freund and coworkers (Freund & Zouloumian, 1981a, b; Zouloumian & Freund, 1981a, b; Freund et al., 1984, 1986).

Method 2

$(La)_{peak}$ occurs almost concomitantly with equilibrium between muscle and blood lactate concentrations during recovery (Freund & Zouloumian, 1981b; Freund et al., 1984). Consequently, the Q_{LaA} (in mmol) in the total lactate distribution space (V_{TLS}) at $(La)_{peak}$ can be estimated according to the following equation

$$Q_{LaA} \text{ at } (La)_{peak} = (La)_{peakMeas} \cdot V_{TLS} \quad [6]$$

However, this estimated quantity is lower than that reached at the end of exercise, because the peak lactate concentration is reached after several minutes of recovery, during which lactate removal is elevated. This Quantity of Lactate Removed (Q_{LaR}, in mmol) from end of exercise [i.e., (La)(0)] to $(La)_{peak}$ can be estimated as follows

$$Q_{LaR} = \left\{\left[(La)_{peakMeas} + (La)(0)\right]/2\right\} \cdot \gamma_2 \cdot t(La)_{peakMeas} \cdot V_{TLS} \quad [7]$$

where $t(La)_{peakMeas}$ is the time to reach the maximal lactate concentration during recovery. Therefore, a second method of estimat-

ing the Q_{LaA} at the end of exercise ($Q_{LaA(m2)}$, in mmol) can be written as follows:

$$Q_{LaA(m2)} = Q_{LaA} \text{ at } (La)_{peak} + Q_{LaR} \quad [8]$$

Method 3

In the bicompartmental model (Freund et al., 1986), total lactate distribution space (V_{TLS}) is composed of V_M and V_S. consequently, the Q_{LaA} at end of exercise ($Q_{LaA(m3)}$ in mmol) can also be estimated as follows:

$$Q_{LaA(m3)} = Q_M + Q_S \quad [9]$$

where Q_M and Q_S (in mmol) are the quantities of lactate accumulated in V_M, and V_S, respectively.

The literature reports that only a minor part of the lactate produced and accumulated in the active muscles during exercise is released into the bloodstream during subsequent recovery. Following supramaximal cycling exercise, this part of lactate release averaged 10% in one study (Hermansen & Vaage, 1977), and ranged between 17 and 29% in another (Freund et al., 1984). The difference between the two findings could be explained by different exercise modes (intermittent vs continuous, respectively). The present study adopted a mean value of 30% (see Discussion) to estimate the Q_{LaA} in active muscles during exercise and released into the bloodstream during recovery. In other words, it could be considered that $NALR_{max}$ represented 30% of the total amount of lactate accumulated in muscle at end of exercise. Thus, Q_M can be estimated as follows:

$$Q_M = (NALR_{max} / 0.30) \quad [10]$$

Furthermore, Q_S can be expressed as follows

$$Q_S = \left[(La)(0) - (La)_{warm\text{-}up}\right] V_S \quad [11]$$

In conclusion, a third method of estimating $Q_{LaA(m3)}$ can be rewritten as

$$Q_{LaA(m3)} = (NALR_{max} / 0.30) + [(La)(0) - (La)_{warm\text{-}up}] \cdot V_S \quad [12]$$

Statistical analysis

Analyses were performed using JMP V7.0.1 (SAS Institute, Cary, North Carolina, USA). Data are expressed as mean ± SD. Linear regression models were fitted by the least squares method. The Wilcoxon test was used for data-group comparison. The statistical significance threshold was set at $P < 0.05$.

Results

Mean $\dot{V}O_{2max}$, Pa_{max} and $\dot{V}O_2La4\%$ values were 5.0 ± 0.3 L/min (68.7 ± 3.8 mL/kg/min), 366.5 ± 29.5 W and 88.7 ± 2.1% of $\dot{V}O_{2max}$, respectively. The relationship between $\dot{V}O_2$ and power output was linear; the mean correlation coefficient was 0.989 ± 0.009 (range: 0.997–1.0). Mean MM was 45.0 ± 2.6 kg, and represented 61.7 ± 2.6% of body mass. Mean V_{TLS}, V_M, and V_S values were 43.7 ± 1.3, 24.9 ± 1.4, and 18.9 ± 1.1 L, respectively.

AOD

Mean P_{AOD} was 435.8 ± 27.1 W (119.3 ± 6.7% of Pa_{max}); mean AOD, 4.1 ± 0.4 L O_2 Eq. (56.0 ± 6.6 mL O_2 Eq/kg); and mean $(La)_{rest}$ and $(La)_{warm\text{-}up}$, 1.21 ± 0.28 and 1.16 ± 0.17 mmol/L, respectively. Mean (La)(0) is displayed in Table 1. Mean $(La)_{peakMeas}$ and $t(La)_{peakMeas}$ were 18.34 ± 2.09 mmol/L and 8.1 ± 2.3 min, respectively.

Recovery blood lactate curves

The mean blood lactate recovery curve is presented in Fig. 1. Eqn. [1] accounted for more than 98% of the variance in the experimental blood lactate recovery curves ($0.994 < r < 0.999$). The mean values of the blood lactate curve parameters A_1, A_2, γ_1, and γ_2 are reported in Table 1.

Mean $(La)_{peakMod}$ and $t(La)_{peakMod}$ did not differ significantly from experimental values (18.32 ± 2.17 mmol/L and 7.9 ± 1.9 min, respectively). The mean time-courses of NLRR and NALR during recovery are shown

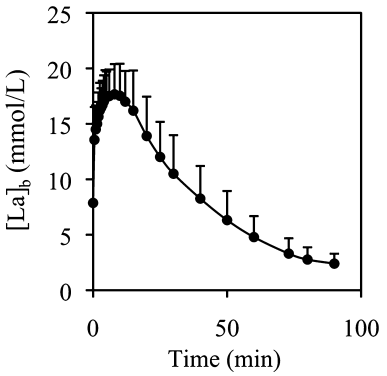

Fig. 1. Mean (±standard deviation) arterialized veinous blood lactate concentration [$(La)_b$] recovery curves after 3-min all-out exercise.

Table 1. Mean values of the blood lactate kinetics parameters obtained during the recovery period of the a 3-min all-out exercise

	(La)(0)	A_1	γ_1	A_2	γ_2	$NALR_{max}$
Mean ± SD	13.37 ± 2.63	18.29 ± 8.41	0.173 ± 0.067	–31.18 ± 10.21	0.0337 ± 0.0074	210.6 ± 55.4

(La)(0) (mmol/L) is the blood lactate concentration at end of exercise; A_1 and A_2 (mmol/L) are the amplitudes of the exponential functions; γ_1 and γ_2 (per min) are the velocity constants; $NALR_{max}$ (mmol) is the maximal value of the NALR from the muscle to the blood during the recovery period.
NALR, net amount of lactate released; SD, standard deviation.

in Fig. 2(a) and (b), respectively. The mean values of Q_{LaA} at $(La)_{peak}$ and Q_{LaR} were 802 ± 89 and 191 ± 73 mmol, respectively. Mean $NALR_{max}$ value is also reported in Table 1. The estimated values of Q_M and Q_S were 842 ± 221 and 229 ± 46 mmol, respectively. The mean estimated values of $Q_{LaA(m1)}$, $Q_{LaA(m2)}$ and $Q_{LaA(m3)}$ were not significantly different (1003 ± 112, 993 ± 149 and 929 ± 213 mmol, respectively).

Correlations between variables

AOD did not correlate with (La)(0) [$r = 0.39$, not significant (*NS*)], $(La)_{peakMeas}$ ($r = 0.61$, *NS*) or $(La)_{peakMod}$ ($r = 0.60$, *NS*). Q_S did not correlate with AOD ($r = 0.46$, *NS*), $Q_{LaA(m3)}$ ($r = 0.45$, *NS*) or Q_M ($r = 0.45$, *NS*). AOD correlated with $Q_{LaA(m1)}$ (Fig. 3(a)), $Q_{LaA(m2)}$ (Fig. 3(b)), $Q_{LaA(m3)}$ (Fig. 3(c)), Q_M ($r = 0.88$, $P < 0.002$), and $NALR_{max}$ ($r = 0.88$, $P < 0.0015$). $Q_{LaA(m1)}$ correlated with $Q_{LaA(m2)}$ ($r = 0.94$, $P < 0.0002$) and $Q_{LaA(m3)}$ ($r = 0.79$, $P < 0.01$). $Q_{LaA(m2)}$ correlated with $Q_{LaA(m3)}$ ($r = 0.90$, $P < 0.001$). Q_M or $NALR_{max}$ correlated with $Q_{LaA(m3)}$ ($r = 0.98$, $P < 0.0001$).

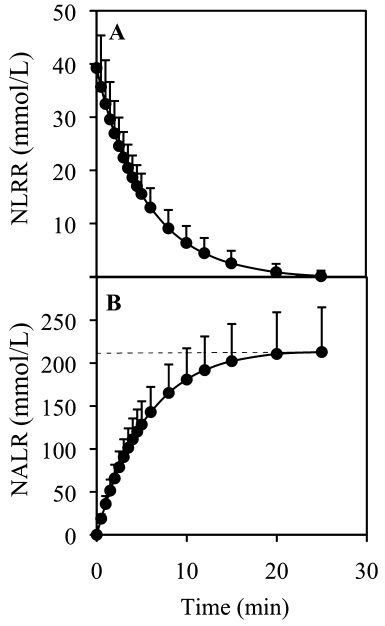

Fig. 2. (a) Mean (±standard deviation) net lactate release rate (NALR) and (b) net amount of lactate release (NALR) recovery curves after a 3-min all-out exercise. Dashed line represents the maximal value of NALR ($NALR_{max}$).

Discussion

The present study investigated whether three different minimally invasive methods of estimating lactate accumulation (Q_{LaA}) in rowers in response to short supramaximal (3-min all-out) exercise correlated with nonoxidative energy supply assessed by AOD.

Peak blood lactate concentration during recovery [$(La)_{peak}$] is usually considered representative of glycolysis involvement in ATP resynthesis during exercise. Nevertheless, and in line with previous studies (Medbø et al., 1988; Scott et al., 1991; Gastin et al., 1995; Pripstein et al., 1999; Bishop et al., 2002), AOD did not correlate with (La)(0) or $(La)_{peak}$ measured experimentally or interpolated from our curve fits. The significant correlation between $Q_{LaA(m1)}$ and AOD (Fig. 3(a)) suggests that $(La)_{peak}$ might be used to estimate nonoxidative glycolytic energy release during supramaximal exercise insofar as blood and whole body water fractions are considered (Margaria's method, see Eqn. [5]). To our knowledge, for the first time we report a correlation between AOD and $Q_{LaA(m1)}$ (Fig. 3(a)). However, it is important to note that $Q_{LaA(m1)}$ accounted for only 48% of variation in AOD, that limits the interest of this method. A possible explanation for this lack of concordance is that a substantial quantity of lactate produced during exercise is removed via oxidation or gluconeogenesis during the first minutes of recovery until $(La)_{peak}$ is reached, after 8 min into recovery in the present study. However, this quantity is not taken into account by Margaria's method.

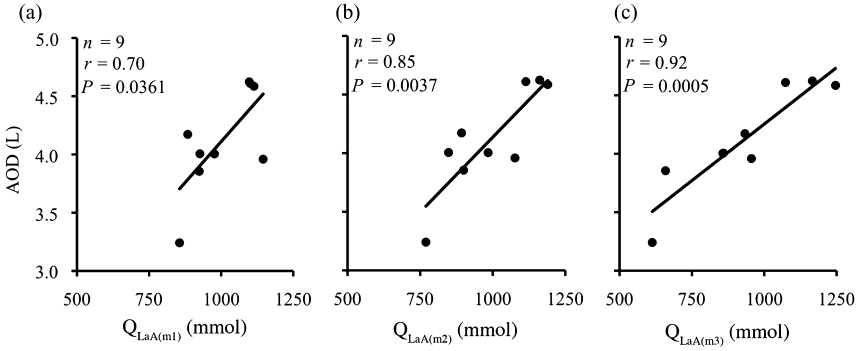

Fig. 3. (a–c) Relationships between the accumulated oxygen deficit (AOD) and the quantity of lactate accumulated estimated from (a) the first method based on Margaria's mathematical approach ($Q_{LaA(m1)}$, a); (b) the second method based on the lactate accumulated and removed at $(La)_{peak}$ ($Q_{LaA(m2)}$, b); and (c) the third method based on the lactate accumulation in muscle and blood compartment ($Q_{LaA(m3)}$, c) during a 3-min all-out exercise.

We propose that the second method takes account of lactate removal in the early phase of recovery. The quantity of lactate removed between (La)(0) and $(La)_{peak}$ (i.e., Q_{LaR}) can be estimated by considering the velocity constant γ_2, which denotes the capacity for lactate removal during recovery (see Eqn. [7]). Notably, the γ_2 values obtained here were comparable to those obtained elsewhere following exhaustive cycling exercise (Oyono-Engéllé et al., 1992; Thomas et al., 2004). Remarkably, Q_{LaR} represented 24% of Q_{LaA} at $(La)_{peak}$ and 19% of $Q_{LaA(m2)}$, which shows the importance of taking account of lactate removal in the early phase of recovery. More interestingly, considering Q_{LaR} in addition to Q_{LaA} at $(La)_{peak}$ in estimating $Q_{LaA(m2)}$ improved the correlation with AOD. In the present study, $Q_{LaA(m2)}$ accounted for 72% of AOD variation (Fig. 3(b)).

Comparing m1 and m2, Q_{LaA} at $(La)_{peak}$ and $Q_{LaA(m1)}$ represented the same parameter. However, Q_{LaA} at $(La)_{peak}$ was significantly lower than $Q_{LaA(m1)}$ (802 ± 89 vs 1003 ± 112 mmol, respectively). The difference is explained by the lactate distribution space values chosen for calculation. Margaria and coworkers considered that the water fraction was 0.6 for the whole body and 0.8 for the blood (Margaria et al., 1963, 1971) in determining lactate distribution volume (i.e., 75% of body mass; see Eqn. [5]). In the present study, on the basis of more recent reports (Di Prampero, 1981; Freund & Zouloumian, 1981b; Zouloumian & Freund, 1981a), lactate distribution space was taken as 600 mL/kg (i.e., 60% of body mass; see Eqn. [6]). Despite this difference, $Q_{LaA(m1)}$ was similar to $Q_{LaA(m2)}$ (1003 ± 112 vs 993 ± 149 mmol, respectively), meaning that adding Q_{LaR} to Q_{LaA} at $(La)_{peak}$ compensates the overestimation of lactate distribution space on the method of Margaria et al. (1963, 1971).

Lactate production is continuous and probably also occurs during recovery. Because $(La)_{peak}$ (used in methods m1 and m2) includes lactate production during the early phase of recovery [between end of exercise and $t(La)_{peak}$], $Q_{LaA(m1)}$ and $Q_{LaA(m2)}$ may have been overestimated. However, considering (a) that NLRR at baseline was ~0.12 mmol/min, and (b) that $t(La)_{peak}$ occurred on average at 8 min into recovery, the amount of the lactate released between end of exercise and $t(La)_{peak}$, which can be attributed to lactate production during recovery was only ~1 mmol, whereas $Q_{LaA(m1)}$ and $Q_{LaA(m2)}$ were approximately 1000 mmol. Because the amount of lactate produced between end of exercise and $t(La)_{peak}$ appeared negligible, we decided to not take account of this production in estimating $Q_{LaA(m1)}$ and $Q_{LaA(m2)}$.

Eqn. [12] proposes a totally different line of reasoning to determine Q_{LaA}. This third method is based on an application of the two-compartmental model of lactate distribution space during recovery. $Q_{LaA(m3)}$ during exercise depends on lactate accumulation in V_M (the previously active muscle) and V_S (the remaining lactate space) (Q_M and Q_S, respectively; see Methods). Interestingly, while Q_M correlated with $Q_{LaA(m3)}$ and AOD ($r = 0.98$, $P < 0.0001$, and $r = 0.88$, $P < 0.002$, respectively), Q_S did not. This lack of correlation clearly demonstrates once again that blood lactate accumulation at the end of exercise is not representative of muscle lactate production or more generally of nonoxidative energy supply during exercise.

Different assumptions were formulated to calculate $Q_{LaA(m3)}$. Both total lactate distribution space and muscle water content are expected to demonstrate weak inter- and intra-individual variation under normal conditions. Also, following Steinacker's estimate (Steinacker, 1993), we assumed that 70% of muscle mass was considered to be involved in rowing. This percentage could be discussed; however, for a given expertise level, the muscle mass involved in rowing should not differ between individuals, which considerably minimizes the impact of the exact percentage attributed, especially on the correlations obtained. The main assumption was formulated for Q_M calculation. As stated in the Materials and Methods section, and in accordance with a previous study (Freund et al., 1984), 30% of the lactate accumulated during exercise in the active muscles was considered as being released into the bloodstream during subsequent recovery. While setting this value at 30% does not affect the correlation between $Q_{LaA(m3)}$ and AOD, it does affect the level of muscle lactate accumulation (Q_M) during exercise. For instance, $Q_{LaA(m3)}$ varies from 929 ± 213 to 1456 ± 360 mmol if, instead of 30%, a value of 17% is applied in Eqn. [10]. However, the ratio Q_M/V_M, which approximates muscle lactate concentration at the end of exercise [$(La)_m$, in mmol/L], provides a mean end-of-exercise $(La)_m$ of 49 and 28 mmol/L with values of 17% and 30%, respectively. This second predicted $(La)_m$ value is very close to that of 27.1 mmol/L obtained from muscle biopsies after an exhaustive maximal leg extension exercise of similar duration (Bangsbo et al., 1991). These results further argue in favor of our choice of 30% for the percentage lactate release from muscle. On the other hand, applying the same value of 30% in all the subjects constitutes one limitation of our approach: it is not self-evident that the proportion of lactate accumulated in the previously active muscle which is then released in V_S during recovery will be the same from one individual to another; indeed, a previous study reported inter-individual differences of 12% (Freund et al., 1984). In spite of these limitations, the present results showed close correlations between Q_M or $NALR_{max}$ and AOD, suggesting that inter-individual differences in the proportion of the Q_{LaA} in the muscle during exercise that is released during recovery would have only a minor impact on our results. More interestingly, the present method provided the highest correlation coefficient between the three Q_{LaA} estimates and AOD ($r = 0.92$, Fig. 3(c)).

In conclusion, the main findings of the present study are that (a) no single post-exercise blood lactate concentration [whether (La)(0), $(La)_{peakMeas}$ or $(La)_{peakMod}$] provides valid and consistent information on nonoxidative

glycolytic energy supply during exercise, and (b) although the original method of Margaria et al. does correlate positively with AOD (Fig. 3(a)), the correlation coefficients using the two new methods described in the present study were much higher (Fig. 3(b) and (c)). Taken together, these results highlight the importance of glycolysis in total ATP resynthesis during a supramaximal exercice of 3-min duration. Finally, the present study provides two interesting, useful and minimally invasive methods for estimating lactate accumulation (Q_{LaA}) in response to supramaximal exercise.

Perspectives

Despite their admitted limitations, the present methods m2 and m3 provide new insights into lactate accumulation during supramaximal exercise, and do so in a minimally invasive way, making them potentially interesting for sport scientists, exercise physiologists and coaches. Performance during supramaximal exercise is highly dependent on the contribution of nonoxidative glycolytic energy. Thus, methods m2 and m3 allow the relationship between lactate accumulation and performance to be explored in different kinds of supramaximal exercise: e.g., sprint running events. The effects of different kinds of training on lactate accumulation could be investigated. And research might also seek to improve the methods by estimating more precisely (a) the different volumes of distribution for lactate (V_{TLS}, V_M and V_S), and (b) the muscle mass involved in exercise, for which magnetic resonance imaging could be useful.

Key words: nonoxidative glycolytic metabolism, accumulated oxygen deficit, mathematical model, rowing.

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