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Arterial versus Venous Blood Lactate Increase in the Forearm during Incremental Bicycle Exercise

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Summary. Eight male subjects were studied during incremental bicycle exercise. From the forearm, arterial and venous blood lactate concentrations were measured every minute until exhaustion. There was a statistically significant difference ($P < 0.01$) in the points at which the arterial and venous blood lactates began to increase above the resting level. The onset of increase of lactate in arterial blood occurred at $1.00 \pm 0.07\,l \cdot min^{-1}$ in $\dot{V}O_2$ (mean ± SEM), which corresponded to $37.0 \pm 1.5\%$ of $\dot{V}O_{2max}$. Its venous counterpart occurred at $1.50 \pm 0.17\,l \cdot min^{-1}$ in $\dot{V}O_2$, $55.0 \pm 3.8\%$ of $\dot{V}O_{2max}$. The arterio-venous lactate difference became greater after the onset of increase in arterial blood lactate (anaerobic threshold), presumably as consequence of lactate utilization by the forearm muscles.

It was concluded that the onset of blood lactate increase differs according to the sites of blood sampling, which should be considered for the interpretation of anaerobic threshold.

Key words: Arterial blood lactate concentration – Anaerobic threshold – Arterio-venous lactate difference

Introduction

Blood lactate concentration during exercise has been used to determine anaerobic threshold (AT) as the point at which lactate begins to increase above the resting level (Williams et al. 1967; Wasserman et al. 1973; Davis et al. 1976; Senay and Kok 1977; Farrell et al. 1979; Reinhard et al. 1979; Yoshida et al. 1981a, b) or it reaches a given value (Londeree and Ames 1975; Mader et al. 1976; Mader and Hollmann 1977; Kindermann et al. 1979; Rusko et al. 1980; Jacobs 1981; LaFontaine et al. 1981). There is, however, inconsistency concerning the site of blood sampling for the determination of lactate concentration (arterial, arterized, or venous blood).

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Table 1. Physical characteristics of subjects

	Age (year)	Height (cm)	Weight (kg)	$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)
Mean	21.4	168.3	68.5	40.3
± SEM	0.5	3.8	4.3	2.6

It is well known that the lactate production is not caused merely by anaerobic metabolism, but is the result of dynamic imbalance between anaerobic glycolysis and the utilization of pyruvate in TCA cycle (Jöbsis and Stainsby 1968). Therefore, the AT determined by the lactate concentration indicates the point at which the rate of lactate production exceeds that of its utilization. The produced lactate has been found to be utilized in the inactive muscles (Stainsby and Welch 1966; Jorfeldt 1970; Issekutz et al. 1976; Poortmans et al. 1978). This fact would vary the lactate concentration obtained by different sites of blood sampling, resulting in different values of AT.

Although lactate uptake in the forearm has been indicated during bicycling exercise (Hollmann 1961; De Coster et al. 1969; Poortmans et al. 1978), less attention has been paid to the onset of increase in the arterial and venous blood lactate. The purpose of this study was to investigate simultaneous changes of the arterial and venous blood lactates in the forearm during bicycle exercise.

Methods

Subjects. Subjects for this investigation were eight healthy college male students who had no previous history of cardiorespiratory disease. Each subject was accustomed to exercise on the bicycle ergometer but none of them took part in any form of supervised training program or stressful recreational activities. Each subject was fully informed of the purpose of this study and possible risks before signing an informed consent form. Prior to this investigation, each subject underwent medical examinations, including an ECG test, blood pressure measurement, spirometric test and general medical check-up. The physical characteristics of the subjects are shown in Table 1.

Test Procedures. Exercise was performed on a Monark bicycle ergometer. Exercise was commenced with 4 min unloaded pedalling "0 watt" and thereafter 25 watt increments were given every minute until the maximal exercise tolerance was reached.

Oxygen uptake ($\dot{V}O_2$) was measured by the Douglas bag method. The subject breathed through a low resistance J-valve and the expired gas was collected into a Douglas bag every succeeding minute. The respiratory minute volume ($\dot{V}_E$) was measured by a gas meter and a portion of the gas sample was dried with $CaCl_2$ and was analyzed for O_2 and CO_2 concentrations by a polarograph oxygen analyzer and an infra-red carbon dioxide analyzer, respectively: these were calibrated against a micro-Scholander gas analyzer prior to each experimental run. Furthermore, before and after each expired gas analysis, air and a standard gas (14.89% O_2, 5.10% CO_2 in N_2) which had previously been analyzed by a micro-Scholander technique were used to test any instrumental drift. $\dot{V}O_2$ was calculated based upon $\dot{V}_E$ (STPD), true O_2 and CO_2 concentrations. Heart rate (HR) was continously monitored by an ECG with CM_5 lead position.

Blood Samplings and Analyses. To obtain the blood samples, two Teflon catheters were inserted into the radial or brachial artery anesthetized with 0.5% xylocaine, and the cubital vein. The arterial and venous blood samplings were simultaneously performed at rest, after the third minute of exercise and thereafter every minute at the times of expired gas collection until the cessation of exercise.

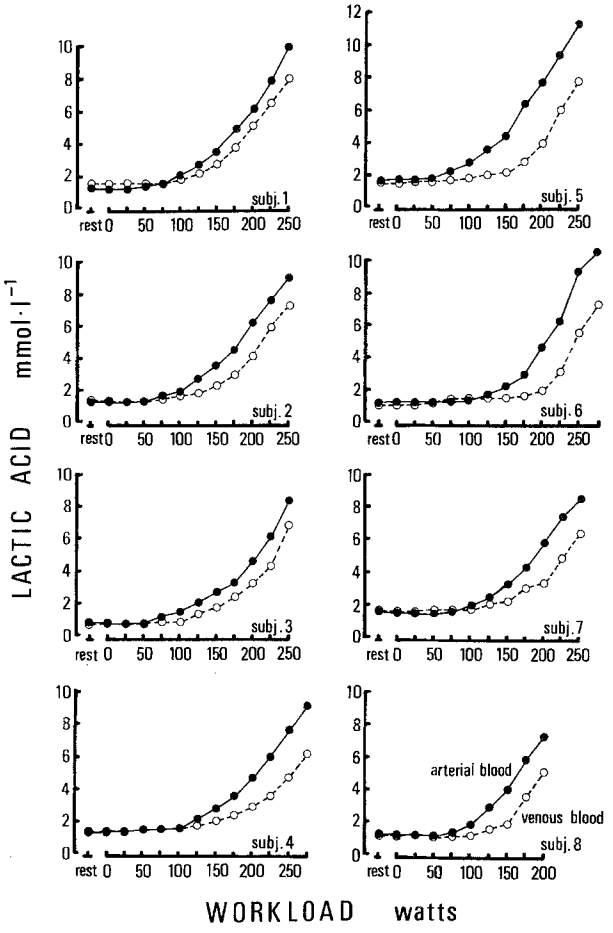

Fig. 1. Arterial and venous blood lactate concentrations during incremental bicycle exercise. The figure gives the individual data. Note that the onset of increase in arterial blood lactate occurred earlier than the venous lactate counterpart. Soild lines denote the arterial blood lactate and dashed lines indicate the venous blood lactate

Blood pH, Po_2, Pco_2 were analyzed by the electrodes at 37° C (Instrumentation Laboratory Model 813). Bicarbonate concentration (HCO_3^-) was calculated by the Henderson-Hasselbach equation. The analysis of blood lactate concentration was performed by the enzymatic method (Boehringer, Mannheim, FRG); chilled 0.6N perchloric acid was used for deproteinization.

Statistical Analyses. Means and SEM were calculated according to standard methods. Regression analysis and correlation analysis were performed on the appropriate data. Paired *t*-tests were used to evaluate differences in the appropriate data.

Results

Figure 1 indicates the changes in the forearm arterial and venous blood lactate concentrations during incremental bicycle exercise. In all subjects, the point at which the arterial blood lactate concentration began to increase above the

Table 2. Descriptive measures associated with the onset of increase in arterial and venous blood lactate concentrations

Measures	The onset of increased in		"t"
	Arterial lactate	Venous lactate	
$\dot{V}O_2$ ($l \cdot min^{-1}$)	1.00 ± 0.07	1.50 ± 0.17	4.76**
% of $\dot{V}O_{2\,max}$	37.0 ± 1.5	55.0 ± 3.8	6.44**
$\dot{V}_E$ ($l \cdot min^{-1}$)	25.6 ± 2.6	36.6 ± 4.9	2.81*
R	0.80 ± 0.01	0.87 ± 0.002	7.84**
Workload (watts)	65.6 ± 8.1	121.9 ± 14.5	5.46**

$\bar{X}$ ± SEM, * $P < 0.05$, ** $P < 0.01$

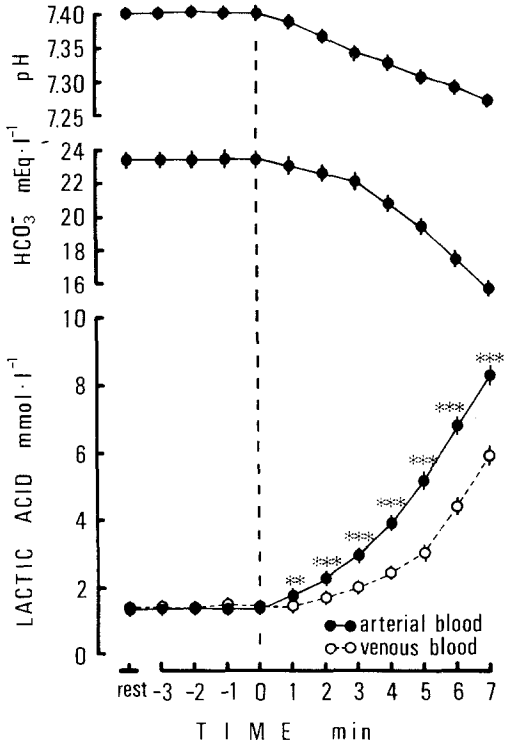

Fig. 2. Changes of arterial and venous blood lactate concentrations, arterial blood bicarbonate concentration, arterial blood pH in the forearm during incremental bicycle exercise (data indicate means ± SEM). Note that time (abscissa) is expressed "zero" when onset of increase in arterial blood lactate in individual subject was occurred (denoted as vertical dashed line). The workload was increased by 25 watts every minute. Symbol (*) denotes significant difference between arterial and venous blood lactate concentrations (** $P < 0.01$, *** $P < 0.001$)

resting level was found at a lower workload than the venous lactate counterpart. As a result, the paired *t*-test applied to gas exchange and workload at the onset of increase in the arterial and venous blood lactates was significant (Table 2).

It is well known that the workload at which arterial blood lactate begins to increase is dependent on the subject's state of conditioning and/or cardiorespiratory endurance fitness, as indicated in Figure 1 (Williams et al. 1967; Farrell et al. 1979; Yoshida et al. 1981a, b). Therefore, we rearranged data in each subject based upon the time at which the arterial blood lactate began to increase above the resting level (AT), as shown in Figs. 2 and 3. This procedure made the

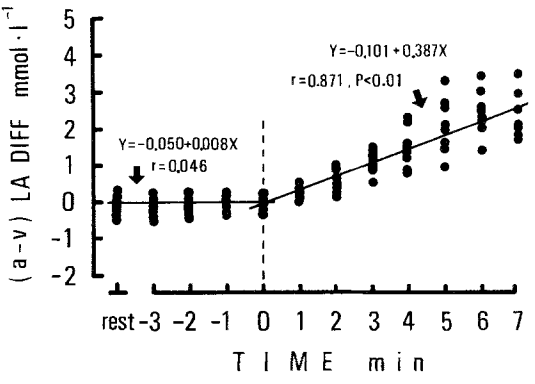

Fig. 3. The arterio-venous lactate difference as the function of time. The time (abscissa) is indicated in the legend of Fig. 2

individual difference very small. Figure 2 indicates the changes in the arterial and venous blood lactate concentrations, arterial blood bicarbonate concentration, and arterial blood pH. The onset of increase in arterial blood lactate coincided with falls in arterial blood bicarbonate and blood pH, but this point did not concur with its venous counterpart.

The difference of arterial and venous blood lactate did not show statistical significance before the onset of increase in arterial blood lactate. After the arterial blood lactate increase, the difference became significant (Fig. 2) and it increased with time (Fig. 3).

Discussion

The major finding of this investigation is that there is not only an arterio-venous lactate difference, but also a statistical difference between the onset of increase in arterial and venous blood lactates in the forearm during incremental bicycle exercise. The former finding supports the results reported by Hollmann (1961), De Coster et al. (1969), and Poortmans et al. (1978). They revealed the positive arterio-venous lactate difference in the forearm during submaximal steady state exercise. However, no study has reported the latter finding.

As shown in Fig. 1, the onset of increase in arterial blood lactate always exceeded the venous lactate counterpart. The onset of increase in arterial and venous blood lactates averaged 37.0% and 55.0% of $\dot{V}O_{2max}$, respectively. The value of 37.0% of $\dot{V}O_{2max}$ was comparable to the AT reported by investigators using the arterial blood lactate concentration (Reinhard et al. 1976; Yoshida et al. 1981a, b). But the value was lower than the AT determined with venous blood lactate in sedentary subjects (Davis et al. 1976; Ivy et al. 1980).

The explanation for the significant difference of onset of increase between the arterial and venous blood lactates during bicycle exercise would be lactate utilization by the inactive forearm muscles. Lactate utilization by the inactive muscles might be proved by the finding that the arterio-venous lactate difference became positive after the onset of increase in arterial blood lactate (Figs. 2, 3). Furthermore, Jorfeldt (1970) indicated that when ^{14}C-lactate was infused into

the brachial artery during light forearm exercise, 30% to 50% of lactate was immediately oxidized into $^{14}CO_2$ by the forearm muscles. Therefore, the venous lactate in the forearm reflects also the result of lactate utilization of arterial blood lactate by the forearm muscles. This process would result in a shift of AT as determined by venous blood lactate levels. Thus, studies on metabolic acidosis during exercise as well as AT should be based on arterial blood.

In conclusion, the AT value differs according to the site of blood sampling, and this fact could lead to errors in AT determination. When discussing AT, therefore, it is necessary to keep in mind that the onset of increase in lactate differs at different blood sampling sites.

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