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Relation between lactic acid steady-state and muscle oxygenation in elite cyclists

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Abstract

Background & Purpose. In cycling, several physiological values are taken into account to evaluate the performance level of each athlete and/or to plan his training program. One of these values is the maximal power output corresponding to the lactic acid steady-state. To find this power value, it is necessary to test the athlete by an incremental protocol to collect all the physiological parameters necessary to program a subsequent square-wave test, during which it is possible to measure directly this power output. This procedure takes a long time. Bellotti et al. showed that the lactic acid steady-state may be determined by measuring the deoxygenated haemoglobin by NIRS technique in healthy subjects (Bellotti et al., 2013: Med Sci Sports Exerc. 45(6): pp1208-16). We wanted to verify if it is possible, in elite cyclists, to detect the power output corresponding to the lactic acid steady-state using the NIRS technique during a single incremental test.

Methods. The experiment was carried on 15 male, elite cyclists participating in international U23 races: 21 ± 2 years, 1.76 ± 0.08 m, 66.1 ± 6.9 kg (avg$\pm$s, n=15). We used an incremental protocol on a stationary bike (Monark 818 equipped with SRM system, Figure 1 left) consisting on five loads of 360 s each, starting from 0.85 w kg^{-1} followed by 1w kg^{-1} increment until the last load at 4.85 w kg^{-1}. Total and oxygenated haemoglobin (tHb and HbO$_2$) were continuously measured in the vastus lateralis of the left quadriceps muscle by NIRS technique (Figure 1 right, NIMO, NIROX, Brescia, Italy), and lactic acid concentration (AL) was measured by Accusport (Bishop, 2011: Sport Med, 22, pp525-530) at 180 s and the end of the last three loads defined as $W_{low} = 2.97\pm0.12$ w kg^{-1}, $W_{medium} = 3.93\pm0.11$ w kg^{-1} and $W_{high} = 4.90\pm0.14$ w kg^{-1} (avg$\pm$s, n=15). Pedalling cadence was 1.5÷1.6 Hz. We calculated the time variation of AL ($\Delta AL/\Delta t$) and the time rate of tHb and HbO$_2$ ($\Delta tHb/\Delta t$ and $\Delta HbO_2/\Delta t$) by a linear interpolation of the NIRS data during the time interval between 180 s and the end of W_{low}, W_{medium} and W_{high}. Twelve participants completed the entire protocol whereas three stopped between 300 and 320 s of W_{high}.

Results and Discussion. Figure 2 shows a typical experimental record. The subject pedals against the incremental load (continuous line) while muscular oxygenation (dotted line) and AL (point and continuous line) were measured during the last three loads. During exercise at W_{low}, all the participants showed an increase both of tHb (upper trace) and HbO$_2$ (intermediate trace) due to peripheral vasodilatation (Grassi et al. 1999: J Appl Physiol; 87, pp348-355). The values of AL at 180 s of W_{low} (3.1 ± 0.8 mM; avg$\pm$s, n=15) were always greater than AL at the end of W_{low} and seemed to be too high for elite athletes. This result may be due to early lactate production; at the end of W_{low}, AL decreased to a value compatible with the mechanical power at that moment (1.7 ± 0.5 mM; avg$\pm$s, n=15). During exercise at W_{medium} the participants reached the AL steady-state: there was no difference between AL measured at 180 s and 360 s (2.3 ± 0.6 vs 2.7 ± 0.7 mM; avg$\pm$s, n=15) and $\Delta AL/\Delta t$ is 1.79 ± 2.19 μM s^{-1} (avg$\pm$s, n=15). NIRS measurements showed that the muscle oxygenation was about constant: $\Delta tHb/\Delta t$ and $\Delta HbO_2/\Delta t$ were -0.0024 ± 0.0081 μM s^{-1} and -0.0061 ± 0.0124 μM s^{-1} respectively (avg$\pm$s, n=15). Figure 3 shows $\Delta AL/\Delta t$, $\Delta tHb/\Delta t$ and $\Delta HbO_2/\Delta t$ plotted as a function of power output; note that the linear function of $\Delta AL/\Delta t$ (continuous line) and $\Delta HbO_2/\Delta t$ (dotted line) reach zero at the same point very near to a W_{medium}. According to Grassi (1999), during and incremental exercise, the onset of lactic acid accumulation is related the beginning of the haemoglobin desaturation. During exercise at W_{high} we measured a sharp increase of AL and a decrease of muscle oxygenation: $\Delta AL/\Delta t$ was 12.76 ± 8.11 μM s^{-1} (avg$\pm$s, n=15) and $\Delta tHb/\Delta t$ and $\Delta HbO_2/\Delta t$ were -0.0097 ± 0.0165 μMs^{-1}; and -0.0150 ± 0.0151 μMs^{-1} respectively; (avg$\pm$s, n=15).

Figure 4 shows $\Delta AL/\Delta t$ as a function of $\Delta HbO_2/\Delta t$. The linear function through all the experimental data passed very close to the axis origin, showing a strong relation between the AL steady-state and the HbO$_2$ steady-state. This point of coincidence is very near to W_{medium}.

Conclusion. We measured the AL steady-state at 3.75 w kg^{-1} and the muscular oxygenation steady-state at 3.74 w kg^{-1}. This suggest that it is possible to determine the power output corresponding to the AL steady-state during in incremental protocol, only measuring the muscular oxygenation by NIRS technique.

Figure 1 Experimental set-up. Left panel: modified Monark 818 modified. Handlebars, saddle and pedals are race bike type. The work load is settled by the mechanical brake and measured by the SRM system. Right panel. The NIRS probe is firmly attached to the shaved thigh by Tensoplast and covered by the race type short.
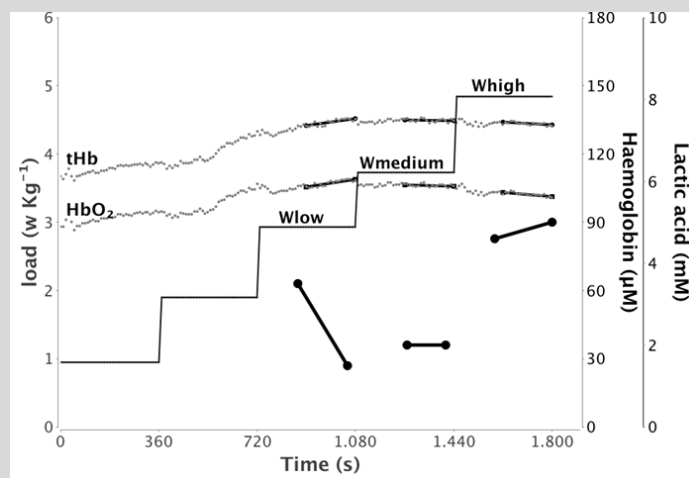

Figure 2. Experimental trace. The cyclist (19 years, 1.87 m, 74.8 kg) pedals against the incremental load (continuous line) while total and oxygenated haemoglobin (tHb upper and HbO2 bottom dotted line) were continuously measured by the NIRS. The slope of the linear interpolation trough the NIRS data gives the time rate of tHb and HbO2 variations during Wlow, Wmedium and Whigh. Point and continuous line are the lactic acid concentration.
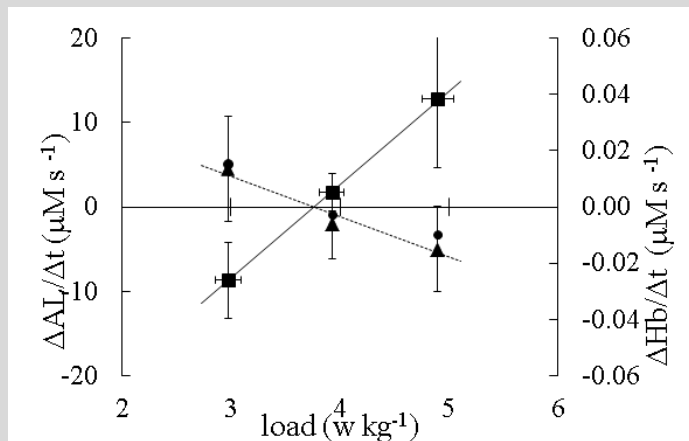

Figure 3. Lactic acid and muscular oxygenation as function of mechanical load. The average value of lactic acid time rate (△AL/△t square symbols, continuous line R2=0.734) and the average value of total (△tHb/△t circles) and oxygenated haemoglobin (△HbO2/△t triangles, dotted line R2=0.355) time rate are plotted as a function of work load. Lines are drawn through all the experimental data (not shown). Error bars are s. (n=15).
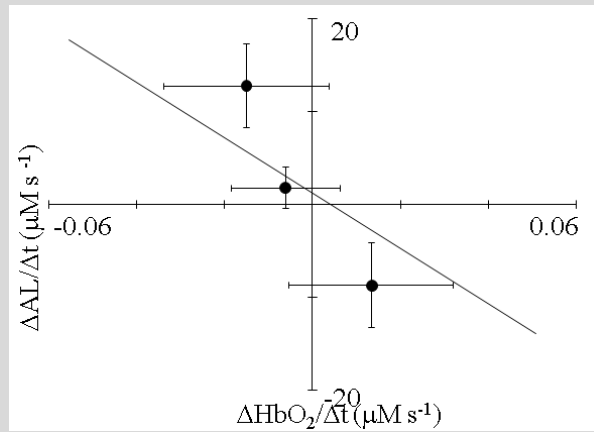
Figure 4. Lactic acid as function of oxygenated haemoglobin. The time rate of lactic acid concentration $\Delta A\Lambda/\Delta\tau$ is plotted as a function of the oxygenated haemoglobin time rate $\Delta H\beta O2/\Delta\tau$. Average value, error bars are s. (n=15). The line is through all the experimental data (not showed)

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