

Preconditioning effect of heavy exercise on O₂ uptake kinetics, determined as MRT (mean response time), in chronic heart failure patients

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It has been demonstrated that oxygen consumption kinetics ($\dot{V}O_{2cin}$) at the onset of aerobic exercise may be speeded up when preceded by a short bout of heavy exercise (*preconditioning heavy exercise*, PHE): PHE is effective only on subsequent high intensity exercise in young physically fit subjects, while it may be effective also in moderate intensity exercise in elderly individuals. It was proposed that the mechanism underlying PHE be related to increased muscle blood flow and heart rate after heavy exercise, which would speed up the enhancement of oxygen transport to active muscles after PHE. Whether this phenomenon operates also in patients with chronic heart failure (CHF) has not been determined, yet, and will be the subject of this presentation. 14 CHF male patients (68yy, 77kg, 172cm) performed a cycle ergometer (Sport Excalibur, Lode, NL) incremental test ($20W+10W \cdot \text{min}^{-1}$) to exhaustion, to determine individual maximal aerobic power ($\dot{V}O_{2max}$) and workload (W_{max}). Workloads corresponding to the first (W_{vt1}) and the second (W_{vt2}) ventilator thresholds were also determined by Wasserman method. $\dot{V}O_{2cin}$ was studied in two subsequent identical sessions (15 days apart) each comprising two moderate load exercises (SW_1 , SW_2) at 80% W_{vt1} , separated by a high intensity exercise (SW_{PHE}), with load equal to $(W_{vt2} + (W_{max} - W_{vt2})/2)$. Each exercise started with 3 min free wheeling at 30 RPM, followed by 6 min loaded pedaling at 70 RPM. Successive exercise periods were separated by 6 min sitting rest. Respiratory gas composition was continuously recorded (Innocor, Innovision, DK) during the entire experiment. Breath by breath oxygen values ($\dot{V}O_{2bxb}$) were used to calculate oxygen deficit (def_{O_2}) as the difference between theoretical and real $\dot{V}O_2$ ($def_{O_2} = (\text{mean steady state } \dot{V}O_2 \cdot 360s - \int_1^{360} \dot{V}O_{2bxb})$). The mean response time (MRT) was calculated as the ratio between def_{O_2} and $\dot{V}O_{2ss}$. The mean workloads were 64w in SW_1 and SW_2 , and 96w in SW_{PHE} . Since there was no difference between the two sessions, we pooled the results. def_{O_2} was 784, 924 and 669 ml at SW_1 , SW_{PHE} and SW_2 , respectively, with a reduction by 14.8% ($p=0.007$) from SW_1 to SW_2 , while $\dot{V}O_{2ss}$ did not change at equal workloads (1.246 and $1.293 \text{ l} \cdot \text{min}^{-1}$) and was $1.521 \text{ l} \cdot \text{min}^{-1}$ in PHE. Thus, MRT, which we used as an index of $\dot{V}O_{2cin}$, was significantly shortened by preconditioning, from 37.1s in SW_1 to 30.6s in SW_2 (-18.4%; $p=0.001$).

Maximal aerobic power is generally hampered in CHF patients, as part of a reduced level of wellbeing. Also $\dot{V}O_{2cin}$ is known to be more sluggish than in their age matched counterparts. Thus, it is important to highlight a means by which $\dot{V}O_{2cin}$ can be speeded up, thus reducing the need to rely on anaerobic mechanisms at the beginning of exercise. Even more important is recognizing that metabolic adjustments in CHF patients, although negatively influenced by the heart pathology, may still be modulated by appropriate physiological stimuli.

It has been demonstrated that oxygen consumption kinetics ($\dot{V}O_{2cin}$) at the onset of aerobic exercise may be speeded up when preceded by a short bout of heavy exercise (*preconditioning heavy exercise*, PHE). It was proposed that the mechanism underlying PHE be related to increased muscle blood flow and heart rate after heavy exercise, which would speed up the enhancement of oxygen transport to active muscles after PHE. This phenomenon operates also in patients with chronic heart failure (CHF) has not been determined, yet. 14 CHF male patients (68yy, 77kg, 172cm, Class II NYHA) performed a cycle ergometer incremental test to exhaustion, to determine individual maximal aerobic power ($\dot{V}O_{2max}$), workload (W_{max}) and workloads at the first (W_{vt1}) and the second (W_{vt2}) ventilator thresholds. $\dot{V}O_{2cin}$ was studied in two moderate load exercises (SW_1 , SW_2) at 80% W_{vt1} , separated by a high intensity exercise (SW_{PHE}), with load equal to $(W_{vt2} + (W_{max} - W_{vt2})/2)$, separated by 6 min sitting rest. Breath by breath oxygen values ($\dot{V}O_{2bxb}$) were used to calculate oxygen deficit ($def_{O_2} = (\dot{V}O_{2ss} \cdot 360s - \int_1^{360} \dot{V}O_{2bxb})$) and the mean response time ($MRT = def_{O_2} \cdot \dot{V}O_{2ss}^{-1}$). def_{O_2} was reduced by 14.8% ($p=0.007$) from SW_1 to SW_2 , while $\dot{V}O_{2ss}$ did not change at equal workloads. Thus, MRT, was significantly shortened by preconditioning (-18.4%; $p=0.001$). The metabolic adjustments in CHF patients, although negatively influenced by the heart pathology, may still be modulated by appropriate physiological stimuli.