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ORIGINAL ARTICLE

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Heart rate deflection compared to 4 mmol · l^{-1} lactate threshold during incremental exercise and to lactate during steady-state exercise on an arm-cranking ergometer in paraplegic athletes

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Abstract The deflection point (DP) of the heart rate in relation to the work rate (WR) of 8 male endurance-trained paraplegics and 11 male physically active sports students was investigated during nonsteady-state incremental arm cranking ergometry (IT) and compared to the 4 mmol · l^{-1} blood lactate concentration threshold and to blood lactate concentration in steady-state exercise (SST). Heart rate, and lactate concentration from capillary blood, were determined at rest, during IT and SST. The DP was calculated by linear regression analysis of the heart rate during IT. The SST consisted of three consecutive exercise intensities over a period of 8 min at exercise intensities of 10 W below, and at 10 W above the work rate at deflection point (WR_{DP}). No difference was found between the paraplegics and non-handicapped subjects regarding heart rate and blood lactate concentration at rest and during exercise. A DP was established in all the paraplegics and in 72.7% of the non-handicapped subjects, but lactate accumulation was observed in 75% of the paraplegics and in 62.5% of the non-handicapped subjects at the lowest intensity of SST. In summary, endurance-trained paraplegics with an injury level below T_5 showed heart rate and blood lactate concentration values comparable to non-handicapped subjects during IT. A linear increase at moderate exercise intensities and a levelling-off at higher to maximal intensities could be identified in all the paraplegics and in 72.7% of non-handicapped subjects. The determination of the anaerobic threshold by DP should be applied with caution, since no causal relationship of DP and the anaerobic threshold was found and the WR_{DP} tended to overestimate threshold values.

Key words Paraplegia · Exercise testing · Anaerobic threshold · Heart rate threshold · Lactate threshold

Introduction

A sigmoidal heart rate (HR) relationship to work rate (WR) curve has been observed during incremental nonsteady-state exercise with a linear increase at moderate to submaximal intensities and a levelling off at higher and maximal intensities in most cases (Bernard et al. 1997; Brooke and Hamley 1972). The transition from a linear to a non-linear increase in HR has been described as the deflection point (DP). It has been found that at low WR the heart adapts initially to the increasing exercise intensity by increasing stroke volume more than heart rate (Astrand and Rodahl 1986). The physiological mechanisms of the heart rate deflection are not completely understood and are still under discussion (see Conconi et al. 1996; Heck et al. 1989; Israel 1982).

It has been assumed to be due to a smaller increase in oxygen uptake ($\dot{V}O_2$) because of the activation of the anaerobic lactate mechanisms of adenosine triphosphate production, or to a less pronounced increase in HR when the absolute muscle force becomes greater, or to a lack of time for HR adjustment at higher intensities of exercise (Israel 1982; Pendergast et al. 1979). It has also been discussed that the metabolic acidosis facilitates the release of oxygen from haemoglobin and improves its extraction from the tissues, so that increases in $\dot{V}O_2$ can exceed increases in cardiac output or HR (Conconi et al. 1996). According to Conconi et al. (1982) the deflection point of the heart rate (HR_{DP}) is a noninvasive, indirect way to determine the anaerobic threshold during incremental exercise. This method has been the subject of controversy (Heck et al. 1989; Sumsion et al. 1989; Tokmakidis and Leger 1992).

Various concepts of the anaerobic threshold have been propounded, e.g. the WR corresponding to a maximal steady state of blood lactate concentration ($[La^-]_{max,ss}$) during a constant workload, a fixed

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threshold at a blood lactate concentration of 4 mmol · l^{-1} (AT_4), the lactate turn point or the ventilatory threshold (Davis et al. 1983; Hofmann et al. 1994a; Mader and Heck 1986; Stockhausen et al. 1995; Wasserman et al. 1991).

It has been reported that complete spinal cord lesion results in a loss of motor and sensory functions conducted via afferent and efferent spinal pathways, but also in an interruption of pathways from the central nervous system to the peripheral sympathetic nervous system, which results in cardiovascular and metabolic alterations at rest and during exercise (Figoni 1992; Hopman et al. 1993; Schmid et al. 1998; Schmid et al. in press). Little information is available on the HR and lactate reaction and on the determination of the anaerobic threshold during incremental nonsteady-state exercise for spinal cord injured persons (Lin et al. 1993; Melton et al. 1988; Vinet et al. 1997).

In this study, HR_{DP} curve was investigated during incremental nonsteady-state ergometry (IT) and compared to AT_4 and to the blood lactate concentration curve in a steady-state exercise test (SST) at the DP. These parameters were studied in paraplegic high-performance athletes and non-handicapped subjects during arm cranking exercise.

Methods

Subjects

A group of 8 male paraplegic high-performance athletes (PP) with spinal cord injuries between T_6 and L_1 (German National Team Cross-Country Sledge and High Performance Wheelchair Athletes) and a group of 11 male, physically active, non-handicapped sports students (NH) participated in this study. Their physical characteristics are summarized in Table 1. All the subjects gave written informed consent for participation.

Protocol

Both groups performed a continuous IT, a so-called modified Conconi test, until they were exhausted and a SST on an electrically braked arm-crank ergometer (Erich Jäger Co., Würzburg). On this ergometer the pedals could be set in different positions and could be replaced by handles. During the arm-exercise PP used their everyday wheelchairs, NH sat straight with the back supported; in order to minimize the use of accessory muscles the legs were stretched out in front without being restrained. In addition, they were given detailed instructions to use only the upper body muscles. The height of the crank axis was adjusted for each individual to heart level and was at a distance which caused a slight flexion in the extended arm. Following a 3-minute warm-up at 20 W, the IT began with an initial intensity of 20 W, increasing by 10 W every minute. Crank frequency was kept constant between 50 and 60 rpm.

In the second part of the study, following a 3-min warm-up at 30 W, SST consisting of three consecutive exercise intensities over a period of 8 min at intensities of 10 W below, at, and 10 W above the work rate at deflection point (WR_{DP}) was carried out. The tests were separated by 3 days during which no physical exercise was performed.

Physiological variables

The HR was calculated continuously with a 5-s storage interval and recorded at rest, every minute during IT and every other minute during SST (Polar Sport Tester, Unilife Co.). The lactate concentration was estimated from capillary blood drawn from the ear lobe made hyperaemic by a cream containing nonyl-acid-vanillyamide and nicotine-acid-butoxyethylester and was determined enzymatically at rest and at 2 min intervals during IT and SST as has been described by Hohorst (1962).

The relationship between WR and HR during the incremental test was calculated by computer and presented graphically (Polar Electro Co.). The HR curve produced an initial curved region, which was excluded from the analysis. The remaining data were divided into two linear segments when possible. The DP was calculated as the intersection of the two regression lines by computer-aided linear regression analysis of the slopes of HR plotted as a function of WR. The analysis was only accepted if at least three points appeared on the second line (Fig. 1).

A steady-state of blood lactate concentration $[La^-]_{ss}$ was assumed in SST if the lactate concentration did not increase more than 0.1 mmol · l^{-1} · min^{-1} between the 4th and 8th min of any level of exercise.

Statistical analysis

The Wilcoxon test was used to compare intraindividual mean differences. The groups were compared using the Mann-Whitney U-test for independent data. A nonparametric method for testing statistical significance was used because, due to the small number of

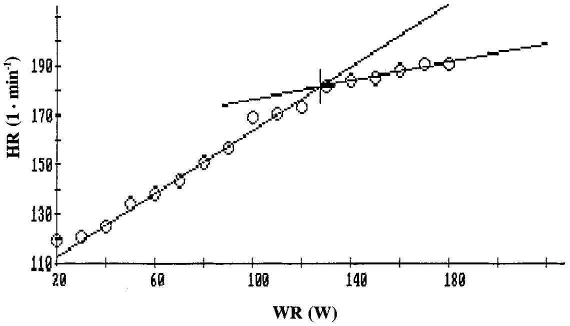

Fig. 1 Example of computer-aided determination of the deflection point of the heart rate (*HR*) curve in incremental arm cranking ergometry. *WR* work rate

Table 1 Mean values of the anthropometric data and amounts of training of the paraplegics (*PP*) and non-handicapped persons (*NH*)

	Number	Age (years)		Height (cm)		Body mass (kg)		Training amount (h · week^{-1})	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
PP	8	35.7	8.1*	175.0	6.5	68.8	7.6*	8.3	3.7*
NH	11	26.4	2.4	182.9	7.9	76.6	8.4	5.6	2.6

* $P < 0.05$ between *PP* and *NH*

Table 2 Maximal work rate (WR_{max}), resting and maximal heart rate (HR_{rest}, HR_{max}) and resting and maximal lactate concentrations ($[\mathrm{La}^-]_{\mathrm{rest}}$, $[\mathrm{La}^-]_{\mathrm{max}}$) of paraplegic (*PP*) and non-handicapped (*NH*) subjects in an incremental arm cranking exercise

	$\mathrm{HR}_{\mathrm{rest}}$ ($1 \cdot \mathrm{min}^{-1}$)		$\mathrm{La}^-_{\mathrm{rest}}$ ($\mathrm{mmol} \cdot \mathrm{l}^{-1}$)		$\mathrm{WR}_{\mathrm{max}}$ (W)		$\mathrm{HR}_{\mathrm{max}}$ ($1 \cdot \mathrm{min}^{-1}$)		$\mathrm{La}^-_{\mathrm{max}}$ ($\mathrm{mmol} \cdot \mathrm{l}^{-1}$)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
PP	81	9	1.4	0.4	145.0	21.3*	182	9	9.8	2.6
NH	89	10	1.6	0.5	160.0	48.3	178	18	10.6	2.7

* $P < 0.05$ between *PP* and *NH*

subjects, it was difficult to prove normal distribution and homogeneous variance. The correlation of $\mathrm{WR}_{\mathrm{DP}}$ and the AT_4 threshold was studied using linear correlation analysis. The minimal level of significance was set at $P < 0.05$.

Results

No statistically significant differences were observed in HR and blood lactate concentrations at rest, at the different exercise intensities and after exercise between PP and NH (Table 2, Fig. 2), as well as at AT_4 (Table 3). The NH subjects achieved a higher maximal work rate ($\mathrm{WR}_{\mathrm{max}}$) than the PP (Table 2). The lactate/WR curve was nonlinear during IT in all the subjects. A $\mathrm{HR}_{\mathrm{DP}}$ could be determined for all PP (100%) and for 8 of the 11 NH (72.7%) during IT. The correlation between $\mathrm{WR}_{\mathrm{DP}}$ and AT_4 was significant in both groups (Fig. 3). The $\mathrm{WR}_{\mathrm{DP}}$ was significantly higher than WR at $4 \mathrm{mmol} \cdot 1^{-1}$ in NH, but not in PP.

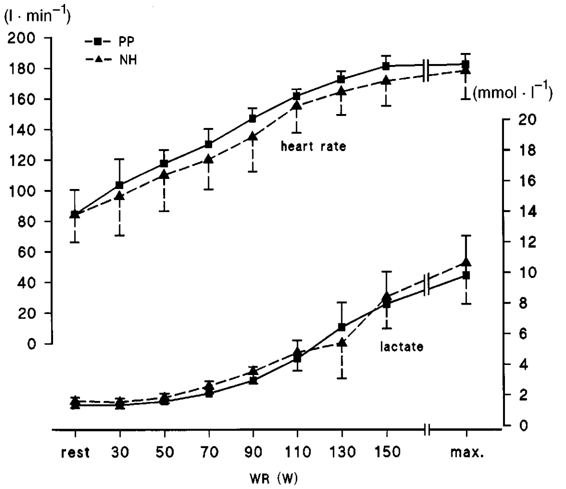

Fig. 2 Heart rate and blood lactate concentration relationships to work rate (*WR*) of paraplegics (*PP*) and non-handicapped persons (*NH*) in incremental arm cranking ergometry

The WR and blood lactate concentrations at DP were significantly lower in PP than in NH beats $\cdot \mathrm{min}^{-1}$. The average maximal HR in NH (with an identifiable DP was 8 beats $\cdot \mathrm{min}^{-1}$ higher than that of the NH without an established DP (182 vs 174 beats $\cdot \mathrm{min}^{-1}$). Lactate accumulation at the first level of SST in 6 PP (75%) and 5 NH (62.5%) led to a premature termination of the exercise (Fig. 4). Thus, the determination of $[\mathrm{La}^-]_{\mathrm{ss}}$ was only possible for 2 PP and 3 NH.

Discussion

In nonsteady-state incremental exercise tests a HR curve with a linear increase at moderate exercise intensities, and a levelling-off at higher and maximal intensities has been observed in NH (Brooke and Hamley 1972). The mechanisms by which these cardiovascular responses may be linked are still unknown. Lacking or even inverse deflections of HR in relation to WR have been reported (Heck et al. 1989; Pokan et al. 1993). This is sometimes explained by the methods that have been used, in particular the lack of attainment of complete exhaustion or differences in the test protocol, e.g. increasing cadence, exercise intensity, or the slope of the increase in exercise intensity (Conconi 1996; Jakob et al. 1987). However, a linear relationship or an inverse deflection of the HR curve can also be a normal biological finding.

In most studies left ventricular ejection fraction, as a parameter of myocardial function, has been shown to increase from rest up to submaximal intensities and to level off or even to decrease at higher intensities during incremental exercise (Hofmann et al. 1994b; Keul et al. 1982). As a possible explanation of the absence of DP, a

Table 3 Work rate and heart rate at $4 \mathrm{mmol} \cdot \mathrm{l}^{-1}$ anaerobic threshold and deflection point (WR_{AT4}, WR_{DP}, HR_{AT4}, HR_{DP}, respectively) and lactate deflection point (La^-_{DP}) of paraplegic (*PP*) and non-handicapped (*NH*) subjects in an incremental arm cranking test

	$\mathrm{WR}_{\mathrm{AT4}}$ (W)		$\mathrm{WR}_{\mathrm{DP}}$ (W)		$\mathrm{HR}_{\mathrm{AT4}}$ ($1 \cdot \mathrm{min}^{-1}$)		$\mathrm{HR}_{\mathrm{DP}}$ ($1 \cdot \mathrm{min}^{-1}$)		$\mathrm{La}^-_{\mathrm{DP}}$ ($\mathrm{mmol} \cdot \mathrm{l}^{-1}$)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
PP (*n* = 8)	108.9	20.2	118.7	15.2^{a}	147.9	5.3	166.5	9.2	5.30	1.40^{a}
NH (*n* = 8)	107.7	12.9^{b}	139.9	22.1	143.0	19.8^{c}	170.2	10.4	6.80	2.20

[a] $P < 0.05$ between *PP* and *NH*, [b] $P < 0.05$ between WR_{AT4} and WR_{DP}, [c] $P < 0.05$ between HR_{AT4} and HR_{DP}

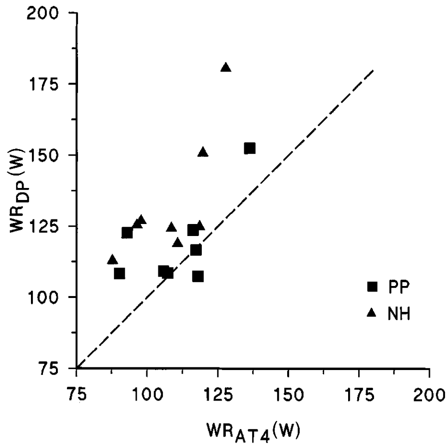

Fig. 3 Relationship between work rate at the deflection point of heart rate (WR_{DP}) and at the 4mmol · l^{-1} lactate threshold (WR_{AT4}) in incremental arm cranking ergometry. *PP* Paraplegics, *NH* non-handicapped persons; *dotted line*, line of identity

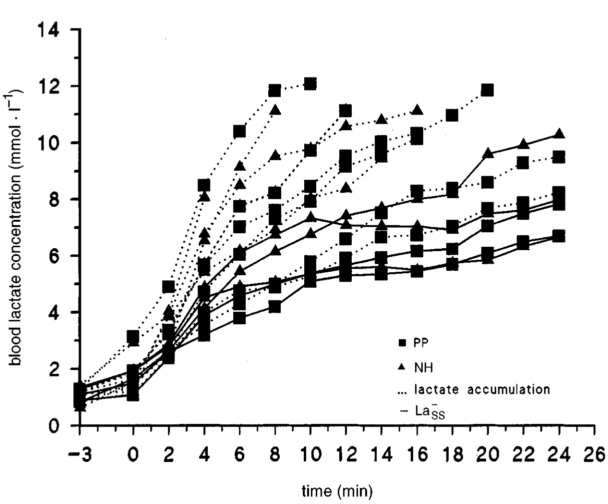

Fig. 4 Blood lactate concentration in the steady-state exercise test on an arm cranking ergometer (−3 to 0 min = warm up, 0 to 8 min = exercise intensity at the deflection point minus 10 W, 9 to 16 min = work rate at the deflection point, 17 to 24 min = work rate at the deflection point plus 10 W). *PP* Paraplegics, *NH* non-handicapped persons, $[La^{-}]_{ss}$ blood lactate concentration at steady state

decreased stress-dependent myocardial function with a diminution of the left ventricular ejection fraction has been observed around the anaerobic threshold in subjects without a deflection in HR during incremental exercise (Hofmann et al. 1994b; Pokan et al. 1993, 1995). Thus, the increase in the cardiac output would be regulated by the increase in HR resulting in a lack of levelling-off at higher intensities of exercise.

Possible reasons for the different responses of the cardiovascular system to incremental exercise have been shown to be the lactate buffer capacity related to muscle fibre composition, the metabolic capacities of skeletal muscles, skeletal muscle acidosis and the attendant drop in blood pH or the sensitivity of cardiac muscle, to catecholamines (Hofmann et al. 1994b). In other studies a poor reproducibility of DP under daily variations or varied nutrition has been described (Jones and Doust 1995; Thorland et al. 1994).

The results of these studies are only partially applicable to people with spinal cord injuries. Depending on the level of injury (aside from motor paralysis and loss of sensation), the impairment of the autonomic nervous system has been shown to affect different cardiovascular and metabolic exercise reactions due to, for example, impaired cardiac and muscle sympathetic innervation or blood pooling in the abdomen and lower limbs (Figoni 1992; Hopmann et al. 1993).

Exercise reactions from trained PP during arm cranking ergometry have been shown to be correlated to those of untrained NH by Davis and Shephard (1988) and Schmid et al. (1996). The higher WR_{max} of the NH in our study were mainly due to the difficulty, even with cooperation, in controlling and limiting extra activity of accessory muscles at maximal exercise intensities. It can be assumed that this had no real influence on the HR and blood lactate curve at submaximal and high exercise intensities.

In contrast to Melton et al. (1998), who have found no or an inverse HR deflection in PP, in our study a HR_{DP} curve was determined for all PP and for the majority of the NH (72.7%). Therefore it has been suggested that the linear relationship as well as the inverse deflection of HR may also be seen as a normal physiological reaction during non-steady state incremental exercise (Francis et al. 1989; Pokan et al. 1993).

The HR curves of PP show the same cardiovascular regulation mechanisms compared to NH even at maximal stress. It can therefore be assumed that in PP athletes with an injury level below T_5 the vast majority of cardiac sympathetic innervation is intact. Moreover, the DP found for all PP during nonsteady-state incremental exercise seems to suggest that decreased myocardial function, in comparison to NH does not occur.

In other literature a greater cardiovascular response and lactate release at a given $\dot{V}O_2$, as a result of a more prominent isometric component and an increased sympathetic nervous activity in arm exercise compared to leg exercise has been well documented (Bernard et al. 1997; Rowell and O'Leary 1990). In our study this had no recognizable influence on the existence of a DP for PP or NH. This corresponds to the findings of Krüger et al. (1988) on arm cranking exercise in NH.

According to Conconi et al. (1982) and other authors, HR_{DP}, if it exists, corresponds significantly to the start of lactate accumulation and shows a high reproducibility with various lactate and ventilatory thresholds (Ballarin et al. 1996; Bunc et al. 1995; Drogheti et al. 1985; Hofmann et al. 1994a; Zacharogiannis and Farrally 1991).

A lack of correlation (Sumsion et al. 1989; Tokmakidis and Leger 1992), a divergence of regression lines (Jakob et al. 1987) and a poor reproducibility (Jones and Doust 1995; Thorland et al. 1994) between

HR_{DP} and different lactate thresholds have been described in other studies. Thus the validity of this method has been questioned and doubts have been raised about a causal relationship between DP and anaerobic threshold. In our study, a significant correlation of WR_{DP} and AT_4 was found. Because HR_{DP} occurs at a particular percentage of maximal oxygen uptake, as do all kinds of thresholds, the correlation does not imply a unifying model for a physiological causal relationship.

The WR_{DP} in NH was significantly higher than AT_4 (32.2 W); no significant difference was found in these parameters for PP. This difference between the groups showed that while considering the comparable HR and lactate curve, a direct causal relationship between AT_4 and deflection of HR could not be established.

Steady-state arm cranking exercise led to lactate accumulation at 10 W below WR_{DP} obtained in the incremental test in 75% of PP and 62.5% of NH. Thus, WR_{DP} obtained in the incremental test in arm cranking ergometry did not correspond to the anaerobic threshold of $[La^-]_{max,ss}$, neither for PP nor for NH. Given the differences in HR and lactate dynamics during nonsteady-state incremental exercise, the relationship between HR data and lactate threshold would seem to be heavily dependent on the design of the individual test, e.g. the dynamics of the test signal.

Krüger et al. (1988) have found similar results with a wide range of lactate values in steady-state arm cranking exercise at selected percentages of WR_{DP} in NH persons. The relationship described in the current literature possibly indicates that a coupling exists of control mechanisms in the cardiovascular and metabolic systems. The increased sympathetic activity involves significant changes in many functional and biochemical parameters and it has been suggested that it could be an explanation for the link between DP and muscle metabolism (Bunc et al. 1995; Pessenhofer et al. 1991).

The intact cardiac sympathetic innervation preserved by spinal cord lesions below T_5 and the partial sympathetic denervation of the muscles, as well as the abdomen, especially the adrenal medulla, could explain the comparable HR but varying lactate concentration at the DP in PP and NH. Despite being a simple method, the determination of the anaerobic threshold by DP should be applied with caution to PP as well as to NH, since no causal relationship of DP and the anaerobic threshold was found and the WR_{DP} tended to overestimate threshold values, in agreement with the findings of other authors (Goodman et al. 1986; Jakob et al. 1987; Krüger et al. 1988; Kuipers et al. 1988; Tokmakidis and Leger 1992).

In summary, endurance-trained PP with a level of injury below T_5 showed HR and blood lactate concentrations comparable with NH control subjects during incremental arm cranking ergometry. A linear increase at moderate exercise intensities and a levelling-off (DP) at higher to maximal exercise intensities could be identified in all PP and in 72.7% of the NH subjects. No causal relationship of DP and the anaerobic threshold was found, the WR_{DP} tending to overestimate threshold values.

Further studies are necessary to investigate the mechanisms of cardiovascular regulation in spinal cord injured and NH persons during nonsteady-state incremental exercise and to determine the anaerobic threshold in PP with various levels of injury. Additional cardiovascular and metabolic changes may be expected as a result of the interruption of cardiac sympathetic innervation from the brain due to cervical lesion (see Figoni 1992; Schmid et al. 1998).

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