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► **To cite this version:**

Nicolas Tordi. Jacobs Journal of Physiotherapy and Exercise Evaluation of Autonomic Function after Concentric and Eccentric Cycling Exercise at Different Pedalling Frequencies. Jacobs Journal of Physiotherapy and Exercic, 2016. hal-04060269

HAL Id: hal-04060269

<https://univ-fcomte.hal.science/hal-04060269>

Submitted on 6 Apr 2023

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Jacobs Journal of Physiotherapy and Exercise

Research Article

Evaluation of Autonomic Function after Concentric and Eccentric Cycling Exercise at Different Pedalling Frequencies

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Received: 03-03-2016

Accepted: 09-02-2016

Published: 12-23-2016

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Abstract

Compared to concentric exercise, eccentric cycling is characterized by a lower cardiovascular and metabolic cost. However, its effects on the autonomic nervous system and the effects of pedalling frequency are little known. Eleven healthy subjects participated in this study. Cardiorespiratory variables were assessed during concentric and eccentric cycling performed with 3 different pedalling frequencies and post-exercise parasympathetic reactivation (heart rate recovery during the first 60 seconds of recovery and its normalized value; HRR60 and nHRR60) was studied. Cardiorespiratory responses were significantly lower during eccentric than concentric cycling. Heart rate recovery metrics were lower after eccentric than concentric cycling. In particular nHRR60 was 0.19 ± 0.11 vs 0.26 ± 0.11 , after eccentric vs concentric exercise at 30 revolutions per minute respectively; 0.13 ± 0.22 vs 0.28 ± 0.11 at 40 rpm and; 0.12 ± 0.14 vs 0.26 ± 0.10 at 60 rpm showing an attenuated post-exercise parasympathetic reactivation after eccentric exercise. While no change was observed during concentric exercise, increasing the pedalling frequencies lowered the cardiopulmonary solicitation during eccentric cycling. A trend for a delayed post-exercise parasympathetic reactivation was also observed when the pedalling frequency increased ($p = 0.057$). These results highlighted that eccentric cycling imposed a greater strain on the autonomic nervous system than concentric cycling at the same power output despite a lower cardiorespiratory requirement.

Keywords: Heart Rate Recovery; Parasympathetic Reactivation; Cardiorespiratory Requirement

Abbreviations:

ANS: Autonomic Nervous System;

CON: Concentric;

ECC: Eccentric;

EEHR: End of Exercise HR;

HR: Heart Rate;

HRR: Heart Rate Recovery;

HRR60: HR Recovery during the first 60 sec of Recovery;
nHRR60: normalized HRR60 metric;
PNS: Parasympathetic Nervous System;
rpm: revolution per minute (pedalling frequency unit);
SNS: Sympathetic Nervous System;
T30: Time constant for the short-term post-exercise decay in HR;
 VCO_2 : Absolute Volume of Carbon dioxide released ($ml.kg^{-1}$);
VE: Respiratory Minute Volume;
 VO_2 : Absolute Volume of Oxygen uptake ($ml.kg^{-1}$);
W: Watts ($J.s^{-1}$)

Introduction

Eccentric (ECC) exercise describes exercise where a muscular contraction is executed while muscle fibres are lengthening. ECC cycling is a training modality often used in the fields of sport performance and trauma rehabilitation [1]. Briefly, previous studies have shown that for the same power output, ECC exercise elicits less central solicitations (e.g. lower oxygen uptake [VO_2], heart rate [HR] and cardiac output) than CON exercise [1,2]. This interesting property explains the recent and growing interest for ECC prescription in patients with chronic health conditions [3,4]. But ECC cycling is still underutilized in the health field potentially for two main reasons. First, ECC exercise can be easily traumatizing, triggering delayed onset muscle soreness and inflammation [5,6]. Secondly, there is a lack of knowledge and understanding of the effects ECC exercise compared to concentric (CON) exercise on metabolic and cardiovascular functions, and on the autonomic nervous system (ANS) balance.

Cardiovascular function, including HR, is modulated by the parasympathetic (PNS) and sympathetic (SNS) branches of ANS. During exercise, both metaboreflex and mechanoreflex are activated resulting in an increased HR, due to withdrawal of PNS and an increase in SNS activity [7]. When the exercise stops, SNS activity decreases and PNS is engaged to restore homeostasis. During CON cycling, ANS activity seems mainly influenced by metaboreflex, in accordance with the slower HR restoration by ANS when metabolic demand is greater [8]. The impact of ECC exercise on the regulation of the cardiovascular function is not well defined but logic dictates a differential response than during CON exercise because of the distinct metaboreflex and mechanoreflex involved. Indeed, a higher SNS activity has been observed during ECC exercise, likely due to the higher muscle tension developed at the same metabolic cost [9]. The same research team underlined the lower dependence of the cardiovascular adjustments to the metabolic demand as well as an increase in SNS activity in muscle, which could suppress PNS reactivation after exercise [1]. These results provide interesting knowledge but as only a 60 and 80 rpm pedalling frequency were studied, any frequency effect on cardiorespiratory variables and ANS control needs to be assessed especially at low frequencies commonly used for clinical purpose [10]. It should also be noted that differences in

the physiological adaptations observed between ECC and CON cycling for a given pedalling frequency are significantly conditioned by the internal work, which would be strongly different according to the frequency for a same power developed [11]. In addition, higher movement frequency is more traumatizing for muscles during elbow ECC exertion [12] and can lead to lead to long-lasting neuromuscular dysfunction [13]. Overall, these results suggest that muscle tension, delayed onset muscle soreness, and cardiorespiratory adaptations to ECC cycling are a function of pedalling frequency and power developed but these observations need to be investigated, especially with regard to the ANS involvement.

As ECC has been recommended as an exercise modality for patients with chronic diseases that contraindicate excessive workloads such as chronic obstructive pulmonary disease [14], its effects and physiological outcomes should be better understood. The aim of the present work was thus to assess the cardiorespiratory and ANS responses to ECC cycling at different pedalling frequencies. To do this, we compared the cardiorespiratory and post exercise PNS reactivation during ECC and CON exercise performed at the same power output at three different pedalling frequencies. Based on the literature, we hypothesized that a lower cardiopulmonary involvement during ECC exercise would be observed together with a delayed PNS reactivation after exercise, at each pedalling frequency.

Material and Methods

Participants

Eleven healthy subjects (no cardiovascular, pulmonary or metabolic chronic diseases, neither musculoskeletal disorders) with a mean age of 25 ± 2 years (mean \pm SD – standard deviation), 2 women (172 ± 1 cm, 61 ± 2 kg) and 9 men (184 ± 8 cm, 77 ± 6 kg), volunteered to participate in the study. They were non-smokers, and they were not taking any medication that could affect our measurements. They participated in a sport at least four hour per week: cycling, athletics, swimming or European football. All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional research committee and the 1964 Declaration of Helsinki. Informed consent was obtained from all individual participants included in the study.

Measures

Ergometer

The ergometer used in this study was a reproduction of the Abbott model [15], two back-to-back bicycles connected by a single chain: since subject A was pedalling “forward” (CON) while subject B was pedalling “reverse” (ECC), they necessarily cycled at the same frequency and force development. The pedalling frequency and the power output were checked and recorded on line by a SRM device (SRM, PowerControl, Jülich, Germany).

Exercise Parameters

Pulmonary gas exchange (VO_2 , VCO_2) and respiratory minute volume (VE) were measured using breath by breath K4b² technology (COSMED, Rome, Italy) calibrated following the manufacturer's recommendations. We also collected beat by beat heart rate data (R-R intervals) using a Polar S810i HR monitor (Polar Electro, Kempele, Finland). Measures were carried out continuously during warming up, CON and ECC exercise bouts, and recovery periods. Respiratory breath-by-breath parameters and HR were averaged over 30 seconds of each bout.

Heart Rate Recovery Metrics Calculation

Three Heart Rate Recovery (HRR) indices were calculated to assess PNS post-exercise reactivation.

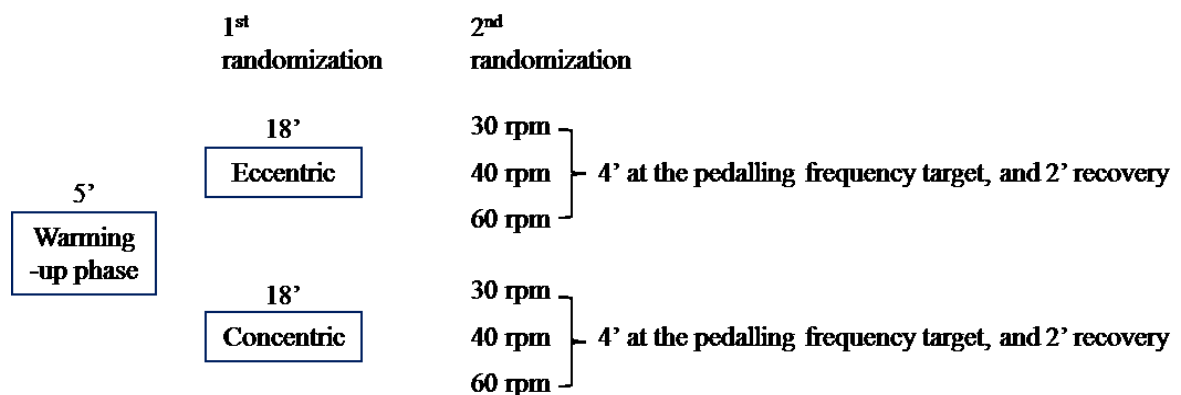


Figure 1. Detailed pedalling protocol

First the absolute difference between the average HR measured during the first 5 sec of the end of exercise (EEHR) and the average HR recorded during the ten last seconds of the first minute following exercise end (HRR60s). Second, a normalization of the first metric was applied (nHRR60s) by dividing HRR60s by the average immediate post-exercise HR, to take into account differences in the HR at the end of exercise.

Finally, a third metric was obtained using semi-logarithmic regression. The natural logarithm of the HR during the interval 10-40 s after exercise was plotted against the length of the period of exercise and linear regression analysis then applied. The negative reciprocal of the slope of this regression line represented the time constant for the short-term post-exercise decay in HR (T30) [16]. Heart rate recovery indices such as HRR60 and T30 are commonly used in the assessment of PNS activity in both health and disease because of their good reliability especially after submaximal exercise [17].

Procedures

Exercise testing on the cycle-ergometer started by 5 minutes of warming up. Then each participant performed two sessions (Figure 1) of either CON or ECC cycling, in a randomised fashion. Each session lasted a total of 18 minutes divided in 3 bouts

of 6 minutes: 4 minutes at 100 Watts followed by 2 minutes of passive recovery in the sitting posture on the cycle ergometer. The three cycling bouts were performed at 30, 40 or 60 rpm in a random order.

Participants were asked not to perform any strenuous exercise or drink alcohol during the 48 hours before their visit. Coffee, tea, energizing drink and food were prohibited during the 5 hours preceding exercise. The laboratory has controlled conditions of temperature, pressure and humidity for human experimentation (20°C, 765 mmHg, 50% relative humidity).

Statistics

Data are presented as Mean \pm SD unless otherwise stated. Data analysis was conducted on SigmaStat (Systat Software Inc, San

Jose, CA, USA). A Two Way Repeated Measures ANOVA (pedalling frequency vs. contraction mode) was applied with Equal Variance Test, to assess differences in each of exercises having first checked, using the Shapiro-Wilk test, that there was no evidence to suggest that these changes violated normality. The Student-Newman-Keuls procedure was applied to perform post-hoc pair-wise comparisons employed for data analysis. Statistical significance was set at $p \leq 0.05$.

Results

Responses to Exercise

The results obtained (Figure 2) showed that HR, VE , VO_2 and VCO_2 were significantly lower during ECC than CON cycling exercise, for the three pedalling frequencies and similar mechanical power developed (102 ± 4 W).

Also, an interaction between exercise mode and pedal frequency was observed with the effect of pedalling frequency being significantly different according to the contraction mode (Figure 2). No pedal frequency effect was observed during CON exercise. On the contrary, a significant lowering of VE , VO_2 , VCO_2 , and HR was observed with the increase in frequency during ECC exercise.

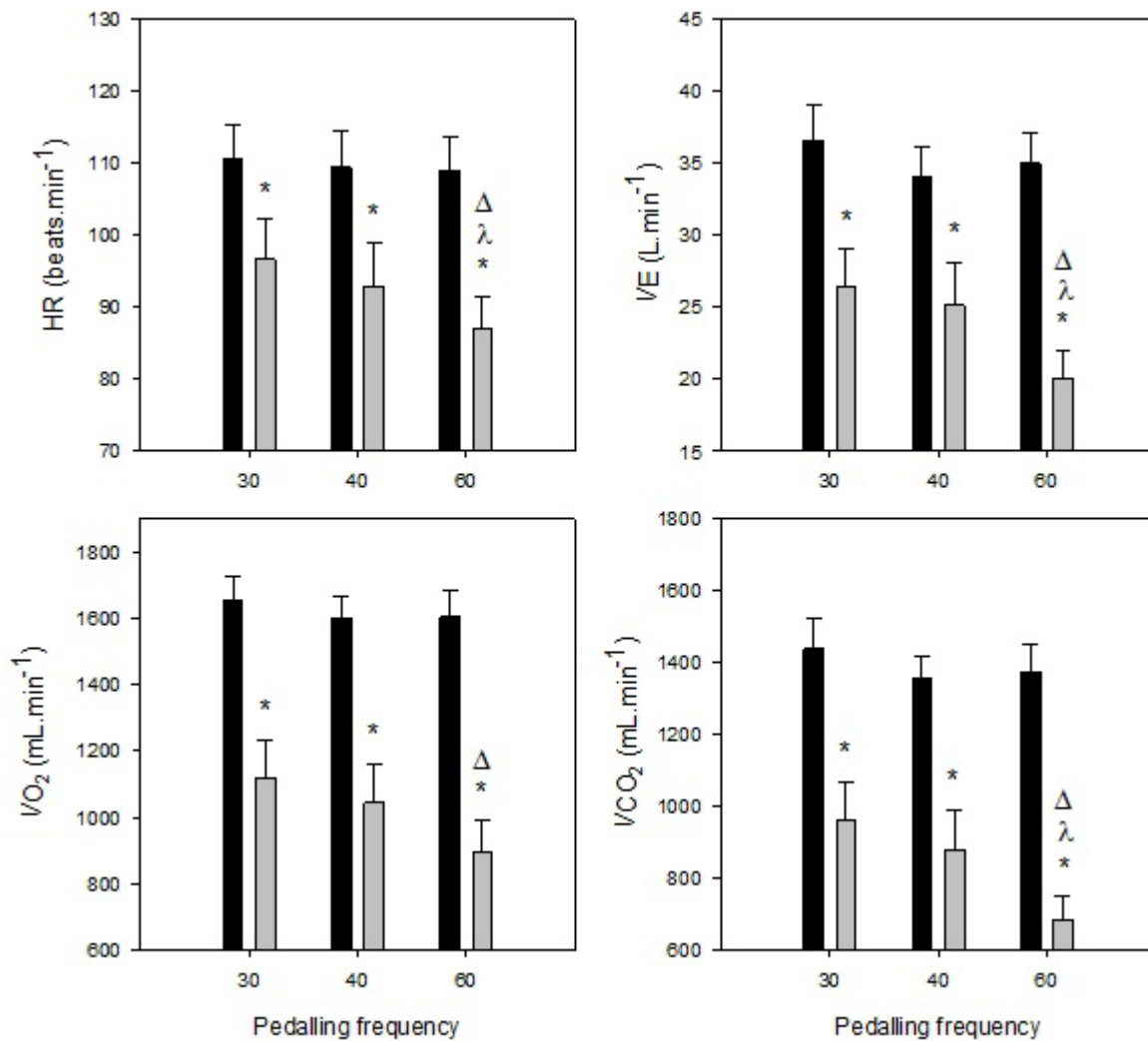


Figure 2. Heart rate (HR), ventilation (VE), oxygen uptake (VO₂) and carbon dioxide release (VCO₂) during the three tested pedalling frequencies for the CON (black bars) and ECC (grey bars) exercises. Values are presented as mean ± SEM.

* = significant difference between CON and ECC exercise ($p < 0.05$)

Δ = significant difference between 30 and 60 rpm ($p < 0.05$)

λ = significant difference between 40 and 60 rpm ($p < 0.05$)

Post-Exercise PNS Reactivation

EEHR, HRR60 and nHRR60 were significantly lower at the end of ECC compared to CON exercise regardless of pedalling frequency (except for nHRR60 at 30 rpm; Figure 3). T30 metric showed no significant differences between ECC and CON exercises.

No pedalling frequency effect was observed for CON exercise. During ECC exercise, a significant lowering of EEHR with the increase of the pedalling frequency was observed (Figure 3), but no effect was found for T30, HRR60 and nHRR60 metrics.

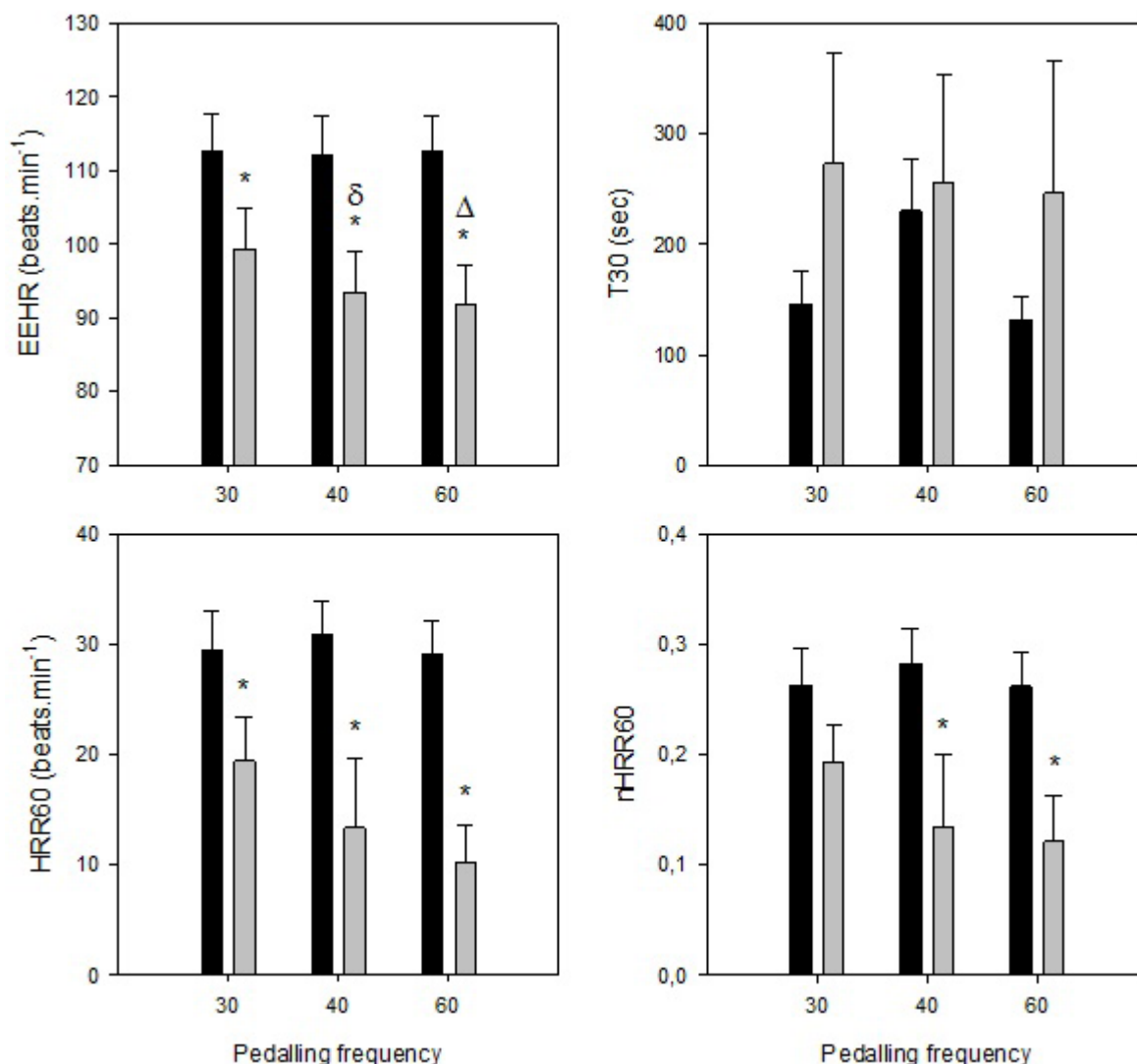


Figure 3. Heart rate (HR) at the end of exercise (EEHR), absolute and normalized HR recovery during the first 60 sec of recovery (HRR60 and nHRR60) and time constant for the short-term post-exercise decay in HR (T30) for the CON (black bars) and ECC (grey bars) exercises. Values are presented as mean ± SEM.

* = significant difference between CON and ECC exercise ($p < 0.05$)

δ = significant difference between 30 and 40 rpm ($p < 0.05$)

Δ = significant difference between 30 and 60 rpm ($p < 0.05$)

Discussion

As hypothesized, lower cardiorespiratory responses to exercise were observed as well as a delayed post-exercise PNS reactivation during ECC cycling when compared to CON exercise at the same power output. Increasing the pedalling frequency also lowered the cardiopulmonary responses as well as HRR60 and nHRR60 during ECC cycling, while no significant changes were observed during CON cycling.

For a given mechanical power output, ECC exercise is known to evoke lesser cardiovascular and respiratory responses [14,15,18]. Our results are in line with these previous researchers with cardiopulmonary values (HR, VE, VO_2 and VCO_2 ; Figure 2) being significantly lower during ECC exercise at 100 W at each pedalling frequency. Importantly, the results of the present study, to our knowledge, represent the first attempt to specifically compare the autonomic function after CON and ECC cycling performed at the same intensity with different pedalling frequencies.

After submaximal CON pedalling exercises PNS reactivation and SNS activity inhibition are observed, resulting in a quick decay of HR to resting values [19]. It is known that during CON exercise, both metaboreflex and mechanoreflex are stimulated [7] with a larger contribution of metaboreflex to HR stimulation [9]. Indeed, post-exercise PNS reactivation is dependent on the intensity of the exercise [17] with a delayed PNS reactivation after high intensity compared to low intensity exercise [20]. For instance, it has been demonstrated that CON exercises with a large anaerobic contribution (i.e. when the chemoreflex and metaboreflex are involved) elicited a SNS post-exercise predominance and slow HR recovery [21]. On the contrary, the effects of ECC exercise on ANS post-exercise are little studied. Here we show a significant difference between ECC and CON cycling on post-exercise PNS reactivity despite the same absolute power output (Figure 3). In line with the lower cardiopulmonary strain during ECC exercise, we observed a lower exercise ending heart rate irrespective of the pedalling frequency (Figure 3). However, heart rate recovery, as evidenced by HRR60, was lower after all pedalling frequencies ECC exercise. This result suggests an attenuated PNS reactivation. To ensure that the difference was not due to methodological aspects (i.e. due to the index used to assess PNS activity) a normalized heart rate recovery, nHRR60 was calculated [22]. Lower values of nHRR60 after ECC compared to CON were still observed at all pedalling frequencies (Figure 3) demonstrating that the delayed PNS reactivation is not dependent on the post-exercise HR but on the exercise modality. It is of note that the T30 metric was not significantly different between the two exercise modalities, even though a trend for an increase, suggestive of a delayed PNS reactivation, was observed after the ECC exercise.

Taken together, these results suggest that the ANS activity after

ECC exercise is perturbed and PNS reactivity is delayed at each of the pedalling frequencies investigated. Although metaboreflex should be superior to mechanoreflex in eliciting circulatory responses during CON cycling, the mechanoreflex could explain specific circulatory adjustments observed during ECC cycling (equal VO_2 , but higher cardiac output and HR) [23]. The greater level of exercise-induced muscle mechanical stimulation during ECC exercise is likely due to the higher tension developed by each muscular fibre for a same power output [1] and the strong muscle mechanoreflex activation could impact PNS reactivation [7]. The attenuation of the PNS has two important corollaries. Firstly, an elevation in resting metabolic rate post eccentric exercise could have implications for exercise programming prescription for weight loss. Indeed, other authors [24,25] have reported elevations in resting metabolic rate post eccentric exercise but have not been able to compare and contrast the effects with concentric exercise due to their experimental designs. Secondly, it should be noted that “transient, unfavorable changes in central macrovascular function” have been reported following eccentric exercise [5]. Elevated vascular resistance have been reported [26] and “adverse effects” in an eccentric training group that led to 2/15 participants failing to complete the experimental protocol despite training at RPEs of 9-11 [3]. “Autonomic imbalance” has also been reported following eccentric training in elderly men with a shift towards SNS dominance and cardiovascular metrics associated with increased morbidity and mortality [27].

Our second aim was to evaluate the effect of pedalling frequency on cardiopulmonary and PNS responses to ECC cycling. During CON cycling, a U-shaped relationship has been shown between VO_2 and pedalling frequency describing an optimum pedalling frequency Barstow et al. (1996) that could be explained by a better elastic energy return with faster pedalling frequency. Our results obtained during a steady-state exercise using a constant power demonstrated a similar VO_2 at each of the pedalling frequencies observed. But the moderate power output (100 W) and low pedalling frequencies used (30, 40 and 60 rpm) may explain why we did not observe a similar response between VO_2 and pedalling frequency during CON cycling. Indeed, the pedalling frequencies used in the present study were slow compared to those usually used during CON exercise studies [28,29], but they were comparable to those used for ECC cycling [4,9,10]. We chose to use a maximal pedalling frequency of 60 rpm in order to avoid muscles traumas and a minimum pedalling frequency of 30 rpm to allow lower limb coordination during the ECC cycling protocol. As preliminary tests showed that with our tandem ergometer, a 15 rpm pedalling frequency induces coordination problems for subjects and difficulties to control their power output. Furthermore, it can be noted that coordination was perceived more difficult by participants at 30 rpm than 60 rpm, probably due to the greater muscle tension (lower frequency so higher force for a same power output) elicited during 30 rpm pedalling.

In contrast to CON cycling, changing the pedalling frequency during ECC cycling elicited significantly different physiological responses. Indeed, the higher the pedalling frequency was, the lower the cardiopulmonary strain observed (HR, VE, VO₂ and VCO₂). This relation has already been described by Wells et al. (1986) with internal work, showing a greater VO₂ during ECC cycling at 30 rpm than 60 or 90 rpm. Chung et al. (1999) used eccentric at 35, 55 and 75 rpm, and their results revealed the same trend with the lowest HR and VO₂ observed at 55 rpm (i.e. comparable to our 60 rpm frequency). Our results (i.e. diminution of VO₂ when pedalling frequency increase) do not allow us to conclude about an optimum pedalling frequency during ECC cycling due to the range of pedalling frequencies examined, but it is clear that the optimum pedalling frequency during ECC cycling differs from CON exercise as suggested. With respect to ANS responses across the different pedalling frequencies, there were no significant differences in HRR60 and nHRR60 within CON cycling. In contrast, after ECC cycling we observed the same trend as for cardiovascular variables (i.e. the higher the pedalling frequency, the slower the heart rate recovery), although differences did not reach statistical significance ($p = 0.057$ for HRR60, 30 vs 60 rpm). We could hypothesize that with faster movements, the involvement of muscle mechanoreflex is more important leading to a delayed HR recovery (HRR60 and nHRR60 decrease). Another explanation could be related to the coordination difficulties experienced when pedalling at 30 rpm. The central command activation during exercise is known to impact ANS activity [30]. Constraints in neuromotor control during slow pedalling ECC exercise could lead to greater central command activity and subsequent post-exercise ANS perturbations.

Conclusion

Our results confirmed that ECC cycling involved a lower cardiopulmonary strain when compared to concentric exercise at the same power output. ECC exercise induced an attenuated PNS post-exercise reactivation likely due to greater mechanoreceptors involvement. This delayed parasympathetic activation may have important ramifications considering the low energy expenditure; this highlights potential contraindications in patient populations. Furthermore, here we demonstrate that changing pedalling frequency between 30 to 60 rpm does not alter the cardiovascular or ANS responses during CON cycling but has a significant impact on analogous responses during and after ECC cycling. Other research should be conducted to confirm our results, especially with different power output and pedalling frequencies, and to study the long-term effects of ECC training on ANS in healthy subjects and patients.

Acknowledgements

This study was supported by the Exercise Performance Health Innovation platform (Franche-Comté University, Besançon, France) with the valuable help of Catherine Capitan who han-

dled logistical matters. The authors wish to thank the participants for their time and cooperation.

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