

## Cardiovascular Responses Between Low Cadence/High Force vs. High Cadence/Low Force Cycling

RANDY G. CANIVEL<sup>†1</sup>, and FRANK B. WYATT<sup>‡2</sup>

<sup>1</sup>Department of Applied Physiology/Wellness, Southern Methodist University, Dallas, TX, USA; <sup>2</sup>Department of Athletic Training & Exercise Physiology, Midwestern State University, Wichita Falls, TX, USA

<sup>†</sup>Denotes graduate, <sup>‡</sup>Denotes professional author

---

### ABSTRACT

*International Journal of Exercise Science 9(4): 419-426, 2016.* The purpose of this study was to determine the cardiovascular responses during sustained power output comparing low cadence (LC) high force (HF) vs. high cadence (HC) Low force (LF) cycle ergometry. Nine participants (N = 9) volunteered for this study. Participants signed a Human Participants consent form. A power output of 150 watts (W) for 20 minutes was estimated as the sustainable workload. Participants were instructed to pedal steadily at 50 rpm (LC) or 100 rpm (HC) with resistance adjusted to sustain 150 W. The following measures were obtained each minute during the 20-minute protocol: average heart rate ( $b \cdot \text{min}^{-1}$ ), blood pressure (mmHg) and Rate Pressure Product (i.e.  $\text{RPP} = (\text{SBP} \cdot \text{HR}) \cdot 100^{-1}$ ). Randomization was used to counterbalance both protocols (HC vs. LC) and demographic controls (N=9). Means and standard deviations (SD) were determined for age ( $36 \pm 13.64$  years), resting heart rate ( $68.83 \pm 11.95$   $b \cdot \text{min}^{-1}$ ), resting blood pressure ( $126.42 \pm 13.27$  mmHg), body fat percentage (male:  $14.7 \pm 4.3$  %; female  $20.6 \pm 1.3$  %) and height ( $157.80 \pm 10.04$  cm). A Repeated Measures Analysis of Variance (RM-ANOVA) was utilized to compare exercising values between test protocols. A Pearson Product Correlation Coefficient was utilized to determine bivariate associations between variables. A Tukey Post Hoc analysis was performed to analyze differences in LC HR and RPP. Statistical significance was set *a priori* at  $p \leq 0.05$ . There were statistical differences among LC  $\pm$ HR ( $130.51 \pm 3.36$ ), HC HR ( $150.83 \pm 6.49$ ), LC RPP ( $204.63 \pm 11.45$ ), and HC RPP ( $245.57 \pm 25.70$ ) Between the HC and LC protocols, the use of HC protocol elicited an increase in HR and RPP.

**KEY WORDS:** Heart rate, systolic blood pressure, rate pressure product, cycle ergometry, hemodynamics

### INTRODUCTION

Power output through cycle ergometry may be accomplished in one of two ways: 1) by utilizing a low gear and high cadence (HC) output; or 2) through high gear and low cadence (LC) output. Resistive forces, metabolic pathway considerations, and Type I or Type II muscle fiber recruitment may differ based on these two choices (1).

While one may utilize a slow twitch, oxidative pathway, the other may use a fast twitch, glycolytic contribution for energy production. Stebbins et al. tested competitive male cyclists ( $35 \pm 2$  yrs.) for 180 minutes at either 80 or 100 rpm on varying intensities based on percent of maximal oxygen capacity (20). They noted no differences between cadence in blood glucose, respiratory exchange ratio (RER),

or rating of perceived exertion (RPE). However, they did find differences in the efficiency of total energy expenditure (i.e., oxygen cost per work output) and maximal power attained. They concluded that the higher cadence (100 rpm) was less efficient than the lower cadence (80 rpm) (19). Yet within this study, the authors may have overlooked consistencies in work output and comparisons of cadence vs. force for the same power output. Thus, fiber recruitment patterns, enzymatic up-regulation, vaso-activity, and cardiovascular parameters may have differed.

Moore et al. investigated the myocardial parameters of heart rate (HR), cardiac output (CO), oxygen utilization ( $\text{VO}_2$ ) and tissue oxygen extraction ( $a\text{-vO}_2 \Delta$ ) (12). Similar to the previous study (1), the authors utilized 80 and 100 rpm as LC vs. HC. Their findings indicated no differences in stroke volume between cadences, yet CO and HR were higher when cyclists were working at lower oxygen demands (i.e., 50%  $\text{VO}_2\text{max}$ ). Additionally, Moore and colleagues concluded that oxygen extraction was facilitated at higher work demands (i.e., 65%  $\text{VO}_2\text{max}$ ) regardless of cadence (12). One inherent issue related to the previous studies was the range of discrepancy between cadences (2, 11, 19). One could argue that differences between 80 and 100 rpm may not be adequate to elicit changes in muscle recruitment patterns, bioenergetic processes or a combination of the two. Thus, cardiovascular alterations between cadences of minimal range could be negligible.

Sarre and Lepers used a large range of cadences at 50 rpm and 110 rpm while

looking at neuromuscular function during sustained cycling efforts (19). The authors noted differences in muscle activation with higher cadences but phasic (i.e., muscle bursts) aspects were not different. They concluded that nervous system activation required by the different cadences were not different but adaptations (i.e., alterations) to sustained efforts were altered with neuromuscular fatigue (19).

In an earlier study, Merrill and White used three different pedal cadences at a constant workload (11). The investigators randomly assigned cadences at 70, 95 and 127 rpm with a constant workload eliciting approximately 70% of their  $\text{VO}_2\text{max}$ . They found that cardio-respiratory demands were greatest in the high cadence setting. In addition, muscular efficiency was lowest in the high cadence pedal rates indicating metabolic insufficiencies by higher pedal rates (11). Compensatory mechanisms of cyclists through metabolic, neuromuscular or cardio-respiratory may exist when sustained power output demands are warranted (2, 11, 18). Alternate gearing choices and cadence during competition may not always benefit a cyclist, hence, leading to early onset of fatigue. The emphasis of past studies have centered on cardiovascular considerations as they relate to oxygen delivery and utilization (4, 11, 19, 20, 21). To the best of the authors' knowledge, past research has not investigated myocardial work per se during sustained power output with substantially different cadence patterns. Consequently, the purpose of this study was to determine the following cardiovascular responses: heart rate, systolic blood pressure (SBP) and Rate Pressure Product (RPP) on sustained power during Low Cadence/High Force

(LCHF) versus High Cadence/Low Force (HCLF) cycle ergometry.

## METHODS

### *Participants*

Nine participants (7 males and 2 females) volunteered for this study. Inclusion criteria included the following: healthy males or females (determined through health questionnaire) age 21-55 years, all participants had cycling experience of 5+ years or were a licensed competitive cyclist with *United States Cycling Federation* Category level 3 or 4. Exclusion criteria included participants who resistance train on a weekly basis, and individuals who were symptomatic based on *The American College of Sports Medicine Risk Stratification* (14). Controls on physiological inclusion (i.e., healthy, trained, years of training, competitive) were primary criteria excluding age and gender.

This study utilized a controlled-crossover design in which low vs. high cadences were examined with high force vs. low force, respectively. This was to allow for a sustained equal power output throughout the testing process. Applying a Latin Squares Design, participants were randomly assigned to a "Low" cadence trial and "High" cadence trial (10). After random placement into the first test procedure, participants were allowed an adequate recovery time of 48 hours then performed the alternative procedure (12). In order to quantify and compare results between the test procedures, minute-to-minute cardiovascular measures were taken (e.g. HR, SBP, and RPP). These cardiovascular responses were measured to determine interactions of myocardial function associated with RPP. Alterations in

these cardiovascular variables would indicate acute myocardial adjustments to altered demands of the two cadence-resistance protocols.

All cyclists were tested in a climate controlled exercise laboratory. The participants were encouraged not to alter (i.e. reduce or add) their current food or fluid intake, and to abstain from training between tests (12,13). In an effort to control any cardiovascular stimulation, participants were asked not to ingest any caffeinated products within three hours of their participation (9). Two days prior to testing, each participant was required to visit the exercise lab, sign an informed consent document approved by the Midwestern State University Human Participants Board, a physical readiness questionnaire (PAR-Q), and a medical-health questionnaire (14). In addition, a familiarization process was conducted with all participants on testing procedures.

Each participant was scheduled within a two-day time frame for testing during the same week. To reduce the possibility of any circadian and/or diurnal effects, all participants reported for their testing on both days at the same time (11,12). Test measures included the following: resting blood pressure (auscultation method), body fat percentage (%) through skin-fold measures (Harpenden™, United Kingdom), height (cm), body mass (kg), and resting heart rate through electronic telemetry (Polar™, Finland). Measurement of body fat was calculated using the Brozek equation:  $((4.570 \cdot Db^{-1}) - 4.142) \cdot 100$ , where Db is body density (6).

A Monark™ 850 (Sweden) cycle ergometer was used and calibrated for each

## CARDIOVASCULAR RESPONSES TO HIGH VS. LOW FORCE CYCLING

participant prior to each test. For each participant, appropriate knee flexion for power stroke was measured at 10 degrees with the use of a goniometer. Individual handlebar and other bike adjustments were marked and recorded for consistency between tests. Participants were allowed to use cycling cleats and pedals at their discretion, but for consistency purposes, participants were instructed to use them on both test days. On both trials, no psychological encouragement was given during the exercise.

### Protocol

After resting measures and bike adjustments were obtained, participants were allowed five (5) minutes of warm-up at a self-selected cadence and workload. A set power output of 150 watts for 20 minutes was estimated to be sustainable for all participants in order to obtain acute cardiovascular responses. In order to provide a greater degree of external validation, an absolute value for power output was utilized, in contrast to relative values based on body weight. Based on the selected protocol, participants were instructed to pedal steadily at 50 rpm or 100 rpm. Exercise measures were taken every minute and included the following: HR ( $b \cdot \text{min}^{-1}$ ), SBP (mmHg) and RPP ( $\text{RPP} = [\text{SBP} \cdot \text{HR}] \cdot 100^{-1}$ ). For reliability, all pre-exercise and exercise measures for (i.e. anthropometric and cardiovascular) were taken at the same anatomical site by the same technician.

### Statistical Analysis

Descriptive statistics (mean  $\pm$  standard deviation) were used to establish group age (y), resting ( $b \cdot \text{min}^{-1}$ ), resting blood pressure (mmHg), body fat percentage (%), body mass (kg) and height (cm) (Table 1).

Repeated Measures Analysis of Variance (RM-ANOVA) was utilized to compare exercising values between test protocols. In addition, a Pearson Product Correlation Coefficient was utilized to verify any bivariate associations: exercising HR, SBP and RPP. A Tukey Post Hoc analysis was performed to determine if any minute-to-minute differences occurred in cardiovascular parameters. Statistical significance was set *a priori* at  $p \leq 0.05$ .

**Table 1.** Descriptive mean (SD) values during rest and cycle ergometer testing.

Descriptive Statistics and Mean (SD) Values During Cycle Ergometer Testing	MEAN	STD. DEV.
Age (y)	36.00	13.64
Height (cm)	157.80	10.04
Weight (kg)	80.91	14.01
LC HR ( $b \cdot \text{min}^{-1}$ )	130.51	3.36
HC HR ( $b \cdot \text{min}^{-1}$ )	150.83**	6.49
LC SBP (mmHg)	156.66	5.17
HC SBP (mmHg)	166.05**	5.60
LC RPP	204.63	11.45
HC RPP	245.57**	25.70
LC RPE (6-20)	11.72	0.60
HC RPE (6-20)	12.98	1.05

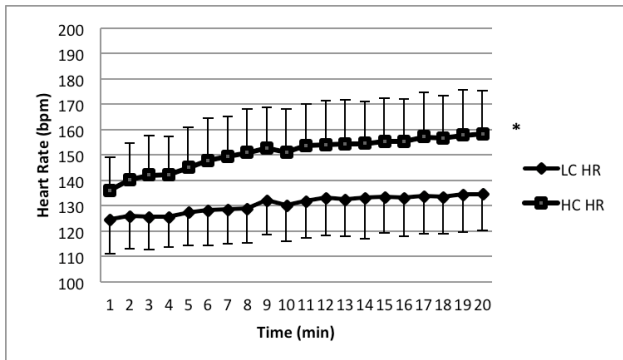
LC= Low Cadence; HC = High Cadence; HR=Heart Rate; SBP = Systolic Blood Pressure; RPP = Rate Pressure Product; RPE = Rating of Perceived Exertion; \*\*Statistically different at  $p < 0.05$ .

## RESULTS

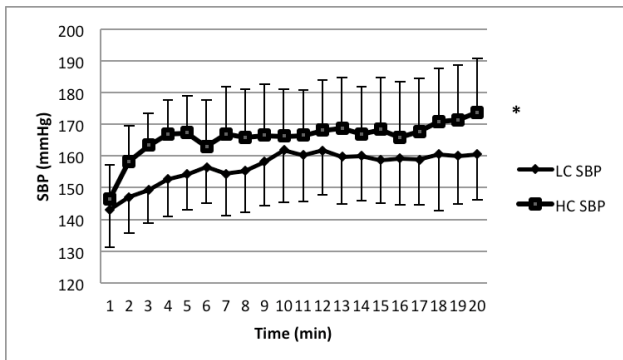
Descriptive statistics including mean (SD) values for all nine (N=9) participants and their ergometer results (e.g. HR, SBP, and RPP) between the two protocols are shown in Table 1; asterisks note statistical differences. Figures 1, 2, and 3 illustrate

# CARDIOVASCULAR RESPONSES TO HIGH VS. LOW FORCE CYCLING

fluctuations in the cardiovascular measures during the 20-minute steady rate at 150 W.



**Figure 1.** Heart rate comparison. LC- (Low Cadence) Heart Rate (HR), HC- (High Cadence) Heart Rate (HR), \*= significant difference between response,  $p < 0.05$ .

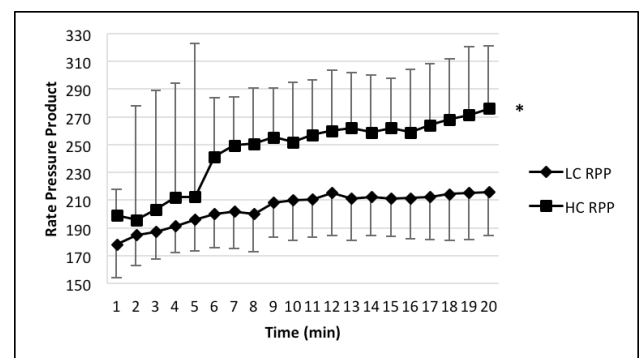


**Figure 2.** Systolic blood pressure comparison. LC- (Low Cadence) Systolic Blood Pressure (SBP). HC- (High Cadence) Systolic Blood Pressure (SBP). \*= significant difference in response,  $p < 0.05$ .

## DISCUSSION

Results from the current study indicate that average HR, SBP and RPP showed a significant increase ( $p < 0.05$ ) when participants utilized the HC protocol compared to the LC protocol during an equal, sustained workload. The findings in this study suggest that future investigations of sustained power output specifically examine chronotropic and inotropic characteristics of the heart. In addition, vaso-activity, hemodynamics, baroreceptor influence, and the exercise-pressor reflex

(i.e. mechanoreceptor and metaboreceptor) are also warranted. During extended steady state work periods, faster, lower body muscle contractions during each cycling pedal stroke (i.e., high cadence) reduce relaxation periods leading to compromised blood flow to the involved leg muscles (2, 21). This pulsatile blood flow, during extended time periods leads to an increase in vascular pressure and subsequent lack of adequate tissue oxygenation (i.e., localized hypoxia). In this study, the high cadence protocol elicited higher heart rates, reduced ventricular filling time, and facilitated an upward rise in heart rate to meet tissue demands during sustained efforts (12, 21).



**Figure 3.** Rate pressure product comparison. LC- (Low Cadence) Rate Pressure Product (RPP). HC- (High Cadence) Rate Pressure Product (RPP). \*= significant difference in response,  $p < 0.05$ .

This study's application of a high cadence cycle ergometry demonstrated that HR, SBP, and RPP were greater compared to low cadence work performed at the same power (W) output. At the onset of exercise, central command and efferent nerves initiate the rise in myocardial and blood pressure responses (4, 14). During a HC protocol, the continued modulation of the cardiovascular system stems from interaction of the arterial baroreceptors and muscle metaboreflex generating an enhanced pulsatile blood flow (8). An enhanced pulsatile flow facilitated by the

HC protocol, triggers baroreceptor response and subsequently resets the exercise-pressor reflex allowing for continued elevations in heart rate during sustained efforts (4,16). Thus, a greater HR, increased SBP and higher RPP were noted during the HC protocol (4, 10, 14). This study is consistent with prior investigations of HC protocols with an increase in blood pressure and a decrease in arterial-venous oxygen difference (5, 11, 12, 13, 19). In addition, there is an increased glycolytic production of adenosine triphosphate (ATP) while yielding hydrogen ions and carbon dioxide stimulating afferent signals to the myocardium (16, 22). Accumulation of these metabolites in the vasculature elevates the metaboreflex and facilitates sympathetic neural activity (4, 6, 8, 22). Whereas normal response to increased peripheral metabolites (i.e., hydrogen ions, carbon dioxide) is an increased vasodilation, past research indicates an increase in mean arterial pressure under a high cadence output (5). This increase in mean arterial pressure could also signify the baroreflex opposing the pressor response elicited by the metaboreflex and resetting itself in a time-dependent manner (i.e. duration and length of contraction). This is supported by past findings that have noted increases in oxygen uptake ( $\text{VO}_2$ ), stroke volume, HR and decreased a-v  $\text{O}_2 \Delta$  (1, 12, 15). Conversely, if HC work leads to increases in HR, SBP, and RPP and reliance on glycolysis, then the LC protocol may differ in these reactions (7).

The results of this study suggest lowered HR, SBP and RPP during the LC protocol in contrast to the HC protocol. Additionally, low cadence (LC) work at the same power output suggests the following: a reduced pressor response, increased blood flow,

increased oxygen extraction by the skeletal muscles with subsequent increase in oxidative phosphorylation leading to a decreased myocardial response (2, 7, 19). Additionally, cadence alterations seem to facilitate altered bioenergetics. Past studies indicate increases in glycolysis, through increased blood lactate, at higher cadence output (12, 15). In a related study, implications of contractile work and blood flow were investigated. Short, fast contractions vs. long, slow contractions were measured through ATPase (i.e. summation of force and non-force processes) to quantify muscle metabolism (7). Their study established that HC have an elevated metabolic cost, but more importantly, flow-mediated vasodilators (adenosine, nitric oxide, lactate, and oxygen) were produced to sustain steady-state blood flow to the working muscles (7). Taken together, this signifies fast, repetitive muscle contractions are closely associated with muscle metabolism rather than absolute contractile work (7). This is consistent with our investigation of a sustained, 20-minute high vs. low cadence protocol. The finding for the current study suggests the LC protocol leads to a reduction in HR and SBP via enhanced vasodilation and reliance on aerobic metabolism.

In conclusion, LCHF vs. HCLF may allow for the same power to be accomplished for an extended time period through varied pedal cadences. The investigators sought to answer the original question, "Does selection of different pedal cadences in cycling affect cardiovascular reactions when work is maintained over time." It was determined that HC pedaling, when compared to LC pedaling, significantly increased cardiovascular parameters of HR,

## CARDIOVASCULAR RESPONSES TO HIGH VS. LOW FORCE CYCLING

SBP, and RPP. Similarly, influencing factors such as baroreceptors, chemoreceptors and metaboreceptors suggest the discrepancy in HC vs. LC (3, 4, 12). A low cadence, high gear application produces lower stress through myocardial characteristics, blood flow, and bioenergetics during sustained power (13).

For the competitive cyclist, this study could be a useful strategy when training for endurance, sprint, or time-trial events. Cycling coaches and riders must recognize, as this study suggests, an appropriate cadence selection is critical; not just for preference of the cyclist, but for avoiding undue stress on the cardiovascular system. With that said, pedal efficiency could assist road cyclists in their overall success by conserving their energy for more intense bouts during a race. Many devoted cyclists and coaches still advocate a HCLF for the fear of leg fatigue from the higher gear selections. However, this study shows increased myocardial demand with a HC selection over time.

### ACKNOWLEDGEMENTS

The authors would like to thank their list of volunteer subjects and undergraduate/graduate students who sacrificed their time to assist in making the study come to life. They would like to thank colleagues and administrators at Midwestern State University and Southern Methodist University who supported the research project from its conception to completion.

### REFERENCES

1. Abbiss CR, Peiffer JJ, Laursen PB. Optimal cadence selection during cycling. *Int SportMed J* 10:1-15, 2009.

2. Ansley L, Cangle P. Determinants of optimal cadence during cycling. *Eur J Sports Sci* 9(2): 61-85, 2009.

3. Boushel R. Muscle metaboreflex control of the circulation during exercise. *Acta Physiol* 199: 367-383, 2010.

4. Fadel PJ. Arterial baroreflex control of the peripheral vasculature in humans: rest and exercise. *Med Sci Sports Exerc* 40 (12): 2055-2062, 2008.

5. Gottshall RW, Bauer TA, Fahmer SL. Cycling cadence alters exercise hemodynamics. *Int SportMed J* 17 (1): 17-21, 1996.

6. Guerra RS, Amaral TF, Marques E, Mota J, Restivo MT. Accuracy of Siri and Brozek Equations in The Percent Body Fat Estimation in Older Adults. *J Nutr Health Aging* 14 (9): 744-748, 2010.

7. Hamann JJ, Kluess HA, Buckwalter JB, Clifford PS. Blood flow response to muscle contraction is more closely related to metabolic rate than contractile work. *J Appl Physiol* 98: 2096-2100, 2005.

8. Ichinose M, Saito M, Kondo N, Nishiyasu T. Baroreflex and muscle metaboreflex: control of muscle sympathetic nerve activity. *Med Sci Sports Exerc* 40 (12): 2037-2045, 2008.

9. Kraemer WJ, Fleck SJ, Deschenes MR. *Exercise Physiology: Integrating Theory and Application*. 2<sup>nd</sup> ed. New York: Wolters Kluwer Publishers; 2016.

10. Krathwohl D.R. *Methods of Educational and Social Science Research: An Integrated Approach*. New York: Longman Publishers; 1993.

11. Merrill EG, White JA. Physiological efficiency of constant power output at varying pedals rates. *J Sport Sci* 2: 25-34, 1984.

12. Moore JL, Shaffrath JD, Casazza GA, Stebbins CL. Cardiovascular effects of cadence and workload. *Int SportsMed J* 29: 116-119, 2008.

## CARDIOVASCULAR RESPONSES TO HIGH VS. LOW FORCE CYCLING

13. Nimmerichter A, Easton R, Bachl N, Williams C. Effects of low and high cadence interval training on power output in flat and uphill cycling time-trials. *Eu J Appl Physiol* 112: 69-78, 2012.
14. Pescatello LS, Arena R, Riebe D, Thompson PD, eds. *ACSM's Guidelines for Exercise Testing and Prescription*. 9th ed. Baltimore: Lippincott Williams & Wilkins Publishers; 2013.
15. Potts JT, Mitchell JH. Rapid resetting of carotid baroreceptor reflex by afferent input from skeletal muscle receptors. *Am J Physiol*. 275 (Heart Circ Physiol 44): H2000-H2008, 1998.
16. Price MJ, Collins L, Smith PM, Goss-Sampson M. The effects of cadence and power output upon physiological and biomechanical responses to incremental arm-crank ergometry. *Appl Physiol Nutr Metab* 32: 686-692, 2007.
17. Raven PB. Recent advances in baroreflex control of blood pressure during exercise in humans: an overview. *Med Sci Sports Exerc* 40 (12): 2033-2036, 2008.
18. Robergs RA. Exercise induced metabolic acidosis: where do the protons come from? *Am J Physiol Regul Integr Comp Physiol* 5:(2), 2001.
19. Sarre G, Lepers R. Neuromuscular function during prolonged pedaling exercise at different cadences. *Acta Physiol Scand* 185: 321-328, 2005.
20. Stebbins L, Moore JL, Casazza GA. Effects of cadence on aerobic capacity following a prolonged, varied intensity cycling trial. *J Sports Sci Med* 13:114-119, 2014.
21. Suzuki M, Ishiyama ST, Nishikawa E, Matsubara S. Cardio-pulmonary responses to increasing workload exercise on a cycle ergometer in healthy men. *Adv Exerc Sports Physiol* 13 (2): 19-24, 2007.
22. Takashi I, Katayama K, Yamazaki K, Yamamoto T. Effect of cycling experience and pedal cadence on the near-infrared spectroscopy parameters. *Med Sci Sports Exerc* 34 (12): 2062-2071, 2002.
23. Wyatt FB. Thresholds of ventilation and heart rate during incremental exercise and venous leg occlusion. *JEPonline* 10 (3): 25-31, 2007.