

The Effect of Active versus Supine Recovery on Heart Rate, Power Output, and Recovery Time

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ABSTRACT

International Journal of Exercise Science 6(3) : 180-187, 2013. The purpose of this research was to compare the effectiveness of two different post-exercise recovery methods, active and passive, on the heart rate (HR), peak power (PP), average power (AP) and time to Baseline Active Heart Rate (BAHR) following three short (10 s) bicycle sprints. Fourteen males (mean age: 21.0±0.7 yrs) participated in the study. Each participant performed two separate trials that included three maximal Wingate rides of 10 s each. In one trial each ride was followed by a two-minute supine recovery. In the second trial each ride was followed by a two-minute active recovery that involved walking on a treadmill at 1.5 mph with a 2.5% grade. Heart rate was recorded every 20 s during the recovery periods, and PP and AP were obtained during the cycle rides. Time to recovery was recorded following the third (and final) ride in each trial to determine the time required to return to a pre-determined recovery heart rate. This HR value was determined in a pre-test by recording the HR of each participant while walking on a treadmill at 1.5 mph with a 2.5% grade. Results showed supine recovery resulted in significantly lower HR at each 20-s interval and overall ($p < 0.01$). Additionally, supine recovery resulted in a significantly shorter time to BAHR (10.8±9.0 min) compared to the active recovery (30.5±18.2 min; $p < 0.001$). There was no difference in PP or AP for any rides between the two recovery modes ($p > 0.05$). Heart rate and time to BAHR were significantly lower following supine recovery compared to active recovery; however, this decreased HR did not have an effect on peak or average power.

KEY WORDS: Wingate, anaerobic, recovery

INTRODUCTION

In many sports, performance is based on maintaining high-level physical outputs during repeated bouts (15,18,21). Declines in force output during subsequent bouts have been associated with several metabolic changes. These changes include the restriction of blood flow (4), a decrease in phosphocreatine (2), accumulation of lactate or H⁺ ions (2,3,23), and the inability

to regenerate ATP (2,3). During recovery, adequate blood flow is the common regulator for both energy regeneration and acid-base recovery (4). Recovery is significant in maintaining performance, as it increases the rate of regeneration of energy, stabilizes the acid-base balance, and decreases fatigue (4,6). The importance of recovery on performance has led to research on a variety of recovery strategies (6). Different strategies include contrasting

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temperature water immersion (6), sports massage (14), and cooling the legs (9). However, authors suggest the use of an active or a passive recovery, seeing as both help facilitate venous blood flow back towards the heart (8). The active and passive recoveries take different approaches to providing the nutrient rich blood that enhances energy regeneration and removal of lactate or H⁺ ions.

In a standing position, gravity significantly affects the distribution of the blood volume in the body (22). The body compensates for this off-balanced distribution by increasing peripheral resistance. Pumping against stronger resistance causes the heart to have a lower left ventricular end-diastolic pressure, lower stroke volume, and higher heart rate (17). When the body is changed from upright to supine, parasympathetic nerve activity increases its affect on the heart, whereas sympathetic nerve activity decreases (22). Additionally, in a supine position, a balanced distribution of blood causes weaker peripheral resistance. Parasympathetic activity and weaker resistance cause the heart to have a lower heart rate and higher stroke volume (5,22). The upright active and supine passive recoveries are able to take advantage of their respective attributes to maintain performance.

Studies have shown that an active recovery is very efficient at the removal of lactate or H⁺ ions (4,10,15,19,20,21). This removal is induced by the higher heart rate associated with the upright position and sympathetic nerve influence. By maintaining a higher heart rate, the body is able to quickly circulate nutrient rich blood and remove unnecessary metabolites. Contracting muscles promote blood circulation by

pumping venous blood back towards the heart (19). An active recovery is more effective at the removal of lactate, while a passive recovery is more efficient at lowering the post-exercise heart rate (6,8,10,12). The heart rate is lower because inactive recovery is associated with the cessation of the primary exercise stimulus from the brain (8,12). Prolonging diastole allows the slower heart rate to circulate blood at a sufficient rate (8). The slower, larger volume of blood being moved during a passive recovery is more efficient at resynthesizing the phosphocreatine energy stores (10,20).

Although the active and passive supine recoveries differ in mechanisms, the subsequent performance outcomes appear to be equal (18). Some studies indicate that an active recovery maintains higher performance (4,21), while others indicate that a passive recovery preserves higher performance (20). Other studies have shown there are no differences between the recovery types (15,18). Many studies have examined only the effects of recovery on power output (1,9,10,15,19,20,21), while others have examined the effects of recovery on heart rate and metabolites (6,8,14,18,22). However, the combination of power output and heart rate during recovery is an understudied area. Additionally, no articles were found that compared the differences in time needed for heart rate to recover to baseline active level between active and passive recoveries. The purpose of this study was to determine the effect that recovery type has on heart rate, power output, and time needed to fully recover after subsequent maximal effort sprints.

METHODS

Participants

This study consisted of fourteen males (21.0±0.7 yrs, 1.83±0.07 m, 77.8±11.9 kg, and 11.7±5.1% body fat). To be included in the study the participants must have been physically active for the four weeks prior to the study, which consisted of 30-min exercise sessions for at least three days a week. Volunteers who had any prior heart complications, who were taking any blood pressure or heart rate medications, or who smoked were excluded from the study. To screen for these exclusion criterion, each participant completed a health history questionnaire. Data collection was completed after written informed consent was obtained from each participant. Approval from Institutional Review Board was obtained prior to any data collection.

Protocol

Each participant performed two counterbalanced trials. The trials consisted of three 10-s Wingate sprints with 2-min recovery intervals in between each sprint. One trial was performed with an Active Recovery (AR) after each Wingate sprint, and the other trial was performed with a Supine Recovery (SR) after each Wingate sprint. The AR consisted of walking on the treadmill at a 1.5 mph pace with a 2.5% grade (7,16). The SR consisted of lying flat in a supine position on a trainer's table (12,15).

Before the data collection began, the participants were informed of the purpose of this study, the timeline of each trial, and familiarized with the equipment used in the study. The order of trials was counterbalanced, so that during the first

trial seven participants performed the AR, and the other seven performed the SR. During the second trial, the participants who initially performed the AR performed the SR, and the participants who initially performed the SR performed the AR.

Prior to testing, the participants had their height and weight measured, and body composition was calculated via bioelectrical impedance analysis using a handheld body fat analyzer (Omron HBF-306, Omron Healthcare, Inc, Illinois, USA). At the beginning of the first trial the Baseline Active Heart Rate (BAHR) was measured. The BAHR was the participant's heart rate after walking on the treadmill for 10 min at a 1.5 mph pace with a 2.5% grade. One goal of this study was to determine how long it took to recover back to a baseline heart rate after exercise. The same body weight and BAHR recorded during the first trial were also used for the second trial. The same body weight was used to ensure that the weighted resistance for the Wingate sprints did not vary between trials. The same BAHR was used for both trials so that the baseline, target heart rate was constant through both trials. The BAHR and heart rate during the recovery intervals were measured using a standard heart rate monitor (Timex T5K537, Timex Corporation, Little Rock, Arkansas).

During the two trials, the participants performed three 10-s Wingate sprints separated by 2-min recovery intervals. Three 10-s sprints were chosen based on published and pilot data. The Wingate sprints were completed on a Monark Ergonomic Peak Bike (Monark Ergonomic 894-E Peak Bike, Monark, Vansbro, Sweden.) The original software for the Monark Ergonomic Peak Bike was used

during the sprints to calculate peak power and average power. The opposing force for each full effort, cycling sprint was 0.075 kg / kg-body-mass (11,21). After completing the Wingate sprint, the participants immediately moved to the assigned recovery position. During the 2-min recovery, the AR participants walked on the treadmill at a 1.5 mph pace and 2.5% grade, while SR participants recovered lying down on a trainer's table. During each 2-min recovery, the participant's heart rate was recorded every 20 s. A total of six heart rate measurements were recorded per 2-min recovery interval. When the first 2-min recovery was over, the participant immediately performed another 10-s Wingate sprint and recovery interval. At the end of the second 2-min recovery, the participants immediately performed the third Wingate sprint. Following the third Wingate sprint, the participants recovered on the table or treadmill and were timed until their heart rate returned to their BAHHR.

The first and second trials for each participant were separated by four to seven days. At the beginning of the second trial, the participants again walked on the treadmill for 10-min at a 1.5 mph pace with a 2.5% grade. The BAHHR was not re-measured; rather, this 10-min walk was used as a warm up, to keep the trials consistent. Following the warm up walk, the second trial was conducted in the same manner as the first. To maximize recoveries, the treadmill and table were short, equal distances away from the bike. The participants had 7 s from the end of their Wingate sprint to be in the correct recovery position, and 7 s from the end of their recovery interval to start cycling.

The participants were instructed not to eat or exercise two hours prior to their trials. They were told not to consume any caffeine, or any type of energy drink, the day of their trials. They were told not to consume any water 30 min prior to their trials. Water consumption was not allowed during the trials. No verbal encouragement was given during either trial. However, at the beginning of both trials the participants were reminded that the Wingate sprints were full effort, cycling sprints. To limit distractions, or any outside influence, during the test, the trials were conducted in a closed exercise physiology lab with only the researchers present.

Statistical Analysis

The alpha value for our statistical analysis was set at 0.05. The means and standard deviations were calculated for the heart rate, peak power, average power and time to BAHHR for both the AR and SR trials. Repeated Measures Analysis of Variance tests were used to determine differences between trials for each of these variables. Differences in HR, peak power and average power between trials were analyzed for each ride. Average HR within each trial over the three rides was also analyzed.

Table 1. Participant characteristics.

<i>Variable (n=14)</i>	<i>Mean ± SD</i>
Age (yrs)	21.0 ± 0.7
Height (m)	1.8 ± 0.1
Weight (kg)	77.8 ± 11.9
BMI (kg/m ²)	23.2 ± 2.6
Body Fat (%)	11.7 ± 5.1

Values are mean±SD. n, number of subjects. BMI, Body Mass Index.

RESULTS

All 14 participants completed the entire study. Descriptive characteristics for the

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participants are provided in Table 1. When averaging HR over the three rides within each trial the HR during the AR trial was significantly higher than the SR trial at each time point and overall ($p < 0.01$) as shown in Figure 1. Time to BAHR was significantly lower in the SR trial compared to the AR trial ($p < 0.001$), as shown in Figure 2. Peak power (Figure 3) and average power (Figure 4) were determined to have no significant difference between or within each trial ($p > 0.05$).

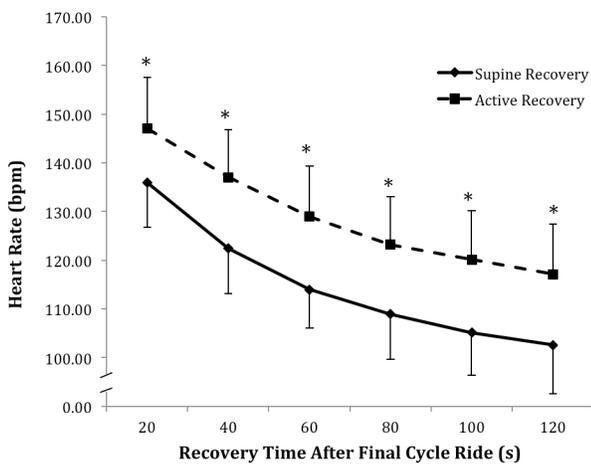


Figure 1. Heart rate averages for supine recovery and active recovery trials. Data points represent average heart rates for the three rides at each time point for each trial. Values are mean \pm SD. *Significantly greater ($p < 0.01$) than supine recovery at all time points.

DISCUSSION

The aim of the present study was to compare the effectiveness of two different recovery methods, active and supine recoveries, on heart rate, power output, and time for heart rate to recover to baseline active level. It was found that a supine recovery resulted in a significantly lower heart rate at every time point during the trials, as well as a faster time for heart rate to recover to baseline active level. However, there was not a significant difference in

power output between the supine and active recovery trials.

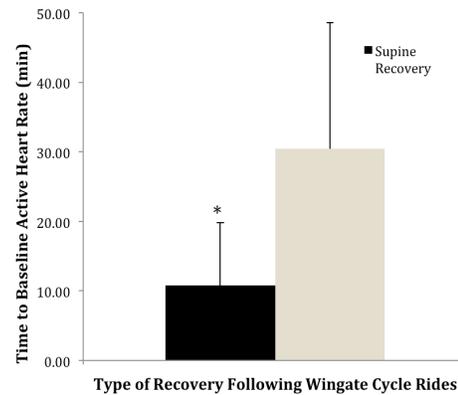


Figure 2. Represents the duration for participants to return to Baseline Active Heart Rate (BAHR) following final Wingate cycle ride for supine and active recovery trials. BAHR was determined by each participant's heart rate during a pre-test while walking at 1.5 mph with a 2.5% grade. *Significantly less than active recovery trial ($p < 0.001$).

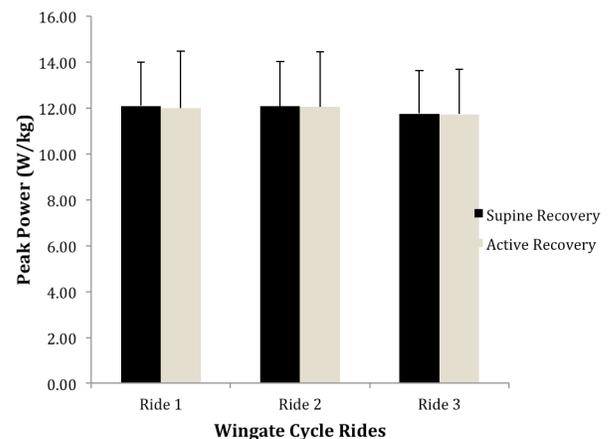


Figure 3. Peak power output from each ride for supine and active recovery trials. No significant difference was found between the trials ($p > 0.05$).

Our finding that a supine recovery maintained a lower post-exercise heart rate is in accordance with a number of other studies (6,8,10,12,). Findings from previous research indicate that it may be reasonable to assume that the heart rate is lower because inactive recovery is associated with an increase in parasympathetic influence on

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the heart. (8,12). The initial increase of parasympathetic influence, which slows the heart rate, is caused by the cessation of exercise, loss of central command, baroreflex activation, and other mechanisms, despite maintained sympathetic activation (12,22). In addition to that, the heart rate is lower because minimizing gravitational affects lowers the peripheral resistance (22). When in a supine position, the majority of the blood in the body is now in the same plane as the heart. This allows easier venous flow back to the heart, reducing the stress placed on the heart. Easier venous flow and a slower heart rate lead to prolonging diastole, which allows for greater ventricular filling (8). An increase in ventricular filling allows an adequate blood supply to be circulated around the body, despite the lower heart rate.

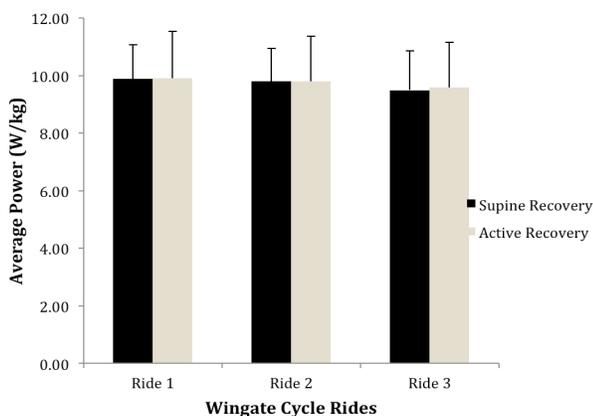


Figure 4. Peak power output from each ride for supine and active recovery trials. No significant difference was found between the trials ($p > 0.05$).

From our findings, it can be deduced that the supine recovery results in a quicker time for heart rate to recover to baseline active level for the physiological reasons that a supine recovery maintained a lower heart rate. Again, those reasons being increase parasympathetic activity and minimizing gravitational affects. However,

while the supine recovery resulted in a significantly lower heart rate and quicker recovery time, the results of our study indicate that for three subsequent, anaerobic sprints, a lower heart rate does not translate into better performance. This finding suggests that recovery of heart rate does not necessarily indicate recovery of metabolic systems for performance.

Our finding that there was no difference for subsequent power output measurements between supine and active recoveries is also in agreement with current research (15,18). We can explain the parallel power outputs, even with the differences in the heart rate and time to recovery, by the fact that our experiment was mainly anaerobic in nature. Anaerobic exercises are shorter and utilize energy regeneration by metabolic pathways that do not require oxygen. When integrating all the findings of this study, we can deduce that for repeated anaerobic bouts, maintaining a lower heart rate does not affect performance. Performance is maintained by providing the anaerobic systems with enough time to be replenished before the next sprint, regardless of heart rate.

Future research could include observing the differences in heart rate, power output, and time to recovery by making the trials more aerobic in nature. This could include making durations of the sprints longer, the recovery intervals shorter, or increasing the number of successive sprints. Additionally, the inclusion of measures of metabolic recovery would be warranted. In conclusion, a supine recovery keeps the heart rate significantly lower and allows for a significantly quicker recovery time for heart rate. However, heart rate does not

affect power performance for three repeated anaerobic sprints.

REFERENCES

1. Billaut F, Giacomoni M, Falgairette G. Maximal intermittent cycling exercise: effects of recovery duration and gender. *J Appl Physiol* 95: 1632-1637, 2003.
2. Bogdanis GC, Nevill ME, Boobis LH, Lakomy HKA, Nevill AM. Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man. *J Physiol* 482(2): 467-480, 1995.
3. Bogdanis GC, Nevill ME, Lakomy HKA, Boobis LH. Power output and muscle metabolism during and following recovery from 10 and 20 s of maximal sprint exercise in humans. *Acta Physiol Scand* 163:261-272, 1998.
4. Bogdanis GC, Nevill ME, Lakomy HK, Graham CM, Louis G. Effects of active recovery output during repeated maximal sprint cycling. *Euro J Appl Physiol* 74(5): 461-469, 1996.
5. Carter III R, Wilson TE, Watenpaugh DE, Smith ML, Crandall CG. Effects of mode of exercise recovery on thermoregulatory and cardiovascular responses. *J Appl Physiol* 93: 1918-1924, 2002.
6. Coffey V, Leveritt M, Gill N. Effect of recovery modality on 4-hour repeated treadmill running performance and changes in physiological variables. *J Sci Med Sport* 7(1): 1-10, 2004.
7. Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MS. Heart-rate recovery immediately after exercise as a predictor of mortality. *N Engl J Med* 341(18): 1351-1357, 1999.
8. Crisafulli A, Orru V, Melis F, Tocco F, Concu A. Hemodynamics during active and passive recovery from a single bout of supramaximal exercise. *Eur J Appl Physiol* 89: 209-216, 2003.
9. Crowley GC, Garg A, Lohn MS, Van Someren N, Wade AJ. Effects of cooling the legs on performance in a standard Wingate anaerobic power test. *Br J Sp Med* 25(4): 200-203, 1991.
10. Dupont G, Moalla W, Matran R, Berthoin S. Effect of short recovery intensities on the performance during two wingate tests. *Med Sci Sports Exerc* 39(7): 1170-1176, 2007.
11. Gotshalk LA, Volek JS, Staron RS, Denegar CR, Hagerman FC, Kraemer WJ. Creatine supplementation improves muscular performance in older men. *Med Sci Sports Exerc* 34(3): 537-543, 2002.
12. Javorka M, Zila I, Balharek T, Javorka K. Heart rate recovery after exercise: relations to heart rate variability and complexity. *Braz J Med Biol Res* 35(8): 991-1000, 2002.
13. Kang, J. *Bioenergetic primer for exercise science*. Champaign, IL: Human Kinetics, 2008.
14. Martin NA, Zoeller RF, Robertson RJ, Lephart SM. The comparative effects of sports massage, active recovery, and rest in promoting blood lactate clearance after supramaximal leg exercise. *J Athletic Training* 33(1): 30-35, 1998.
15. McAinch AJ, Febbraio MA, Parkin JM, Zhao S, Tangalakis K, Stojanovska L, Carey MF. Effect of active versus passive recovery on metabolism and performance during subsequent exercise. *Int J Sport Nutr Exerc Metab* 14: 185-196, 2004.
16. Nishime EO, Cole CR, Blackstone EH, Pashkow FJ, Lauer MS. Heart rate recovery and treadmill exercise score as predictors of mortality in patients referred for exercise ECG. *JAMA* 284(11): 1392-1398, 2000.
17. Poliner LR, Dehmer GJ, Lewis SE, Parkey RW, Blomqvist CG, Willerson JT. Left ventricular performance in normal subjects: a comparison of the responses to exercise in the upright and supine exercises. *Circulation* 62(3): 528-534, 1980.
18. Siegler JC, Bell-Wilson J, Mermier C, Faria E, Robergs RA. Active and passive recovery and acid-base kinetics following multiple bouts of intense exercise to exhaustion. *Int J Sport Nutr Exerc Metab* 16: 92-107, 2006.
19. Signorile JF, Ingalls C, Tremblay LM. The effects of active and passive recovery on short-term, high intensity power output. *Can J Appl Phys* 18(1): 31-42, 1993.
20. Spencer M, Bishop D, Dawson B, Goodman C, Duffield R. Metabolism and performance in repeated cycle sprints: active versus passive recovery. *Med Sci Sports Exerc* 38(8): 1492-1499, 2006.
21. Spierer DK, Goldsmith R, Baran DA, Hryniewicz K, Katz SD. Effect of active vs. passive recovery on work performed during serial supramaximal exercise tests. *Int J Sports Med* 25(2): 109-114, 2004.

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22. Takahashi T, Okada A, Satioh T, Hayano J, Miyamoto Y. Difference in human cardiovascular response between upright and supine recovery from upright cycle exercises. *Eur J Appl Physiol* 81(3): 233-239, 2000.

23. Tomlin DL, Wenger HA. The relationship between aerobic fitness and recovery from high intensity intermittent exercise. *Sports Med* 31(1): 1-11, 2001.