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*Original Research*

# Acute Changes in Arterial Function Post High-Intensity Lower Extremity Cycling

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ABSTRACT

***International Journal of Exercise Science 14(2): 1363-1374, 2021.*** The purpose of this study was to assess the acute arterial blood flow velocity of the lower extremity (LE) immediately after a bout of high-intensity LE cycling exercise. Twenty-eight healthy males ($n$ = 14) and females ($n$ = 14) aged 20.9 ± 1.7 years participated in this study. All subjects performed a single bout of high-intensity cycling (70% of HRR) for 45 min. The subjects' LE blood flow velocity, heart rate (HR), systolic blood pressure (SBP), and oxygen saturation ($SpO_2$) were measured at rest, immediately post-, post-15 min., and post- 30 min. intervention. A repeated-measures ANOVA with a Bonferroni adjustment was used for each measure to compare the responses at each time point. Resting blood flow velocity (37.5 ± 11.3 cm/s) and HR (64.9 ± 11.8 bpm) measures were significantly different ($p < 0.05$) compared to measures of immediately post cycling (44.8 ± 13.7 cm/s; 118.3 ± 17.2 bpm), post-15 min. (50.1 ± 15.0 cm/s; 80.1 ± 12.0 bpm) and post-30 min. (52.7 ± 18.1 cm/s; 73.9 ± 11.9 bpm). SBP measures were significantly different ($p < 0.05$) at immediately post (118.2 ±17.0 mmHg) compared to post-15 min. (108.1 ± 13.6 mmHg). Resting $SpO_2$ (98.2 ± 1.3 %) measures were significantly different ($p < 0.05$) compared to measures immediately post (96.5 ± 1.1 %) and post-15 min. (96.9 ± 1.2 %). This study indicates that LE blood flow velocity was increased, and HR was elevated following a single bout of high-intensity LE cycling up to 30 min.-post. Additionally, SBP was elevated, while $SpO_2$ dropped following a bout of exercise to 15 min.-post activity.

KEY WORDS: Last bout effect, acute effect, aerobic, cardiovascular function, blood flow velocity

## INTRODUCTION

Exercise is medicine, and regular aerobic exercise is widely used to prevent and/or treat cardiovascular disease (19, 24). The acute effects of exercise indicate the physiological changes that occur during a single bout. In contrast, the chronic effects of regular aerobic exercise appear

over weeks or months and result in physiological adaptations that allow the cardiovascular system to function more efficiently (18, 19, 24, 29, 30).

The chronic effects of exercise include the central adaptation of increased cardiac muscle cross-sectional area, which results in increased heart contractility and stroke volume to allow more blood to be pumped out into the peripheral arteries at rest and during exercise (19, 24, 29, 30). Peripheral adaptations include improved blood flow, increased blood return, decreased blood pressure via decreased total peripheral resistance, and increased blood volume to skeletal musculature (19, 24). Additionally, there is a reduction in inflammation and low-density lipoproteins, which all lower cardiovascular disease risk (19, 24, 29, 30).

Another critical adaptation of exercise to lower cardiovascular disease risk is known as the last-bout effect, which refers to physiological changes between single bouts of physical activity (18). The last-bout effect has important implications for exercise prescription, specifically the importance of regular exercise. Each bout of exercise provides protective effects that carry over to the next bout; however, the effects dissipate quickly. Thus, the closer the next bout, the more protection the individual has against cardiovascular disease.

Common adaptations from the last-bout effect include post-exercise hypotension, improved lipid profile (i.e., increased high-density lipoprotein) (32), and improved insulin sensitivity (13). Additionally, the brachial and carotid arteries have elevated blood flow velocity and vasodilation, which result in a transitory decrease in brachial blood pressure (2, 8, 15, 29, 30). The decrease in blood pressure can last up to 11 hours post-activity (23) and is more pronounced in individuals with hypertension (11).

To date, most studies investigating the acute adaptations to aerobic exercise have used various modes, intensities, and time intervals. Also, although these studies have utilized lower extremity (LE) aerobic exercise, some authors have assessed arterial blood flow velocity in the upper extremities (UE) while others have assessed it in the LE (2, 3, 8, 15, 27, 29, 30). Specifically, Bailey and colleagues assessed the acute effect of moderate and high-intensity LE cycling while assessing the arterial blood flow velocity in the UE. The authors found that UE arterial blood flow velocity decreased for individuals with lower fitness levels and increased for those with higher fitness levels post-session (2). Siasos et al. demonstrated that moderate-intensity and high-intensity LE interval cycling sessions resulted in increased LE arterial blood flow velocity post-session (30). Brito et. al., used single-leg extension for 45 min. and assessed LE arterial blood flow velocity post-exercise and found a decrease in LE arterial blood flow velocity post-session (3). Additionally, Rogno et al. used high-intensity interval running on a treadmill and assessed arterial blood flow velocity in the UE. The authors found that the trained individuals showed increased blood flow velocity following the session while the untrained individuals showed no significant changes post-session (27). Therefore, uncertainty remains regarding the general

understanding of continuous high-intensity LE exercise and the acute response of arterial blood flow within the LE.

Given that exercise modes, intensity, and time intervals could stimulate different acute adaptations, assessing arterial blood flow velocity changes associated with different modes and intensities would be meaningful. Also, it would seem most appropriate to assess LE arterial blood flow velocity changes after a bout of LE exercise. Thus, the primary purpose of this study was to examine the acute effects of a single bout of high-intensity LE cycling exercise on LE arterial blood flow velocity immediately post, post-15, and post-30 minutes (min.). The secondary purpose of this study was to assess the acute effects of heart rate (HR), systolic blood pressure (SBP), and oxygen saturation ($SpO_2$) immediately post, post-15, and post-30 min. It was hypothesized that arterial blood flow velocity would be elevated post-exercise up to 30 min. The secondary hypothesis was that HR would be elevated, SBP would be lowered, and $SpO_2$ would not change post-exercise up to 30 min.

## METHODS

*Participants*

The participants included 29 males and females aged 18-30 years recruited via convenience sampling from local colleges. Participants were excluded in this study if they had any of the following: greater than one cardiovascular disease risk factor or known cardiovascular disease, neurological disorders, any LE musculoskeletal disorder that affects gait, use of any medication that would affect exercise response, use of tobacco, and exercise for more than 150 min. per week. After completion of a health screening questionnaire, informed consent was signed and witnessed by the primary investigators. The University of Southern Indiana's Institutional Review Board approved this study for the protection of human participants. This research was carried out entirely under the ethical standards of the International Journal of Exercise Science (21).

The subjects reported to the lab for a single session lasting approximately 120 min. All measurements were performed in a quiet, temperature-controlled room (22–24°C) after at least 4 hours of fasting and caffeine abstinence, without any medications within 24 hours, as well as the absence of vigorous exercise. Initially, subjects underwent a health history screening for CVD risk factors or any known disorders. Determination of any known disorders excluded subjects from the study. Subsequently, the subjects had their height, weight, and age recorded. Height and weight were measured using a mechanical scale with an attached telescoping height rod (Detecto Eye Level, Detecto, Webb City, MO).

*Protocol*

Participants rested in the supine position on a table for 5 min. before the resting blood flow velocity, HR, SBP, and $SpO_2$ were measured. Arterial blood flow velocity was measured on the left side using a mobile ultrasound (Esaote MyLab™25Gold; Esaote, Florence, Italy) equipped with a Doppler probe positioned at a 60° angle isolating the popliteal artery at a frequency of 10 MHz. To obtain resting and post-exercise blood flow velocity measures, subjects were

positioned prone on an examination table. The ultrasound probe was placed in the popliteal fossa behind the knee, with the popliteal artery identified in a transverse image. After the popliteal artery was identified, the transducer was rotated perpendicularly to see the vessel image longitudinally. A line was then drawn with a surgical marker to mark the vessel image's location to ensure that the image would be the same at each measurement time point. Doppler tracing was obtained to get peak systolic velocity (PSV) to assess popliteal artery blood flow velocity. The ultrasound technique used to assess the popliteal artery blood velocity was derived according to Hwang and Olowoyeye et al. (14, 22). An experienced investigator (AW) was responsible for imaging and measuring all popliteal artery blood flow velocities. It has been found that the intra-and inter-rater reliability of peripheral arterial blood flow velocity by Doppler ultrasound ranges from 0.501 to 0.866 (10). HR and $SpO_2$ were taken on the left index finger of the participant's body using a pulse oximeter (Nellcor™ Portable $SpO_2$ Patient Monitoring System, PM10N, Medtronic, Minneapolis, MN). Brachial systolic BP was measured on the left side via Digital Fourier Oscillometer (Wallach Summit Doppler Vantage ABI™, Wallach Surgical Devices, Trumbull, CT).

After the subject had their resting measures completed, the cycle ergometer (Monark 828E Ergomedic, Monark Exercise AB, Vansbro, Sweden) was set up to ensure that the seat height was based on the individual's height and leg length. Additionally, during cycling, the knee extension was slightly flexed at 5°-10 °, and pedaling was performed lightly with no observed hip rocking (9). After the seat height was adjusted, the subject started a 5-min. warm-up of pedaling at the cadence of 50 RPM without resistance. After a 5-min. warm-up, the subject pedaled on a cycle ergometer (Monark 828E Ergomedic, Monark Exercise AB, Vansbro, Sweden) with the cadence set at 50 RPM. While the subject pedaled, the investigator progressively added resistance and adjusted as necessary to reach and maintain the subject's HR reserve (HRR) of 70% for 45 min., which has been shown to cause post-exercise hypotension (7, 29). HRR is calculated as follows: (predicted HRmax – HRresting) x (70%) + (HRresting) (17).

Immediately following the exercise session, blood flow velocity, HR, SBP, and $SpO_2$ measures were taken while the participant remained seated on the cycle ergometer. The subject placed the left foot on the floor with the knee flexed to ~10 degrees for the blood flow velocity measurement immediately after the exercise session. Then the same steps were performed as the resting measurements (SEE Pre-intervention). Measurements were also taken post-15 min. and post- 30 min. of the exercise session while the subject was lying supine on a table. Figure 1 provides a graphic overview of the experimental design.

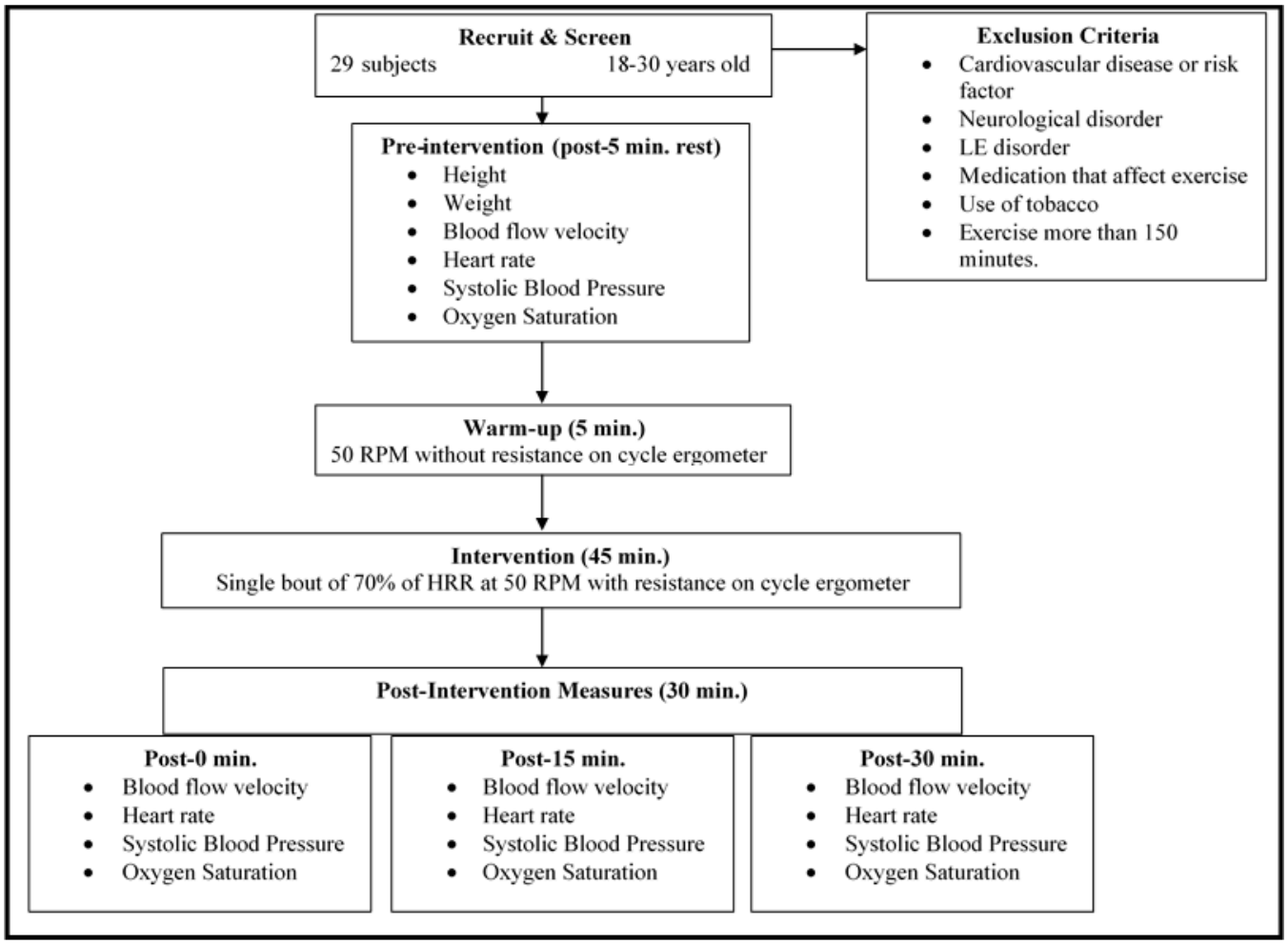


**Figure 1.** Schematic of the experimental study design.

*Statistical Analysis*

Subjects' descriptive statistics were reported as mean ± standard deviation (SD). A repeated-measures ANOVA with a Bonferroni adjustment was used for each measure to compare the responses at baseline, immediately post-, post-15 min., and post-30 min. intervention. An alpha level of .05 was used to determine statistical significance with IBM SPSS Statistics (v24; SPSS Inc., Armonk, NY). An experimental power analysis was conducted with G*POWER 3.1.9.7 (Universitat Kiel, Germany), which determined that the 28 participants included in this study had a power of .99, with an effect size of 0.5 and an $a$ = 0.05. The effect size of 0.5 was determined based on previous research studies on arterial blood flow velocity having similar study designs (1, 26-30). Also, it is suggested that when designing an exercise study, .05 is used for a moderate size effect (31).

## RESULTS

Of the twenty-nine subjects recruited, twenty-eight healthy males ($n$ = 14) and females ($n$ = 14) completed the study and were analyzed. One subject dropped out at the end of the study session due to time constraints which enabled the individual to complete the post-30 min. measures. Table 1 shows the demographics of the subjects included in this study.

**Table 1.** Demographic characteristics.

| Gender (M:F) | 14:14 |
|---|---|
| Age (yr) | 20.9 ± 1.7 |
| Height (cm) | 178.7 ± 9.5 |
| Weight (kg) | 69.5 ± 13.8 |
| BMI (kg/m²) | 24.4 ± 3.2 |

Resting blood flow (37.5 ± 11.3 cm/s) and HR (64.9 ± 11.8 bpm) measures were significantly different ($p$ < 0.05) compared to measures of immediately post cycling (44.8 ± 13.7 cm/s ;118.3 ± 17.2 bpm), post-15 min. (50.1 ± 15.0 cm/s; 80.1 ± 12.0 bpm) and post-30 min. (52.7 ± 18.1 cm/s; 73.9 ± 11.9 bpm) (Figure 2 & 3). SBP measures were significantly different ($p$ < 0.05) at immediately post cycling (118.2 ± 17.0 mmHg) compared to post-15 min. (108.1 ± 13.6 mmHg) (Figure 4). Resting $SpO_2$ (98.2 ± 1.3 %) measures were significantly different ($p$ < 0.05) compared to measures immediately post cycling (96.5 ± 1.1 %) and post-15 min. (96.9 ± 1.2 %) (Figure 5). All other SBP and $SpO_2$ measures were not significantly different ($p$ > 0.05).

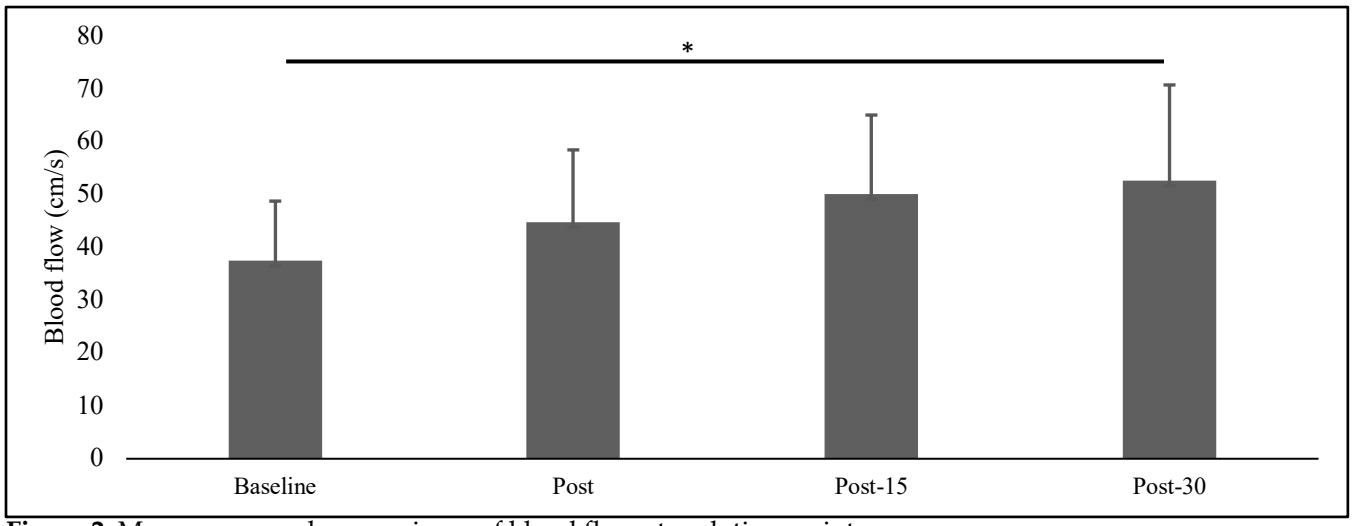


**Figure 2.** Mean scores and comparisons of blood flow at each time point.
*indicates significant differences from each time point (p<0.05).

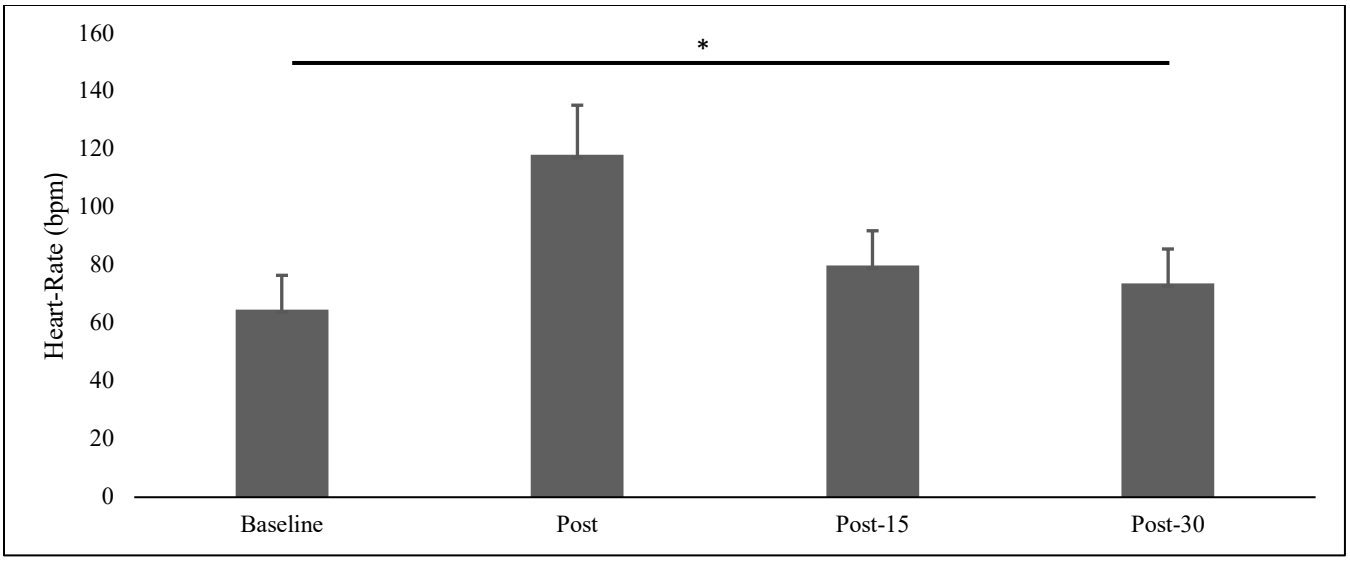


**Figure 3.** Mean scores and comparisons of heart rate at each time point.
*indicates significant differences from each time point ($p<0.05$).

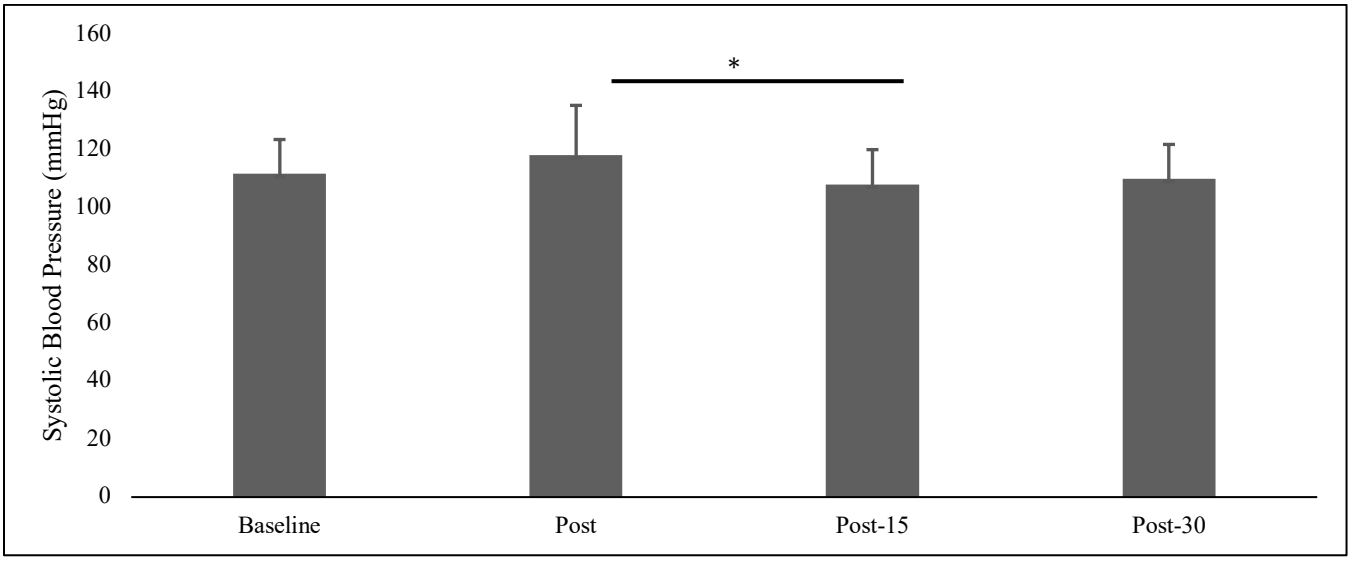


**Figure 4.** Mean scores and comparisons of systolic blood pressure at each time point.
*indicates significant differences from each time point ($p<0.05$).

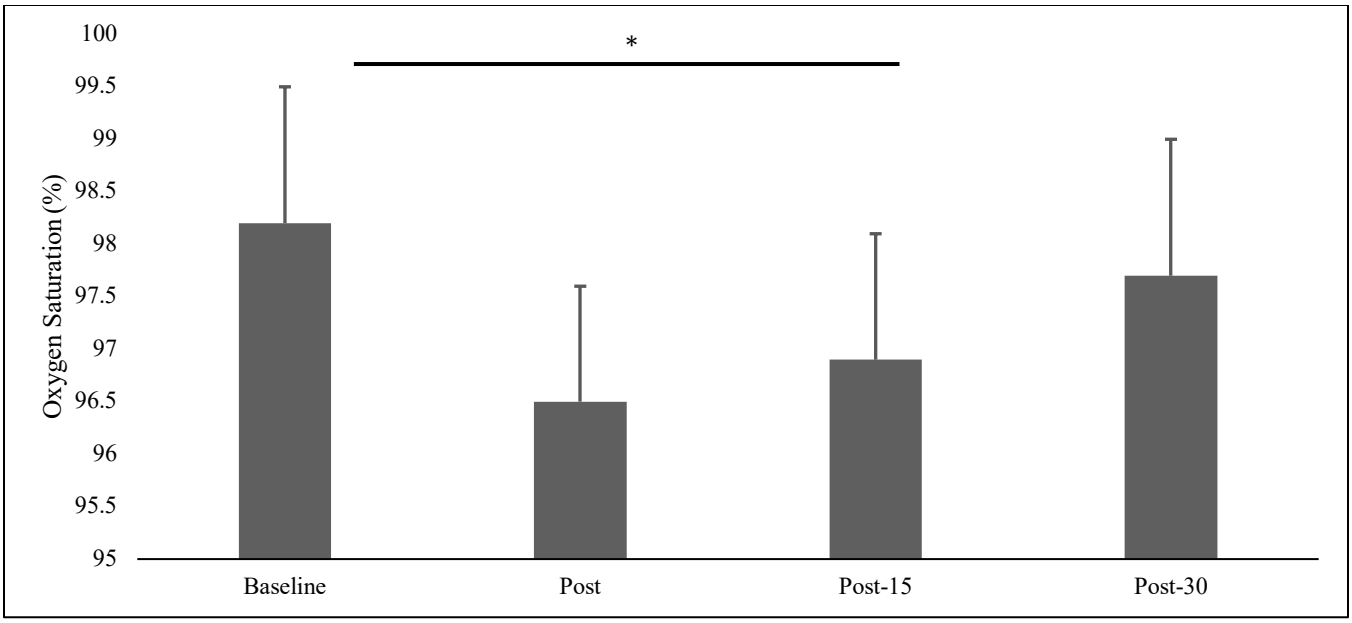


**Figure 5.** Mean scores and comparisons of oxygen saturation at each time point.
*indicates significant differences from each time point ($p<0.05$).

## DISCUSSION

The primary purpose of this study was to examine the acute effects of a single bout of high-intensity LE cycling exercise on LE arterial blood flow velocity immediately post, post-15, and post-30 min. The secondary purpose of this study is to assess the acute effects of HR, SBP, and $SpO_2$ immediately post, post-15, and post-30 min. The present study indicated a significant increase in LE arterial blood flow velocity and elevated HR following a bout of aerobic exercise. The arterial blood flow velocity and HR changes were present from baseline to 30 min.-post activity. Additionally, SBP was significantly elevated following a bout of exercise to 15 min.-post activity, while $SpO_2$ dropped following a bout of exercise to 15 min.-post activity. These data on SBP, $SpO_2$, and HR measures following a bout of aerobic exercise were consistent with the previous studies' findings (2, 15, 25, 30). This is the first study to assess arterial blood flow velocity in the LE following a continuous bout of high-intensity LE cycling to the best of our knowledge. Although most studies assessing LE blood flow velocity after a bout of aerobic exercise measured it at the femoral or tibialis arteries (2, 3, 16, 26, 30), the popliteal artery was utilized in the current study.

Previous work found that UE peripheral artery blood flow velocity decreased after LE aerobic training, whereas LE blood flow velocity increased compared to baseline (2, 4, 6, 16, 26). These findings imply that the arterial blood flow velocity appears to be affected differently at these various arterial trees post aerobic training. Differences in the literature regarding arterial blood flow velocity following an acute bout of exercise can be attributed to the muscles utilized during exercise, the site where the arterial blood flow velocity is measured, and fitness levels.

For example, Siasos and colleagues assessed arterial blood flow velocity in the LE after continuous moderate-intensity and high-intensity interval LE cycling (30). The authors found

that arterial blood flow velocity in the LE increased following these LE cycling sessions. However, Bailey and colleagues assessed arterial blood flow velocity in the UE after high and moderate-intensity LE cycling (2). The authors found that UE arterial blood flow velocity decreased following these LE cycling sessions in individuals with lower fitness levels. However, the participants with higher fitness levels showed an increase in UE arterial blood flow velocity.

Some mechanisms that may explain the difference in arterial blood flow velocity at the various arterial tree segments include the arterial wall properties and central nervous system response (3, 16). During aerobic exercise, the heart responds to the increased demand on the vasculature by increasing cardiac output via increased HR, stroke volume, and SBP, which increases blood supply to the working muscles. Thus, during LE exercise, the arteries of the LE are likely supplied with the majority of the blood. The additional blood flow to the LE increases the shear stress on the arteries' walls, resulting in a shift in collagen fibers to elastin fibers (3, 16). Concurrently, there could be a shift in elastin fibers to collagen fibers in the walls of the UE peripheral arteries to constrict blood flow and divert it to the working muscles of the LE. Additionally, sympathetic inhibition at the LE peripheral arteries occurs at the central nervous system to allow vessel dilation at the working muscles (3, 16). Therefore, this could explain why some studies have found different arterial blood flow velocity changes at the various arteries compared to our study (2, 4, 6, 16, 26).

It is vital to establish acute arterial blood flow velocity adaptations to aerobic exercise in apparently healthy individuals for later comparison to clinical populations where pathology may limit blood flow to the working tissues. Also, it is essential to assess blood flow changes in the LE for scientific discovery and the clinical implications for individuals with peripheral arterial disease. Although walking is the preferred mode in this population, cycling has been recommended as a supplement or adjunct (12). Thus, determining the effect of cycling on LE blood flow is vital to this population.

There are several strengths to this study. First, our study used ultrasound with Doppler to measure peak systolic arterial velocity at the popliteal artery. Second, our study resembles a typical cardiovascular exercise setting and session for cardiovascular risk factors or disease. Third, we assessed arterial blood flow velocity and HR up to 30 min. following the exercise session, which addressed the acute effects or last-bout phenomenon identified as an essential component of maintaining healthy cardiovascular function through regular exercise.

Although the study possesses several strengths, it also has some limitations. A detailed exercise history of the subjects was neither asked nor reported; however, all participants reported histories consistent with the inclusion criteria that all participated in exercise activities less than 150 min. per week. We cannot exclude the potential for variability in the subjects' exercise participation to contribute to our results. Additionally, our study participants included both males and females, and we acknowledge that sex-related differences in arterial function may limit our data comparison. However, it should be noted that we had the same number of females and males who participated in our study. Again, the lack of a control group in our study may limit the strength of our results; however, there is relatively strong evidence regarding the effect

of our intervention. Finally, the present findings may have value for application in clinical populations such as those with intermittent claudication, as it stands to reason those individuals conceivably have the most to gain from regular participation in this type of exercise intervention. However, caution or restraint must be used until this, or a similar study is replicated in such a clinical population. Therefore, these results may not apply to a different population or individuals with a known disease.

In conclusion, the acute effects of high-intensity LE cycling demonstrated an increase in LE blood flow velocity, and elevated HR was elicited up to 30 min. -post activity. Additionally, SBP was elevated, while $SpO_2$ dropped, following a bout of exercise to 15 min. -post activity. Our results suggest that high-intensity LE cycling could be a potential pre-conditioning activity or adjunct modality to progressive walking to improve LE arterial blood flow. The data of this study may have clinical implications and therapeutic benefits regarding the protective effects associated with the last-bout effect. The effects of successive sessions of acute exercise may be superimposed, resulting in temporal summation. This summation may contribute to the chronic adaptations seen with exercise training, including improved vascular function, leading to reduced cardiovascular disease. Therefore, based on the results of this study, practitioners should consider more frequent exercise sessions for their clients/patients.

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