

Autonomic Function in Indoor Versus Mountain Bike Riding

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AUTONOMIC FUNCTION IN MOUNTAIN VERSUS INDOOR CYCLING

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Abstract

AUTONOMIC FUNCTION IN INDOOR VERSUS MOUNTAIN BIKE RIDING

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Road cycling and cross country mountain biking are common cycling disciplines, involving long distances over flat roads and short distances over loose, rough terrain respectively. Both stress the cardiovascular system, but cross country biking creates an adrenaline rush, which leads to stimulation of the sympathetic nervous system, which can increase heart rate, though the peripheral nervous system is more dominant. The activity level of the sympathetic and parasympathetic nervous systems are analyzed to determine cardiovascular autonomic function. Heart rate variability is the difference in time between many consecutive heart beat intervals, and is measured to assess nervous system activity. It has been reported that chronic stimulation of the sympathetic nervous system can impair autonomic function and lead to sudden cardiac death. **Purpose:** The purpose of this study was to assess autonomic function before and after road and cross country mountain biking. **Methods:** Four healthy, college-aged males, experienced in road and cross country biking were recruited. Subjects completed a baseline, pre, and post ride tilt-table test while electrocardiograph and blood pressure data were collected. Subjects performed a stationary,

indoor ride to simulate a road ride and cross country mountain bike ride in a randomized fashion. Two minutes of electrocardiograph data and one minute of blood pressure data were processed via WinCPRS software to assess overall autonomic function. **Results:** Speculative analysis indicates that after the cross country ride diastolic blood pressure increases compared to before, whereas following the indoor ride, cardiac output is elevated.

Discussion: The observed cardiac output increase may be a result of the immediately preceding exercise. The unexpected diastolic blood pressure increase may be caused by sympathetic nervous system stimulation from the outdoor cross country mountain bike ride.

Conclusion: An outdoor cross country ride may potentially increase sympathetic nervous system activity more than an indoor ride.

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Introduction

The participation in recreational physical activities has increased as people in the general population look for new and additional ways to get healthy. Bicycle riding has long been enjoyed by many, whether as a recreational activity or competitive sport. Among the many variations of bicycle riding, road and mountain biking are the two most popular. Road cycling is understood to be a strenuous, aerobically-demanding activity with races ranging from criteriums of short laps around a flat track, to long, multi-day stages over hilly terrain (El Helou et al., 2010; Lucia, Hoyos, & Chicharro, 2001; Mujika & Padilla, 2001).

Conversely, mountain biking is a relatively young sport which has grown immensely over the past few decades, developing many sub disciplines such as downhill, slope style, and cross country (Berto, 1999). The mountain biking sub discipline cross country (XC), is one of the most popular and physically demanding variations, involving steep climbs and descents, rocks, roots, drops, banked turns and jumps. Studies have shown that road and XC biking both place high stress on the cardiovascular system; however, XC biking also includes high skeletal muscle stress, through different types of contractions for stabilization, and an 'adrenalin rush' from a higher probability of falling (Curtis & O'Keefe, 2002; Impellizzeri & Marcora, 2007; Lee, Martin, Anson, Grundy, & Hahn, 2002; Wang & Hull, 1997).

An 'adrenaline rush' is an adrenergic stimulation caused by experienced exhilaration (Curtis & O'Keefe, 2002). Adrenergic stimulation is a function of the sympathetic nervous system (SNS) that results in large changes in the cardiovascular system such as increases in

heart rate (HR), blood pressure (BP), and cardiac output (CO) (Curtis & O'Keefe, 2002). Although the SNS is involved, the parasympathetic nervous system (PNS) is the primary regulator of HR and CO via the vagus nerve (Krassioukov & West, 2014). The autonomic nervous system (ANS) encompasses both the SNS and PNS, and the activity of each is used to assess overall cardiovascular autonomic function (ANF) (Da Silva, Verri, Nakamura, & Machado, 2014; Malik et al., 1996; Seals, Monahan, Bell, Tanaka, & Jones, 2001). Testing ANF requires the assessment of heart rate variability (HRV), which is defined as the change in duration between consecutive heartbeat intervals, also known as R-R intervals (Malik et al., 1996; Oliveira et al., 2013). High variations in R-R intervals represent a high HRV, which indicates a strong capability of the ANS to adjust HR and SV in response to external stimuli (Freeman & Chapleau, 2013; Malik et al., 1996; Oliveira et al., 2013). By continuously measuring arterial wave forms via an arm and finger cuff, data such as HR, BP, CO, and TPR (total peripheral resistance) can be obtained via algorithms and combined with frequency data from an electrocardiogram (ECG) to assess ANS activity (Freeman & Chapleau, 2013; Malik et al., 1996).

Autonomic function is being used more often to test healthy and athletic populations during and after exercise (Freeman & Chapleau, 2013; Heffernan, Kelly, Collier, & Fernhall, 2006; La Rovere, Pinna, & Raczak, 2008; Molina, Porto, Fontana, & Junqueira, 2013; Sztajzel, Jung, Sievert, & Bayes De Luna, 2008). Moreover, studies reported increases in HR, CO, and sympathetic activity, as well as decreases in parasympathetic activity (based on power spectral analysis of HRV) during exercise (Murrell et al., 2007). Because XC biking

involves additional skeletal muscles for stabilization over the rigorous terrain, it is likely that XC biking is of higher exercise intensity than road cycling for a given speed and cadence. Research indicates that exercise intensity influences how much SNS activity increases during and immediately after exercise (Goldstein & Kopin, 2008; Terziotti, Schena, Gulli, & Cevese, 2001). However, the only study to assess HRV in XC bikers tested elite competitors at rest and reported no significant differences in overall HRV, SNS or PNS activation compared to non-bikers (Molina et al., 2013). This may be from an increased ability of the ANS to regulate HR in the XC bikers from chronic cardiovascular exercise.

Since both road and XC biking are predominantly cardiovascular exercises, leisurely participation in either could elicit potential health benefits such as lower resting BP and HR (Baron, 2001; Forjaz et al., 2000; Gonzalez-Camarena et al., 2000; Molina et al., 2013). It has been shown that chronic high mental stimulation of the SNS increases the risks of cardiovascular events like sudden cardiac death and negatively affects overall ANF (Curtis & O'Keefe, 2002), but it is unclear whether high SNS stimulation from exercise will have a similar result. The chronic cardiovascular benefits of XC biking may actually outweigh potential ANF decrements when performed at the elite level. Therefore, the purpose of this study was to assess changes in cardiovascular autonomic function at rest, before and after mountain and road biking. The experimenters hypothesized that changes in cardiovascular autonomic function will be greater following a cross country mountain bike ride than a road ride when compared to resting function.

Review of Literature

Introduction

The sport of bicycle riding includes thousands of participants and numerous disciplines such as road cycling and XC mountain biking. Road cycling is a well-known, cardiovascular activity which scientists have been researching for decades (El Helou et al., 2010; Lucia et al., 2001; Mujika & Padilla, 2001). Though almost equally as popular as road cycling, XC mountain biking has been minimally researched. One area of research being studied in physical activities that has been gaining interest is ANF.

ANF refers to how the two branches of the ANS, (PNS & SNS) collectively monitor and regulate cardiovascular functions in the body. Assessment of these two processes can be completed in a variety of ways including: pharmacological administration, intra-arterial catheters, and non-invasive blood pressure and heart rate recordings (Jasson et al., 1997; La Rovere et al., 2008; Malik et al., 1996; Parati et al., 2003). Autonomic function has traditionally been used to assess the relationship between the ANS and mortality in various clinical populations such as myocardial infarct patients; however, it has also been used to test athletes and healthy populations during and after exercise (Arai et al., 1989; Cleroux, Kouame, Nadeau, Coulombe, & Lacourciere, 1992; Freeman & Chapleau, 2013; Molina et al., 2013; Smit, Halliwill, Low, & Wieling, 1999). Physical activities, such as XC biking,

result in the release of catecholamines, including epinephrine (adrenaline), which typically stimulate the SNS, though the acute effects on the autonomic nervous system after participation in XC biking have not been studied. Chronic, high stimulation of the SNS may cause cardiovascular events like sudden cardiac death (Freeman & Chapleau, 2013). Additionally, activities such as XC biking may cause a high stimulation of the SNS, which could negatively affect cardiovascular autonomic function over time.

The Autonomic Nervous System

The ANS is a branch of the nervous system that regulates involuntary functions, including HR and BP (Freeman & Chapleau, 2013). Anatomically, this system is made up of parts of the medulla oblongata, the vagus nerve and several pre and postganglionic fibers that branch off the spinal cord and brain stem to the heart (Krassioukov & West, 2014). The ANS is made up of two subparts, the PNS and SNS, which are determined as more or less active when assessing ANF based on the interpretation of cardiovascular data. The SNS is typically activated during exercise and other forms of stimulation like cognitive tasks, emotional and fearful stimulants, pain, and grip strength tasks (Beissner, Meissner, Bar, & Napadow, 2013). Many of these stimuli activate the SNS because they involve a higher level of fear or nervousness compared to rest (Goldstein & Kopin, 2008). Conversely, the PNS is dominant during calm, relaxing situations like watching television or eating (Goldstein & Kopin, 2008). The heart and blood vessels are controlled in complement to

each other by the ANS; however, much can be learned about the ANS through HR alone. HRV is a process involving regulation of HR and SV by both the SNS and PNS (Krassioukov & West, 2014). Testing HRV is one of the best ways to assess the two autonomic nervous system divisions; and determine overall ANS control over the cardiovascular system.

Heart Rate Variability

HRV is a non-invasive method to assess the ANS, and is understood to be the difference between successive R-R intervals. Since the ANS is anatomically difficult to measure directly, the responses of its effectors (i.e. the heart) to different stimuli are measured (Freeman & Chapleau, 2013). HRV can be assessed via two main methods, time domain, using statistical or geometric analyses, and frequency domain, which uses parametric or nonparametric analyses (Patel et al., 2013). The time domain analysis is said to be better for long term readings (24 hours or more), whereas the frequency domain method is better for short term readings (5-10 min) (Malik et al., 1996).

History:

HRV fluctuations have been measured as early as 1733 by Steven Hales, and again in 1760 by Albrecht Von Haller (Freeman & Chapleau, 2013). However, in 1960 HRV became understood as a way of testing the autonomic nervous system when it was reported that HRV

could be used to measure ANF, and its decrease was accompanied with autonomic failure (Sharpey-Schafer & Taylor, 1960). Since then, HRV has been used to help diagnose certain disorders like congestive heart failure and orthostatic hypotension, as well as predict mortality in patients with acute myocardial infarctions (Malik et al., 1996).

Physiology:

The anatomy of the ANS pertaining to HRV includes preganglionic PNS neurons (nerve cells), with cell bodies located in the medulla oblongata of the brain stem. These neurons extend down to the cardiac ganglia on the heart via the vagus nerve (Freeman & Chapleau, 2013). On the heart, there are postganglionic fibers in the cardiac ganglia that contain nicotinic cholinergic receptors. These receptors are stimulated by a chemical neurotransmitter called acetylcholine (ACh) released from the preganglionic fibers (Freeman & Chapleau, 2013). The contractions of the heart are paced by two nodes, the sinoatrial (SA) and atrioventricular (AV) nodes, which are innervated by the postganglionic PNS fibers. This innervations involves Ach, from the postganglionic PNS fibers, binding to muscarinic receptors in the nodes to decrease contractility and HR (Freeman & Chapleau, 2013). Due to the rapidity of these interactions, cardiovagal nerve activity can modulate R-R intervals in a beat-to-beat manner (Freeman & Chapleau, 2013).

Testing:

Time domain analysis uses the distance between one R wave of a QRS complex from the following adjacent R wave on an ECG; this distance is considered an R-R interval and is

measured in time (ms) (Malik et al., 1996). This interval measurement is compared to the next consecutive R-R interval, which is compared to the next consecutive interval, and so on throughout the continuous recording. Based on these measurements, variables such as average HR and average R-R interval can be determined. In addition to direct R-R measurements, other statistical measures can be calculated such as RMSSD and pNN50. RMSSD stands for the root mean square of successive differences which means the square root was taken of the mean of squared differences in R-R intervals (Malik et al., 1996). The measurement pNN50 represents the total R-R interval differences that are greater than 50ms divided by all R-R intervals. RMSSD and pNN50 can be calculated from small segments of ECG data (less than 5 minutes), and are used to estimate HF variations or PNS activity (Malik et al., 1996).

Frequency domain, also known as power spectral analysis, takes the variance recorded and translates it as a function of frequency through specific algorithms (Freeman & Chapleau, 2013; Malik et al., 1996). This method of analysis forms prominent peaks which are then divided into frequency bands (Freeman & Chapleau, 2013). The highest frequency peak is known to mirror oscillations that can be quantified to measure the response of the heart to vagal nerve activity (Freeman & Chapleau, 2013). Conversely, the low frequency peak reflects primarily efferent sympathetic activity (Freeman & Chapleau, 2013).

Cardiac Autonomic Function

Background:

It is widely accepted that autonomic function represents SNS and PNS activity and is analyzed via HRV; however, many scientists continue to question the various methodologies and their validity (Pinna et al., 2007). Frequently, SNS and PNS activation are analyzed by HRV alone and how it changes in response to certain stimuli. These stimuli have included pharmacological administrations, specific respiration and body positioning (Freeman & Chapleau, 2013). While measuring HRV, much information about the autonomic nervous system can be obtained when observing the cardiovascular changes after applying a stimulus. Though these methods appear valid, recent studies have shown that the non-invasive finger clamp technique provides equally accurate and consistent results (Freeman & Chapleau, 2013; Kurki, Smith, Head, Dec-Silver, & Quinn, 1987; Parati, Casadei, Groppelli, Di Rienzo, & Mancia, 1989).

At rest:

During a resting state, it is generally understood that healthy individuals have high activation of the PNS, and low activation of SNS (O'Brien, O'Hare, & Corral, 1986; Seals et al., 2001). Specifically, PNS dominance at rest causes HR to decrease to a resting value of

about 70 bpm, when it would otherwise be around 100 bpm as a result of SNS dominance (Malik et al., 1996; Patel et al., 2013). In certain instances, there is an additional resting PNS influence on the heart causing resting values to get as low as 40 bpm, similar to that seen in endurance athletes (Martinelli et al., 2005). When analyzing HRV, this high PNS activity is interpreted as a higher vagal tone, meaning a high level of efferent vagal activity (Freeman & Chapleau, 2013; Malik et al., 1996).

HRV and BP changes in response to body position changes are examined to gain an understanding of how the ANS adjusts to a stimulus while the body is in a resting state. Cooke et al. (1999) studied the effects of different body positions on autonomic function at rest when tilting at supine, 20°, 40°, 60°, 70°, and 80° tilts and found significant increases in systolic and diastolic BP, and mean arterial pressure (MAP). Also, significant decreases were shown in end tidal carbon dioxide concentrations, respiratory rate, and R-R intervals (meaning higher HR) during high tilts (Cooke et al., 1999). These same investigators also reported that 3 of their subjects experienced pre-syncope or early signs of a loss of consciousness, within a few minutes at either 60° or 80° tilts. These data collectively show that changing body position via head-up tilt elicits an ANF shift towards increased sympathetic stimulation and decreased vagal stimulation (Cooke et al., 1999).

Other studies have assessed the autonomic nervous system at rest in athletic populations in addition to clinical populations and healthy controls. For example, one investigation attempted to assess HRV in XC bikers at rest (Molina et al., 2013). Unfortunately, the findings of that study were inconclusive; the bikers did show obvious

bradycardia, but no clear indication of it being caused by either enhanced PNS or decreased SNS activity. On the other hand, Furlan et al. (1993) compared the HRV of elite swimmers at the peak of their yearlong program to their HRV after a month and a half of rest, (detraining). What they found was that both groups had bradycardia, but the detrained athletes had a greater high frequency power spectrum (HF) component, and the trained athletes showed higher low frequency power spectrum (LF) values (Furlan et al., 1993). The HF and LF components represent efferent vagal (PNS) and efferent sympathetic (SNS) activity, respectively (Malik et al., 1996). In other words, the detrained athletes had strong PNS stimulation at rest, explaining the observed bradycardia, but the trained athletes showed bradycardia and high SNS stimulation, indicating that a complex relationship develops between the cardiovascular and nervous systems after chronic aerobic training (Furlan et al., 1993).

However, a different study showed that endurance athletes had higher vagal stimulation at rest compared to sedentary individuals (Sacknoff, Gleim, Stachenfeld, & Coplan, 1994). Unfortunately, this study also reported conflicting results from two different methods used to measure HRV, indicating there may have been experimenter error. Martinelli et al. (2005) also studied HRV in elite cyclists and sedentary controls via the same two, commonly used, methods (time and frequency domains), at supine and during a 70° tilt. The results of their study were that athletes showed significantly greater HRV, both at rest and when tilted, than healthy control subjects (Martinelli et al., 2005). Other assessments of HRV at rest and during seven minutes of cycling exercise following a 12 week aerobic

training program, showed greater HRV in the endurance trained individuals both at rest and during exercise (Carter, Banister, & Blaber, 2003). Even in elite endurance athletes, HR and SNS activity both increase in response to stressors such as exercise, mental stress, or fear, though the responses typically differ based on training status (Goldstein & Kopin, 2008). This led to the conclusion that regular cycling training does not result in any negative alterations in ANF as suggested by data from previous studies (Martinelli et al., 2005).

Not only has ANF been shown to be greater in endurance athletes compared to controls, but several studies have also compared ANF to more specific measures of exercise performance, mainly VO_{2max} . For example, Boutcher and Stein, (1995) showed that individuals with greater HRV at rest made about 20% more improvements in VO_{2max} after 12 weeks of training compared to subjects with lower HRV. Other data indicate that about 27% of the VO_{2max} increases made after 8 weeks of training are results of greater resting HRV (A. J. Hautala et al., 2003). Furthermore, a study assessed ANF in elite athletes and reported a strong positive correlation between SNS control and specific field-based measures of exercise performance (West, Romer, & Krassioukov, 2013). Since XC bikers have shown to go above lactate threshold for many brief periods during races and have similar VO_{2max} values to road bikers, these data provide justification for the need to assess the acute ANF effects after the two forms of cycling. Hence, it seems there is a clear connection with HRV and aerobic performance, which is a key factor for XC and road biking success. However, many of the aforementioned studies used methods of measuring HRV that are not very

accurate or reliable, like portable polar HR monitors, and therefore their validity is in question.

During Exercise:

Recently, many studies have been published involving accurate and reliable assessments of ANF during and in response to various forms of exercise. Unfortunately, formulating conclusions from the literature involving ANF during exercise is difficult because of the variety of protocols that can be used to administer exercise. For instance, one study reviewed many articles in which ANF was recorded before and after different exercise interventions and concluded that, in general, aerobic exercise can improve ANF in post myocardial infarction patients (Oliveira et al., 2013); though the exact mode, duration, and intensity necessary for improvements could not be determined. However, Oliveira et al. (2013), noted that most exercise studies focus on aerobic exercise bouts, mainly because of the stress that is placed on the cardiovascular system, though others have used dynamic resistance and isometric exercises as well. On the contrary, it was also reported that some studies showed no changes in HRV after regular cycling at 70% HR reserve (Oliveira et al., 2013). Still, these studies were based on clinical populations and would expectedly show different results compared to healthy or athletic populations.

There have been a growing number of research articles analyzing ANF during exercise in healthy and individuals, most with relatively short exercise interventions. For instance, 10-min cycling bouts have been used with intensities at 50% VO_{2max} to assess HRV

compared to standing upright and passive recovery (Kamath, Fallen, & McKelvie, 1991). Kamath et al. (1991) reported that even with a relatively light exercise protocol, the ratio of LF to HF hertz decreased during exercise, and exercise was the only condition to lower LF values. This means that SNS activity actually seemed to decrease during exercise, contrary to what one would anticipate. Another study also measured HRV during an incremental exercise test and reported that only PNS indication changed during most exercise (Yamamoto, Hughson, & Nakamura, 1992). In other words, PNS indications increased as exercise intensity increased, up to about 60% ventilatory threshold, whereas SNS indicators showed no change until 110%. Additional studies used healthy males and both static and dynamic exercise bouts at varying intensities. One study in particular had subjects perform isometric knee extensions at 30% maximum voluntary contraction, and two different dynamic exercises, one at 30% VO_{2max} , the other at 60% VO_{2max} so that BP was the same as the recorded values during static exercise. The results showed that static exercise increased HRV significantly more than both forms of dynamic exercise, while increasing both SNS and PNS influence (Gonzalez-Camarena et al., 2000). The authors also noted vagal withdraw and indications of increased SNS modulation during the higher intensity dynamic exercise. However, there were problems in the overall interpretation of the spectral analysis data collected at an exercise intensity of 60% VO_{2max} , hindering the validity of the reports of the study (Gonzalez-Camarena et al., 2000). Other studies also analyzed ANF during isometric contractions; one in particular reported a significant increase in muscle sympathetic activity after only one min of performing an isometric hand grip exercise (Seals, Chase, & Taylor,

1988). The reason for noting the results of isometric hand grip studies is because such activity is said to be present with mountain biking and not road biking (Wang & Hull, 1997). In addition, such activity has been known to increase HR and BP systemically (Helfant, De Villa, & Meister, 1971).

After Exercise:

Many authors have demonstrated long term effects of exercise and acute effects during exercise on ANF, but fewer have assessed ANF after exercise. It is known that HR and BP return to baseline after exercise and at differing rates for different individuals; however, the mechanisms behind these rates of decline are still being investigated. Furthermore, one study reported that, even after BP and VO_{2max} had returned to baseline, the ANS was still being modulated for at least 30 min after maximal exercise (Hayashi, Nakamura, & Muraoka, 1992). Another study tested HRV for 30 min after exercise and reported a lack of full ANF recovery with an intensity of only 65% VO_{2max} for 30 min (Heffernan et al., 2006). In addition to this, it has also been stated that ANF remains altered as long as 1 hr after 20 min of cycling exercise at high intensity (80% anaerobic threshold) compared to moderate intensity (50%) (Terziotti et al., 2001). Another investigation had subjects cycle for a relatively long duration of 60 min, at a moderate intensity (65% VO_{2max}) and measured HR via surface ECG recordings from limb leads for 10 continuous min at 1, 3, 6, and 22 hr post exercise (Poher, Braun, & Freedson, 2004). The results showed that one hour of acute endurance exercise causes greater PNS control of HR for all time points

measured. The authors also pointed out that this shift was similar in magnitude as seen when comparing endurance trained athletes to healthy controls (Poher et al., 2004). However, this protocol still used an indoor, stationary lower body ergometer, and eliminated any potential sources of additional stressors or muscle contractions that occur during activities like off-road cycling.

When examining studies involving the ANS and outdoor, adventure-like activities more closely related to XC biking, there are only a couple that contribute valuable information. One study was conducted in which ANF was assessed in subjects before and 30 min, 24, and 48 hr after a 46 km rocky trail run (Bernardi, Passino, Robergs, & Appenzeller, 1997). Trail running is to long distance road running, what XC mountain biking is to road cycling; meaning mode and volume are similar, but terrain is very different. The authors reported that acutely (30 min post) HRV, SBP, and DBP all dropped significantly, and then only SBP and DBP remained lower until the 48 hr time point. The changes in HRV were said to indicate that sympathetic modulation was still preserved though exercise had ceased, but not enough to counter the hypotension illustrated by the decreases in systolic and diastolic BP (Bernardi et al., 1997). The biggest issue with this study, though, is that they used an activity that is far greater in exercise duration (average running time per subject was 6 hr) than nearly all others and therefore is not closely comparable with other literature. One other study assessed HRV before and after a prolonged outdoor activity in cross country skiers and found that vagal outflow was attenuated for several hours after exercise, meaning a decrease in PNS stimulation (Hautala et al., 2001). Unfortunately, the methods used to

measure HR data (portable polar HR monitors later interpreted via spectral analysis) are subpar compared to the commonly used ECG in other articles, (Hautala et al., 2001). In addition, the exercise duration used by Hautala et al. (2001) was also much greater than most ANF studies at an average time per subject of 4.5 hrs.

Mountain biking

The sport of mountain biking has grown in popularity to the point where mountain bikes consistently outsell road bikes annually. Mountain biking became recognized as a sport in the mid 1970's, however there is still a relatively small pool of scientific literature on the sport. More recently, some researchers have examined the resting and exercising physiology of elite XC bikers. For instance, Impellizzeri & Marcora (2007) tested the aerobic capacity of elite XC bikers and found they have VO_{2max} values averaging between 66 to 78 $ml \cdot kg^{-1} \cdot min^{-1}$. Mountain bikers have also produced maximum power output values of 413 watts (W), and relative power outputs of 6.3 W per kg of body weight ($W \cdot kg^{-1}$) at the end of a VO_{2max} test (Lee et al., 2002). To put this in perspective, elite road cyclists have been shown to have similar VO_{2max} and max power values (73 $ml \cdot kg^{-1} \cdot min^{-1}$ and 430 W on average, respectively), but significantly lower relative power values averaging 5.8 ($W \cdot kg^{-1}$) (Lee et al., 2002; Prins, Terblanche, & Myburgh, 2007). As expected, these performance test values match observed physiological characteristics during acute XC biking. Impellizzeri & Marcora (2007) showed that during an elite XC mountain bike race, a total of about 80% of

the duration is spent above lactate threshold, which is when exercise intensity becomes too great to effectively renew cellular energy and eliminate metabolites from the cells (Faude, Kindermann, & Meyer, 2009). In endurance exercises, such as cycling, these processes need to be maintained for prolonged periods of time so athletes can maintain their pace without slowing down in a race. If an athlete goes above lactate threshold, metabolic waste products start to accumulate in the blood and muscle, leading to an eventual decline in performance if the intensity does not decrease (Faude et al., 2009). Because lactate threshold is a limiting factor of endurance performance, competitive endurance athletes aim to stay at or directly below lactate threshold to prevent performance decreases during competitions. Since most of a XC biking race is spent above lactate threshold, it can be argued that the activity profile intensity of XC biking is higher than traditional road cycling. Hence, based on the physiological characteristics measured in mountain and road bikers, it is likely that these two forms of cycling stress the cardiovascular system differently.

Summary:

Both XC biking and road cycling are physically demanding cardiovascular activities, each with their own set of challenges. Elite athletes in both sports have shown similar high aerobic capacities and power outputs, but mountain biking includes isometric contractions and a higher adrenaline stress. Autonomic function tests allow researchers to analyze how the SNS and PNS both control HRV. At rest, PNS influence lowers HR to 70 bpm and as

low as 40 bpm in elite endurance athletes. Studies that tested ANF in XC bikers showed inconclusive results for PNS and SNS activation. However, most studies on elite athletes showed enhanced vagal tone at rest compared to untrained controls. Studies of ANF during dynamic exercise have produced mixed results of whether the PNS or SNS is more active, but most agree that PNS activation is significantly higher post exercise compared to rest. It is still unclear what the autonomic function and blood pressure responses will be after an activity, such as XC mountain biking, which includes isometric contractions, and heavy cardiovascular demands.

Methods

Subjects

A total of four subjects were recruited for the study through email lists and word of mouth. All subjects were males between the ages of 18 and 24, who regularly participate in road and mountain biking. Subjects were excluded if they did not have a minimum of two years of experience in road and mountain bike riding, and did not complete at least two road and mountain bike rides within 30 days of the first visit. Subjects were cleared for physical activity in accordance with the American College of Sports Medicine (ACSM) health screening questionnaire. The study was approved by the Appalachian State University Institutional Review Board.

Experimental Design

Subjects made three visits to the lab, all during the same time of day to avoid any diurnal variations and separated by at least 48 hr. Subjects were also instructed to avoid any caffeine or alcohol for 3 hr and any riding for 24 hr prior to each visit. ANF was measured on a separate day (Baseline) and before and after two modes of cycling. The modes were an indoor cycling ride, and an outdoor XC mountain bike ride using the subjects' own bikes and

equipment. During visit one subjects completed the informed consent process, health screening, and inclusion/exclusion criteria questionnaires followed by height, weight, and the Baseline ANF measurement via head-up tilt. Resting HR and BP measures were taken during the supine phase of the Baseline tilt test. For visits two and three, subjects completed either the indoor (Road) or outdoor (MTN) ride in a randomized fashion, with ANF measured before and as soon as possible after, making up the Road pre, Road post, MTN pre and MTN post measures.

Autonomic Function Testing

Each head-up tilt ANF test involved the subjects being secured to a table while hooked up to a finger and upper arm blood pressure cuff. In addition, a single lead ECG, composed of three electrodes, was placed just underneath the mid line of each clavicle and at the 5th intercostal space on the subject's left side. The test consisted of three phases: supine, tilted (80°), then back to supine, with all measurements recorded continuously for no more than 10 min per phase. Beat-to-beat BP data were collected by a Finometer™ from finger plethysmography (FMS, Amsterdam, Netherlands), while the ECG data was transmitted and saved to a nearby laptop in real time. After entering subject height, weight, and sex into the Finometer™, it was then calibrated in accordance with standard operating procedures immediately prior to the start of the test. Brachial BP was obtained by the Finometer™ through the use of the integrated brachial BP cuff and reconstruction of brachial BP

waveforms from finger arterial waveforms through the application of an inverse transfer function, a level correction, a waveform filter, and a level calibration (Guelen et al., 2003). BP data was averaged for every minute and the 2nd to last minute per each phase was used, while a period of at least two minutes of ECG data was used from the end each tilt phase. All data was processed via WinCPRS software (Absolute Aliens, Turku, Finland).

Exercise Protocols

The indoor ride consisted of 30 min of cycling at 70% age calculated HR_{max} on a stationary cycle ergometer (Lode Excalibur; Medgraphics, St Paul, Minnesota, USA) which was determined as an applicable method to elicit potential ANF changes based on previous literature (Gonzalez-Camarena et al., 2000; Terziotti et al., 2001). The bike was adjusted to match the exact feel of the subjects' road bikes prior to the pretest to avoid discomfort during the ride. Subjects maintained a cadence between 90 and 100 rpm and were allowed water ad libitum, while intensity was maintained by adjusting the wattage. HR was monitored continuously during the indoor ride via Polar Heart Rate Monitor (Polar Electro, Inc., Woodbury, NY, USA). Ambient temperature during the indoor ride and tilt tests was maintained at a constant 75°F.

The outdoor ride was performed offsite at Rocky Knob Bike Park in Boone, NC. The trails ridden were self-selected by the subjects based on what loop, or combination of loops, they were able to complete between 20 and 40 min. Subjects recorded the start and finish

time via their own Garmin GPS ride trackers. Since the ride took place offsite, the time between the completion of the outdoor ride and start of the MTN post tilt test was recorded.

Results

Subject characteristics are reported in Table 1. Average riding experience was greater than two years for both road and mountain biking. MTN ride duration, outdoor temperature, and time gap between ride completion and test start are shown in Table 2. The MTN ride averaged 32.8 min in duration, with an average ambient temperature of 40.8°F.

Table 1: Subject characteristics. ($n = 4$)

	Mean \pm SEM
Height (m)	1.8 \pm 0.02
Weight (kg)	68.0 \pm 3.29
Age (yr)	20.8 \pm 1.25
Resting HR (bpm)	50.8 \pm 0.48
Resting SBP (mmHg)	121.3 \pm 3.77
Resting DBP (mmHg)	68.3 \pm 2.10
Road Exp (yr)	>2 \pm 0
MTN Exp (yr)	>2 \pm 0.25

Table 2: MTN ride characteristics.

	Mean \pm SEM
Duration (min)	32.8 \pm 1.1
Temp (°F)	40.8 \pm 7.4
Time to test (min)	14.5 \pm 1.9

Mean SBP measures during each tilt test phase for all trials are illustrated in Figure 1. Other than MTN Pre, all trials appear to decrease noticeably during tilt and increase during return. For both MTN and Road, pre ride values are elevated from Baseline and post ride

are decreased from pre-ride, though MTN post measures are only slightly below pre measures at return, whereas Road post remains considerably lower.

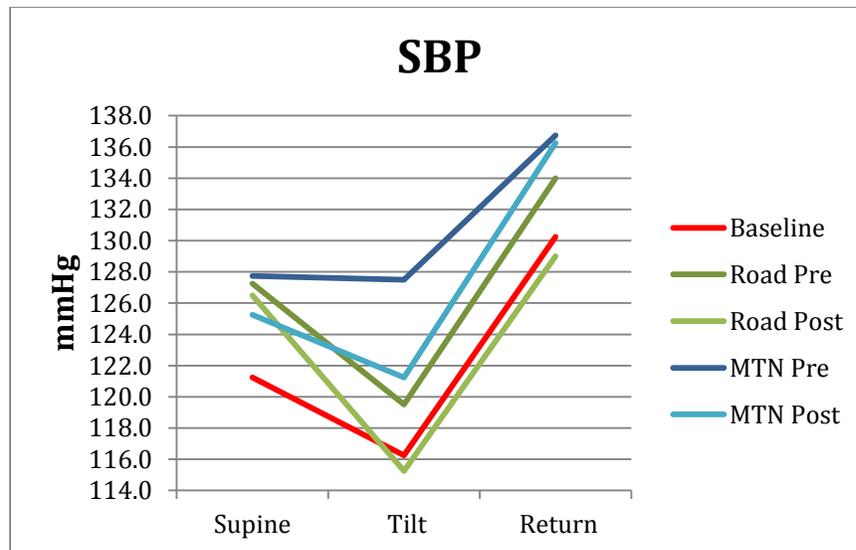


Figure 1: Systolic blood pressure during tilt test for all trials.

Mean DBP measures during each tilt phase for all trials are outlined in Figure 2. All trials appear to increase at tilt and decrease at return, except baseline which slightly increases at return. Both MTN and Road pre measures increased from Baseline. MTN post increases from pre, whereas Road post decreases from pre except during supine where it elevated.

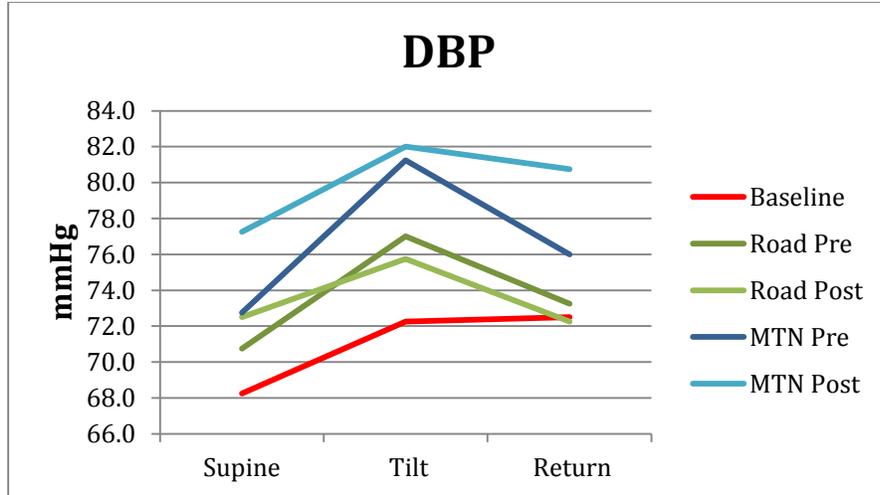


Figure 2: Diastolic blood pressure during tilt test for all trials.

Average values for MAP at each tilt test phase for all trials are shown in Figure 3.

Baseline MAP is lower than all other trials at supine and tilt. Other than Road Post and MTN Pre, MAP for all trials increased steadily throughout the test. Except during supine, Road Post decreases largely from Road Pre. MTN Post is largely increased at supine compared to Pre, then goes below at tilt, and increases again at return.

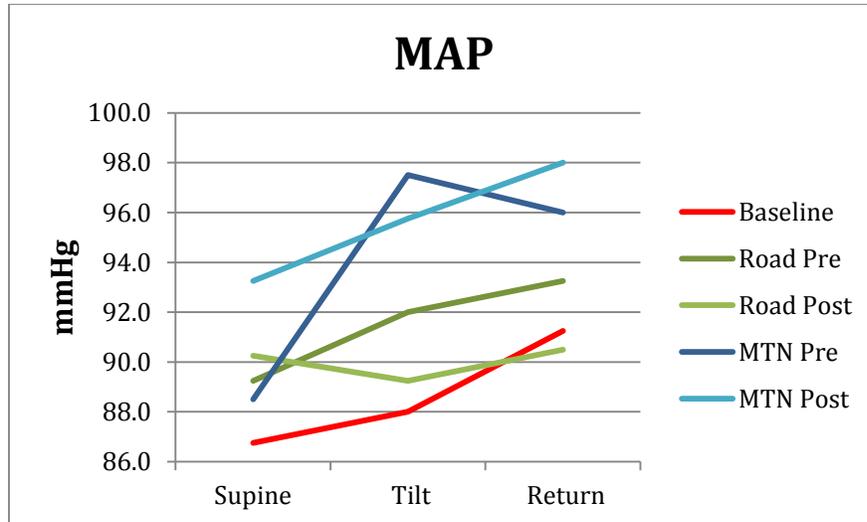


Figure 3: Mean arterial pressure during tilt test for all trials.

All mean heart rate measures increased from supine to tilt and decreased below supine at return (Figure 4). Baseline, MTN Pre, and Road pre appear the same for all time points. Both Road and MTN Post values increased similarly compared to Pre values for all time points, though Road Post appears consistently higher than MTN post.

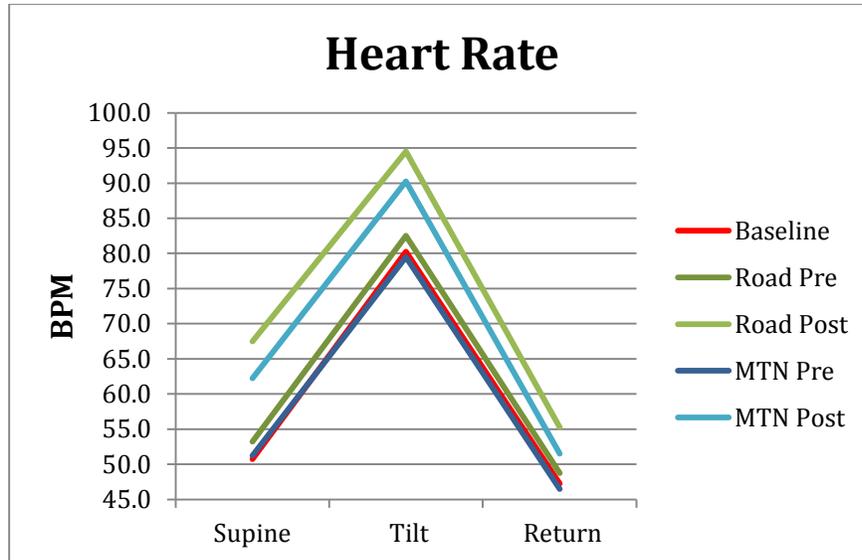


Figure 4: Heart rate during tilt test for all trials.

Average stroke volume values for Baseline, Road and MTN Pre are similar for all time points and show a large decrease during the tilt phase compared to supine and return as seen in Figure 5. Road Post is also similar to Baseline, and Road and MTN Pre values except at the tilt time point where it is noticeably lower. The Road Post trial appears to have a small, but consistent decrease from the Road Pre trial throughout all time points. MTN Post is considerably lower from all other trials during all 3 time points.

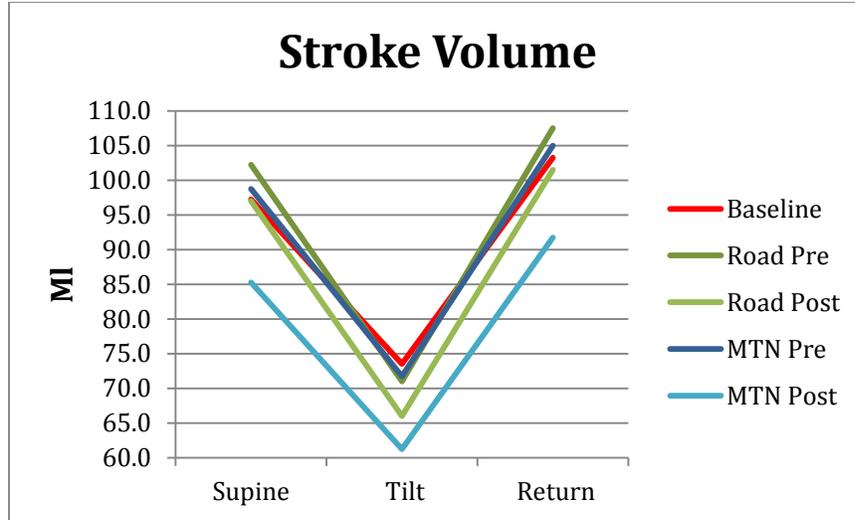


Figure 5: Stroke volume during tilt test for all trials.

Mean cardiac output values for all trials appear to increase during the tilt phase and become lowest at the return phase except for the Road Post trial which is the only trial to show a decrease from supine to tilt (Figure 6). Other than during supine, the MTN Post trial appears slightly lower than MTN Pre. Road Post is noticeably higher throughout the test than all other trials including Road Pre. During the supine phase, both MTN and Road Post measures were largely elevated from Pre measures.

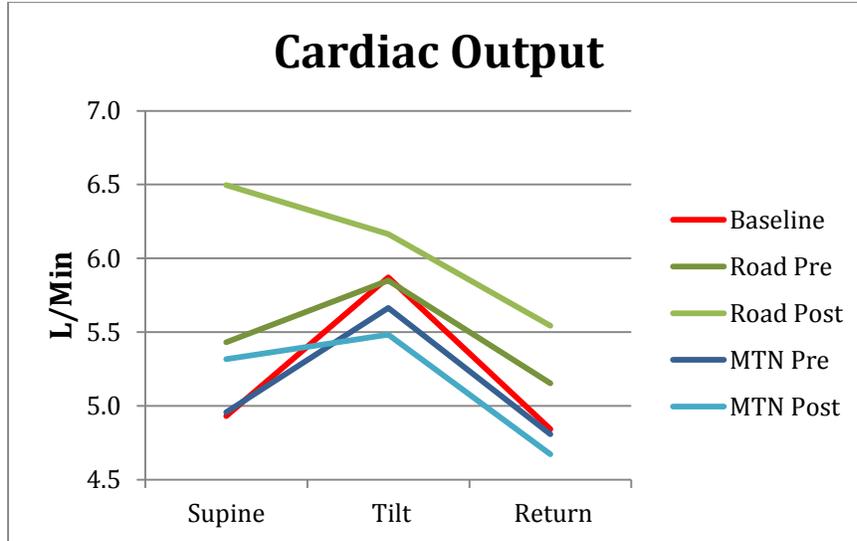


Figure 6: Cardiac output during tilt test for all trials.

Except for the Baseline trial, average total peripheral resistance values for all trials show little to no change between supine and tilt, followed by an increase from tilt to return (Figure 7). Though MTN Pre was largely greater for all time points than Baseline, MTN Post showed minimal decreases compared to Pre, whereas Road Post showed considerable decreases for all time points compared to Pre.

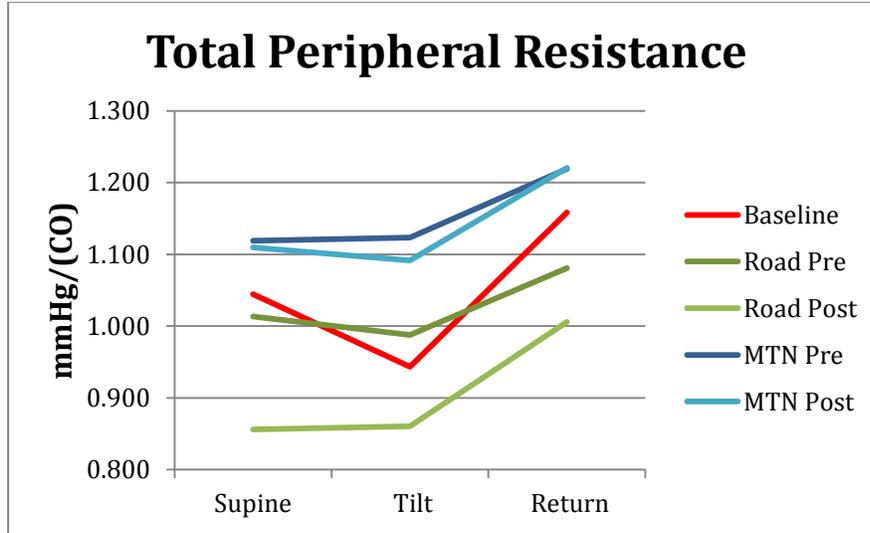


Figure 7: Total peripheral resistance during tilt test for all trials.

All trials show total power decreases of varied magnitudes during tilt compared to supine and return (Figure 8). Road Post shows little to no change from Road Pre throughout all time points. MTN Pre and Post appear the same at supine, though MTN Post decreases before largely increasing at the tilt and return time points respectively. Baseline appeared higher than all other trials during supine and tilt, and lower than all trials at return.

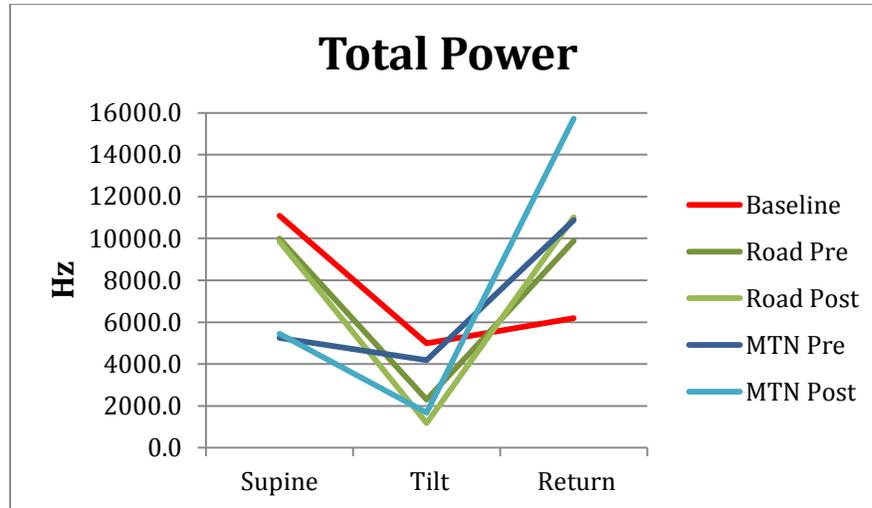


Figure 8: Total power during tilt test for all trials.

Except for the Baseline and MTN Pre trials, mean LF for all other trials decrease noticeably at tilt before increasing at return (Figure 9). Road and MTN Post measures both start considerably higher than Pre measures at supine, then decrease below pre measures at tilt. MTN Post measures increase largely more than all other trials between tilt and return.

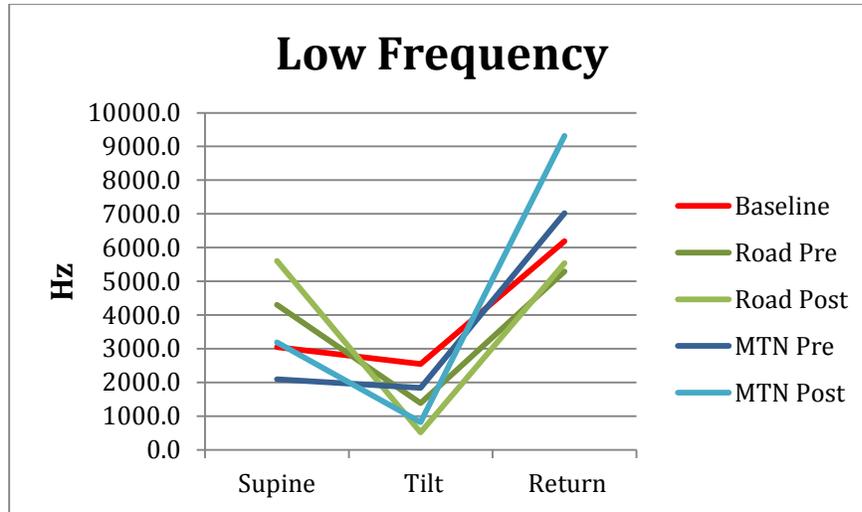


Figure 9: Low frequency power during tilt test for all trials.

Mean HF measures for all trials decrease at tilt compared to supine and return (Figure 10). Both Road and MTN Post measures are largely lower at supine relative to Pre measures. For both Road and MTN, Post measures appear to be the same as Pre measures for the tilt time point. Despite starting below Pre measures at supine, MTN Post rises considerably above MTN Pre at return.

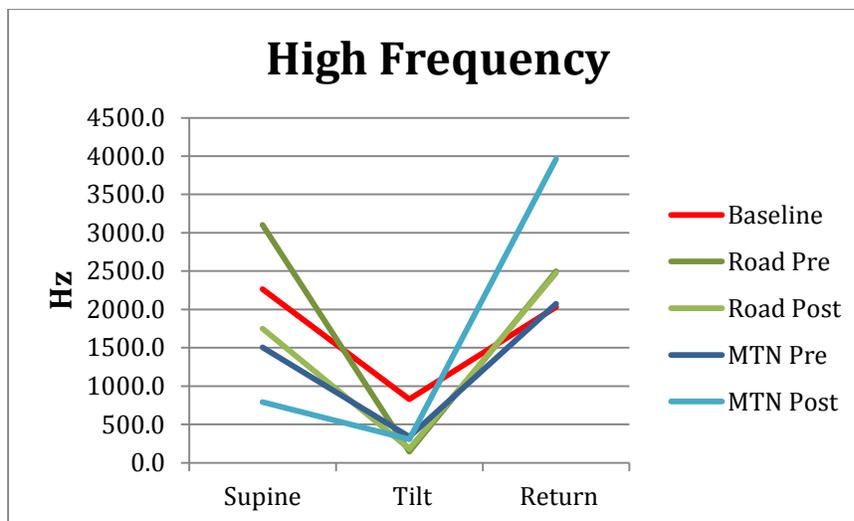


Figure 10: High frequency power during tilt test for all trials.

Mean ECG data recorded from all subjects for each tilt phase is represented in Table 3. Root mean square of successive R-R differences (RMSSD) decreased during the tilt phase then increased above tilt and supine values during the return phase. The amount of successive R-R intervals that differ by 50ms or more over the total number of R-R intervals (pNN50), also decreased in all trials except for Road Pre during tilt and increased above tilt and supine values during return. For all trials except MTN Post, the LF/HF ratio increased at tilt before decreasing below tilt and supine levels at return. MTN Post is the only trial in which the LF/HF ratio decreased during tilt compared to supine. The normalized low frequency data (nLF) increased during tilt before decreasing to near supine levels in all trials except Road Post where it slightly decreased at tilt. Besides Road Post which stays the same for supine

and tilt, the normalized high frequency data (nHF) decreased during tilt and increased at return, though the decrease in the MTN Post trial is miniscule.

Table 3: Tilt test ECG data represented as mean \pm SEM.

	Supine	Tilt	Return
	Baseline		
RMSSD	122.8 \pm 16.3	54.3 \pm 29.1	134.5 \pm 10.0
pNN50 (%)	56.8 \pm 12.7	17.6 \pm 12.4	56.9 \pm 15.2
LF/HF	152.3 \pm 79.3	1304.8 \pm 406.5	127.2 \pm 47.2
nLF (Hz)	0.47 \pm 0.13	0.85 \pm 0.09	0.50 \pm 0.08
nHF (Hz)	0.50 \pm 0.13	0.15 \pm 0.09	0.48 \pm 0.08
	Road Pre		
RMSSD	104.0 \pm 13.4	21.5 \pm 5.6	112.3 \pm 23.8
pNN50 (%)	55.0 \pm 8.7	5.5 \pm 3.8	51.3 \pm 13.1
LF/HF	180.0 \pm 114.1	1831.2 \pm 781.7	179.9 \pm 82.9
nLF (Hz)	0.48 \pm 0.14	0.88 \pm 0.06	0.54 \pm 0.12
nHF (Hz)	0.49 \pm 0.13	0.11 \pm 0.06	0.45 \pm 0.12
	Road Post		
RMSSD	70.8 \pm 23.1	33.3 \pm 13.7	116.5 \pm 19.9
pNN50 (%)	36.2 \pm 13.6	2.1 \pm 0.9	58.7 \pm 11.6
LF/HF	492.9 \pm 184.5	1116.1 \pm 690.5	247.7 \pm 100.9
nLF (Hz)	0.76 \pm 0.07	0.72 \pm 0.14	0.62 \pm 0.10
nHF (Hz)	0.23 \pm 0.07	0.23 \pm 0.11	0.37 \pm 0.10
	MTN Pre		
RMSSD	101.5 \pm 16.0	32.3 \pm 8.4	116.5 \pm 29.0
pNN50 (%)	54.8 \pm 6.8	8.2 \pm 4.1	59.9 \pm 12.1
LF/HF	153.8 \pm 55.6	686.5 \pm 234.7	376.8 \pm 268.4
nLF (Hz)	0.54 \pm 0.10	0.81 \pm 0.07	0.57 \pm 0.14
nHF (Hz)	0.45 \pm 0.10	0.18 \pm 0.07	0.41 \pm 0.13
	MTN Post		
RMSSD	62.5 \pm 9.0	30.8 \pm 17.4	152.3 \pm 33.9
pNN50 (%)	32.8 \pm 9.0	3.4 \pm 1.8	70.9 \pm 4.5
LF/HF	557.0 \pm 311.2	492.4 \pm 262.8	226.9 \pm 131.8
nLF (Hz)	0.71 \pm 0.11	0.74 \pm 0.07	0.57 \pm 0.10
nHF (Hz)	0.28 \pm 0.11	0.24 \pm 0.06	0.42 \pm 0.10

Discussion

Despite the lack of statistical significance, there exist several observations of the present study worth mentioning. The main findings of this study show that, following an indoor cycling ride, CO appears consistently greater than after a MTN ride, while TPR appears consistently lower. Other noteworthy findings include DBP being elevated following an outdoor XC ride for all phases of the tilt test, whereas measures following the indoor ride had shown decreases for the later two time points.

Overall appearances among the trials during the tilt test were increases in DBP, HR, and CO during tilting and decreases in SBP, SV, TP, LF, HF, RMSSD, pNN50, and nHF though changes in SBP, CO, and nHF were not consistent for all trials. Aside from an unusual increase in the MTN Post trial in the return time point, there were no distinguishable changes in TP between Pre and Post for either Road or MTN. TP represents global index of cardiovascular autonomic modulation, and is typically seen elevated and lowered during high vagal and sympathetic activation respectively (Sanudo et al., 2013). These trends are in disagreement with previous studies, which reported a decrease in TP immediately following exercise (Gonzalez-Camarena et al., 2000; Heffernan et al., 2006). This discrepancy, when combined with the lack of consistent responses between phases, indicates a high variability between subjects, and hence low validity of the total power results.

The increased DBP and HR trends during tilt in the present study match the results of Bahjaoui-Bouhaddi, Henriet, Cappelle, Dumoulin, & Regnard (1998) who reported increases

in HR, systolic, diastolic, and mean arterial pressure increases when tilted. However, our results indicate a decrease in SBP at tilt which is contrary to other studies that measured BP changes with tilting (Bahjaoui-Bouhaddi et al., 1998; Cooke et al., 1999). This phenomenon is difficult to explain especially when it is taken into account that Bahjaoui-Bouhaddi et al. (1998) also used elite cyclists but it may be from large variability as a result of our small sample size.

The LF/HF ratio increased greatly during tilting in all trials except MTN Post where it decreased. Although minimally in MTN Post, nLF also increased during tilting in all trials except Road Post where it decreased. These findings are unique since other studies showed significant increases in LF/HF and nLF during the tilt phase (Martinelli et al., 2005). LF and the LF/FH ratio for MTN and Road Post during supine were also considerably higher than Pre trials. Our results also show that HF was decreased in the Road and MTN Post trials compared to Pre for the supine time point. Other studies reported decreases in HF, LF, and LF/HF ratio during dynamic exercise compared to resting values (Gonzalez-Camarena et al., 2000; Kamath et al., 1991). Furthermore, Heffernan et al. (2006) reported a lack of ANF recovery for up to 30 min post exercise. As a result, one could expect the ANF changes seen in the Post measures during the supine phase to be from exercise. Since HF and LF represent parasympathetic (vagal) and sympathetic tone respectively, our results match those of Murrell et al. (2007) who reported a significant increase in sympathetic tone coupled with a decrease in parasympathetic tone following exercise (Murrell et al., 2007). Other authors reported increases in SNS activity after as little as one minute of isometric exercise at

intensities as low as 25% (Seals et al., 1988). This could explain the increased LF values seen during the supine phase in the MTN Post group since XC biking requires many isometric contractions (Impellizzeri & Marcora, 2007). However, the similar change also seen in the Road Post trial indicates this is likely not the case.

The observed decrease in SV during tilting throughout the trials is likely due to a decrease in venous return compared to supine which is expected based on previous studies (Takahashi, Hayano, Okada, Saitoh, & Kamiya, 2005). HR was shown to increase during tilting here and in other studies in order to maintain CO and overall blood flow in response to the decrease in SV (Cooke et al., 1999; Martinelli et al., 2005; Takahashi et al., 2005). After the road ride, however, subjects appeared to show a substantial increase in CO compared to other trials, especially during the supine phase, even though the MTN Post trial also showed increased CO during supine compared to Pre. This could partially be a result of the test starting almost immediately after completion of the road ride, compared to 14 min, on average, after the completion of the MTN ride. Under normal circumstances exercise would increase CO mainly by increasing HR, explaining the increases seen in the post measures during supine (Takahashi et al., 2005). Though the MTN Post trial showed greater changes in HR and SV from pre ride measures, Road Post showed higher overall HR averages than all trials, and higher SV values compared to MTN Post. Based on the equation: $CO = SV \times HR$, the high CO values for Road Post can be explained from the high HR and relatively similar SV values (Thiele, Bartels, & Gan, 2015).

The large decrease in TPR from Road Pre to Road Post can be explained by the natural decrease in vascular resistance during exercise and delayed increase during recovery (Coats et al., 1989; Halliwill, Taylor, & Eckberg, 1996). This delay in TPR recovery is further illustrated by the slight increases seen in all but Baseline towards the later points of the tests. The different TPR response observed between the MTN Pre and Post trials may in part be explained by the time delay between ride completion and the test start. This is unlikely, however, because of the large difference in TPR between Road Pre and Post still present in the later time points, while MTN Pre and Post remain similar. Moreover, the visual presence of an exercise effect in the last time point for Road Post means even with a time delay, there should be noticeable changes seen in the MTN Post trial. As a result the exact reasoning for this lack of TPR changes following XC biking is unknown.

One peculiar finding in the present study was a consistent increase in DBP in the MTN Post trial compared to Pre. During exercise, SBP typically increases as a result of increased SNS activity, while DBP stays the same or slightly decreases (Goldstein & Kopin, 2008; Kokkinos, 2014). Typically, vascular resistance and vasodilation recover slowly following many forms of exercise, causing a drop in BP that has shown to take several hours to return to pre exercise values (Halliwill et al., 1996; Jones, George, Edwards, & Atkinson, 2007; Kenney & Seals, 1993; Pescatello, Fargo, Leach, & Scherzer, 1991). However, Goldstein et al. (2008) stated that various physiological and psychological stressors including fear and anxiety can cause increased activation of the SNS. The noticeable DBP increase in the MTN Post trial seen in the present study may be a result of mental stressors from the

nature of the ride influencing additional SNS activity than the exercise itself. This also could have been an effect of the ambient temperature during the outdoor ride, which was drastically lower at 40°F compared to the temperature of the indoor ride which was 75°F. The lower temperature could have caused large BP changes through responses like shunting of blood from extremities and shivering. However, SBP and MAP did not show changes consistent enough to validate this possibility.

Mean SV values were distinctly lower in the MTN Post trial compared to all others throughout all time points. This observation matches other reports of decreased SV after exercise (Coats et al., 1989; Jones et al., 2007). Jones et al. (2007) attributed this decrease in SV to a sudden lack of contracting muscles which would have facilitated venous return. In our study however, there is a possible effect of different body positions between the two rides. Although no exact angles were measured, body position during the indoor ride was monitored, with all subjects leaning forward on the handlebars throughout the test. It is unknown however if the body positions of the subjects were more upright or flat during the outdoor ride which could potentially impact SV since it is known that SV decreases when upright compared to supine (Cooke et al., 1999). The possibility of subjects being more upright during the MTN ride combined being upright for the drive back to the lab gives reason to believe the MTN Post SV averages may be a result of postural changes.

Our data did not show any consistent change in MAP between phases. Under normal circumstances, MAP is expected to increase during the tilt phase, which our results only show in the MTN Pre trial (Bahjaoui-Bouhaddi et al., 1998). Similar to TP, the lack of

expected responses in MAP in all trials is likely a result of the small sample size and relatively large variability.

It is difficult to formulate definitive conclusions with the limited data in the present study; however the results do provide small indications of a possible increase in SNS activity following a XC mountain bike ride compared to an indoor road ride. This was mostly illustrated by the increase in DBP seen during the tilt test in the MTN Post trial. This finding is significant because chronic activation of the SNS increases the risk for potentially life-threatening cardiovascular events (Curtis & O'Keefe, 2002). Although endurance exercise has been shown to improve ANF, XC biking may not be the most beneficial form of aerobic exercise to improve cardiovascular and autonomic nervous system health, though additional research is needed before making this conclusion (Ribeiro et al., 2012; Seals et al., 2001; Shin, Minamitani, Onishi, Yamazaki, & Lee, 1995).

There are several limitations to this study that may have affected our data interpretation. First, the particularly small sample size ($n=4$) lowered statistical power significantly to the point where any statistical results would not be of use. As a result, it was decided among the researchers to forgo statistical analysis and instead graph out means to illustrate any potential changes between trials. Second, the subjects were not grouped by physiological parameters in any way, resulting in a large uncontrollable variability within the already small sample size. Third, there were no preliminary tilt tests to allow subjects to acclimatize to the laboratory testing, which could have resulted in increased anxiety felt by the subjects from the testing procedures. In addition, little time was provided prior to the

start of the Pre and Baseline tilt tests to eliminate any possible ANF effects from outside stressors experienced by the subjects just prior to entering the lab. Also, there was a large time between the cessation of the MTN ride and the post ride test that did not occur after the indoor ride. This hindered an accurate comparison of time points throughout the tilt test between the two post trials. Additionally, the inability to control and monitor the subjects' body positions during the outdoor ride and drive back to the lab, which could have affected heart and blood vessel activity. Finally, the use of age calculated HRmax to establish the indoor ride intensity would not have accounted for physiological differences between subjects. Though the subjects were screened for riding experience, there may still have been differences in various performance measures, such as VO_{2max} , which would have altered the effect of the intensity of the indoor ride on the subjects.

Conclusion

In conclusion, the present study discovered that TPR considerably increased following an indoor ride compared to resting, whereas no change appeared following a MTN ride. Also, DBP was consistently increased more following the MTN ride, potentially representing a greater increase in SNS activity compared to the indoor ride. As a result, XC mountain biking may not be the most beneficial form of aerobic exercise to improve ANF and cardiovascular health, though further research is needed to support this claim.

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Vita

Shaun Woerner was born in Summit New Jersey. Shaun attended William Paterson University and graduated with a Bachelor of Science Degree in Exercise Science with a Concentration in Exercise Physiology. After graduating, Shaun pursued his Master of Science Degree from Appalachian State University and graduated in August of 2015. Shaun plans to one day earn a Ph.D. in Exercise Physiology with a focus on Skeletal Muscle Physiology. Shaun's remaining parent, Paul J. Woerner Jr., and brothers Evan, Brendan and Paul Woerner live in New Jersey.

