

Cardiovascular Effects of Passive Exercise: A Comparative Study

By Ingrid Brenner*, Sai Gayathri Chakrala[±] & Holly Bates[°]

Despite its clinical use for rehabilitation, little is known about the cardiovascular effects of passive exercise. This study examined the cardiovascular response of passive compared to active exercise. Twelve healthy individuals (7 M and 5 F, aged 21 ± 1 yrs) participated in the study. Participants were randomly assigned to perform 30 min of passive or active exercise, each separated by a one-week interval. Each session involved three, 10-minute bouts of pedalling performed either passively on a Theracycle or actively on a Monark cycle ergometer. Heart rate (HR), heart rate variability (HRV), blood pressure (BP), rate pressure product (RPP), and ratings of perceived exertion (RPE) were evaluated throughout each exercise session. HR and RPP were significantly increased from baseline with passive exercise, at all pedaling frequencies. At the highest pedaling frequency, diastolic blood pressure and RPE were significantly higher in passive exercise, compared to baseline. However, these measures were significantly lower than that observed with active exercise. Sympathetic nervous system (SNS) activation and parasympathetic nervous system (PNS) withdrawal was greater with active exercise. The results of this study indicate that passive exercise places minimal stress on the cardiovascular system and may be useful to promote mobility in people with chronic diseases.

Keywords: Cardiovascular response, passive exercise, active exercise

Introduction

Passive (or forced) exercise involves movement of a body part without voluntary contraction of skeletal muscle and can be accomplished with help from an individual (e.g., a physical therapist, kinesiologist, or a caregiver) or by using a mechanized device (i.e., continuous motion machine, motion splints or motorized exercise equipment) to move a patient's joints throughout their range of motion (Shirzad et al. 2022). These devices are commonly used to treat patients with a variety of neurological conditions (for example, Parkinson's disease, stroke, spinal cord injury, multiple sclerosis and post-traumatic brain injury) or for patients following orthopedic surgery. This form of movement is usually applied as an early form of physical therapy for patient mobilization (Llano-Diez et al. 2012).

Several studies have demonstrated that passive exercise training can induce numerous physiological benefits depending upon the clinical population examined. Patients with Parkinson's disease experience a reduction in tremors immediately post-exercise as well as an improvement in overall physical fitness following training (Sackner 2012). Stroke and spinal cord-injury patients experience a reduction

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in edema, a decrease in spasticity, improved balance, increased cognition, enhanced muscle strength and an increased range of motion (ROM) (Brenner 2018). In patients with multiple sclerosis, continuous passive motion exercise (CPM) has been shown to increase mobility, gait speed, stride length and balance (Heesen et al. 2006).

As described above, most studies examining the effects of passive exercise training focus on neuromuscular effects. Perhaps, since the goal of passive therapy does not normally target the cardiovascular system, minimal information is available on the cardiovascular responses to an acute bout of passive exercise. By recruiting young, healthy participants, this preliminary investigation has been designed to determine the feasibility of the use of passive exercise as a mode of training for both healthy and clinical populations. The focus of this study is to examine the cardiovascular responses of passive exercise performed on a Theracycle (a passive exercise ergometer) in comparison to a Monark cycle ergometer (an active exercise ergometer) performed at the same pedalling frequency.

Literature Review

A review of the literature was done to examine the history of passive exercise as a treatment modality as well as the effects of one bout of passive exercise on the cardiovascular responses in healthy and clinical populations. Minimal information was available on the effects of one bout of passive exercise on the cardiovascular system and no information was available on the effects of passive exercise compared to active exercise on heart rate variability. To date, most of the research has focused on the effects of regular passive exercise training on neuromuscular function (i.e., motor function and performance) in stroke patients and/or patients with Parkinson's disease. Other studies have examined the effects of continuous passive motion following knee replacement surgery. These areas are outside the scope of this study and are not included in the literature review which follows.

The History of Passive Exercise

Historically, passive exercise treatment was introduced to enhance circulation. Herrmann and Reid (1934) describe a method known as passive vascular exercise or "pavaex" treatment that was used to treat patients with peripheral vascular disease. Use of this device involved placing the effected leg into a treatment boot made of Pyrex glass. With the patient in the supine position, the foot and leg were elevated slightly above the heart. Positive and negative pressure were rhythmically applied to the lower leg which in-turn influenced circulation. Post-pavaex treatment, patients displayed greater blood flow to the lower limb, a decrease in pain and a reduction in the incidence and extent of gangrene.

Roberts et al. (1971) investigated the effect of rhythmic passive foot flexion in patients undergoing surgery for varicose veins using a motorized foot mover. Patients performed passive flexion movements (24 to 50 flexions per minute) through a 20-to-50-degree range of motion (ROM) (depending upon flexibility) for 30 minutes prior to their surgery. These patients experienced an increase in femoral vein blood

flow, faster recovery times, and a reduction in the occurrence of postoperative deep vein thrombosis (DVT).

While these earlier studies focused primarily upon the effects of passive exercise on the vascular system, later work extended these findings to the cardiovascular system in healthy and clinical populations. However, inconsistencies are reported regarding the effects of passive exercise on the HR response. Some studies indicate no change in HR whereas others reported a slight decrease following passive exercise.

Healthy Participants

Nagaya et al. (2015) recruited 17, healthy elderly females (aged 69-83 years) to examine an intervention to counteract orthostatic hypertension. Participants performed foot-flexion exercise (60 flexes per minute) for two, 1-minute bouts in the supine position. One bout was performed passively, and 1-bout was performed actively. Participant allocation to each bout was randomized. As little as 1-min of active foot flexion led to an increase in heart rate (HR) and mean arterial pressure (MAP). However, heart rate and blood pressure did not change with passive exercise, although both active and passive exercise increased cerebral oxygenation (as measured by near-infrared spectroscopy [NIRS]). Active exercise was thought to increase blood velocity in the femoral and popliteal veins by activating the skeletal muscle pump, increasing venous return and cardiac output. This in turn led to an increase in cerebral perfusion. Passive exercise was thought to produce a similar type of mechanical stimulation on the vasculature which also led to increased cerebral perfusion.

Nóbrega et al. (1994) compared active and passive exercise in 10 (7 male and 3 female) healthy volunteers (aged, 24-36 yrs) using a tandem bike with the second rider performing the active movements. Their results support the findings that HR and blood pressure (BP) are influenced differently in active and passive exercise. Study participants performed unloaded (no load placed on the bike) active cycling for 5 minutes at 40 rpm, rested for 5 minutes, then cycled for 5 minutes at 60 rpm, with passive revolutions performed by a second rider. Both active and passive pedalling on the tandem bike invoked similar leg movements. Nóbrega et al., (1994) reported that HR increased during active exercise but did not change during passive exercise. The increase in HR that occurred during active exercise was thought to be caused by stimulation of cardiac control centre by the motor cortex and subsequently activation of the sino-atrial node of the heart, increasing HR. Further to this, peripheral vascular resistance and mean arterial pressure decreased during active exercise. In contrast, mean arterial pressure increased in passive exercise due to stimulation of muscle mechanoreceptors.

Clinical Populations

Studies involving clinical populations have also demonstrated similar effects of passive and active exercise on the cardiovascular system. Ballaz et al. (2007) examined the effects of 10 minutes of passive cycling at 40 revolution per minute (RPM) in 15 paraplegic patients. Twelve males and three females (aged 39 - 55

years) with spinal cord injuries (SCI) in the thoracic region (T3 through T12) volunteered to participate in the study. One single bout of passive leg cycling increased the velocity of leg vascular blood flow, however, heart rate did not change.

Burns et al. (2018) recruited nine paraplegic (T3-T11) patients (aged 40 – 52 years) to participate in five, 1-minute bouts of knee flexion and extension exercise. Participants performed 50 extensions/flexions per minute on a single leg using the Biodex System 4 Pro dynamometer and maintained an 80° range of motion. These exercises were performed in the up-right position, and a 1-minute recovery period took place between each bout. The authors found that although neither HR nor MAP was altered by the passive exercise bouts, skin and femoral blood flow increased. These results have implications for the prevention of decubitus pressure ulcers, deep-vein thrombosis, cardiovascular disease and poor wound healing from occurring in the lower extremities of paraplegics through improving tissue perfusion with regular passive exercise bouts.

Lastly, Camargo Pires-Neto et al. (2013) examined the effects of 20 minutes of passive rotational leg motion performed at 30 rpm (using a Flexo-motor device) by intensive care unit (ICU) patients lying in bed. Participants included eighteen (8 male, 10 female), newly admitted patients (within 72 hours), aged 55 ± 17 years who were on ventilators. During the exercise session, these patients were under deep sedation and 68% required nor-epinephrine to sustain their blood pressure. No significant hemodynamic, metabolic, nor respiratory changes were observed during exercise. However, there was a significant 3% reduction in HR following exercise compared to pre-exercise values which the authors deemed clinically irrelevant. Although, the reduction in heart rate (HR) following exercise in these mechanically ventilated, critically ill patients possibly suggest an interaction with nor-epinephrine. This study also demonstrated that acute bouts of passive exercise could be safely performed by ventilated ICU patients, with the goal of patient mobilization, early discharge and a reduction in readmission. Since these patients were highly sedated, central command by the motor cortex was negated explaining the minimal effect on heart rate. Moreover, the limited range of motion utilized did not stretch the leg muscles nor induce a passive muscle pump sufficient to increase venous return or stimulate muscle mechano-reflexes further explaining the minimal cardiovascular effects observed.

In summary, minimal information is available on the cardiovascular effects of one bout of passive exercise, compared to active exercise, performed at similar pedalling frequencies. Moreover, there has not been any studies that have compared heart rate variability (HRV) in response to both types of exercise. Therefore, this study examined the cardiovascular responses (including HRV) of passive and active exercise performed at similar pedalling frequencies on two different cycle ergometers (the Theracyle and the Monark ergometer). Our research question was: what is the cardiovascular response in young, healthy individuals to passive and active exercise performed at the same pedaling frequency? Our hypothesis was that the cardiovascular response to passive and active exercise would vary; HR, BP and SNS tone (as reflected through HRV measures) would increase with active exercise and not change with passive exercise.

Materials and Methods

Participants

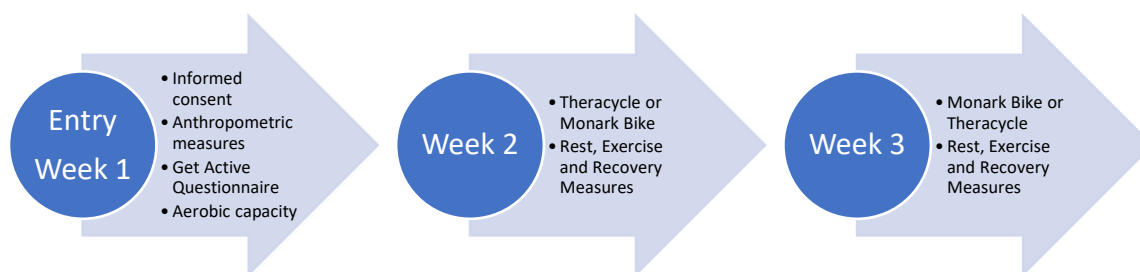
Following local research ethic board (REB) approval (REB file #: 28277), twelve University students (7 male and 5 female) were recruited to participate in the study through announcements and word of mouth. Participants were required to be healthy, between the ages of 18 and 30 years and English speaking to be eligible for the study. The study took place through the months of March and April of 2023.

Testing

A flow-chart of the testing procedures used in this study is displayed below (Figure 1). Participants visited the exercise laboratory on three occasions (each separated by a one-week interval). Each visit was approximately 1 hour in duration and occurred at the same time and day of the week to account for circadian variation. At the first visit, participants signed consent forms and completed the “Get Active Questionnaire” to assess their ability to perform exercise safely. Anthropometric measurements such as height (cm) and weight (kg) (Health-o-Meter Professional Scale - Model: 402KL) were made and body mass index (BMI) (kg/m^2) was subsequently determined ($\text{weight (kg)} / (\text{height (m)})^2$).

Maximal aerobic capacity was assessed using the submaximal Åstrand-Rhyming cycle ergometer test (Acevedo & Starks, 2011). For this test, HR was measured throughout all stages of exercise using a POLAR V800 GPS sports watch (Model: 90060773). Participants initially warmed-up by cycling on a Monark cycle ergometer for 3-minutes at 50 revolutions per minute (rpm) and 0 resistance. They then continued pedalling at 50 rpm for 6 minutes with the resistance set at an intensity to sufficiently to elicit an average target HR between 125 and 170 beats per minute (bpm). Based upon the average HR attained during the last two minutes of exercise, the participants’ maximum aerobic capacity ($\dot{V}\text{O}_2 \text{ max}$) was predicted from sex-specific tables.

Figure 1. Flow Chart of testing Procedures



The following two visits consisted of either a passive exercise procedure using the Theracycle (Theacycle 100 - RSS Industries, Franklin, MA) or an active exercise procedure using the Monark Exercise Bike (Ergomedic 828E, Sweden) (Figure 2).

Figure 2. Exercise Equipment used for the Study

a) Position on the Theracycle.



b) Position on the Monark cycle ergometer.



Participants were randomly assigned to perform either the passive or active exercise for the first visit and performed the other procedure in the second visit. HR was measured continuously using the POLAR V800 GPS sports watch (Model: 90060773) at rest and throughout each exercise bout. BP was measured every 5 minutes using a BPTru monitor (Model: BPM-200 - BPTru Medical Devices, BC, Canada). Borg's, 6-20 rating of perceived exertion (RPE) scale was used to determine RPE every 5 minutes. For each procedure, measurements were first taken for 10 minutes at rest, with HR recorded continuously and BP measured every 5 minutes in a supine position on an examination bed. The participant then moved to the Theracycle or the Monark cycle ergometer. For the Theracycle, the participant used the machine for 10 minutes per pedalling frequency (low - level 4, medium - level 8, high - level 12) for 30 minutes total. For the Monark bike, the participant cycled for 10 minutes at each pedalling frequency (low, medium and high) for 30 minutes total while matching the revolutions per minute (rpm) that was (or would be) performed on the Theracycle to the Monark cycle ergometer (level 4 = 23 rpm, level 8 = 47 rpm, level 12 = 67 rpm). The participant returned to the supine position for 10 minutes for recovery measurements on an examination bed. Following the collection of all measurements, rate pressure product (RPP) was calculated according to the formula $\text{HR (bpm)} \times \text{systolic blood pressure (mmHg)}$ to assess the workload of each exercise bout on the heart. Heart rate variability (HRV) data was analyzed based upon the HR recordings obtained during the last 7 minutes of each stage (i.e., once steady state had been achieved).

Data Analysis

Polar Flowsync was used to download the POLAR watch data (R-R intervals) and the downloaded data was put into the Kubios HRV Scientific software. The Kubios HRV software measured differences in the R-R intervals to provide a detailed overview of HR and variability at each of the exercise stages using the Fast Fourier transformation (FFT) spectral analysis (Tarvainen et al. 2013). Data was extracted from each Kubios HRV spectral analysis output for the values of low-

frequency power (LF), high-frequency power (HF) and total power in milliseconds squared (ms^2), along with the LF/HF ratio (sympathetic nervous system (SNS) tone indicator) (Bernardi et al. 2011). The parasympathetic nervous system (PNS) indicator tone indicator was determined from the HF and total power indicator (HF/total). The SNS and PNS tone were determined to assess the autonomic effects of active and passive exercise.

Statistical analysis was performed using Microsoft Excel for Mac, version 16.101.3. All statistical analyses used the measurements taken at the ten-minute mark for each stage and variable, except for HRV data which was analyzed over the last 7 minutes of each data collection point (rest, low, medium and high frequency and recovery). Differences in descriptive characteristics between males and females were assessed using an independent sample t-tests with a significance level of $p < 0.05$. A repeated measure, two-factor analysis of variance (ANOVA) was performed for the rest, exercise and recovery data obtained with a significance level set at $p < 0.05$. Two between-group measures (passive versus active exercise) and a within group measure (exercise frequency stage) were compared (Haverkamp & Beauducel, 2019). Two-tailed paired t-tests were used to determine differences between the two exercise modes at each exercise stage and for within-group measures. The values for PNS tone (HF/total power) were log-transformed to normalize the data for statistical analysis. All data is presented as means and standard deviation (SD).

Results

Descriptive Characteristics

The descriptive characteristics of the 12 participants are displayed in Table 1. There were no significant differences between the sexes for age, BMI or aerobic capacity ($\dot{V}\text{O}_2 \text{ max}$). The male participants were significantly taller and heavier than the female participants.

Table 1. *Descriptive Characteristics (mean $\pm$ SD) of the Participants*

Variable	Male (n=7)	Female (n=5)	Total (n=12)
Age (years)	20.7 $\pm$ 0.8	20.8 $\pm$ 0.8	20.8 $\pm$ 0.8
Height (cm)	177.1 $\pm$ 6.1 *	162.5 $\pm$ 6.1	171.0 $\pm$ 9.5
Weight (kg)	89.0 $\pm$ 24.6 *	63.7 $\pm$ 14.6	78.4 $\pm$ 24.0
Body mass index (kg/m^2)	28.3 $\pm$ 7.5	24.1 $\pm$ 5.4	26.6 $\pm$ 6.8
$\dot{V}\text{O}_2 \text{ max}$ ($\text{mL}/\text{kg}/\text{min}$)	28.3 $\pm$ 9.1	28.4 $\pm$ 4.7	28.3 $\pm$ 7.3

Note: * = significant difference between males and females ($p < 0.05$).

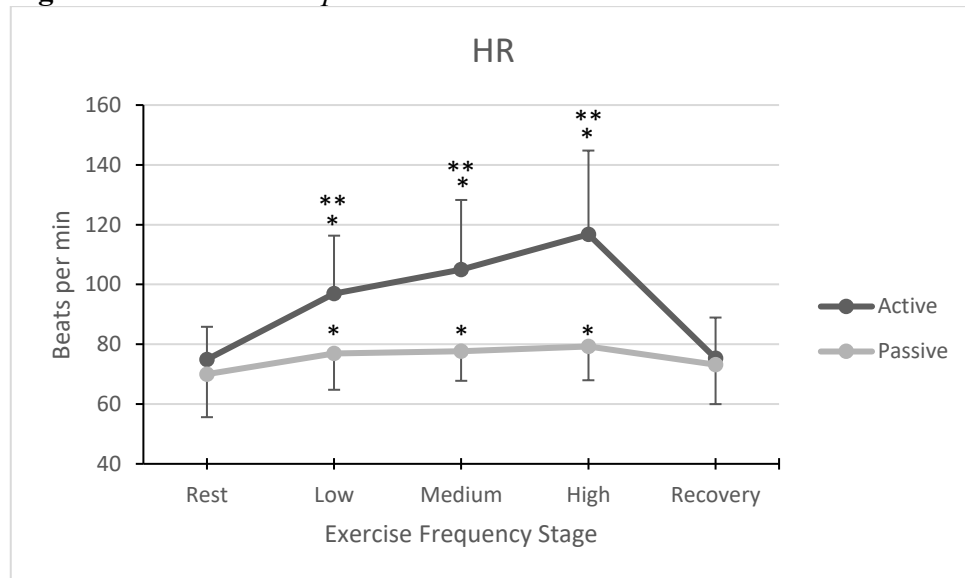
Cardiovascular Responses

A two-way analysis of variance for repeated measures revealed no significant differences in the cardiovascular responses of males versus females in response to each exercise mode. Thus, data from both sexes were combined and are presented as follows.

Heart Rate

Heart rate (HR) significantly increased from rest in response to exercise during the low, medium and high pedalling frequency exercise stages for both active and passive exercise and returned to pre-exercise baseline values at recovery (Figure 3). HR was significantly higher for active compared to passive exercise at all three of the exercise stages.

Figure 3. Heart Rate Response

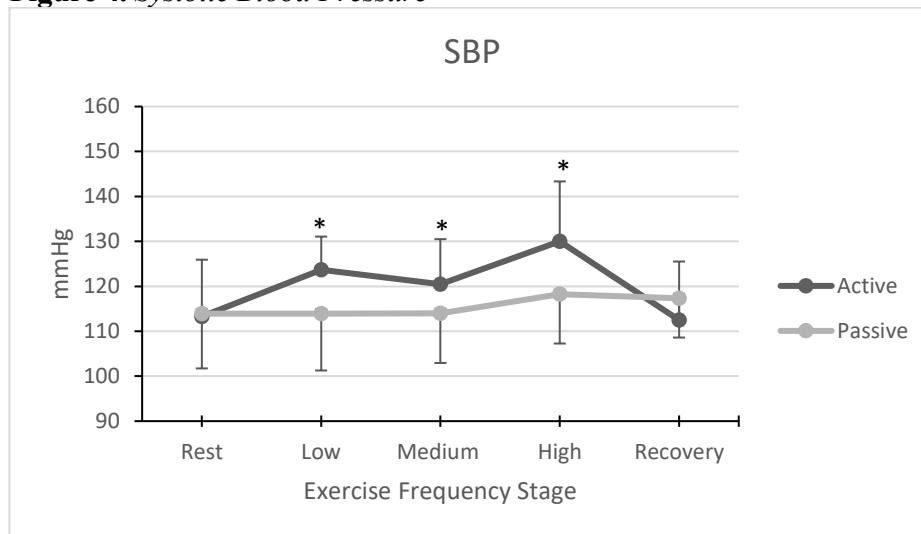


Note: Mean ($\pm$ SD) heart rate responses during active and passive exercise at 3 pedalling frequencies and recovery. Data were recorded at the end of each 10 min exercise stage. * = significant difference for specific exercise stage compared to rest ($p < 0.05$). ** = significant difference between exercise modes at each exercise stage ($p < 0.05$).

Blood Pressure

Systolic Blood Pressure (SBP)

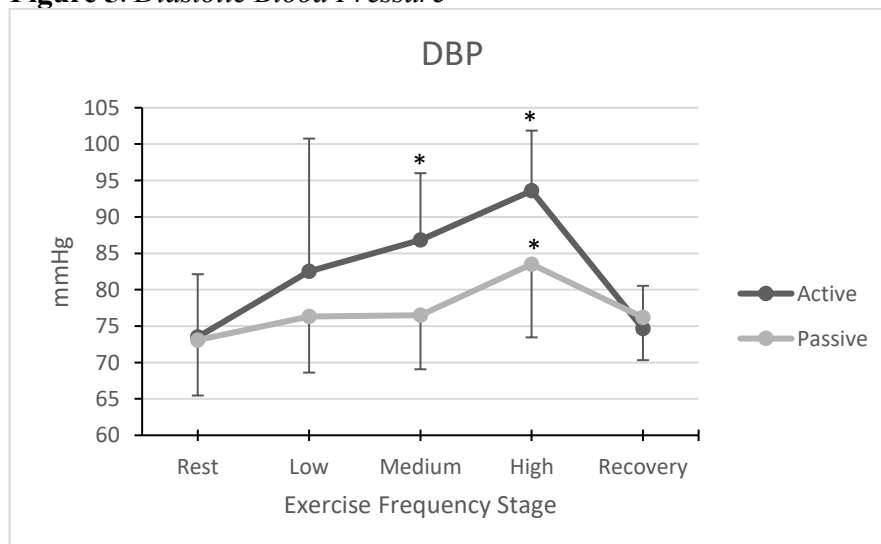
SBP significantly increased from resting measures during the low, medium, and high pedalling frequency exercise stages of active exercise and returned to pre-exercise baseline values at recovery (Figure 4).

Figure 4. Systolic Blood Pressure

Note: Mean ($\pm$ SD) systolic blood pressure responses during active and passive exercise at 3 pedalling frequencies and recovery. Data were recorded at the end of each 10 min exercise stage. * = significant difference for specific exercise stage compared to rest ($p < 0.05$).

Diastolic Blood Pressure (DBP)

DBP significantly increased from resting measures during the medium and high pedalling frequency exercise stages of active exercise and returned to pre-exercise baseline values at recovery (Figure 5). During passive exercise, DBP significantly increased from rest in response to the high frequency pedalling stage and returned to pre-exercise baseline values at recovery.

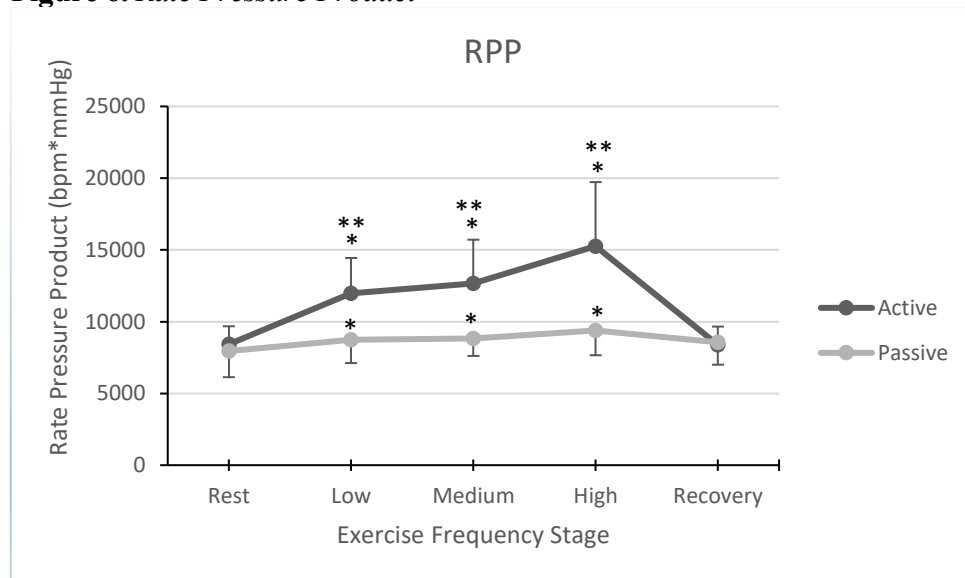
Figure 5. Diastolic Blood Pressure

Note: Mean ($\pm$ SD) diastolic blood pressure responses during active and passive exercise at 3 pedalling frequencies and recovery. Data were recorded at the end of each 10 min exercise stage. * = significant difference for specific exercise stage compared to rest ($p < 0.05$).

Rate Pressure Product (RPP)

During both active and passive exercise, RPP significantly increased from resting measures during the low, medium and high pedalling frequency exercise stages and returned to pre-exercise baseline levels at recovery (Figure 6). RPP was significantly higher for active compared to passive exercise at all three pedalling frequency exercise stages.

Figure 6. Rate Pressure Product

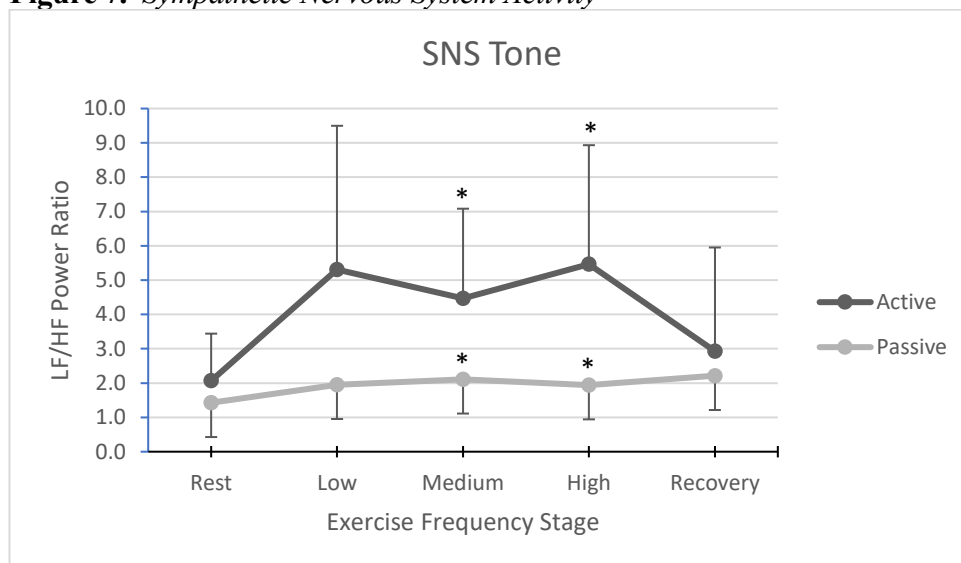


*=Note: Mean ($\pm$ SD) rate-pressure pressure responses during active and passive exercise at 3 pedalling frequencies and recovery. Data were recorded at the end of each 10 min exercise stage. * = significant difference for specific exercise stage compared to rest ($p < 0.05$). ** = significant difference between exercise modes at each exercise stage ($p < 0.05$).

Autonomic Effects

Sympathetic Nervous System (SNS) Tone

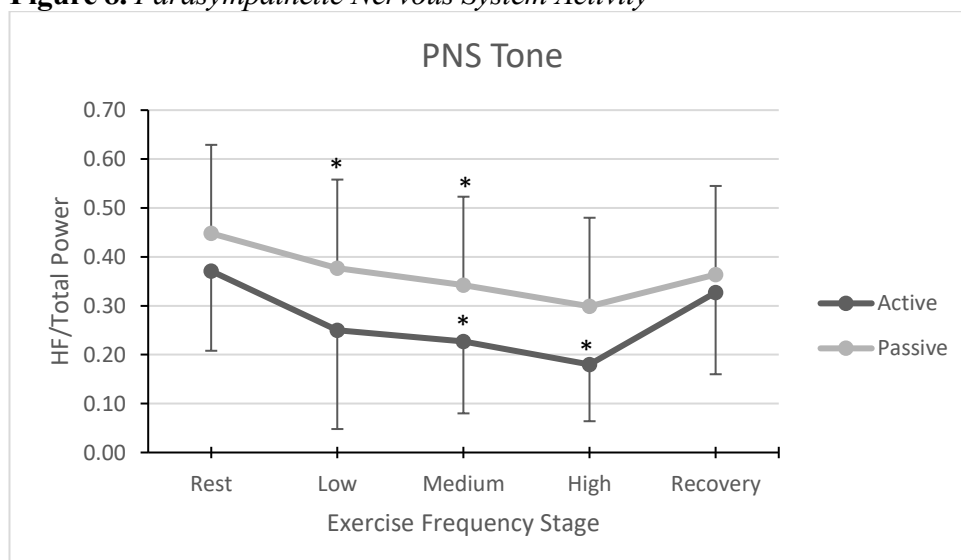
During both active and passive exercise, SNS tone significantly increased from resting measures during the medium and high pedalling frequency stages and returned to pre-exercise baseline values at recovery (Figure 7).

Figure 7. Sympathetic Nervous System Activity

Note: Mean ($\pm$ SD) sympathetic nervous system responses during active and passive exercise at 3 pedalling frequencies and recovery. Data were recorded and analyzed for the last 7 min of each exercise stage. * = significant difference for specific exercise stage compared to rest ($p < 0.05$).

Parasympathetic Nervous System (PNS) Tone

During active exercise, PNS tone significantly decreased from resting measures during the medium and high pedalling frequency exercise stages and returned to pre-exercise baseline measures at recovery (Figure 8). In passive exercise, PNS tone significantly decreased from rest during the low and medium pedalling frequency exercise stages and returned to pre-exercise baseline measures at recovery.

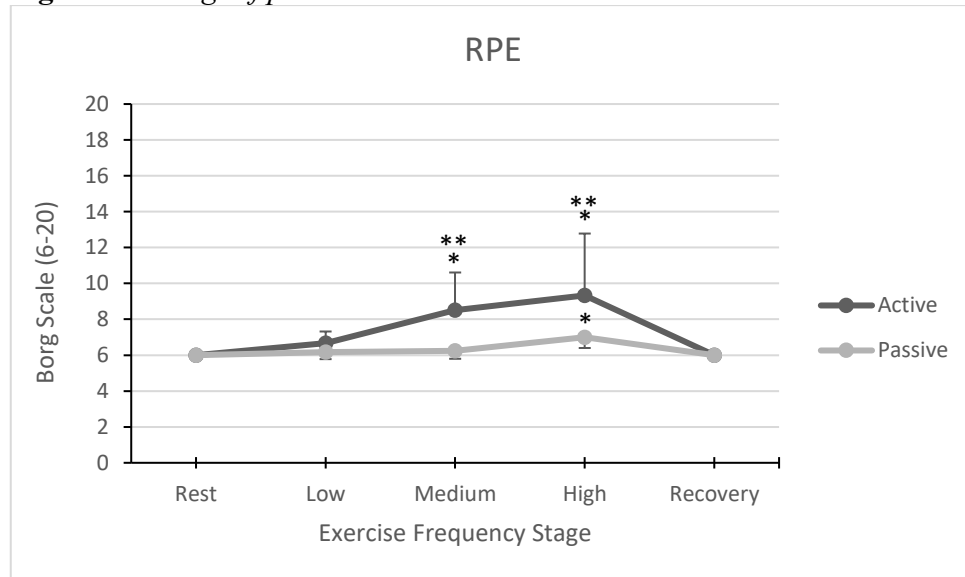
Figure 8. Parasympathetic Nervous System Activity

Note: Mean ($\pm$ SD) parasympathetic nervous system responses during active and passive exercise at 3 pedalling frequencies and recovery. Data were recorded and analyzed for the last 7 min at each exercise stage. * = significant difference for specific exercise stage compared to rest ($p < 0.05$).

Ratings of Perceived Exertion (RPE)

During active exercise, RPE significantly increased from resting measures during the low, medium and high pedalling frequency exercise stages and returned to pre-exercise baseline values at recovery (Figure 9). In passive exercise, RPE significantly increased from rest in response to the high frequency pedalling and returned to baseline values at recovery. RPE was significantly higher for active compared to passive exercise during the medium and high pedalling frequencies.

Figure 9. Ratings of perceived Exertion



Note: Mean ($\pm$ SD) ratings of perceived exertion during active and passive exercise at 3 pedalling frequencies and recovery. Data were recorded at the end of each 10 min exercise stage. * = significant difference for specific exercise stage compared to rest ($p < 0.05$). ** = significant difference between exercise modes at each exercise stage ($p < 0.05$).

Discussion

This study examined the cardiovascular responses to passive and active exercise performed at similar pedalling frequencies. The results of this study supported our hypothesis that cardiovascular responses to exercise would vary depending on whether the exercise is performed passively or actively. A significant main effect of exercise modality and the different pedalling frequencies was observed for the cardiovascular measures as well as for RPE. As predicted, HR, RPP, and RPE were significantly greater for active exercise compared to passive exercise performed at similar pedalling frequencies. SBP and DBP increased in response to active exercise but was generally unaffected by passive exercise. Significant changes in SNS and PNS tone also occurred whereby the SNS tone increased and PNS tone decreased to a greater extent in active exercise compared to passive exercise. However, our findings that HR and RPP was significantly greater compared to pre-exercise baseline

values for passive cycling was unexpected. Based upon our literature review, we had expected these values to not change.

This is one of the first studies to compare the cardiovascular responses and autonomic balance (as assessed through HRV analysis) to both passive and active exercise performed at identical pedalling frequencies using two different exercise modalities (a Theracycle versus a Monark cycle ergometer, respectively). To date, there has been only one other study whose investigators have examined the cardiovascular responses to similar pedalling frequencies (Nóbrega et al., 1994). Nóbrega et al. (1994) examined the effects of participants performing exercises on an adaptive tandem bicycle. One person actively pedalled, whereas the other person had their legs moved passively at the same time. Participants performing the passive exercise reported that they did not conscientiously contract their exercising muscles. Oxygen uptake, cardiac output and HR significantly increased from resting baseline measures during active exercise performed at both 40 and 60 rpm. Peripheral vascular resistance significantly decreased with each exercise stage during active exercise. However, HR did not change but mean arterial pressure as well as stroke volume significantly increased during passive exercise. The results obtained for HR differed from those reported in our study and may be explained by the possible postural differences associated with the different exercise modalities used in our study. Nóbrega et al. (1994) had their participants in identical postural positions (using a tandem bike) when comparing the responses of passive to active exercise. Thus, one would assume that there were no additional muscle groups used for the passive exercise. In our study, the participants were positioned slightly different during the two modes of exercise, with a slight semi-recumbent posture and greater hip flexion occurring during the passive exercise performed on the Theracycle. The increase in HR and RPP from resting measures during the low, medium and high pedalling frequency exercise stages for both active and passive exercise may be attributed to central command driven by the motor cortex and changes in autonomic balance.

The cardiovascular changes that occurred in response to passive exercise in our study was also contrary to that reported in the literature for both healthy and clinical populations (Ballaz et al., 2007; Burns et al., 2018; Nagaya et al., 2015; Nóbrega et al., 1994). This may have been due to postural difference as explained above or by our participants actively contracting their muscles while exercising. Even though, in our study, the participants were asked to not exert pressure with their legs when they were pedalling, our results suggest that they were. Moreover, we observed PNS (vagal) withdrawal to occur in our study. PNS withdrawal normally occurs at the start of exercise through feedback provided to the central nervous system, from the metabolic and mechanoreceptors in the exercising muscles (Miyamoto et al., 2006). SNS activation, which tends to occur at approximately 50% of maximal aerobic capacity (Goldsmith et al., 2000) would have contributed to the greater rise in HR that was observed during active exercise. This is important to note, as passive exercise utilized in our study did not engage the SNS to the same degree as active exercise. Hence, patients who clinically are encouraged to minimize the amount of stress imposed on the body can still perform passive exercise without inducing substantial cardiovascular responses.

The effects of active and passive exercise on the autonomic nervous system were measured through HRV analysis. SNS and PNS tone responded differently to each exercise modality and the various pedalling frequency stages, but with no interactions. Malhotra et al., (2023) studied HR variability in healthy untrained adults who performed an acute bout of mild-intensity exercise on a cycle ergometer and saw that SNS tone increased as PNS tone decreased at the beginning of exercise and throughout cycling. During recovery, PNS tone increased. Similarly, this study reported an increased SNS tone from resting measures only for the medium and high pedalling frequency exercise stages of active and passive exercise. The PNS tone also significantly decreased from resting measures during the medium and high frequency exercise stages for participants performing active exercise and during the low and medium pedalling frequency stages for the passive exercise while increasing again during the recovery stages for both exercise modalities.

The prediction that blood pressure would be different in response to the different types of exercise performed at the same pedalling frequency was partially supported. SBP and DBP were significantly affected by the exercise modality and the different exercise stages performed but with no significant interactions between the effects of active and passive exercise and the three exercise stages. Wielemborek-Musial et al. (2016) examined the normal BP responses to submaximal, multistage exercise tests using a cycle ergometer in healthy adults. Participants displayed significantly increased SBP with an increase in exercise workload and intensity. Like these results, the results of our study demonstrated a significant increase in SBP from rest during the low, medium and high pedalling frequency stages for active exercise only. It is interesting to note that the SBP response mirrored the pattern of SNS activation, and an increase in SNS tone may have contributed to the increased SBP observed.

In addition, DBP significantly increased from rest during the medium and high pedalling frequency stages for active exercise and only during the high pedalling frequency stage for passive exercise. These results are aligned with the work of Wielemborek-Musial et al. (2016), who also reported that DBP increased significantly from resting measures during exercise. However, DBP is a relatively stable value that normally remains unchanged during an acute bout of exercise. The higher DBP observed in this study for the various exercise stages may be attributed to a decrease in vasodilation of the resistance vessels of the actively exercising muscles (Brett et al. 2000). However, the DBP results for active and passive exercise should be carefully evaluated, primarily due to the small sample size of this study.

RPP is an indicator of myocardial exertion used to approximate increases in the heart's metabolic requirements (workload) that can be caused by exercise (Adams et al. 2010). The RPP for participants in this study was significantly affected by the exercise modality and the various exercise stages, with a significant interaction between the effects of active and passive exercise and the three exercise stages. Kim et al. (2013) examined the effects of RPP on a graded cycle ergometer exercise test in healthy men and saw that RPP increased from resting measures during exercise, with greater RPP values correlating with increased exercise intensities. Similarly, RPP increased from rest during the low, medium and high pedalling frequency stages during active exercise and passive exercise. Although, RPP was greater during active

than passive exercise for all three pedalling frequency stages. The differences in RPP observed for the active compared to the passive exercise, and for the different exercise stages can be attributed primarily to the increases in HR and SBP.

Borg's RPE is a subjective measurement that estimates exercise intensity based upon the exercise load applied during exercise and is associated with a homeostatic imbalance of the body (Day et al. 2004, Mann et al. 2017). The RPE of participants in this study was significantly affected by the exercise modality and the different exercise stages performed, with an interactive effect between the effects of active and passive exercise and the three exercise stages. These results align with the work done by Nóbrega et al. (1994), as the RPE of their participants was significantly higher for active exercise compared to passive exercise. Mann et al. (2017) suggested that psychological factors may also affect the RPE of an individual at any given point during exercise. RPE also has a known correlation between HR and blood lactate levels, as exercise induced changes in HR, depletion of energy stores and reliance on anaerobic metabolism has been suggested to make an individual perceive greater exertion. Thus, participants in this study may have displayed significant changes in RPE with both active and passive exercise at the different pedalling frequency stages due to the combined psychological and physiological effects.

Limitations

Due to the small sample size and the participation of young, apparently healthy individuals, this study can be considered a preliminary investigation on the cardiovascular responses to passive exercise on a Theracycle. With only twelve participants (five female and seven males) the applicability of the study findings is limited. Moreover, without consideration of menstrual phase of the female participants, the interpretation of the results could be problematic. Since the data was collected over a two-week period, the data collected could have been influenced by the phase of the menstrual cycle the female participants were in. Also, the participants in this study were all healthy and did not have any health complications.

Furthermore, the results obtained for the passive exercise are applicable for use of the Theracycle only. The Theracycle (Theracycle, 100) used in this study was pre-programmed with specific levels that had pre-selected speeds (rpm). The Monark cycle ergometer was standardized by instructing participants to maintain a similar rpm speed (at a minimal load) corresponding to each selected pedalling frequency. This study only explored the cardiovascular responses to one bout of active versus passive exercise performed at different pedalling frequencies.

Future Studies

Future studies should be designed to recruit more participants. It would be advisable to also have a greater range in participant ages. Female participants should be tested in the same phase of their menstrual cycle. Examining and comparing the responses of regularly active individuals, sedentary participants and individuals with neurological impairments to passive exercise would increase the applicability of the results. Additional studies should also be designed to use the same cycle ergometer

by making modifications to the cycle ergometer (or to use a tandem bike) to allow for both active exercise and passive exercise to be performed in the same body position at the same pedalling frequency. Collecting oxygen consumption measures during exercise would allow us to determine exercise intensity more accurately. Training studies designed to compare the effects of passive exercise training on the cardiovascular system are also needed. Overall, these modifications to the design of the study would increase the reliability of the results.

Conclusions

In conclusion, the results of this study supported our hypothesis that the cardiovascular responses to exercise vary depending on whether the exercise was performed actively or passively. The significantly different HR, RPP, and RPE responses observed between passive and active exercise at similar pedalling frequencies in a young and generally healthy population suggest that passive exercise may be a suitable form of activity/exercise for individuals beginning an exercise program or for those who have health limitations as it places minimal stress upon the CV system. These findings can inform exercise guidelines for individuals with chronic disease for use of passive exercise modalities and to promote the use of passive exercise as a mode of training for both sedentary healthy and clinical populations.

Acknowledgements

The authors acknowledge the generous contribution of the Kinesiology Dept. at Trent University for allowing use of their Monark cycle ergometer for exercise testing for this study.

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