EFFECT OF CARDIOVASCULAR DRIFT ON MAXIMAL OXYGEN UPTAKE DURING WALKING AND CYCLING IN WOMEN

by

LAURA JANE HINES

(Under the Direction of Kirk Cureton)

ABSTRACT

This study investigated whether the progressive rise in heart rate (HR) and fall in stroke volume (SV) during prolonged, moderate-intensity exercise (cardiovascular drift) in a thermoneutral environment is associated with a reduction in $\dot{V}O_{2max.}$ CV drift was measured on nine moderately-trained women (age = 23.3±1.8 yr, $\dot{V}O_{2max}$ = 43.0±5.5 ml/kg/min) between 15 and 45 min of cycling and treadmill walking at 60% $\dot{V}O_{2max,}$ with $\dot{V}O_{2max}$ measured immediately thereafter without cessation of exercise. During a separate trial, $\dot{V}O_{2max}$ was measured after 15 min submaximal exercise, so that change in $\dot{V}O_{2max}$ that occurred between 15 and 45 min could be associated with CV drift. This protocol was performed during two different modes of exercise, cycling and walking. There was no significant effect of exercise mode on the maginitude of CV drift or change in $\dot{V}O_{2max.}$ In cycling and walking, respectively, a 5.5% increase in HR (150±14 vs 158±17 bpm, $p<0.05$) and a 10% decrease in SV (77±10 vs 68±9 ml, $p<0.05$) between 15 and 45 min was associated with a 5% reduction in $\dot{V}O_{2max}$ (2.70±0.40 vs 2.52±0.57 l/min, $p<0.05$). There was a significant relationship between ΔHR and Δ %$\dot{V}O_{2max}$ ($r=0.47$, SEE=0.32 l/min, $p<0.05$). Submaximal $\dot{V}O_2$ was unchanged over time ($p<0.05$), but

%$\dot{V}O_{2max}$ increased 5.2%. It is concluded that CV drift, characterized by the rise in HR and decline in SV during prolonged, moderate-intensity exercise, is associated with a decrease in $\dot{V}O_{2max}$ during walking and cycling. Increased HR reflects increased relative metabolic intensity during prolonged cycling and walking. This study supports the validity of using HR as an indicator of relative metabolic intensity during prolonged cycling and walking when CV drift occurs.

INDEX WORDS: heart rate, stroke volume, cardiovascular drift, exercise mode, cycling, walking, oxygen consumption

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Chapter 1

Introduction

Prolonged, moderate-intensity, constant-rate exercise leads to a downward drift in central venous pressure, stroke volume (SV), and pulmonary and systemic arterial pressures, while during this time a rise in heart rate (HR) maintains nearly constant cardiac output (CO) (38). These slow progressive changes have been termed cardiovascular drift (CV drift). CV drift has been shown to occur in thermoneutral (13, 16, 32, 33, 40) and hot environments (20, 22, 31, 39), beginning after about 10 min of dynamic exercise and continuing for several hours.

The cause of CV drift is unknown, but two theories have been proposed. Rowell and colleagues speculated that the cause of CV drift was a progressive rise in cutaneous blood flow and venous volume, reducing ventricular filling pressure, thus decreasing end diastolic volume limiting SV (25, 38). HR increases to maintain blood pressure (34). An alternative hypothesis is that the increase in HR due to increased body temperature and increased sympathetic nervous activity causes a decline in stroke volume due to decreased diastolic time (8, 16).

The consequences of CV drift for physical work capacity are uncertain. It is not known whether the decrease in SV during prolonged exercise reflects a decrease in cardiovascular capacity and maximal oxygen uptake ($\dot{V}O_{2max}$) or not. Two studies have investigated this problem. Saltin and Stenberg (40) had four subjects exercise for 195 min under thermoneutral conditions at 75% $\dot{V}O_{2max}$. During this time, they saw a progressive increase in HR by about 10% and a progressive decrease in SV of about 15%, indicating CV drift occurred. After a 90-min

rest, subjects resumed submaximal exercise and then three subjects completed a maximal load. They found $\dot{V}O_{2max}$ was decreased by 5%. These data suggest that for a brief period of time, the cardiovascular alterations characterizing CV drift, principally reduced SV, can be overcome and that cardiovascular capacity and $\dot{V}O_{2max}$ are minimally reduced.

Ekblom (12) measured $\dot{V}O_{2max}$ immediately after 1 h of cycling at 75% $\dot{V}O_{2max}$ under thermoneutral conditions before and after aerobic training in seven male subjects when CV drift occurred. SV measured during maximal exercise immediately after the 60 min of submaximal exercise was unchanged from that at 60 min, indicating that the SV changes that occurred with CV drift persisted during maximal exercise. $\dot{V}O_{2max}$ was reduced 6% following the 60 min of submaximal exercise before training and 12% after training, compared to control. These data suggest that the decrease in SV during prolonged exercise persisted during maximal exercise and caused a reduction in $\dot{V}O_{2max.}$ Additional research is needed, however, on the consequences of CV drift during shorter periods of time that do not include rest, with different modes of exercise.

Nassis and Geladas (32) compared CV drift in 90 min of running and cycling at 60% of mode-specific $\dot{V}O_{2max}$ in thermoneutral conditions. They found a larger decrease in SV but similar increase in HR in cycling compared to running, in spite of greater dehydration and hyperthermia during running. Cardiac output decreased in cycling, but not in running. The authors suggested that there may be a more efficient muscle pump during running than cycling due to greater muscle relaxation time and lower compressive forces. Whether the different degree of cardiovascular drift during prolonged running and cycling would be reflected in different effects on $\dot{V}O_{2max}$ is unknown.

Purpose

The specific aims of this study were to:

1. To determine if the magnitude of cardiovascular drift during prolonged exercise is different during cycling compared to walking on a treadmill and
2. To determine the association between cardiovascular drift and $\dot{V}O_{2max}$ during prolonged exercise on the cycle ergometer and the treadmill.

Hypotheses

The research hypotheses for this study were:

1. There is less increase in heart rate and decline in stroke volume during prolonged treadmill walking compared to cycling.
2. The association between cardiovascular drift and $\dot{V}O_{2max}$ is the same during prolonged stationary cycling and treadmill walking.

Significance of the Study

The extent to which CV drift affects maximal oxygen uptake has important implications for exercise prescription. Intensity of exercise is commonly prescribed using heart rate, based on the strong relation between percent of maximal heart rate (%HRmax) or percent of HR reserve (%HRR), and percent of maximal oxygen uptake ($\%\dot{V}O_{2max}$) or percent of oxygen uptake reserve ($\%\dot{V}O_2R$)(1). However, the use of heart rate as an indicator of relative metabolic intensity is based on validation studies that employed short-term exercise of progressively increasing intensity (9, 47). Whether heart rate is a valid indicator of relative metabolic intensity during more prolonged exercise is unknown. If the decline in SV and increase in HR over time during prolonged exercise is associated with a proportional decrease in $\dot{V}O_{2max}$ and no change in

submaximal $\dot{V}O_2$, then relative exercise intensity would increase. If this occurs, change in heart rate could be considered a good indicator of change in relative exercise intensity during prolonged exercise. However, if CV drift is not associated with altered submaximal $\dot{V}O_2$ or $\dot{V}O_{2max}$, then an increase in HR over time would be dissociated from, and not be a good indicator of, change in relative metabolic intensity. The answer has implications for individuals who exercise for prolonged periods of time and use heart rate as the marker for exercise intensity.

Another unique aspect of this study was that the experiments were performed on women. Previous studies on CV drift have neglected to study women, in part because the menstrual cycle confounds effects on experimental treatments. Although there is no apparent reason gender should influence the effect of CV drift on maximal oxygen uptake, it is important to have data on women so that results can be generalized to them.

Limitations of the Study

The use of women as subjects introduces possible confounding effects of the menstrual cycle. Because of the difficulties in scheduling, it was not possible to test all women in the same phase of the cycle. Instead, an effort was made to perform all experimental trials within either the follicular or luteal phase of the cycle. The menstrual cycle does not affect $\dot{V}O_{2max}$ (6), so performing the control tests of $\dot{V}O_{2max}$ in a different phase did not influence the results. The primary variables affected by the menstrual cycle that were used in this study were core body temperature and heart rate during submaximal exercise (45). These measures were only taken during the experimental trials, which were conducted within one phase of the cycle.

Chapter 2

Review of Literature

Cardiovascular Drift

During prolonged moderate intensity (50-75% $\dot{V}O_{2max}$) exercise, slow progressive changes in cardiovascular function occur though work rate is unchanged. These changes have been termed cardiovascular drift (CV drift), which is characterized by the downward drift in central venous pressure, stroke volume, pulmonary and systemic arterial pressures, while at the same time a rise in heart rate maintains nearly constant cardiac output (38). CV drift occurs during exercise in a thermoneutral and hot environment during different modes of exercise (32). The magnitude of CV drift is affected by the exercise intensity (17), exercising posture (14), and the subject's level of dehydration and hyperthermia (20).

Though the mechanisms underlying CV drift are unknown, two hypotheses have been proposed. One hypothesis is that increased cutaneous blood flow displaces blood volume from central circulation to the periphery, reducing venous return to the heart thus reducing stroke volume (25, 38). According to this theory, the rise in heart rate is presumably in response to the drop in stroke volume and arterial pressure. The alternative theory is that an increase in heart rate is due to the increased body temperature and increased sympathetic nervous activity causing time at diastole to be reduced thus reducing stroke volume (8, 16).

During exercise in a comfortable environment (~22°C), studies have shown that SV declines and HR increases progressively over time. In an early study, Saltin and Stenberg (40)

found that during prolonged cycling and running (195 min) at moderate intensity (75% $\dot{V}O_{2max}$), HR increased 20% and SV decreased about 8% between min 15 and min 195 during submaximal exercise. After 195 min of exercise, subjects rested for 90 min and then completed a maximal workload immediately following. Values at the end of the additional bout of exercise remained the same as at the end of the 195 min of submaximal exercise. During maximal exercise, SV remained unchanged from the end of 195 min of submaximal exercise. $\dot{V}O_2$ was decreased 5% compared to a control measured at a later date. The average decrease in body weight was approximately 5% and rectal temperatures were between 38.0-38.6°C. Their data indicated that the reduced SV observed following prolonged exercise persisted during maximal exercise and was related to a slightly lower $\dot{V}O_{2max.}$ Although the rest period did not alter the factors causing CV drift during submaximal exercise, it may have increased the ability to overcome reduced SV during maximal exercise.

Ekelund and Holmgren (14) had subjects cycle for 50-60 min at a workload expected to elicit a heart rate of 140-150 bpm. After 10 min, heart rate rose about 15% and stroke volume declined approximately 14%. The decrease in SV was compensated for by an increase in HR, which kept CO unchanged during exercise. Rectal temperature increased during exercise to an average of 38.6°C. They suggested that the decrease in SV may be partially explained by decreased filling and/or emptying of the heart. Filling of the heart is dependent on the time allowed for filling and the amount of energy, potential and kinetic, present in the capacitance vessels in front of the heart. The emptying also is dependent on myocardial contractility, not measured during this investigation. Also, they propose that increased pulmonary minute ventilation ($\dot{V}E$), due to increased respiratory rate and increased tidal volume could cause an

increase in dead-space ventilation. If perfusion of the upper lungs is reduced because of increased blood volume to the legs, then this is compatible with falling SV.

Ekblom (12) measured $\dot{V}O_{2max}$ immediately after 1 h of cycling at 75% $\dot{V}O_{2max}$ under thermoneutral conditions before and after aerobic training in seven male subjects. Central circulatory measures were obtained during submaximal exercise and at $\dot{V}O_{2max.}$ They found that HR increased 9 bpm (~5%) between min 15 and min 60 before training and 7 bpm (~4%) after training. SV decreased 4 ml (~3%) between min 15 and min 60 before training and 8 ml (~8%) after training. SV measured during maximal exercise immediately after the 60 min of submaximal exercise was unchanged from that at 60 min, indicating that the SV changes that occurred with CV drift persisted during maximal exercise. $\dot{V}O_{2max}$ was reduced 6% following the 60 min of submaximal exercise before training and 12% after training, compared to control. These data suggest that the decreases in SV that occur during prolonged exercise persist during maximal exercise and cause decreased $\dot{V}O_{2max}$.

Johnson and Rowell (25) examined forearm skin and muscle vascular responses to exercise under thermoneutral conditions (~24 °C). Five subjects cycled for 60 min at a constant workload to elicit a HR of ~130 bts/min for the first 10 min. During this experiment they measured forearm blood flow (FBF) in one or both arms, or muscle blood flow (MBF) throughout the control, exercise and recovery periods. They found a progressive rise in FBF during the entire 1 h of exercise and a progressive decline in MBF at the beginning of exercise which was sustained throughout exercise. These findings suggest that rising cutaneous blood flow and a reduction in venous tone during exercise (37), can lead to a reduction in peripheral

resistance and an increase in peripheral blood volume, leading to reduction in central blood volume, thus reducing SV.

Shaffrath and Adams (42) studied the effects of airflow and work load on CV drift and skin blood flow. Eight subjects performed four bouts of prolonged exercise under two different power outputs (40% and 60% $\dot{V}O_{2max}$), with a fan or without. Subjects exercised for 70 min and cardiovascular and metabolic measures were assessed at 10-min time intervals. At the lower workloads, CV drift did not occur regardless of fan use. However, at 60% $\dot{V}O_{2max}$, HR was increased and SV was decreased to a greater extent at higher workloads when no fan was used and was not significantly different when a fan was used. Also they found that FBF increased during both workloads without the fan, but remained fairly constant when the fan was used. This evidence supports the hypothesis that CV drift occurs because of displacement of blood volume into cutaneous capacitance vessels. CV drift occurred only in conditions that combined the higher power output with no fan, reducing ability to dissipate heat. During the higher intensity exercise when the fan was used, increased heat dissipation and cooling of the skin reduced pooling of blood in the skin.

Fritzsche et al. (16) investigated the role of sympathetic stimulation of the heart in increasing HR or SV during prolonged exercise. Subjects cycled for 60 min at a moderate intensity (~57% $\dot{V}O_{2max}$). HR increased by 11% and SV declined by 13%. Cardiac output was maintained constant throughout the exercise bout. Rectal temperature was 37.8°C at the end of the exercise. In another trial, the same subjects, were given atenolol, a β-blocker, prior to performing the same exercise HR was kept constant and they found that when HR did not rise,

SV remained constant. This supports the theory that HR is the driving force causing changes in SV.

Nassis and Geladas (32) investigated whether the mode of exercise affected cardiac output decline during prolonged exercise. Subjects either ran or cycled for 90 min at 60% of mode-specific $\dot{V}O_{2max}$ and found that during cycling SV declined 22% and HR rose 12%. Cardiac output declined approximately 11%. Rectal temperature rose to approximately 38.8°C. During treadmill running stroke volume declined 12%, less than during cycling, and heart rate rose 11%. Cardiac output was maintained during running. Rectal temperature was higher at the end of running (39.6°C) than at the end of cycling (38.7°C). They speculated that these changes were due to the more efficient muscle pump during running compared to cycling, which may blunt the drop in SV associated with CV drift (23). They also speculated that during cycling there are higher intramuscular forces than during running (11), which could compress or even partially occlude the blood vessels in the leg reducing venous return back to the heart (28). During the study, they found that despite higher levels of hyperthermia during treadmill running, there was less of an increase in skin blood flow, leading to a greater central blood volume during running compared to cycling.

Some studies have investigated cardiovascular drift under warm temperatures (~35°C). Montain and Coyle (31) examined if fluid ingestion during exercise would attenuate CV drift and hyperthermia by reducing levels of dehydration. The subjects were given no fluid, small volume of fluid, moderate volume of fluid and large volume of fluid during 110 minute bouts of exercise at 62-67% $\dot{V}O_{2max}$. During the no-fluid (NF) trial SV declined 27% during exercise. Fluid

ingestion attenuated stroke volume decline. HR increased progressively during the exercise in which no fluid was given. HR rise was blunted with fluid ingestion and the magnitude of change was directly related to the amount of fluid that was ingested. Cardiac output was directly related to the level of dehydration accrued with the greatest decline in CO occurring during the no fluid trial.

Gonzalez-Alonso et al. (20), investigated the extent to which dehydration, hyperthermia, or a combination of both dehydration and hyperthermia contributed to cardiovascular drift. Subjects exercised for 100 min at approximately 72% $\dot{V}O_{2max}$ in either 23°C or 35°C conditions and were either given fluid to maintain euhydration or they were dehydrated. The net effect from dehydration and hyperthermia was a decline in SV by 20% along with a 9% rise in HR. Dehydration and hyperthermia alone decreased SV by 7% and increased HR by 5%. Cardiac output was maintained during hyperthermia and dehydration alone, but during the combination of hyperthermia and dehydration, CO was reduced. They also determined whether or not reversing the effect of dehydration attenuated CV drift. By infusing a dextran solution, they eliminated the detrimental effect of dehydration on plasma volume and blood volume, and there was no decline in SV or increase in HR over time during prolonged cycling. The findings of this study suggest that it is not prolonged exercise alone that contributes to CV drift, but the synergistic effects of dehydration combined with hyperthermia reducing CO during exercise.

Gonzalez-Alonso et al. (22) determined the effect of environment and hydration on stroke volume. They had four different trials that consisted of exercising in the heat for 2 h at 60% $\dot{V}O_{2max}$ during which they manipulated hydration status. They elicited euhydration, 1.5% dehydration, 3% dehydration, and 4.2% dehydration by having subjects ingest different amounts

of fluid. After completion of 2 h of exercise in a hot environment, subjects rested and then completed two more 30 min exercise bouts in both the cold (~8°C) or hot (~35°C) environment. During euhydration, SV was similar in the cold and hot environments. SV declined in both environments in direct proportion to the degree of dehydration. For every 1% dehydration in the heat, SV declined ~6.4 ml and for every 1% of dehydration in the cold, SV declined ~3.4 ml. HR was significantly higher (~11-14 bpm) in the hot environment than in the cold environment. In the heat, the HR rise per degree of dehydration was slightly higher in the heat than in the cold. Although CO was significantly higher in the heat compared to the cold under euhydration, it was lowered in proportion to the degree of dehydration. In the cold environment, CO was not significantly reduced under any degree of dehydration. Skin blood flow was also measured and was found to be significantly higher during exercise in the heat compared to the cold environment. Esophageal temperature in the heat was ~0.21°C higher at each level of dehydration compared to the cold. These data suggest that the reduction in SV with dehydration during moderate intensity exercise is associated with lower blood volume and increased core temperature.

Cardiovascular Drift and Exercise Mode

In most studies investigating cardiovascular drift, the mode of exercise used was cycling on an ergometer. However, Nassis and Geladas (32) investigated CV drift during cycling on an ergometer and running on a treadmill. The purpose was to determine if cardiac output decline associated with CV drift during prolonged exercise was affected by exercise mode. Eight men exercised on the cycle ergometer or treadmill at 60% $\dot{V}O_{2max}$ for 90 min or until volitional exhaustion. While performing the exercise, $\dot{V}O_2$ was measured, HR was recorded every five min

and CO was measured using CO_2 rebreathing method. SV was determined from HR and CO. Forearm blood flow and core body temperature were also measured. Absolute $\dot{V}O_2$ during treadmill exercise was higher than during cycling. During running, there was a progressive rise in $\dot{V}O_2$ until the 43rd min, whereas on the cycle, $\dot{V}O_2$ remained constant. CO was higher during running than cycling and remained constant, whereas during cycling it progressively declined over time. HR increased progressively over time by ~11-12% in both modes of exercise, however SV declined to a greater extent in cycling (~22%) compared to running (~12%). Forearm blood flow declined during running, however it remained the constant during cycling. Rectal temperature increased progressively during both modes of exercise, but was higher in running compared to cycling.

In another study, Nassis and Geladas (33) determined if water ingestion during prolonged exercise affected cardiovascular and thermal responses during cycling and running. Eleven men performed four trials of exercise on either a treadmill and cycle ergometer at 60% $\dot{V}O_{2max}$ for 90 min. Two trials were performed on the cycle and two on the treadmill. Under each mode of exercise, subjects either exercised with no fluid intake or they were given tap water to replace 60% of fluids lost. During exercise, $\dot{V}O_2$, CO and HR were measured, and SV was determined from CO and HR. Changes in forearm blood flow and core body temperature were recorded. $\dot{V}O_2$ was not affected by the levels of hydration, though it was higher during running than cycling. Cardiac output was higher during running than cycling at 25 minutes of exercise. With no fluid intake, CO progressively declined during both modes of exercise, but remained higher in running than cycling. Water ingestion blunted the decline in CO in both exercise modes. SV declined progressively during both modes of exercise when fluid was ingested, though the

decline was less than without fluid ingestion. During the no-fluid trial, SV declined significantly in both trials after 45 min of exercise though the decline during cycling was more profound than running. During both modes of exercise, forearm blood flow rose during the first 20 min of exercise and then remained fairly constant, though it was significantly higher when no fluid was ingested during running than when fluid was ingested. Rectal temperature rose progressively over time during all trials, but it was significantly higher in running when no fluid was ingested than when fluid was ingested. These data suggests that water ingestion prior to prolonged exercise may help to improve physical and cardiovascular function.

$\dot{V}O_{2max}$ *during Treadmill and Cycle Exercise*

In studying the relation of CV drift to $\dot{V}O_{2max}$ during treadmill walking and cycling, it is important to know if there are any fundamental differences in cardiovascular and metabolic responses during these two exercise modes. A number of studies have compared physiological responses to treadmill running or walking, and cycling on an ergometer, to determine if there are differences in cardiovascular responses to exercise during cycling and running. Faulkner et al. (15) studied whether or not there were differences during submaximal and maximal cycling and running. Eight men performed 70 tests on the bicycle ergometer and 45 tests on the treadmill. Half of the tests were performed at 100% physical work capacity. Each test was completed until exhaustion or until a maximum of 10 min. Three workloads were completed on a single day with 15 min of rest between each exercise session. During each test, measures of HR, SV, CO, $(a\text{-}v)O_2$ difference and $\dot{V}O_2$ were taken during submaximal and maximum exercise. The results showed that $\dot{V}O_{2max}$ was higher (~11%) during running than cycling. HR was not different during the exercise modes however SV was lower during cycling than running. This resulted in

a lower CO during cycling and a lower $\dot{V}O_{2max}$, compared to running. There was no difference in (a-v)O_2 difference between the two modes.

Shephard (43) assessed the difference in maximal oxygen uptake during step exercise, cycling and running. Measures of heart rate, $\dot{V}O_2$, and CO were taken during exercise. Maximum CO and SV in all subjects were significantly higher during treadmill running than cycling. There was no significant difference in (a-v)O2 difference. The $\dot{V}O_{2max}$ from treadmill running on a grade was 6.6% higher than during cycling. No significant difference was found in HR_{max} between modes of exercise.

A study by Hermansen and Saltin (24) investigated the oxygen uptake during maximal treadmill and bicycle exercise. Fifty-five male subjects ranging from athletes to sedentary individuals exercised at a variety of exercise intensities and performed 2-4 maximal determinations on each ergometer. During the exercise, HR, $\dot{V}O_2$ and measurements of blood lactate were taken. The results of this investigation showed that $\dot{V}O_{2max}$ was higher (~7%) in all groups on the treadmill compared to the cycle. Despite this difference they found no difference in the mean maximal values for pulmonary ventilation, heart rate or blood lactate concentration. During submaximal exercise, heart rate, pulmonary ventilation and blood lactate were higher during cycling that running at the same work rates.

A similar study investigating the physical work capacity and $\dot{V}O_{2max}$ in treadmill and cycle exercise by McArdle and Magel (30) had 23 men perform $\dot{V}O_{2max}$ tests on the cycle and treadmill. Subjects either walked on the treadmill or cycled and expired gases were collected. The results of this study showed about 10% reduction in $\dot{V}O_{2max}$ on the cycle compared to the treadmill. $\dot{V}O_2$ was lower during submaximal exercise on the cycle compared to the treadmill at

the same heart rate. There were no differences in HR at PWC_{max}. Also, minute ventilatory volume (VE) was similar at all work levels during both modes of exercise and respiratory exchange ratio (RER) was significantly higher during bicycle exercise than treadmill during submaximal and maximal exercise.

Kamon and Pandolf (27) investigated the differences in maximal aerobic power during climbing, uphill running and cycling. Twelve females and eleven males, ranging from inactive to vigorously active were given cycle, running and climbing tests that resulted in the measurement of maximal aerobic capacity. Across all levels of activity in the men and women, a running test achieved a greater $\dot{V}O_{2max}$ than the other forms of exercise. Also, there was a significant difference between men and woman across all activity levels, men having a much higher $\dot{V}O_{2max}$. During this study, they found an 11% difference in $\dot{V}O_{2max}$ during cycling and running in men and a 7% difference in females, with running producing the higher $\dot{V}O_{2max}$.

Effect of Prior Prolonged Exercise on Maximal Oxygen Uptake

One concern with determining $\dot{V}O_{2max}$ immediately after prolonged exercise as in the current study is whether or not the prior exercise will affect the ability of one to reach $\dot{V}O_{2max}$. Stamford et al. (44), investigated whether severe prior exercise affected $\dot{V}O_{2max}$. Five moderately-trained men performed 14 continuous $\dot{V}O_{2max}$ tests on a treadmill separated by 10, 20, 30 or 40 minutes of rest. They found that performance time for each test was reduced, however, there was no significant reduction in $\dot{V}O_{2max}$.

Saltin and Stenberg (40) had four subjects exercise for 195 min under thermoneutral conditions at 75% of maximal oxygen uptake. During this time, they saw a progressive increase in HR by about 10% and a progressive decrease in SV of about 15%, indicating CV drift

occurred. After a 90 min rest, subjects resumed submaximal exercise and then three subjects completed a maximal load. They found that $\dot{V}O_{2max}$ was decreased by only 5%. These data suggest that for a brief period of time, the cardiovascular alterations characterizing cardiovascular drift, mainly reduced SV, can be overcome an that cardiovascular capacity and $\dot{V}O_{2max}$ are not reduced much.

Ekblom (12) measured $\dot{V}O_{2max}$ immediately after 1 h of cycling at 75% $\dot{V}O_{2max}$ under thermoneutral conditions before and after aerobic training in seven male subjects when CV drift occurred. SV measured during maximal exercise immediately after the 60 min of submaximal exercise was unchanged from that at 60 min, indicating that the SV changes that occurred with CV drift persisted during maximal exercise. $\dot{V}O_{2max}$ was reduced 6% following the 60 min of submaximal exercise before training and 12% after training, compared to control. These data suggest that the decrease in SV during prolonged exercise persists during maximal exercise and causes decreased $\dot{V}O_{2max.}$

Costill et al. (7) studied the effect of exhaustive running on muscle glycogen utilization. Five well trained male subjects completed a 10-mile treadmill run, at approx ~77-80 %$\dot{V}O_{2max,}$ and then performed a $\dot{V}O_{2max}$ test after a 45 min rest. During the exercise HR, rectal temperature and respiratory measures were attained. In all subjects, HR rose progressively during the 10-mile run, and $\dot{V}O_{2max}$ was reduced by 8% following submaximal exercise, compared to the preliminary $\dot{V}O_{2max}$ test. They found no differences in HR_{max} and conclude that further information is needed to establish the cause for the reduction in aerobic capacity.

Astrand et al. (3) investigated blood lactate concentrations after prolonged severe exercise. Six female and eighteen male athletes, competed in a cross country skiing competition,

and following competition a finger-tip blood sample was taken from each athlete 1-3 min post competition. Measures of blood lactate and glucose were taken. Also athletes performed maximal work on a bicycle for 3 min and a blood sample was taken and analyzed for lactate concentrations. A measure of metabolic rate was determined by having the skiers keep a constant speed around a 300-m track for 10 laps. $\dot{V}O_2$ was measured during the last min of skiing. When $\dot{V}O_{2max}$ tests were performed after competition, there was a reduction ~10% from normal values. They assume that a reduction in anaerobic capacity, as reflected by blood lactate concentration, caused a reduction in maximal work load.

Chapter 3

EFFECT OF CARDIOVASCULAR DRIFT ON MAXIMAL OXYGEN UPTAKE DURING WALKING AND CYCLING IN WOMEN[1]

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Abstract

This study investigated whether the progressive rise in heart rate (HR) and fall in stroke volume (SV) during prolonged, moderate-intensity exercise (cardiovascular drift) in a thermoneutral environment is associated with a reduction in $\dot{V}O_{2max.}$ CV drift was measured on nine moderately-trained women (age = 23.3±1.8 yr, $\dot{V}O_{2max}$ = 43.0±5.5 ml/kg/min) between 15 and 45 min of cycling and treadmill walking at 60% $\dot{V}O_{2max,}$ with $\dot{V}O_{2max}$ measured immediately thereafter without cessation of exercise. During a separate trial, $\dot{V}O_{2max}$ was measured after 15 min submaximal exercise, so that change in $\dot{V}O_{2max}$ that occurred between 15 and 45 min could be associated with CV drift. This protocol was performed during two different modes of exercise, cycling and walking. There was no significant effect of exercise mode on the maginitude of CV drift or change in $\dot{V}O_{2max.}$ In cycling and walking, respectively, a 5.5% increase in HR (150±14 vs 158±17 bpm, $p<0.05$) and a 10% decrease in SV (77±10 vs 68±9 ml, $p<0.05$) between 15 and 45 min was associated with a 5% reduction in $\dot{V}O_{2max}$ (2.70±0.40 vs 2.52±0.57 l/min, $p<0.05$). There was a significant relationship between ΔHR and $\Delta\%\dot{V}O_{2max}$ ($r=0.47$, SEE=0.32 l/min, $p<0.05$). Submaximal $\dot{V}O_2$ was unchanged over time ($p<0.05$), but $\%\dot{V}O_{2max}$ increased 5.2%. We conclude that CV drift is associated with a decrease in $\dot{V}O_{2max}$ during walking and cycling. Increased HR reflects increased relative metabolic intensity during prolonged cycling and walking. This study supports the validity of using HR as an indicator of relative metabolic intensity during prolonged cycling and walking when CV drift occurs.

INDEX WORDS: heart rate, stroke volume, cycling, walking, oxygen consumption

Introduction

Prolonged moderate-intensity (50-75% $\dot{V}O_{2max}$) exercise leads to a downward drift in central venous pressure, stroke volume, and pulmonary and systemic arterial pressures, while during this time a rise in heart rate maintains nearly constant cardiac output (38). These slow progressive changes have been termed cardiovascular drift (CV drift). CV drift occurs in thermoneutral (13, 16, 32, 33, 40) and hot environments (20, 22, 31, 39), beginning after about 10 min of dynamic exercise and continuing for several hours. The cause of CV drift is unknown, but two theories have been proposed. One hypothesis is that the cause of CV drift is a progressive rise in cutaneous blood flow and venous volume in response to increasing hyperthermia, reducing ventricular filling pressure, thus decreasing end diastolic volume limiting stroke volume (25, 38). According to this theory, the rise in heart rate is presumably in response to the drop in stroke volume and arterial pressure. An alternative hypothesis is that the increase in heart rate due to increased body temperature and increased sympathetic nervous activity causes a decline in stroke volume due to decreased diastolic time (8, 16).

The consequences of cardiovascular drift for physical work capacity are uncertain. It is not known whether the decrease in stroke volume during prolonged exercise reflects a decrease in cardiovascular capacity and maximal oxygen uptake ($\dot{V}O_{2max}$) or not. Three studies in which CV drift occurred during prolonged exercise in a thermoneutral environment have observed 5-12% declines in $\dot{V}O_{2max}$ following exercise (12, 40, 41). However, in these studies, $\dot{V}O_{2max}$ was not measured at the same points in time as the variables characterizing CV drift. $\dot{V}O_{2max}$ after exercise usually was measured after a rest period. Additional research is needed on the

consequences of CV drift during shorter periods of time that do not include rest, with different modes of exercise.

Mode of exercise appears to affect the magnitude of CV drift. Nassis and Geladas (32) compared CV drift in 90 min of running and cycling at 60% of mode-specific $\dot{V}O_{2max}$ in thermoneutral conditions. They found a larger decrease in SV but similar increases in HR during cycling compared to running, in spite of greater dehydration and hyperthermia with running. Cardiac output decreased during cycling, but not in running. The authors suggested that there may be a more efficient muscle pump during running than cycling due to greater muscle relaxation time and lower compressive forces. Whether the different degree of CV drift during prolonged running and cycling would be reflected in different effects on $\dot{V}O_{2max}$ is unknown.

The purpose of this study was to compare the magnitude of cardiovascular drift during treadmill walking and cycling and to determine whether CV drift is associated with a reduction in $\dot{V}O_{2max}$. This was accomplished by measuring CV drift between 15 and 45 min of cycling or walking and then immediately measuring $\dot{V}O_{2max}$. $\dot{V}O_{2max}$ was also measured immediately after 15 min of cycling and walking on separate days so that a change in $\dot{V}O_{2max}$ between 15 and 45 min of exercise could be associated with the CV drift that occurred during the same time interval.

Methods

Subjects:

Nine healthy women volunteered as subjects. Sample size was estimated based on the number of subjects needed to have adequate statistical power to detect a meaningful (5%) change in $\dot{V}O_{2max}$, using a one-way ANOVA with paired observations (29). Nine subjects were

sufficient to detect a 5% decrease in $\dot{V}O_{2max}$ with statistical power of .80 using a two-tailed test and alpha =0 .05, assuming individuals have a mean $\dot{V}O_{2max}$ of 43 ml/kg/min with a SD of 5.5 ml/kg/min and that the correlation is .90 between repeated tests of $\dot{V}O_{2max}$. Subjects physical characteristics (means ± SD) were: age = 23.3±1.8 yr, mass = 62.5±8.5 kg, height =166.1±4.5 cm, and percent body fat estimated by skinfolds = 20.8±5.4%. Five women were taking a tri-phasic oral contraceptive and four were not. Six subjects were tested during the mid-follicular phase and three were tested in luteal phase of the menstrual cycle. Subjects were moderately trained and engaged in regular or structured aerobic exercise for at least 30-60 min three times a week. The study was approved by the University's Institutional Review Board and written informed consent was obtained prior to testing.

***Experimental Design*:**

A repeated measures experimental design, in which subjects served as their own control was used. The dependent variables were cardiovascular measures characterizing cardiovascular drift and maximal oxygen uptake. CV drift was defined as the changes in HR and SV that occured between min 15 and 45 during a 45 min bout of exercise at 60% $\dot{V}O_{2max}$. $\dot{V}O_{2max}$ was measured immediately after 15 and 45 min of cycling and walking on separate days. The independent variable was exercise mode. The association between CV drift and change in $\dot{V}O_{2max}$ over the same time was determined during walking and cycling. The order of the walking and cycling tests, and the tests of $\dot{V}O_{2max}$ following 15 and 45 min for each mode, was randomly assigned. Each subject was tested at the same time of day to minimize effects of circadian rhythm on heart rate and core body temperature. The trials were separated by one day.

Protocol and Procedures:

Prior to reporting for testing, subjects were instructed to fast for 3 h prior to testing and to ensure adequate hydration by drinking plenty of fluids. They were also instructed to refrain from consuming alcohol, caffeine, or non-prescription drugs the day before or the day of testing. On the morning of each test, they completed a 24-h history questionnaire designed to determine proper adherence to pretest instructions.

Control $\dot{V}O_{2max}$. A control $\dot{V}O_{2max}$ was measured on the cycle ergometer and treadmill on separate days. Testing was conducted on an electronically-braked cycle ergometer (Lode Excalibur Sport, Lode B.V., Groningen, NL) or on a treadmill (Trackmaster, TMX 3030C, Eastlake, OH) in an environmental chamber maintained at room temperature, approximately 22°C, 44% relative humidity. Subjects warmed up on either the cycle or treadmill for 10 min prior to beginning the test. A continuous, progressive, load-incremented protocol was used for the cycle and treadmill tests. Following a brief rest, the subjects cycled at 40 W, with the load increased 25 W every 2 min, or walked at 3.8 mph at 5% grade, with the grade increased by 2.5% every 2 min until subjects were unable to maintain power output. $\dot{V}O_2$ and related gas exchange measures were determined by indirect calorimetry over 30-sec intervals using a Parvo Medics TrueOne 2400 Metabolic Measurement System (Parvo Medics, Inc., Salt Lake City, UT) Prior to each test, the gas analyzers were calibrated using gases of known concentrations, and the flowmeter was calibrated using a 3-1 syringe. HR and ratings of perceived exertion (RPE) were measured each min. HR was measured with a Polar® Vantage XL heart rate monitor (Polar Electro, Inc. Woodbury, NY, model 145900). RPE was measured by the Borg 15-point category scale using standardized instructions (5). Three min after completing the test a finger stick blood

sample was obtained for determination of blood lactate concentration. Blood lactate concentration was measured using a YSI 2300 Stat Plus Analyzer (Yellow Springs Instruments, Inc., Yellow Springs, OH). After completion of the $\dot{V}O_{2max}$ test, subjects completed an additional bout of exercise following 20 min of rest. Subjects exercised to exhaustion at a power output equivalent to the last workload performed during the initial test (if <1 min was completed during the last stage of the graded test) or at a power output 2.5% higher if on the treadmill, or 25W higher on the cycle (if ≥ 1 min was completed during the last stage of the graded test). This was to ensure that a plateau in maximal oxygen uptake was obtained. The next day, $\dot{V}O_{2max}$ was measured on the other ergometer.

Attainment of $\dot{V}O_{2max}$ (average of the two highest consecutive 30-sec values) was determine using a modification of the plateauing criterion described by Taylor et al. (48). The criterion for determining plateau was an increase in $\dot{V}O_2$ (l/min) between the last two stages of less than half of the expected increase (~0.150 l/min) based on the American College of Sports Medicine metabolic equations (1). Using this protocol, all subjects demonstrated a plateau in $\dot{V}O_2$ during the initial bout of exercise.

Practice Trials. The next two pre-experimental trials were used to determine the power output appropriate to elicit 60% $\dot{V}O_{2max}$. Subjects completed 45 min of exercise on the treadmill and cycle ergometer at 60% $\dot{V}O_{2max}$, and practiced the rebreathing technique that was used to measure cardiac output. The workrate eliciting 60% $\dot{V}O_{2max}$ was estimated using the control $\dot{V}O_{2max}$ test results as well as a modified version of the American College of Sports Medicine metabolic equation (1):

$$W = M * ((0.6 * \dot{V}O_{2max})-7)/10.8 \quad \text{(For Cycling)}$$

$$\dot{V}O_2 = 0.1(\text{speed}) + 1.8(\text{speed})(\text{frac grade}) + 3.5 \text{ ml/kg/min} \quad \text{(For Treadmill)}$$

$\dot{V}O_2$ was measured with the Parvo Medics metabolic system for the first 10 min of exercise to make sure that workrate elicited 60% $\dot{V}O_{2max}$. If workrate was not correct, then adjustments were made after min 5 and then again after min 10 (if needed).

Experimental Trials. Prior to exercise, subjects followed the same pretest instructions as during the control $\dot{V}O_{2max}$ tests. To make sure that subjects followed instructions, subjects completed a 24-hour history questionnaire. Tympanic temperature (T_t) and urine specific gravity (USG) were measured to verify that subjects did not have a fever (T_t<37.8°C) and that they were euhydrated (USG<1.030). After this, subjects measured their nude body weight on a platform scale (A&D Co., Ltd., Tokyo, model FW-150KA1) and inserted a rectal temperature probe. A Teflon® venous catheter was inserted into the antecubital vein and was kept patent by 0.5 ml of 10 USP units/ml heparin lock flush. Subjects then sat on the cycle ergometer or stood on the treadmill for about 20 min, allowing plasma volume to stabilize. Thermistors were placed on the chest, deltoid, thigh, and calf for determining skin temperature; resting metabolic, cardiovascular, and thermal measures were taken; and a 6-ml blood sample drawn. Subjects then began to walk or cycle at prescribed intensity estimated to elicit 60% $\dot{V}O_{2max}$. During the 45 min trials on both ergometers, beginning at min 8 and min 38, subjects were connected to the Parvo Medics system for 2 min for a measurement of $\dot{V}O_2$ and $\dot{V}CO_2$. Heart rate was then measured using the Polar® HR monitor, systolic (SBP) and fourth-phase diastolic (DBP) blood pressures were measured by auscultation, and a rating of perceived (RPE) was assessed. A 6-ml blood sample was drawn for determination of hematocrit, and hemoglobin, blood lactate concentration. Then, two trials of CO_2-rebreathing were performed for a measurement of CO.

At the end of the 15 and 45 min trials, subjects immediately began a graded exercise test to measure $\dot{V}O_{2max}$, with no cessation of exercise. The protocol involved increasing exercise intensity until subjects could no longer continue. On the cycle, power output was initially increased 25 W above the power output maintained during the submaximal exercise (60% $\dot{V}O_{2max}$), with additional 25 W increments every 2 min. On the treadmill, speed remained the same but percent grade was increased 2.5% every two min above the grade maintained during submaximal exercise. $\dot{V}O_2$ and other metabolic measures were measured over 30 s intervals with the Parvo Medics system, HR was measured using the Polar® monitor and RPE was obtained every two min during the $\dot{V}O_{2max}$ test until exhaustion. After the completion of the test, a 6-ml blood sample was taken. Then, subjects toweled off and re-measured nude body weight. The measure of $\dot{V}O_{2max}$ on the experimental trials was considered valid if (1) the plateauing criteria used for the control test was met, or (2) if a maximal HR within 5 bpm of that on the control test was obtained. If one of these criteria was not met, then the test was repeated.

Cardiac output was measured using the "indirect" Fick method of CO_2 rebreathing, described by Jones et al. (26), using the Parvo Medics metabolic system and software. During rebreathing, subjects breathed in a content of a medical gas mixture of CO_2 and $\dot{O}_2$ from a sterile anesthesia bag 3 times in succession beginning at min 10 and 40 for about 12 sec. Contents of CO_2 in mixed venous and arterial blood are calculated from estimates of the respective partial pressures using a CO_2 dissociation curve. $PaCO_2$ is estimated from the end-tidal PCO_2 and $PvCO_2$ is estimated using the Collier CO_2-rebreathing method. The reliability of values from the two trials was high (interclass correlation = 0.93). SV was calculated by dividing cardiac output

by HR. Mean arterial pressure (MAP) was estimated as MAP = DBP+0.33(SBP-DBP). Systemic vascular resistance (SVR) was calculated by dividing MAP by cardiac output.

Blood samples were taken from the venous catheter into tubes containing EDTA. Hemoglobin concentration was measured in duplicate using a HemoCue B-Hemoglobin Photometer (HemoCue AB, Angelholm Sweden). Hematocrit was measured in triplicate using the microcapillary technique. Plasma volume change from rest was estimated from measures of hemoglobin and hematocrit using the equation of Dill and Costill (10).

Rectal temperature (T_{re}) was measured using a temperature probe (Ellab, Inc., Arvada, CO, model MOV-55044-A) inserted 10 cm past the anal sphincter. Skin temperature was measured using thermocouples (Ellab, Inc., Arvada, CO, model MHF-18058-A) attached to the chest, deltoid, thigh and calf. The temperature probes were connected to a temperature data acquisition system (Ellab, Inc. model TM9608 with Eval 2.1 software), which collects and stores data continuously. Mean skin temperature ($\overline{T}_{sk}$) was calculated according to the formula of Ramanathan (36).

$$\overline{T}_{sk} = 0.3(T1+T2) + 0.2(T3+T4)$$

With T1, T2, T3 and T4 representing the chest, deltoid, thigh and calf respectively. Mean body temperature ($\overline{T}_b$) was calculated from Tre and Tsk with the formula of Baum et al. (4):

$$\overline{T}_b = 0.87 * T_{re} + 0.13 * \overline{T}_{sk}$$

To evaluate the comparability of changes in HR during submaximal exercise and changes in relative metabolic intensity caused by possible changes in $\dot{V}O_2$ during submaximal exercise or $\dot{V}O_{2max}$, %HRR and %$\dot{V}O_2$R utilized during submaximal exercise were calculated. %HRR was

calculated as: $[(HR_{ex} - HR_{rest})/(HR_{max} - HR_{rest})] \times 100$. $\%\dot{V}O_2R$ was calculated as: $[(\dot{V}O_{2ex} - 3.5/(\dot{V}O_{2max} - 3.5)] \times 100$.

Statistical Analysis:

Data was analyzed using SPSS v. 11 for Windows (SPSS Inc., Chicago, IL). Data is reported as means ± SD for all variables by mode of exercise and time point. Measures associated with CV drift and $\dot{V}O_{2max}$ were analyzed using a two-factor (Mode x Time) ANOVA with repeated measures on both factors. Post hoc repeated measures t tests using a Bonferroni adjusted alpha level at 0.05 was used to test the significance of change between time points within each mode and the difference between modes at each time point.

Results

Responses to submaximal exercise. Changes in cardiovascular and metabolic measures during submaximal exercise between min 15 and min 45 during both modes of exercise are contained in Table 1. There were few significant differences between responses to cycling and walking; however, there was a main effect for time across conditions for most measures. There was no difference in submaximal $\dot{V}O_2$ between min 15 and min 45. The average relative metabolic intensity was ~61% of control $\dot{V}O_{2max.}$ Blood Lactate was not significantly different between cycling and walking and there was no change over time, averaging ~1.9 mmol/l. There was a significant CV drift in cycling and walking as reflected by an increase in HR of 8 bpm (~5.5%; $p<0.05$) and a decrease in SV of 8.5 ml (~11%; $p<0.05$)(Figure 1). A reduction in O_2 pulse of 8% paralleled the decrease in SV.

Table 1: Responses during submaximal exercise (means±SD)

	Walking		Cycling	
Variable	15-min	45-min	15-min	45-min
$\dot{V}O_2$ (l/min)	1.66 ± 0.33	1.62 ± 0.34	1.56 ± 0.36	1.61 ± 0.38
% of control $\dot{V}O_{2max}$	62.6 ± 3.7	61.3 ± 7.7	58.4 ± 4.3	60.5 ± 3.7
CO (l/min)*	11.7 ± 1.9	10.9 ± 2.2	11.3 ± 2.3	10.7 ± 2.2
SV (ml/bt)*	79.4 ± 10.6	68.7 ± 7.7	78.1 ± 11.5	68.3 ± 9.2
HR (bpm)*	152.1 ± 12.3	159.4 ± 15.3	147.2 ± 15.9	156.2 ± 19.6
O_2 pulse (ml/bt)	17.7 ± 3.3	16.3 ± 2.2	17.1 ± 2.8	16.8 ± 2.9
MAP (mm Hg)*	93.1 ± 5.5	94.4 ± 6.7	92.1 ± 7.0	95.4 ± 6.7
TPR (mm Hg/L/min)*	8.1 ± 1.4	9.0 ± 1.9	8.4 ± 1.8	9.2 ± 1.9
Blood Lactate (mmol/l)	1.6 ± 0.7	1.7 ± 0.7	1.9 ± 1.3	2.3 ± 1.6
T_{re} (°C)*	37.8 ± 0.2	38.3 ± 0.3	37.7 ± 0.4	38.1 ± 0.3
$\overline{T}_{sk}$ (°C)	31.0 ± 1.2	32.3 ± 2.0	31.9 ± 1.4	32.6 ± 2.1
$\overline{T}_b$ (°C)*	36.6 ± 0.3	37.5 ± 0.4	36.9 ± 0.4	37.4 ± 0.3
RPE‡	11.8 ± 0.7	12.7 ± 1.1	11.4 ± 0.9	13.1 ± 2.3
Δ PV from Rest (%)‡	-5.5 ± 3.6	-3.7 ± 3.0	-7.8 ± 3.5	-7.6 ± 3.4

$\dot{V}O_2$ = oxygen uptake, CO = cardiac output, SV = stroke volume, HR = heart rate, MAP = mean arterial pressure, TPR = total peripheral resistance, T_{re} = rectal temperature, $\overline{T}_{sk}$ = mean skin temperature, $\overline{T}_b$ = mean body temperature, RPE = rating of perceived exertion, * = Time effect $p<0.05$, ‡ = Condition × Time interaction $p<0.05$.

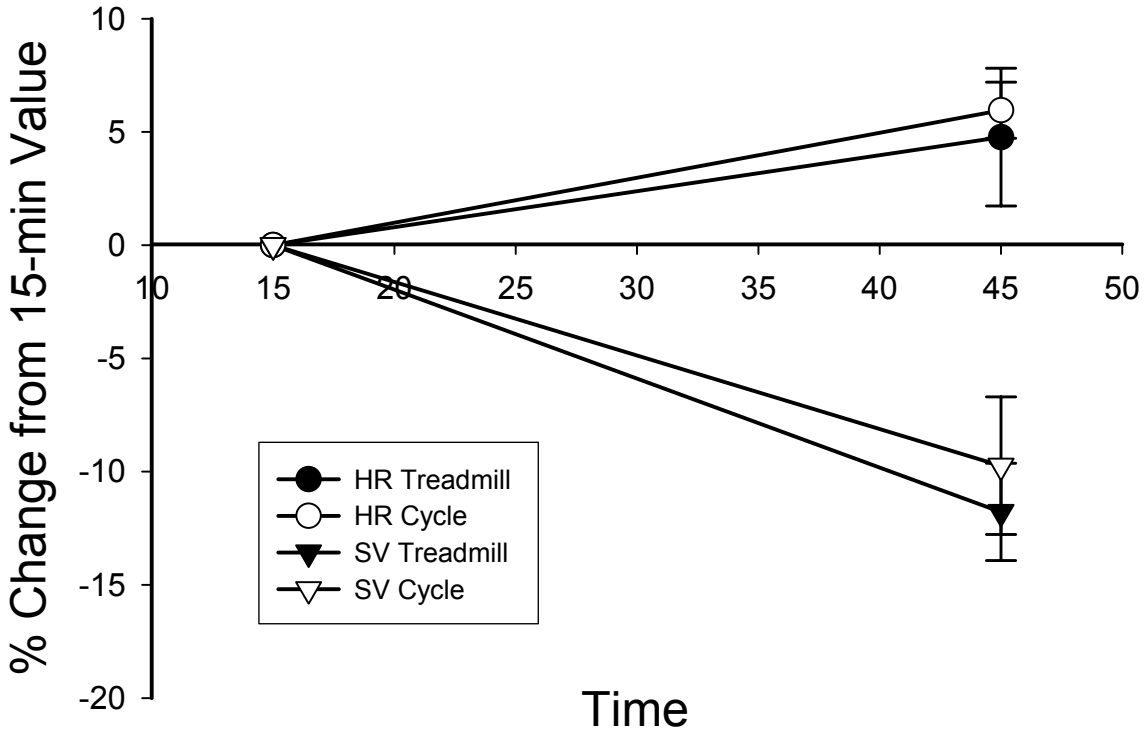

Figure 1. Changes in heart rate (HR) and stroke volume (SV) between 15 and 45 min of treadmill walking and stationary cycling.

Cardiac Output was reduced by approximately 0.75 l/min (~6%; $p<0.05$), and mean arterial pressure and total peripheral resistance were increased ($p<0.05$). T_{re} and $\overline{T}_b$ increased by 0.5 and 0.7 °C ($p<0.05$), respectively, but there was no difference in $\overline{T}_{sk}$ between min 15 and min 45.

There was a significant ($p<0.05$) Condition × Time interaction for RPE; the increase between min 15 and min 45 was not significantly different during treadmill walking, but it was significant during cycling. There also was a significant ($p<0.05$) Condition × Time interaction for percent change in plasma volume from rest. There was a significantly greater difference ($p<0.05$) in plasma volume from rest during cycling compared to treadmill walking at 45 min than 15 min. There was no significant difference in plasma volume reduction from rest during walking at min 15 and min 45, whereas during cycling, there was a greater decline ($p<0.05$) in plasma volume from rest at min 15 than at min 45.

Responses to maximal exercise. Data on $\dot{V}O_{2max}$ and related measures during the control and experimental tests are presented in Table 2 and in Figure 2. $\dot{V}O_{2max}$ measured during cycling and walking after 15 and 45 min of submaximal exercise was not significantly different from that measured during the control $\dot{V}O_{2max}$ test with the same mode of exercise. There was no difference from control in $\dot{V}E$, RER, HR, and RPE measured at $\dot{V}O_{2max}$ for either mode of exercise. However, $\dot{V}O_{2max}$ measured following 45 min of submaximal exercise was significantly lower ($p<0.05$) than that measured following 15 minutes of submaximal exercise by 0.19 l/min (6.9%) in walking and 0.16 l/min (6.0%) in cycling. A significant time effect also was evident for test duration, maximal power output, T_{re} and change in body mass. There were no significant differences among experimental tests on indicators of maximal effort, RER, RPE

and HR during cycling and walking. The only significant difference between test modes was that Δ%PV from rest was significantly ($p<.05$) greater across both time points during cycling compared to treadmill walking.

Relation of CV drift to $\dot{V}O_{2max}$. Change in HR was significantly correlated with $\Delta\dot{V}O_{2max}$ for both treadmill walking ($r=-0.49$, $p<0.05$) and for cycling ($r=-0.66$; $p<0.05$) (Figure 3). Change in SV was significantly correlated to $\Delta\dot{V}O_{2max}$ during cycling ($r=79$; $p<0.05$), but not during treadmill walking ($r=0.21$; $p>0.05$).

Consequences of altered $\dot{V}O_{2max}$ *for exercise prescription.* The metabolic consequences of CV drift are summarized in Figure 4. As mentioned, there was no significant difference in submaximal $\dot{V}O_2$ between 15 and 45 min of exercise during cycling and treadmill walking. But, $\dot{V}O_{2max}$ was reduced by 5% after 45 min, and, therefore, the calculated relative metabolic intensity, i.e., $\%\dot{V}O_{2max}$, was increased 5.2 points ($p<0.05$). The change in HR between 15 and 45 min for both exercise modes was significantly related to the increase in $\%\dot{V}O_{2max}$ ($\Delta\%\dot{V}O_{2max} = 0.68\ \Delta HR + 0.47$, $r=0.47$, SEE=7.9%, $p<0.05$). Based on the regression coefficient, for every 1 bpm change in HR, there was approximately a two-thirds unit change in $\%\dot{V}O_{2max}$. The increase in the %HRR of 6 points (from 65±9 to 71±9) was the same as the increase in mean $\%\dot{V}O_2R$ (from 56±7 to 62±14). Individual changes in %HRR and $\%\dot{V}O_2R$ were significantly related ($\%\dot{V}O_2R = 1.18\ \Delta\%HRR + 0.48$, $r=0.48$, SEE=10.2%, $p<0.05$). Based on the regression coefficient, for every 1% change in HRR there was a little over a 1% change in $\dot{V}O_2R$.

Table 2. Responses to maximal exercise (mean ± SD).

			Treadmill		Cycle	
Variable	Control(Treadmill)	Control(Cycle)	15-min	45-min	15-min	45-min
$\dot{V}E$ STPD (l/min)	83.5 ± 10.5	86.3 ± 13.5	85.2 ± 14.6	83.3 ± 10.8	86.5 ± 17.1	81.3 ± 13.3
$\dot{V}O_{2max}$ (l/min)	2.72 ± 0.45	2.65 ± 0.51	2.74 ± 0.47	2.55 ± 0.54†	2.65 ± 0.51	2.49 ± 0.59†
$\dot{V}O_{2max}$ (ml/kg/min)	43.6 ± 5.1	42.5 ± 6.1	44.2 ± 5.3	41.0 ± 6.90†	42.6 ± 5.7	40.3 ± 7.84†
RER	1.1 ± 0.1	1.1 ± 0.1	1.1 ± 0.1	1.1 ± 0.1	1.1 ± 0.0	1.1 ± 0.0
RPE	20.0 ± 0.0	19.8 ± 0.7	20.0 ± 0.0	20.0 ± 0.0	20.0 ± 0.0	20.0 ± 0.0
HR (bpm)	192.3 ± 9.1	191.9 ± 11.2	191.6 ± 8.8	193.7 ± 10.0	189.1 ± 9.1	191.7 ± 10.1
O_2 Pulse (ml/bt)	22.7 ± 3.1	22.2 ± 3.5	23.1 ± 3.1	21.3 ± 4.0	22.6 ± 3.4	21.2 ± 4.6
Blood Lactate (mmol/l)	7.1 ± 1.8	7.4 ± 0.9	6.0 ± 1.6	5.4 ± 1.5	7.3 ± 1.3	6.3 ± 1.0
Δ PV from rest (%)			-10.0 ± 4.5	-8.3 ± 3.2	-15.7 ± 1.83‡	-13.8 ± 4.9‡
T_{re} (°C)			38.3 ± 0.4	38.5 ± 0.30†	38.1 ± 0.3	38.3 ± 0.30†
$\overline{T}_{sk}$ (°C)			31.9 ± 1.5	32.2 ± 2.0	32.8 ± 1.9	32.3 ± 2.0
$\overline{T}_b$ (°C)			37.5 ± 0.4	37.6 ± 0.4	37.4 ± 0.4	37.5 ± 0.3
Δ Body Mass (%)			-0.5 ± 0.3	-0.9 ± 0.41†	-0.3 ± 0.6	-0.9 ± 0.28†
Test Duration (min)	10.2 ± 1.9	14.6 ± 2.9	7.2 ± 1.02*	7.1 ± 1.11*	7.9 ± 2.23*	6.9 ± 2.21*
Work Rate	15.6 ± 2.4	201.1 ± 39.7	14.1 ± 2.3	13.6 ± 2.3	185.2 ± 45.4	179.8 ± 40.1

$\dot{V}E$ = minute ventilation, $\dot{V}O_{2max}$ = oxygen uptake, RER = respiratory exchange ratio, RPE = rating of perceived exertion, HR = heart rate, T_{re} = rectal temperature, $\bar{T}_{sk}$ = mean skin temperature, $\bar{T}_b$ = mean body temperature, * = $p<0.05$ from control, † = $p<0.05$ from 15-min within exercise mode, ‡ = $p<0.05$ between exercise modes.

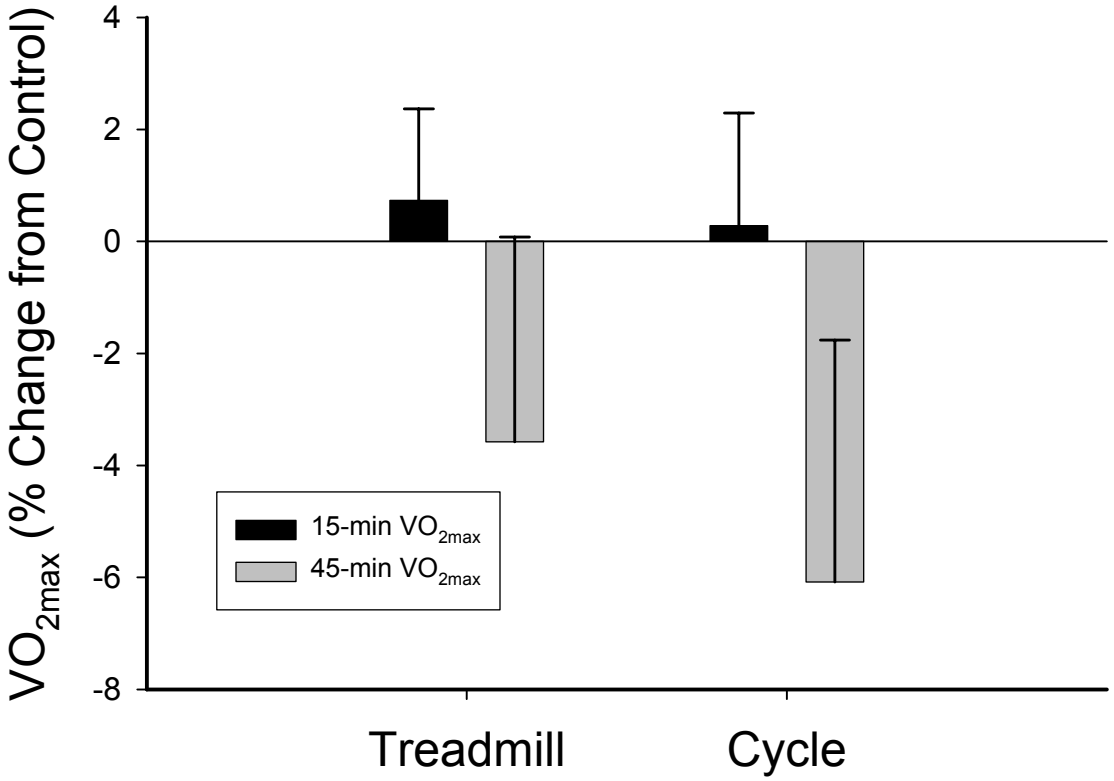

Figure 2. $\dot{V}O_{2max}$ measured following 15 and 45 min of treadmill walking and cycling.

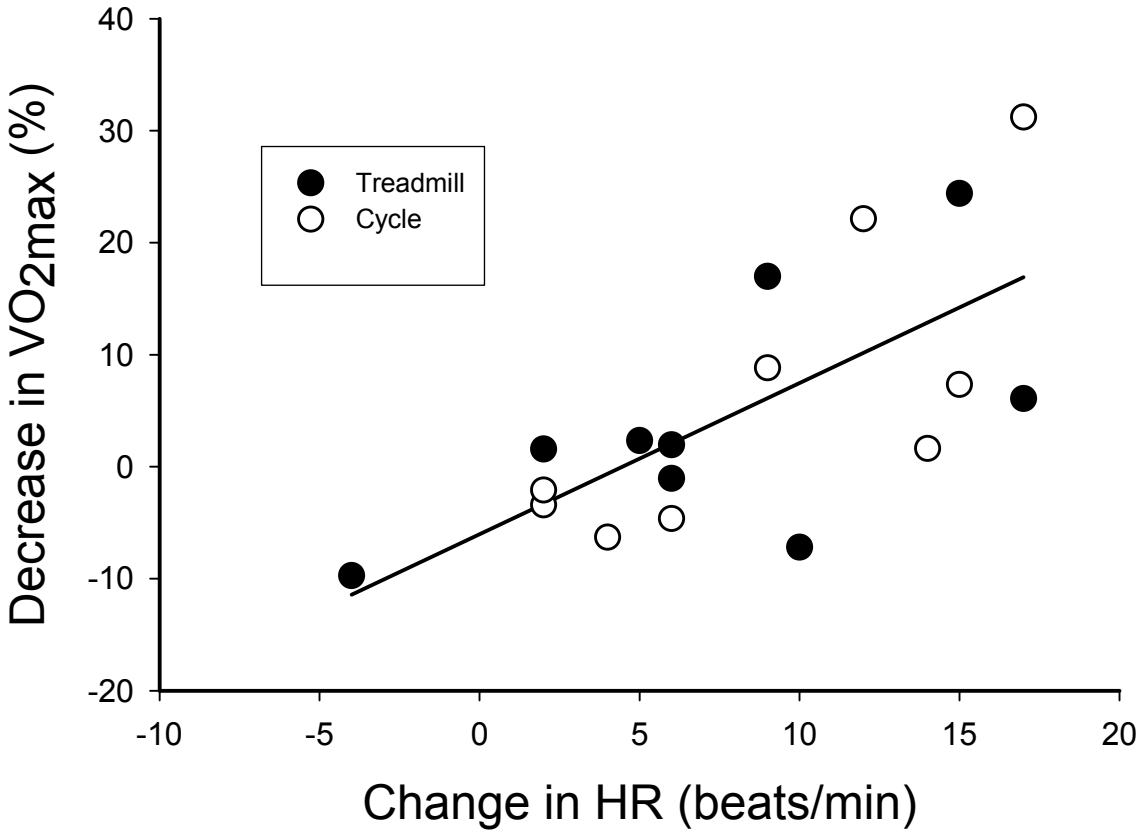

Figure 3. Relation of the change in heart rate (HR) between 15 and 45 min of treadmill walking and cycling to the decrease in $\dot{V}O_{2max}$ measured immediately thereafter.

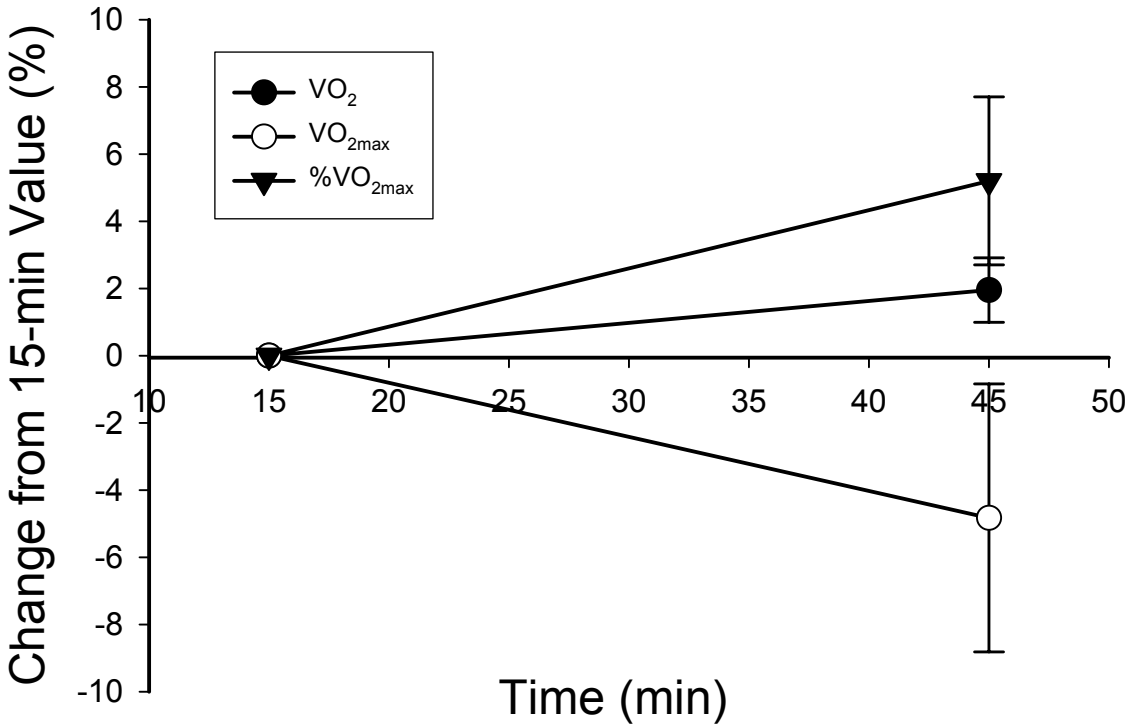

Figure 4. Metabolic consequences of cardiovascular drift.

Discussion

The primary findings of this study was that CV drift, as defined as the progressive rise in HR and progressive decline in SV, was not different between cycling and treadmill walking. During both modes of exercise, HR increased 8 bpm (~5.5%) and SV declined 8.5 ml (~10%). And these changes were associated with a decrease in $\dot{V}O_{2max}$ of 6-7% in both exercise modes over the same time period. Because $\dot{V}O_2$ was unchanged between 15 and 45 minutes, the relative metabolic intensity (%$\dot{V}O_{2max}$ and %$\dot{V}O_2$R) was increased 5-6 percentage points. Thus, the rise in HR over time appeared to reflect a proportional rise in relative metabolic intensity. These data support the practice of using HR as a marker of relative metabolic intensity during prolonged exercise.

The magnitude of CV drift that occurred during min 15 and min 45 of prolonged cycling and walking, falls within the range of values reported in other studies (12, 14, 16-18, 20, 21, 25, 31, 32, 34, 40, 42). Factors affecting the magnitude of CV drift include exercise intensity (42), duration (40), ambient temperature (17), hyperthermia (31), and dehydration (22, 31). During this study, subjects exercised at a modest intensity for a relatively short duration, typical of an exercise session for improving fitness, such that only moderate hyperthermia and minimal dehydration occurred. Because of these conditions, we expected only a modest CV drift.

The finding that there was no difference between exercise modes in the magnitude of CV drift is inconsistent with a previous study by Nassis and Geladas (32). They found that there was greater CV drift during cycling compared to running. HR rose ~11-12% and SV declined progressively during both exercise modes, but the decline was greater during cycling (~22%)

compared to running (~12%). They hypothesized that these changes were due to the more efficient muscle pump during running compared to cycling, which could blunt the drop in SV associated with CV drift (23). Also, they hypothesized that during cycling there are higher intramuscular forces (11), which could compress or partially occlude the blood vessels in the leg reducing venous return back to the heart (28). They found that despite higher levels of hyperthermia during treadmill running, there was less of an increase in skin blood flow, leading to greater central blood volume during running compared to cycling.

We also found that CO was significantly less after 45 min of exercise compared to 15 minutes of exercise in both exercise modes. Nassis and Geladas (32) found that during cycling CO dropped by 11%, but remained unchanged during running. They hypothesized that the decrease in CO during cycling was related to the greater drop in SV during cycling compared to walking. However, in our study, a decline in SV of ~10% was associated with a significant drop in CO by ~6%, regardless of the exercise mode. The differences between their study and ours could be that we used walking instead of running.

The other important finding of our study was that the reduced SV associated with CV drift was associated with a decrease in $\dot{V}O_{2max}$ in both walking and cycling. The decline in $\dot{V}O_{2max}$ was consistent with other studies in which $\dot{V}O_{2max}$ declined 5-12% following prolonged strenuous exercise in thermoneutral conditions (3, 7, 12, 40, 41). The decrease in $\dot{V}O_{2max}$ following 45 min of exercise during which CV drift occurred was not due to premature fatigue. Measures reflecting degree of effort and attainment of cardiovascular capacity indicated $\dot{V}O_{2max}$ was reached. During the experimental tests, subjects either attained the same or slightly greater

$\dot{V}O_{2max}$ compared to the control condition, or achieved HR_{max} within 5 bpm of control tests. Mean HR_{max} on the tests following 45 min of submaximal exercise were slightly higher by ~2 bpm than on the test following 15 min of submaximal exercise, and not different from the mean HR_{max} on the control tests, which suggests that cardiovascular capacity had been attained during experimental trials. Mean RPE at the point of exhaustion during all trials was at or near 20, indicating that near maximal effort was given. $\dot{V}E$, RER, and blood lactate accumulation were lower during the experimental trials following 45 min of submaximal exercise, but were not significantly different from control. The lower values most likely reflect the lower power output at $\dot{V}O_{2max}$ rather than lack of effort.

We found that $\dot{V}O_{2max}$ was decreased after 45 min of exercise in both modes of exercise in direct correlation with the increase in HR that occurred between 15 and 45 min of exercise at 60% $\dot{V}O_{2max.}$ Since submaximal $\dot{V}O_2$ was unchanged during prolonged cycling and walking, an increase in HR could have dissociated HR from relative metabolic intensity. However, since there was a reduction in $\dot{V}O_{2max,}$ relative metabolic intensity increased in concert with the rise in HR. This was consistent for both modes of exercise. A rise in HR (~5.5%) paralleled an increase in relative metabolic intensity (~5.2%). These data provide support for the validity of using changes in heart rate to reflect changes in relative metabolic intensity during prolonged cycling or treadmill walking.

The cause of reduced $\dot{V}O_{2max}$ following 45 min compared to 15 min of submaximal exercise is uncertain. HR_{max} was not reduced; therefore, according to the Fick principle, the decrease in $\dot{V}O_{2max}$ following 45 min must have been due to lower arteriovenous oxygen

difference [(a-v)O_2 difference] or SV, as reflected by the reduced O_2 pulse. Since it is unlikely that (a-v)O_2 difference was reduced (19, 39, 49), the lower SV associated with the reduction in $\dot{V}O_{2max}$ probably reflected a reduction in maximal SV and CO. Although a rise in HR during prolonged exercise has been hypothesized to cause reduced SV (8) during prolonged exercise, this could not have caused a reduced maximal SV and $\dot{V}O_{2max.}$ At $\dot{V}O_{2max}$ in our study, HR was not different following 15 and 45 min of submaximal exercise. If the reduced SV that existed at the end of 45 min of submaximal exercise persisted as exercise intensity increased to $\dot{V}O_{2max}$, it means that factors other than increased HR contributed to the reduced SV or that the expected increases in myocardial contractility and vasoconstriction in splanchnic, nonactive muscle and skin vascular beds did not occur as exercise intensity was increased during the $\dot{V}O_{2max}$ test.

Since SV was not measured at $\dot{V}O_{2max}$, we are not certain the SV decline during prolonged exercise (CV drift) caused the reduction in $\dot{V}O_{2max.}$ However, this seems plausible. The correlation of SV change to reduction in $\dot{V}O_{2max}$ was not significant during treadmill walking ($r=.21$, $p<0.05$) but was strong for cycling ($r=.79$, $p<0.05$). Two studies (12, 40) have reported that the decrease in SV during prolonged cycling persisted during maximal exercise and caused a reduction in $\dot{V}O_{2max,}$ supporting this deduction.

Dehydration and reduced blood volume did not appear to contribute to the greater decline in SV and $\dot{V}O_{2max}$ following 45 min of submaximal exercise. PV change from rest at $\dot{V}O_{2max}$ was similar after the 15 min and 45 min of submaximal exercise within mode. There a significantly greater change in PV from rest during cycling, but this did not appear to affect $\dot{V}O_{2max}$ or CV drift.

Greater hyperthermia at $\dot{V}O_{2max}$ following 45 min compared to 15 min of exercise could have caused the reduction in $\dot{V}O_{2max}$. T_{re} and $\overline{T}_b$ at $\dot{V}O_{2max}$ were higher after 45 min compared to 15 min. The progressive increase in core body temperature during prolonged exercise increases skin blood flow and venous volume, which reduces central blood volume, ventricular filling pressure and end-diastolic volume (39). If these changes cannot be overcome as exercise intensity is increased during the measurement of $\dot{V}O_{2max}$, then $\dot{V}O_{2max}$ will be reduced. Other studies have reported modest reductions in $\dot{V}O_{2max}$ at similar levels of hyperthermia (2, 35) following exercise-induced preheating.

Our data has important implications for the use of HR to prescribe exercise intensity. HR is widely used as an indicator of exercise intensity, based on the strong relation of HR to relative metabolic intensity reported during graded exercise tests (9, 46). It has been assumed this relationship applied during prolonged exercise, but whether the progressive rise in HR accompanying CV drift was associated with altered relative metabolic intensity had not been investigated. We found that CV drift is accompanied by reduced $\dot{V}O_{2max}$ and increased relative metabolic intensity. These changes were directly related to the increase in HR that accompanied CV drift. On average, we found that a 1 bpm increase in HR corresponded to a two-thirds unit change in $\%\dot{V}O_{2max}$ and a slightly greater than 1 unit change in $\%\dot{V}O_2R$ during both cycling and treadmill walking. This suggests that the increase in HR is a valid indicator of increased relative metabolic intensity during prolonged cycling or treadmill walking.

We conclude that CV drift, as reflected by the rise in HR and decline in SV over time between 15 and 45 min of prolonged treadmill walking and stationary cycling, is associated with

a decrease in $\dot{V}O_{2max.}$ The increase in HR reflects an increase in relative metabolic intensity. These data support the use of HR change as the indicator of change in relative metabolic intensity during prolonged exercise in which CV drift occurs.

Chapter 4

Summary and Conclusion

Prolonged, moderate-intensity, constant-rate exercise leads to a downward drift in central venous pressure, SV, and pulmonary systemic arterial pressures, while during this time a rise in HR maintains nearly constant CO (38). The cause of CV drift is unknown, but two theories have been proposed. One hypothesis is that CV drift is caused by a progressive rise in cutaneous blood flow and venous volume, reducing ventricular filling pressure, thus decreasing end diastolic volume limiting stroke volume (25, 38). An alternate hypothesis is that the increase in heart rate due to the increased body temperature and increased sympathetic nervous activity causing a decline in SV due to decreased diastolic time (8, 16). It was not known whether CV drift caused a reduction in physical work capacity, or $\dot{V}O_{2max}$. Nassis and Geladas (32) found that CV drift was different in cycling and treadmill running, but whether this was associated with proportional changes in $\dot{V}O_{2max}$ was unknown. Therefore, the purpose of the study was to compare the magnitude of CV drift and its association with $\dot{V}O_{2max}$ during prolonged cycling and walking.

Nine moderately trained subjects performed four experimental trials, two on the cycle and two on the treadmill. Each subject served as their own control and performed a maximal graded exercise test on the cycle and on the treadmill before beginning the experimental trials for comparison. During the experimental trials, subjects exercised at 60% $\dot{V}O_{2max}$ specific to exercise mode for 15 and 45 min. Upon completion of submaximal exercise subjects immediately performed a $\dot{V}O_{2max}$ test. During the 45 min experimental trials, cardiorespiratory

and metabolic measures were taken to measure CV drift. $\dot{V}O_{2max}$ also was measured after 15 min of submaximal exercise so that the change in $\dot{V}O_{2max}$ between 15 and 45 min could be attributed to CV drift.

The data from this study indicate that, contrary to earlier findings, there was no significant difference in CV drift between cycling and walking. HR increased (~5%) and SV declined (~10%) during cycling and walking between min 15 and min 45. These changes were associated with a 5% reduction in $\dot{V}O_{2max}$ (2.70±0.40 vs 2.52±0.57 l/min, $p<0.05$).

There was a significant relationship between ΔHR and $\Delta\%\dot{V}O_{2max}$ ($r=0.47$, SEE=0.32 l/min, $p<0.05$) and between Δ%HRR and $\Delta\%\dot{V}O_2R$ ($r=0.54$, SEE=10.1 ml/kg/min, $p<0.05$). Submaximal $\dot{V}O_2$ was unchanged over time ($p<0.05$), but $\%\dot{V}O_{2max}$ increased 5% and $\%\dot{V}O_2R$ increased 6%. The progressive rise in HR between 15 and 45 min of submaximal exercise reflected the rise in relative metabolic intensity during both walking and cycling. For every 1 bpm increase in HR there is approximately a two-thirds unit increase in $\%\dot{V}O_{2max}$ and for every 1 point increase in %HRR there was a 1.1 unit increase in $\%\dot{V}O_2R$ during both walking and cycling.

The cause of the reduced $\dot{V}O_{2max}$ after 45 min of prolonged exercise compared to 15 min is uncertain. It is likely it is due in part to lower SV, as reflected by O_2 pulse. Reduced SV may be caused by hyperthermia, increasing cutaneous vasodilation and increased venous volume, reducing central blood volume, ventricular filling pressure and end-diastolic volume (39). T_{re} and $\overline{T}_b$ at $\dot{V}O_{2max}$ was higher after 45 min compared to 15 min, and was not significantly different between exercise modes.

In conclusion, there are no significant differences in the magnitude of CV drift in cycling and walking at 60% $\dot{V}O_{2max}$ in a thermoneutral environment. CV drift during prolonged cycling and walking is associated with a reduction in $\dot{V}O_{2max}$ and an increase in the relative metabolic intensity. The results support the validity of using changes in HR as a guide to changes in relative metabolic intensity during prolonged cycling and walking in a thermoneutral environment during which CV drift occurs.

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