

EFFECT OF CARDIOVASCULAR DRIFT ON MAXIMAL OXYGEN UPTAKE AT TWO
AMBIENT TEMPERATURES

by

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ABSTRACT

This investigation determined whether the progressive rise in heart rate (HR) and fall in stroke volume (SV) during prolonged, moderate-intensity exercise (cardiovascular drift, CV drift) in hot and cool environments is associated with a reduction in $\dot{V}O_{2max}$. CV drift was measured in hot (H, 35°C) and cool (C, 22°C) ambient temperatures in ten male endurance athletes (age = 23 ± 3 yr, $\dot{V}O_{2max} = 64.7 \pm 8.7$ ml/kg/min) between 15 and 45 min of cycling at a power output that elicited $59.2 \pm 1.9\%$ $\dot{V}O_{2max}$, followed immediately by measurement of $\dot{V}O_{2max}$. $\dot{V}O_{2max}$ in both conditions also was measured on separate days after 15 min at the same power output used in the 45 min trials. All four experimental trials included fluid replacement that prevented any significant change in body weight ($0.3 \pm 0.4\%$). CV drift and the associated decrease in $\dot{V}O_{2max}$ was greater ($p < 0.05$) in H than C. Between 15 and 45 min of cycling in H, an increase in HR of 16 ± 4 bpm (11%, $p < 0.05$) and decrease in SV of 12 ± 3 ml (11%, $p < 0.05$) was associated with a 15% decrease in $\dot{V}O_{2max}$ (4.7 ± 0.6 vs 4.0 ± 0.5 l/min, $p < 0.05$). Between 15 and 45 min of cycling in C, HR increased only slightly (3 ± 2 bpm, 2%, $p < 0.05$), SV decreased only slightly (2 ± 1 , 2%, $p < 0.05$) and there was no significant decrease in $\dot{V}O_{2max}$ (4.7 ± 0.6 vs 4.5 ± 0.5 l/min, 5%, $p > 0.05$). Nevertheless, there was a significant relationship between Δ HR and $\Delta\dot{V}O_{2max}$ in H ($r=0.94$, $SEE=0.15$ l/min, $p < 0.05$) and C ($r=0.94$, $SEE=0.09$ l/min, $p < 0.05$). Submaximal $\dot{V}O_2$ was unchanged over time ($p > 0.05$), but $\% \dot{V}O_{2max}$ increased from $57 \pm 4\%$ at 15

min to $67 \pm 8\%$ at 45 min in H ($p < 0.05$), and from $54 \pm 3\%$ at 15 min to $57 \pm 3\%$ at 45 min in C ($p < 0.05$). It is concluded that CV drift during prolonged exercise (1) is greater in a hot than in a cool environment, (2) is associated with a decrease in $\dot{V}O_{2\max}$ in the heat and (3) reflects an increase in relative metabolic intensity.

INDEX WORDS: circulation, cycling, heart rate, oxygen consumption, stroke volume, heat stress

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CHAPTER I

INTRODUCTION

After about 10 minutes of prolonged, moderate-intensity, constant-rate exercise (50-75% $\dot{V}O_{2max}$), cardiovascular drift (CV drift) occurs (10; 39). CV drift is characterized by progressive decreases over time in stroke volume (SV) and pulmonary and systemic mean arterial pressures, and a progressive increase in heart rate (HR) (42). The cause of the reduced SV is multifactorial, with increased core body temperature, dehydration and reduced blood volume, and increased HR, skin blood flow and cutaneous venous volume all being possible contributing factors (21). Whether the reduction in SV during prolonged submaximal exercise reflects reduced cardiovascular capacity and physical work capacity is unknown. We found that subjects cycling at 60% $\dot{V}O_{2max}$ in the heat (35°C) showed a reduction of stroke volume of 16% between 15 and 45 minutes that was associated with a 19% decrease in $\dot{V}O_{2max}$ (unpublished data). The results suggest that CV drift may reflect a reduction in cardiovascular capacity and $\dot{V}O_{2max}$ in a warm environment. Whether there is a similar association between CV drift and $\dot{V}O_{2max}$ in cool ambient temperatures is unknown.

CV drift occurs in comfortable (10; 13; 24; 35; 45) and hot (19; 23; 32; 42) environments. Previous studies have reported SV decreases of 7-13% in thermoneutral (23-27°C) conditions during moderately-prolonged cycling (7; 10; 17; 35; 45), whereas SV decreases of 15-27% were observed in subjects who performed prolonged exercise in warm or hot ambient temperatures (33-43°C) without maintaining fluid loss (19; 23; 32; 39). Only one study has compared CV drift in cool and hot ambient conditions in the same subjects. Gliner et al. (14) found CV drift was greater at 35°C than at 25°C. Although these studies suggest

cardiovascular drift may be greater in the heat, more research is needed on the effect of different ambient temperatures on the magnitude of CV drift and its consequences.

Specific Aims

The specific aims of the study were to determine if ambient temperature (1) affects the magnitude of cardiovascular drift and reduction in $\dot{V}O_{2\max}$ over time during prolonged exercise, and (2) alters the relation of cardiovascular drift to $\dot{V}O_{2\max}$. The results will provide insight into whether changes in heart rate during prolonged exercise can be used as a marker of changes in relative metabolic intensity under different ambient temperatures.

Hypotheses

The research hypotheses for the study are:

1. The magnitude of cardiovascular drift and reduction of $\dot{V}O_{2\max}$ are greater at 35°C than at 22°C.
2. The relation of cardiovascular drift to the reduction in $\dot{V}O_{2\max}$ is the same at 35°C and 22°C.

Significance

Whether or not cardiovascular drift is associated with reduced $\dot{V}O_{2\max}$ has implications for prescription of exercise intensity using HR. Although the relation of heart rate to $\% \dot{V}O_{2\max}$ has been established during brief bouts of exercise of progressively increasing intensity (1; 1; 12; 29), it is not known whether the relation holds during prolonged exercise when heart rate increases over time. Cardiovascular changes that occur during short-term exercise in the heat dissociate the normal HR to $\% \dot{V}O_{2\max}$ relationship used for exercise prescription (1). A similar dissociation could occur with CV drift during prolonged exercise in comfortable and hot conditions. If CV drift is not associated with altered submaximal $\dot{V}O_2$ or altered $\dot{V}O_{2\max}$, then an increase in heart rate over time would not reflect a change in relative metabolic intensity

(% $\dot{V}O_{2max}$). This would suggest that HR should not be used as a proxy of relative metabolic intensity during prolonged exercise. On the other hand, if CV drift is associated with a reduced $\dot{V}O_{2max}$, changes in HR over time during prolonged exercise would be an important marker of changes in $\dot{V}O_{2max}$ and work capacity. If CV drift occurs independently of a change in submaximal $\dot{V}O_2$, then HR would be an indicator of the changes in relative exercise intensity (% $\dot{V}O_{2max}$). Knowledge of the relative magnitude of CV drift and any associated effect on $\dot{V}O_{2max}$ during prolonged exercise at different ambient temperatures is particularly important because heart rate is used as a guide for adjusting exercise intensity during exercise in the heat when work capacity is reduced (25).

CHAPTER II

REVIEW OF THE RELATED LITERATURE

Cardiovascular Drift

Cardiovascular drift (CV drift) is described as occurring when “CV responses begin a continuous time-dependent change, or drift, after ~10 minutes of prolonged moderate-intensity exercise (e.g., 50-75% $\dot{V}O_{2max}$) in a neutral or warm environment” (10). CV drift is characterized by a progressive decrease in stroke volume (SV) and pulmonary and systemic mean arterial pressures (MAP), and an increase in heart rate (HR), while cardiac output (CO) shows little or no change (10). The magnitude of CV drift is affected by several variables, including: dehydration(19), thermal stress(14), mode of exercise (34) exercise intensity (48) and duration (45). While the magnitude of CV drift increases as environmental conditions place greater thermal stress on the individual (14), many studies report significant CV drift in cool or thermoneutral conditions (11; 13; 34; 45).

One of the first studies of changes in hemodynamic variables during prolonged exercise in a thermoneutral environment was done by Cobb et al. (5) in 1963. A group of 14 sedentary and 7 physically active males walked on a treadmill for 30-60 min at an intensity that elicited a $\dot{V}O_2$ of about 1.1 l/min. Cardiac output (CO) was measured by dye-dilution at the beginning (4 to 6 min) and end of exercise. HR increased progressively, while SV decreased 14-16% in both groups from 4 to 6 min until the end of exercise. They concluded that during prolonged exercise in a thermoneutral environment, CO remains constant while SV declines over time, with a corresponding increase in HR to maintain CO.

Ekelund et al. (11) found similar decreases in SV (14%) and systemic arterial blood pressure, and an increase in HR (11%) between 10 and 60 min in subjects who cycled at

70% $\dot{V}O_{2\max}$ in comfortable ambient conditions (23°C). They suggested that the decline in SV might be due to a decrease in central blood volume caused by either gravitational shifts due to a change in tone of the capacitance vessels, a decrease in the total blood volume due to increased filtration pressure in the capillaries, or shifts due to vasodilation caused by a heat-regulating mechanism (11). Further, they proposed that the decrease in SV may be related to the increase or decrease in mechanical systole across time, or displacement of the blood volume peripherally in response to increased body temperature, lower effective filling of the heart caused by a decrease in the vasomotor tone, or a change in the mechanical properties of the myocardium (10).

The displacement of blood to the peripheral circulation as a cause of the decline in SV was investigated further by Saltin and Stenberg (45). They reported a decrease in SV (15%) and an increase in HR (10%) in four subjects who cycled and ran at 75% $\dot{V}O_{2\max}$ for 195 min at 24°C (45). They hypothesized that a reduction in total peripheral resistance (TPR) and a decrease in blood volume was responsible for the impaired cardiovascular function (45). SV did not decline in the same conditions when subjects exercised in the supine position when preload was unaffected, suggesting that the cause may be a reduction in intrinsic pump function (45). There have been no subsequent studies supporting the hypothesis that any mechanical functions of the myocardium are reduced during prolonged exercise. Therefore, the decline in SV appears to be simply related to the Frank-Starling relationship, or a fall in filling pressure (39).

A review by Ekelund (10) summarized the relationship between change in SV and change in HR during prolonged exercise in three previous studies. A significant correlation was found between decrease in stroke volume between 10 min and end of exercise and the corresponding change in heart rate: $\Delta SV_{10\text{-end}}(\text{ml}) = 1.66 + 0.663 \cdot \Delta HR_{10\text{-end}}$, $r=0.60$, $SEE = 7.98$ (10).

Johnson and Rowell (27) studied the cutaneous and muscle vascular responses to prolonged exercise. HR, esophageal temperature, total forearm blood flow (FBF) using plethysmography, and total forearm muscle blood flow using [^{125}I] antipyrine clearance were measured in five subjects during 60 min of cycling. FBF progressively increased between 10 and 40 min, while muscle blood flow decreased at the onset of exercise and either decreased further or remained constant during the rest of exercise. They concluded that the progressive decreases in central venous pressure, SV, and arterial pressure seen in previous studies were due to progressive increases in cutaneous blood flow and volume.

Shaffrath et al. (48) manipulated the magnitude of CV drift and change in skin blood flow by having subjects cycle for 70 min at two power outputs (43 and 62% $\dot{V}\text{O}_{2\text{max}}$) and two conditions which either provided significant airflow over the skin or no airflow. No changes occurred in HR, FBF, SV and MAP at the lower power output in both airflow conditions, while at the higher work load, HR and FBF increased (16% and 105% respectively), and SV and MAP decreased (12% and 11% respectively) in the no airflow condition, but did not change significantly in the higher airflow condition (48). The results appear to support the hypothesis that CV drift is caused by an increase in cutaneous blood flow. CV drift was present in this study only when both high work load and thermal stress were present. When evaporative cooling of the skin is increased, changes in cutaneous blood flow and venous volume were decreased, reducing or preventing the occurrence of CV drift. The influence of skin temperature on skin blood flow and cutaneous venous tone has been shown previously (26).

In his book (39), Rowell expands on his theory that increased blood flow causes CV drift. He argues that increased cutaneous blood flow leads to an increase in cutaneous venous volume, which reduces ventricular filling pressure, end-diastolic volume, and stroke volume during moderate-intensity exercise. Upon beginning exercise, the cutaneous vascular response is

constriction, gradually followed by the withdrawal of vasoconstriction and initiation of active vasodilation in response to a rise in core body temperature. The continuous rise in cutaneous blood flow may cause the progressive decline in ventricular preload and afterload seen in prolonged exercise. Because muscle blood flow remains constant, and blood flow to splanchnic and the kidneys declines as heart rate increases, the increase in skin blood flow must account for the decrease in mean arterial pressure. The rise in cutaneous blood flow to compliant cutaneous veins decreases central blood volume which may lead to an increase in heart rate in order to maintain cardiac output.

Skin temperature and associated venous pooling has been strongly related to the degree of decline in SV during exercise (43). For example, Rowell et al. (43), had subjects cycle at 64% $\dot{V}O_{2max}$ at 24°C and changed skin temperature every 30 min. When skin temperature was raised to 38.7°C, a decrease in SV (14%) occurred along with an increase in cardiac output (19%), but upon rapidly cooling the skin, SV was restored to control levels (43). The results suggest that the decrease in SV is due to a decline in ventricular preload caused by an increase in cutaneous blood flow.

More recent studies also have shown reductions in SV and increases in HR at comfortable ambient temperatures, but have challenged the traditional theory of cutaneous blood flow causing the decline in SV. Gonzalez-Alonso et al. (21) had subjects cycle for 30 min in hot and cold conditions while euhydrated or dehydrated. In order to study the effects of skin blood flow on the magnitude of CV drift, they had subjects exercise in the heat and cold keeping exercise intensity, dehydration, reduction in blood volume, and increases in serum electrolytes and osmolality similar in both conditions. When subjects were euhydrated, SV and core temperature were similar in the hot and cold conditions, while skin blood flow was much greater in hot versus cold. In addition, with each 1% body weight loss, SV declined 4.8% and 2.5% in

hot and cold, respectively. The reduced SV was independent of skin blood flow, but showed high correlations with increased HR and reduced blood volume. They concluded that it could not be increased cutaneous blood flow that caused the CV drift. Rather, they concluded that CV drift was related to a change in core temperature. As a result of this and other studies done around this time, an alternative mechanism for CV drift was proposed.

The alternate explanation for CV drift developed by Coyle and Gonzalez-Alonso (8) proposes the decline in stroke volume during prolonged exercise is due primarily to increased HR, which decreases ventricular filling time and end-diastolic volume (8). This explanation is supported in a study by Fritzsche et al. (13) in which subjects ingested a small dose of β -blocker prior to exercise and performed prolonged exercise that would elicit classical CV drift. The β -blocker prevented the increase in HR and decline in SV after 15 minutes. This was despite the presence of normal cutaneous blood flow. All variables that could be related to the decline in SV, such as blood volume, cutaneous blood flow, esophageal temperature, and skin temperature were similar in the control and experimental trials. They speculated that the rise in HR may be due to the increase in core temperature and accompanying sympathetic nervous system activity.

The greatest reduction in SV and increase in HR have been in studies which took place in the heat. Several early studies measured the circulatory responses to exercise in the heat. Asmussen (2) reported one of the first studies showing CV drift in high ambient temperatures when subjects cycled at 50% $\dot{V}O_{2max}$ in a neutral environment for 2 h and until exhaustion in a hot environment. Subjects showed a decline in SV (20%) during the last half of exercise in the hot environment, while SV did not decline in the neutral environment.

Cardiovascular responses to prolonged (210 min), low-intensity (35% $\dot{V}O_{2max}$) exercise at two different temperatures (25°C and 35°C) were studied by Gliner et al. (14). Subjects walked on a treadmill during 4 sessions of 50 min at 35% $\dot{V}O_{2max}$ separated by 10 min of rest. Expressed

as % difference from 1-h average of mean values, there was a much greater decline in SV (18% vs. 10%) and increase in HR (24% vs 12%) in hot compared to comfortable conditions. They attributed the greater increases in HR in 35°C to the higher mean body temperature, which reflects higher mean skin temperatures in the heat.

Gonzales-Alonso et al. (20) studied whether CV drift in a hot environment associated with dehydration and hyperthermia could be attenuated or prevented by elevating central blood volume (CBV) during supine exercise. Subjects cycled for 30 min in the upright and supine position at 35°C. At the same level of dehydration and hyperthermia, supine exercise did not cause the reduction in MAP or cutaneous vascular conductance that is observed in upright exercise. In addition, supine exercise had 60% less decline in SV and 30% less increase in HR compared to the upright exercise. They associated the decline in cutaneous vascular conductance and increased plasma catecholamine levels with dehydration during upright cycling with a reduction in central blood volume and lower arterial blood pressure. They attributed the smaller decline in SV and increase in HR during supine exercise to a higher left ventricular end-diastolic volume, suggesting the importance of central blood volume on baroreflex control of skin circulation and the sympathetic nervous system.

Another study by Gonzalez-Alonso et al. (22) showed a strong correlation between increases in HR and core temperature, especially at core temperatures above 38°C when cutaneous blood flow reaches a steady state. In addition, when core temperature was not elevated, a decline in SV did not occur. Fritzsche et al. (13) showed a similar correlation between an increase in core temperature and HR ($R^2 = 0.95$).

Hamilton et al. (23) were among the first to study the effects of fluid replacement on CV drift. Subjects cycled for 2 h at 70% $\dot{V}O_{2max}$ in 22°C during three trials, one without replacing fluid, one that replaced body weight loss with water, and one with continuous intravenous

infusion of glucose in water. The no-fluid-replacement trial resulted in a decline in SV of 15% and an increase in HR of 10%, while the fluid replacement trial resulted in no change in SV and an increase in HR of 5% (23). Glucose infusion completely prevented the increase in HR that occurred in the other two trials. They concluded that fluid replacement throughout exercise attenuates hyperthermia and prevents the decline in SV and CO in prolonged moderate exercise. In addition, the results of this study suggest that hypoglycemia is responsible for a small part of the increase in HR and any CV drift associated with it may be prevented by glucose and water infusion.

Montain et al. (32) were the first to investigate the relationship between levels of dehydration and CV drift during exercise. Subjects cycled for 2 hours in a warm (33°C) environment at 65% $\dot{V}O_{2\max}$ and received either no fluid or small, moderate, or large volumes of water that replaced 20, 48, and 81% of fluid lost in sweat during exercise (32). The magnitude of dehydration after 2 hours was linearly related to the increase in core body temperature, the increase in HR, and the decline in SV. They attributed the prevention in hyperthermia in large fluid replacement to higher skin blood flow compared to the other two trials.

Dehydration of just 3-5% body weight during exercise has been shown to cause a reduction in blood volume that results in dehydration-induced hyperthermia (32). Blood volume expansion studies have shown that dehydration alone accounts for about one-half of the reduction in SV during hyperthermia (32). However, the SV reductions resulting from the effects of dehydration and hyperthermia (about 7-8% each) do not account for the extreme SV reductions of 20-28% from the combination of dehydration-induced hypovolemia and hyperthermia (19). The cardiovascular instability associated with the large declines in SV is caused by a synergistic effect of dehydration and hyperthermia, on CO (15). Coyle and Gonzalez-Alonso (8) proposed that the decreased filling pressure from dehydration-induced

hypovolemia in addition to a shortened diastolic filling time from increased core temperature and heart rate cause a disproportionately large decrease in end-diastolic volume, and therefore, SV.

When hyperthermia is not present in dehydrated subjects during exercise in a cold environment, the reduction in SV (7%) appears to be due to reduced blood volume, because SV is restored when blood volume is restored to euhydrated levels, even with the presence of extravascular dehydration (17). In dehydrated, hyperthermic subjects with restored blood volume, stroke volume was only partially restored, even with reduced skin blood flow and declining skin temperature (32). Gonzalez-Alonso et al. (19) found similar results when they reported that hyperthermia alone reduces stroke volume, although total blood volume and cutaneous blood flow was the same as in the control. These findings indicate that hyperthermia decreases stroke volume in both euhydrated and dehydrated subjects in a way that is independent of an increase in skin temperature and skin blood flow, and reduced blood volume (19).

In a different study, Gonzalez-Alonso et al. (21) investigated the interaction of environment and hydration. Endurance-trained male cyclists cycled in the heat (35°C) or the cold (8°C) for 30 minutes when euhydrated or dehydrated by 1.5, 3.0, or 4.2% of their body weight. They found that when euhydrated, the subjects had the same SV and core temperature in the heat and cold environments, even though skin blood flow was significantly higher in H than in C ($365 \pm 64\%$ higher) (21). In addition, with each 1% loss in body weight, SV declined 4.8% in the heat and 2.5% in the cold (21). Their findings indicate that SV can be maintained in subjects that are trained and euhydrated, even with a large increase in skin blood flow. This suggests that the decline in SV with dehydration is largely related to the increase in HR and reduction in blood volume.

The magnitude of CV drift reported by previous studies seems to be dependent on many factors related to exercise intensity, environmental stress, mode of exercise and training state of

the subjects. The magnitude of CV drift has been shown in at least two studies to be related to the relative exercise intensity. Ekelund et al. (10; 11) showed that untrained subjects cycling for one hour in the upright position at 50% $\dot{V}O_{2max}$ had a SV decline of 7%, while at 75% $\dot{V}O_{2max}$, SV declined 17% between 10 and 60 min. Nadel et al. (33) reported similar results with subjects cycling at 40% and 70% $\dot{V}O_{2max}$ at different ambient temperatures for 20 min. For all ambient temperatures, SV declined progressively at 70% $\dot{V}O_{2max}$ but not at 40% $\dot{V}O_{2max}$.

CV drift is affected by mode of exercise. Nassis et al. (35) compared cardiovascular responses to prolonged submaximal exercise while running and cycling. Subjects either cycled or ran at 60% $\dot{V}O_{2max}$ for 90 minutes in thermoneutral conditions (24°C). They found a greater decline in stroke volume and cardiac output in cycling than in running, even with greater dehydration and hyperthermia occurring during cycling. A subsequent study by Nassis et al. (35) tested the effect of water ingestion on cardiovascular responses in the two modes of exercise. Water ingestion attenuated the decline in CO and SV during both cycling and running. However, the mechanism was different in the two modes of exercise. Water ingestion during cycling resulted in the maintenance of blood volume, while not affecting core temperature (35). Water ingestion during running resulted in preventing hyperthermia and did not affect dehydration to the same degree as in cycling.

Endurance-training has been suggested to attenuate or prevent CV drift in some conditions, but the results of studies are not consistent. Saltin et al. (44) tested sedentary subjects before and after eight weeks of training and reported smaller changes in SV and HR after training. The attenuation or prevention of CV drift in that study could be due not only to endurance training, but also improved heat-acclimation, and better familiarization with the testing device.

Eklblom (9) measured cardiovascular changes and $\dot{V}O_{2\max}$ immediately after 1 h of cycling at 75% $\dot{V}O_{2\max}$ under thermoneutral conditions before and after aerobic training in 7 subjects. 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 was unchanged from that at 60 min, suggesting that changes in SV persisted during maximal exercise. $\dot{V}O_{2\max}$ was reduced 6% following the 60 min of submaximal exercise prior to training and 12% after training.

Effect of Temperature on CV Responses to Exercise in the Heat

Cardiovascular variables have been shown to change when heat stress is combined with metabolic stress. Elevated ambient temperatures results in dilation of skin blood vessels caused by increased skin and core temperatures, shifting a greater proportion of the blood volume to the skin. A shift in blood volume to the periphery causes a reduction in the central blood volume and stroke volume, and increased heart rate during submaximal exercise (42; 50). Moderate-intensity exercise in the heat also causes MAP to decrease, while CO remains unchanged (10). Moderate-to-heavy exercise in the heat causes both renal blood flow and splanchnic blood flow to be reduced compared to cool environments. The vasoconstriction increases progressively over time during prolonged exercise and increased hepatic glucose release, hepatic lactate production and a fall in hepatic venous O₂ content is seen during prolonged exercise in the heat when core temperature reaches high levels (40°C) (40). $\dot{V}O_2$, CO and (a-v)O₂ difference generally remain unchanged at submaximal work levels in the heat compared to cool environments (50). Blood lactate levels have been shown to be increased at submaximal work rates in the heat (50) due to reduced extraction of lactate from the blood, probably caused by less blood flow to the liver (36). Increased skin and core temperature causes increased cutaneous blood flow in the heat, while muscle blood flow remains unchanged (27). Increased core temperature (hyperthermia)

accentuates the magnitude of CV drift, with the greatest reductions in SV associated with the greatest increases in HR. The magnitude of CV drift, and increase in HR, is directly related to the degree of thermal strain placed on the individual (18).

Heart rate in a thermoneutral environment increases linearly as a function of exercise intensity and is related to the percentage of maximal oxygen uptake ($\% \dot{V}O_{2\max}$) elicited (4). During exercise in the heat, blood temperature directly affects the sinoatrial node and autonomic nervous system control of HR (28). This results in an increase in HR at rest and at submaximal exercise intensities. Kamon (29) showed a shift in the regression line of HR on $\dot{V}O_2$, with a given $\dot{V}O_2$ associated with an increased HR of 10 bpm with an increase in ambient temperature of 10°C. If $\dot{V}O_2$ during submaximal exercise is unchanged, but $\dot{V}O_{2\max}$ is reduced in the heat, then submaximal $\dot{V}O_2$ would represent a higher $\% \dot{V}O_{2\max}$, and the HR- $\% \dot{V}O_{2\max}$ relation would be dissociated less than if $\dot{V}O_{2\max}$ is assumed to be unchanged in the heat (1). The elevation in HR during submaximal exercise performed in the heat has been shown to be related to the increase in $\% \dot{V}O_{2\text{peak}}$ elicited in the heat. Arngrimsson et al. (1) showed that during sustained submaximal exercise in high ambient temperatures, the dissociation of HR from $\% \dot{V}O_{2\text{peak}}$ is less than that predicted by assuming $\dot{V}O_{2\max}$ is unchanged in the heat.

Effects of Prior exercise on $\dot{V}O_{2\max}$

Previous studies have investigated the effects of severe prior exercise on $\dot{V}O_{2\max}$. Astrand et al. (3) observed an 11% decrease in $\dot{V}O_{2\max}$ in 5 cross-country skiers 90 min following an 85 km race. Maximal heart rates were unchanged, and maximal blood lactate levels were 78% lower compared to control $\dot{V}O_{2\max}$ tests with the same subjects.

Stamford et al. (49) showed that attainment of a valid and reliable $\dot{V}O_{2\max}$ is possible even with a subject in different stages of exhaustion. Subjects were given a $\dot{V}O_{2\max}$ test after participating in trials consisting of three maximal tests in each trial with a varying amount of

rest: 10, 20, 30 or 40 min. Despite a reduction in performance time, $\dot{V}O_{2\max}$ was not significantly different between the trials (49).

Saltin and Stenberg (45) obtained central circulatory measures on three subjects during 195 min of cycling and running at 75% $\dot{V}O_{2\max}$, and following 90 min of rest during an additional 15 min bout of submaximal exercise proceeding directly into a $\dot{V}O_{2\max}$ test. HR increased 20% and SV decreased 8% from min 15 to min 195 of submaximal exercise. Cardiorespiratory values measured at the end of the 15 min of submaximal exercise remained the same as at the end of the 195 min of submaximal exercise. $\dot{V}O_{2\max}$ was decreased 5% compared to the control. The results suggest the reduction in SV observed following prolonged exercise persisted during maximal exercise and was related to slightly reduced $\dot{V}O_{2\max}$. While 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.

Costill et al. (6) measured circulatory and metabolic responses during a 16.1 km run at 80% $\dot{V}O_{2\max}$ and during a maximal exercise protocol following 45 min of rest after the 16.1 km run. $\dot{V}O_{2\max}$ decreased 8% after the 16.1 km run, while maximal blood lactate levels decreased 51%. Muscle biopsies obtained from the vastus lateralis, and gastrocnemius before and after the 16.1 km run showed substantial quantities of muscle glycogen after the prolonged and maximal test, suggesting that glycogen depletion most likely does not contribute to the decrease in $\dot{V}O_{2\max}$ after prolonged exercise. They hypothesized that glycolysis may be inhibited during prolonged exercise, causing a reduction in anaerobic power and maximal blood lactate levels. The decreases in aerobic capacity were attributed to the individual's inability to reach high work outputs due to decreases in anaerobic power.

Sawka et al. (46) compared metabolic and cardiopulmonary responses during submaximal and maximal exercise before and after prolonged running. Seven endurance-trained

subjects performed a graded $\dot{V}O_{2\max}$ test several days prior to, and 45 min following, 21 miles of high-intensity treadmill running. Submaximal $\dot{V}O_2$, O_2 pulse, and V_E were similar during the pre and post-test. Submaximal HR was higher and RER was lower during the post-test. Maximal work time (12%), $\dot{V}O_{2\max}$ (6%), and maximal blood lactate concentration (47%) were all lower during the post-test. The authors concluded that elevated HR responses during subsequent submaximal exercise are due to increased thermal stress, and aerobic power and anaerobic capacity are reduced following prolonged running, causing a reduction in total work time during subsequent exercise (46).

Maximal Oxygen Uptake in the Heat

Physical work capacity (performances) has been shown to consistently decrease in the heat (31), but research into whether $\dot{V}O_{2\max}$ decreases in the heat is conflicting. Previous studies have shown no reduction (42), modest (10%) reduction (47), or quite large (25%) reduction (38). The differences appear to be due to the differences in the magnitude of increases in body core and skin temperatures. Many of the studies have used only relatively brief exposure to the heat at rest prior to measuring $\dot{V}O_{2\max}$, which does not produce an increase in core body temperature (30). The studies of no or minimal change in $\dot{V}O_{2\max}$ in the heat suggest that for short periods of maximal exercise, vasoconstriction of blood vessels in the skin and gut can shunt blood to active skeletal muscles and maintain muscle blood flow and oxygen delivery, increasing stroke volume to levels of those found in thermoneutral conditions. It has been suggested that during prolonged work in the heat, vasodilation of skin blood vessels dominates and $\dot{V}O_{2\max}$ is reduced (41). Studies that have shown large reductions in $\dot{V}O_{2\max}$ in the heat usually have some combination of core and skin temperature that may prevent vasoconstriction of skin blood vessels during high intensity exercise in the heat.

One of the first studies measuring metabolic variables in the heat was by Williams et al. (50) in 1962. Three physically active male subjects cycled at five different power outputs and performed a $\dot{V}O_{2\max}$ test in comfortable (21°C) and hot (36°C) conditions. $\dot{V}O_2$ was lower in the hot compared to comfortable conditions at all five submaximal work rates. An increase in HR and decrease in SV was present across all submaximal work rates in hot but not in comfortable conditions. CO and (a-v) O_2 difference were not different during submaximal work rates in either condition. $\dot{V}O_{2\max}$ was not significantly different between the two conditions. In addition, CO and (a-v) O_2 difference were not significantly different between the two conditions. They concluded that a portion of CO is diverted to vasodilated blood vessels in skin at submaximal levels of oxygen uptake in the heat, resulting in anaerobic metabolism in working muscles and higher blood lactate concentrations.

Pirnay et al. (38) reported a 25% decrease in $\dot{V}O_{2\max}$ in subjects after exercise at a temperature of 46°C. $\dot{V}O_{2\max}$ was measured following 20 min of low-intensity exercise that elevated core temperature (preheating). Without preheating, a 7% reduction in $\dot{V}O_{2\max}$ was shown in another set of subjects at the same temperature.

Nybo et al. (37) reported a 16% decrease in $\dot{V}O_{2\max}$ following preheating that elevated core temperature to 39°C and skin temperature to 37°C. Performance time during a constant-load test was reduced by one-half. The results suggest that $\dot{V}O_{2\max}$ is markedly compromised when both an increase in core and skin temperature occur.

Arngrimsson et al. (1) showed that $\dot{V}O_{2\max}$ was significantly reduced in men and women under conditions that elevated both core and skin temperatures to very high levels. They found that $\dot{V}O_{2\max}$ was reduced in the heat in men and women in direct proportion to the increase in mean body temperature, which directly related to core and skin temperatures. It would be expected that during exercise in high ambient temperature, skin blood flow would be increased,

and stroke volume would be reduced at maximal exercise. If maximal heart rate and arteriovenous oxygen difference (a-v) O_2 difference are unchanged, then $\dot{V}O_{2max}$ and cardiac output should be reduced (39). Angrimsson's study showed that while $\dot{V}O_{2max}$ was reduced, maximal heart rate was not lower, and was actually higher in most subjects. The decrease in $\dot{V}O_{2max}$ was either due to a lower (a-v) O_2 difference or stroke volume. Their results included a strong relationship between reduction in $\dot{V}O_{2max}$ and a lower oxygen pulse.

Lower maximal cardiac output, as seen in CV drift with prolonged moderately-intense exercise in the heat, may cause lower maximal skeletal muscle blood flow and reduced oxygen delivery to the active muscles, which would limit $\dot{V}O_{2max}$ (1). Additionally, any reduction in muscle perfusion at maximal exercise would reduce $\dot{V}O_{2max}$, if both blood flow to muscle and its oxygen extraction are maximal (39).

Gonzalez-Alonso et al. (16) investigated the primary limitations of $\dot{V}O_{2max}$ with and without heat stress by having eight subjects perform intense upright cycling exercise until exhaustion with either high or normal skin and core temperatures (+10°C and +1°C). When both skin and core temperature was elevated, $\dot{V}O_{2max}$ decreased 8% and time to fatigue decreased 28%, despite HR and core temperature levels similar to the no heat stress trial. In both heat stress and normal conditions, CO, MAP, and leg blood flow measured before exhaustion declined significantly. They concluded that the reduced $\dot{V}O_{2max}$ present during heat stress was due to the greater decline in CO and MAP, leading to decreases in skeletal muscle blood flow. The inability to obtain control levels of $\dot{V}O_{2max}$ and work capacity may be directly related to the inability of the heart to maintain CO and O_2 delivery to skeletal muscle (16). During very short periods (3-5min), $\dot{V}O_{2max}$ is not lower in hot conditions when compared to cool conditions (41). This shows the cardiovascular system's ability to sacrifice cutaneous blood flow to maintain muscle oxygen supply. There appears to be a critical level of core temperature, or a combination

of core and skin temperatures at which the body does not sacrifice cutaneous blood flow and $\dot{V}O_{2\max}$ is reduced in the heat. Rowell et al. (41), studied whether it was rectal or skin temperatures, or both that affected the change in $\dot{V}O_{2\max}$ during exercise. Subjects cycled for 15 min at 85% $\dot{V}O_{2\max}$ under conditions meant to increase only core temperature, only skin temperature, and both core and skin temperature. $\dot{V}O_{2\max}$ declined only when there was an increase in \bar{T}_b , representing skin and core body temperature, and did not change in the other conditions.

CHAPTER III
EFFECT OF CARDIOVASCULAR DRIFT ON MAXIMAL OXYGEN UPTAKE
AT TWO AMBIENT TEMPERATURES¹

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ABSTRACT

This investigation determined whether the progressive rise in heart rate (HR) and fall in stroke volume (SV) during prolonged, moderate-intensity exercise (cardiovascular drift, CV drift) in hot and cool environments is associated with a reduction in $\dot{V}O_{2\max}$. CV drift was measured in hot (H, 35°C) and cool (C, 22°C) ambient temperatures in ten male endurance athletes (age = 23 ± 3 yr, $\dot{V}O_{2\max}$ = 64.7 ± 8.7 ml/kg/min) between 15 and 45 min of cycling at a power output that elicited 59.2 ± 1.9 % $\dot{V}O_{2\max}$, followed immediately by measurement of $\dot{V}O_{2\max}$. $\dot{V}O_{2\max}$ in both conditions also was measured on separate days after 15 min at the same power output used in the 45 min trials. All four experimental trials included fluid replacement that prevented any significant change in body weight (0.3 ± 0.4%). CV drift and the associated decrease in $\dot{V}O_{2\max}$ was greater (p<0.05) in H than C. Between 15 and 45 min of cycling in H, an increase in HR of 16 ± 4 bpm (11%, p<0.05) and decrease in SV of 12 ± 3 ml (11%, p<0.05) was associated with a 15% decrease in $\dot{V}O_{2\max}$ (4.7 ± 0.6 vs 4.0 ± 0.5 l/min, p<0.05). Between 15 and 45 min of cycling in C, HR increased only slightly (3 ± 2 bpm, 2%, p<0.05), SV decreased only slightly (2 ± 1, 2%, p<0.05) and there was no significant decrease in $\dot{V}O_{2\max}$ (4.7 ± 0.6 vs 4.5 ± 0.5 l/min, 5%, p>0.05). Nevertheless, there was a significant relationship between Δ HR and $\Delta\dot{V}O_{2\max}$ in H (r=0.94, SEE=0.15 l/min, p<0.05) and C (r=0.94, SEE=0.09 l/min, p<0.05). Submaximal $\dot{V}O_2$ was unchanged over time (p>0.05), but % $\dot{V}O_{2\max}$ increased from 57 ± 4% at 15 min to 67 ± 8% at 45 min in H (p<0.05), and from 54 ± 3% at 15 min to 57 ± 3% at 45 min in C (p<0.05). It is concluded that CV drift during prolonged exercise (1) is greater in a hot than in a cool environment, (2) is associated with a decrease in $\dot{V}O_{2\max}$ in the heat and (3) reflects an increase in relative metabolic intensity.

INDEX WORDS: circulation, cycling, heart rate, oxygen consumption, stroke volume, heat stress

INTRODUCTION

After about 10 minutes of prolonged moderate-intensity exercise (50-75% $\dot{V}O_{2\max}$), cardiovascular drift (CV drift) occurs (33). CV drift is characterized by progressive decreases in stroke volume (SV), and pulmonary and systemic mean arterial pressures, and a progressive increase in heart rate (HR) (35). The cause of the reduced SV is multifactorial, with increased core body temperature, dehydration and reduced blood volume, increased skin blood flow and cutaneous venous volume, and increased sympathetic nervous system activity and HR all being possible contributing factors (19). CV drift has been shown to occur in comfortable (11; 14; 21; 28; 37) and hot (18; 20; 27; 35) environments. Previous studies have found SV decreases during moderately-prolonged, upright cycling of 7-13% in thermoneutral (23-27°C) conditions (8; 11; 17; 28; 37) and decreases of 15-27% in hot ambient temperatures (33-43°C) without maintaining fluid loss (18; 20; 27; 33). One study has directly compared the magnitude of CV drift in different ambient temperatures. Gliner et al. (15) found greater declines in SV (18% vs. 10%) and increases in HR (24% vs. 12%) in hot (35°C) compared to comfortable (25°C) conditions.

Whether the reduction in SV during prolonged submaximal exercise reflects reduced cardiovascular capacity and physical work capacity is unknown. In an unpublished study, we recently found that a SV decline of 16% between 15 and 45 minutes was associated with a 19% decrease in $\dot{V}O_{2\max}$ in subjects cycling at 60% $\dot{V}O_{2\max}$ in the heat (35°C). The results suggest that CV drift may reflect a reduction in cardiovascular capacity and $\dot{V}O_{2\max}$ in the heat. Whether there is a similar association between CV drift and $\dot{V}O_{2\max}$ in cool ambient temperatures is unknown.

Whether or not cardiovascular drift is associated with reduced $\dot{V}O_{2\max}$ has implications for exercise prescription. Although the relation of HR to % $\dot{V}O_{2\max}$ has been established during brief bouts of exercise of progressively increasing intensity (2; 13; 24), it is not known whether

the relation holds during prolonged exercise when heart rate increases over time. Cardiovascular changes that occur during short-term exercise in the heat dissociate the normal HR to $\% \dot{V}O_{2\max}$ relationship used for exercise prescription (2). A similar dissociation could occur with CV drift during prolonged exercise in comfortable and hot conditions. Knowledge of the consequence of CV drift and any associated affect on $\dot{V}O_{2\max}$ during prolonged exercise at different ambient temperatures is particularly important because HR is used as a guide for adjusting exercise intensity during exercise in the heat when work capacity is reduced (22).

The objective of this study was to determine (1) whether ambient temperature affects the magnitude of CV drift and reduction in $\dot{V}O_{2\max}$ over time during prolonged cycling and (2) whether the relation of CV drift to reduction in $\dot{V}O_{2\max}$ is the same in cool and hot ambient conditions. The objective was accomplished by measuring CV drift between 15 and 45 min of cycling in two different ambient temperatures (22°C and 35°C). In addition, $\dot{V}O_{2\max}$ was measured immediately after 15 and 45 min to determine the relationship between CV drift and reduction in $\dot{V}O_{2\max}$. We hypothesized that the magnitude of CV drift and reduction of $\dot{V}O_{2\max}$ are greater at 35°C than at 22°C, but the relation of CV drift to the reduction in $\dot{V}O_{2\max}$ is the same.

METHODS

Subjects. Ten healthy endurance-trained male subjects volunteered for this study. This sample size is sufficient to detect a 5% decrease in $\dot{V}O_{2\max}$, assuming a mean $\dot{V}O_{2\max}$ of 55 ml/kg/min with SD of 7 ml/kg/min and test-retest correlation for $\dot{V}O_{2\max}$ of 0.95, using a two-tailed t-test for dependent samples at $\alpha = 0.05$ and statistical power of 0.8 (26). The subjects were trained cyclists, runners or triathletes averaging 119 ± 54 km/wk cycling, 50 ± 13 km/wk running, or cycling and running for a combined 6 hours/wk during the previous 6 mo. Selection criteria also included a history of competitive experience in cycling/running and no contraindications to participation in strenuous exercise in the heat as determined by a medical history questionnaire. Subject physical characteristics (mean \pm SD) were: age = 23 ± 3 yr, mass = 68.6 ± 6.2 kg, height = 177.8 ± 7.3 cm, and percent body fat estimated from skinfolds = $7.6 \pm 4.2\%$. The study was approved by the University's Institutional Review Board, and written informed consent was obtained prior to testing.

Research Design

A repeated-measures experimental research design was used in which subjects were tested under all conditions. CV drift and changes in $\dot{V}O_{2\max}$ between 15 and 45 min of cycling were determined under two experimental conditions: (1) with an ambient temperature of 22°C, 40% relative humidity (RH) (C) and (2) with an ambient temperature of 35°C, 40% RH (H). There were two trials completed under each condition. In one trial, subjects cycled at 60% $\dot{V}O_{2\max}$ for 15 min followed immediately by the measurement of $\dot{V}O_{2\max}$. In the other, subjects cycled at 60% $\dot{V}O_{2\max}$ for 45 min with CV drift measured between 15 and 45 min, followed immediately by the measurement of $\dot{V}O_{2\max}$. A control $\dot{V}O_{2\max}$ test, along with two practice rides were performed on separate days prior to beginning the four experimental trials. The order of conditions and trials within each condition was randomized, but the two trials for each condition

were performed in succession. Each subject was tested at the same time of day to minimize the effects of circadian rhythms on HR and core temperature, and trials were separated by one day. All four experimental trials included fluid replacement of equal volumes of tap water at room temperature just prior to beginning exercise and at 10 min, 25 min, and 35 min. The volume of water ingested was designed to prevent dehydration and was based on the sweat rate measured during the two practice rides plus the estimated additional sweat loss that would occur during the $\dot{V}O_{2max}$ test portion of the protocol.

Protocol and Procedures

Control $\dot{V}O_{2max}$. A continuous, load-incremented cycling graded exercise test (GXT) was used to measure $\dot{V}O_{2max}$. Subjects reported to the laboratory following a 3-h fast, and were well hydrated (urine specific gravity ≤ 1.020). They were instructed not to consume alcohol, caffeine, or non-prescription drugs the day before and the day of testing. On the morning of the test, a 24-h history questionnaire was given to determine adherence to pretest instructions. Testing was conducted on an electronically-braked cycle ergometer (Lode Excalibur Sport, Lode B.V., Groningen, NL) in an environmental chamber maintained at 22°C, 40% relative humidity. Subjects warmed up by cycling at a comfortable intensity for about 15 min. After a brief rest, the GXT began with subjects cycling at 200 W. The resistance was increased 25 W every 2 min until the subjects could no longer maintain the power output. $\dot{V}O_2$ and related gas exchange measures were determined by indirect calorimetry over 30-s intervals using a Parvo Medics TrueOne 2400 Metabolic Measurement System (Parvo Medics, Inc., Salt Lake City, UT). Gas analyzers were calibrated using gases of known concentrations, and the flowmeter was calibrated using a 3-l syringe. HR was measured each minute during the test using a Polar Vantage XL heart rate monitor (Polar Electro, Inc., Woodbury, NY, model 145900). RPE was measured the last 10-sec of each stage using the Borg 15-point category scale using standardized instructions

(6). Three minutes post test, a finger-stick blood sample was obtained for determination of blood lactate. Blood lactate was measured using a YSI 2300 Stat Plus Analyzer (Yellow Springs Instruments, Inc., Yellow Springs, OH).

To ensure a plateau in $\dot{V}O_2$ occurred, the subjects performed a follow-up test after resting for 20 min. The subjects cycled until exhaustion at a workload equivalent to the last workload performed during the graded test (if < 1 min was completed during the last stage of the graded test) or at a workload 25 W greater than the last workload achieved in the graded test (if ≥ 1 min was completed during the last stage of the graded test).

Attainment of $\dot{V}O_{2max}$ was determined by using a modification of the plateauing criterion described by Taylor et al. (41). The criterion was an increase in $\dot{V}O_2$ (l/min) of less than one-half of the expected increase (0.15 l/min) between the last two test stages (1). Using this protocol, all subjects demonstrated a plateau in $\dot{V}O_2$, either during the GXT (5 subjects) or during the follow-up bout (5 subjects).

Practice rides. Subjects performed two practice sessions on separate days after completion of the control $\dot{V}O_{2max}$ test and before starting the experimental trials. These trials were used to determine individual sweat rates and to familiarize subjects with the rebreathing techniques used in two of the experimental trials. Subjects cycled for 1 h at 60% of the $\dot{V}O_{2max}$ measured during the GXT test. One practice trial was performed at an ambient temperature of 22°C and the other was at 35°C, 40% humidity, with the order randomized. Nude body weight was taken before and after each session to determine sweat rate used to estimate fluid volume required for ingestion during the four experimental trials.

Experimental trials. Subjects arrived at the laboratory after following the same 3-h fast and hydration instructions as for the control GXT. The 24-h history questionnaire was given to verify adherence to instructions. Urine specific gravity (USG) and tympanic temperature (T_t)

were measured to ensure adequate hydration (urine specific gravity ≤ 1.020) and absence of a fever ($T_t < 37.8$). Subjects were instructed to measure their nude body weight and inserted a rectal temperature probe. A Teflon® venous catheter was inserted into the antecubital vein and flushed with .5ml of 10 USP units/ml heparin lock flush. Subjects then entered the environmental chamber and sat on the cycle ergometer for 20 min while skin temperature probes, were attached. Measurements of resting HR, rectal temperature (T_{re}), and skin temperatures (\bar{T}_{sk}) were taken. Subjects then began the trial cycling on the Lode ergometer at a power output previously determined to elicit 60% $\dot{V}O_{2max}$. During the two 45 min trials, metabolic and cardiovascular measures were taken between min 8 and min 15 and between min 38 and 45. Measurements included: systolic (SBP) and fourth-phase diastolic (DBP) blood pressures via auscultation, RPE, a blood sample, $\dot{V}O_2$ and $\dot{V}CO_2$ using the Parvo Medics system, HR using Polar® HR monitor, and two trials of CO_2 rebreathing to measure CO . Measurements were performed in the order listed for each subject and time point.

At the completion of the 15 or 45 min of submaximal exercise in the four experimental trials, subjects immediately performed a GXT without stopping exercise. The GXT protocol consisted of increasing the power output 25 W above the power output maintained during the submaximal exercise (60% $\dot{V}O_{2max}$), with additional 25 W increases in power output every 2 min until exhaustion. $\dot{V}O_2$ and related gas exchange measures were recorded continuously and averaged over 30-s intervals using the Parvo Medics system. HR using the Polar monitor and RPE were recorded during the last 10 seconds of each 2 min stage and at the time of exhaustion. A blood sample was drawn 3 min after the conclusion of the GXT and nude body weight was re-measured to determine fluid loss. $\dot{V}O_{2max}$ was considered valid for each experimental trial if one of the following criteria were met: (1) the plateauing criteria used during the control test was met or (2) maximal HR was within 5 bpm of that during the control test.

CO was measured using the indirect-Fick CO₂ -rebreathing method, described by Jones (23), which uses the $\dot{V}CO_2$, end-tidal CO₂ concentration, and the equilibrium CO₂ concentration after rebreathing to determine CO. SV was calculated by dividing cardiac output by HR. Mean arterial pressure (MAP) was calculated as $MAP = DBP + 0.33(SBP - DBP)$. Systemic vascular resistance (SVR) was calculated by dividing MAP by CO. T_{re} was measured using a thermocouple (Ellab, Inc., Arvada, CO, model MOV-55044-A) inserted 10 cm past the anal sphincter. Skin temperature was measured with thermocouples (Ellab, Inc., Arvada, CO, model MHF-18058-A) secured to the chest, deltoid, calf, and thigh. Rectal and skin temperature were recorded continuously over time using a computerized data acquisition system (Ellab, Inc., model TM9608 with Eval 2.1 software). Mean skin temperature (\bar{T}_{sk}) was calculated using the formula of Ramanathan (32) :

$$\bar{T}_{sk} = 0.3 (T_1 + T_2) + 0.2 (T_3 + T_4),$$

where T_1 , T_2 , T_3 , and T_4 are chest, deltoid, thigh, and calf skin temperatures. Mean body temperature (\bar{T}_b) was calculated from T_{re} and \bar{T}_{sk} with the formula of Baum et al. (5) :

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

Blood samples were drawn into vacutainers containing EDTA. Hemoglobin concentration was measured in duplicate using a HemoCue B-Hemoglobin photometer (HemoCue Inc., Mission Viejo, CA) and hematocrit was measured in triplicate using the microhematocrit method. Plasma volume (PV) change from rest during exercise was estimated from measures of hemoglobin and hematocrit using the Dill-Costill equation (9).

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 $\dot{V}O_{2max}$, percent heart rate reserve (%HRR) and percent $\dot{V}O_2$ reserve (% $\dot{V}O_{2R}$) 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

Statistical analyses were performed using SPSS v. 11 for Windows (SPSS, Inc., Chicago, IL). Data are reported as means \pm SD unless specified otherwise. A one-way ANOVA with repeated measures with post-hoc paired t-tests with Bonferroni α correction was performed to test mean differences between the control $\dot{V}O_{2max}$ test and the means from the four experimental tests. A 2×2 (Condition \times Time) ANOVA with repeated measures on both factors was used to test the significance of mean differences between temperature conditions and between 15 and 45 min in time, and their interaction. Simple regression and correlation were used to describe the relationship between cardiovascular drift and change in $\dot{V}O_{2max}$. Differences between regression equations describing relationships in H and C were tested using the method of Pedhazur (30). An α level of 0.05 was used for all significance tests.

RESULTS

Responses to submaximal exercise. Cardiovascular and gas exchange measures during submaximal exercise are contained in Table 1. $\dot{V}O_2$ at 15 min and 45 min was higher by 6% in H than C ($p < 0.05$), but there was no significant change over time in either condition. $\dot{V}O_2$ averaged $58 \pm 2\%$ and $61 \pm 3\%$ of the control $\dot{V}O_{2max}$ in C and H, respectively. Blood lactate remained low at ~ 2 mmol/l, but was higher in H than C at both time points. CV drift, as reflected by the changes in HR and SV, was significantly greater in H than C. In H, HR increased 17 bpm (11%, $p < 0.05$) and SV decreased 12 ml (11%, $p < 0.05$), but changes during C (3 bpm, 2%, $p < 0.05$ and 2.1 ml, 2%, $p < 0.05$) were much lower (Figure 1). There was a similar Condition \times Time interaction for O_2 pulse, with a decrease of 1.5 ml (8%, $p < 0.05$) in H, but no change during C ($p > 0.05$). There were no differences among means for CO, SVR, or PV change from rest

($p>0.05$), but MAP increased over time ($p<0.05$) at both temperatures. Increases in T_{re} , \bar{T}_{sk} , and \bar{T}_b over time were greater ($p<0.05$) in H than in C by 0.6, 0.8, and 1.0°C, respectively. RPE increased more in H (1.3 points, 11%, $p<0.05$) than in C (0.6 points, 5%), respectively.

Responses to maximal exercise. $\dot{V}O_{2max}$ and associated measures from the four experimental trials and control trial are contained in Table 2. $\dot{V}O_{2max}$ following 15 min of submaximal exercise in C and H was not different from control ($p>0.05$). Following 45 min of submaximal exercise, $\dot{V}O_{2max}$ was lower than following 15 min of submaximal exercise in H (15%, $p<0.05$), but not in C (5%, $p>0.05$). A similar pattern of findings was obtained for O_2 pulse, peak power output, test duration, and blood lactate. Likewise, T_{re} and \bar{T}_b at $\dot{V}O_{2max}$ were higher following 45 min than following 15 min of submaximal exercise in H ($p<0.05$), but not in C ($p>0.05$). \bar{T}_{sk} at $\dot{V}O_{2max}$ was not different following 15 and 45 min of submaximal exercise, but was higher in H than C as expected. Maximal HR was significantly ($p<0.05$) higher and maximal RER was significantly ($p<0.05$) lower in H than in C at both time points. There were no differences in maximal RPE, PV change from rest, or body mass change among the experimental trials.

Table 1: Responses during submaximal exercise (mean \pm SD).

Variable	22°C		35°C	
	15 min	45 min	15 min	45 min
$\dot{V}O_2$, (l/min) †	2.54 \pm 0.24	2.55 \pm 0.24	2.68 \pm 0.24	2.69 \pm 0.26
$\dot{V}O_2$, % Control	57.5 \pm 1.8	57.7 \pm 1.7	60.7 \pm 2.7	60.9 \pm 2.7
$\dot{V}O_{2max}$ †				
Blood lactate, (mmol/l) †	1.8 \pm 0.9	1.7 \pm 0.9	2.3 \pm 1.2	2.3 \pm 1.2
\dot{Q} , (l/min)	16.3 \pm .75	16.5 \pm 0.8	16.4 \pm 0.99	16.4 \pm 1.1
SV, (ml/beat) * †‡	117.4 \pm 9.4	116.9 \pm 9.1	110.1 \pm 12.1	98.2 \pm 9.4
HR, (bt/min) * †‡	140 \pm 6	142 \pm 6	151 \pm 12	168 \pm 11
O ₂ pulse, (ml/bt) * †‡	18.3 \pm 2.5	18.3 \pm 2.2	18.0 \pm 2.1	16.5 \pm 2.1
MAP, (mmHg) *	94.6 \pm 4.4	96.05 \pm 6.45	96.29 \pm 10.0	98.28 \pm 8.1
SVR, (dyn·cm·s ⁻⁵)	5.8 \pm 0.2	5.8 \pm 0.4	5.9 \pm 0.8	6.0 \pm 0.8
Δ PV from rest, %	-6.1 \pm 3.8	-4.6 \pm 3.8	-5.5. \pm 3.8	-6.8 \pm 2.4
T _{re} , (°C) * †‡	37.7 \pm 0.3	38.3 \pm 0.3	37.9 \pm 0.2	39.1 \pm 0.2
\bar{T}_{sk} , (°C) †	33.4 \pm 1.2	33.5 \pm 1.5	35.6 \pm 0.3	36.5 \pm 0.5
\bar{T}_b , (°C) * †‡	37.2 \pm 0.3	37.7 \pm 0.4	37.2 \pm 0.2	38.7 \pm 0.2
RPE*†‡	11.5 \pm 0.7	12.1 \pm 0.7	11.7 \pm 0.7	13.0 \pm 1.1

$\dot{V}O_2$ = oxygen uptake, \dot{Q} = cardiac output, SV = stroke volume, HR = heart rate, MAP = mean arterial pressure, SVR = systemic vascular resistance, T_{re} = rectal temperature, \bar{T}_{sk} = mean skin temperature, \bar{T}_b = mean body temperature, RPE = rating of perceived exertion, * = Time effect p < 0.05, † = Condition effect p < 0.05, ‡ = Condition \times Time interaction p < 0.05.

Table 2: Responses to maximal exercise (mean \pm SD).

Variable	Control	22°C		35°C	
		15-min	45-min	15-min	45-min
\dot{V}_E (STPD), l/min	132.9 \pm 12.35	139.4 \pm 10.5*†	137.7 \pm 9.1*‡	134.4 \pm 12.2*‡	122.5 \pm 11.9*†
$\dot{V}O_2$, l/min	4.42 \pm 0.49	4.70 \pm 0.62*	4.47 \pm 0.49	4.71 \pm 0.62*	4.02 \pm 0.54*†
$\dot{V}O_2$, ml/kg/min	64.7 \pm 8.7	68.9 \pm 11.3*	65.18 \pm 9.2	69.3 \pm 11.3*	59.2 \pm 10.2*†
Blood Lactate, mmol/l	8.93 \pm 2.23	7.8 \pm 1.3*	7.6 \pm 1.7*	7.6 \pm 1.6*	5.7 \pm 2.2*†
RER	1.08 \pm 0.06	1.05 \pm 0.04†	1.05 \pm 0.04	0.99 \pm 0.05*‡	.97 \pm 0.03*
RPE	19.1 \pm 0.6	19.2 \pm 0.4	19.1 \pm 0.3	19.0 \pm 0.5	19.2 \pm 0.4
HR, beats/min	188 \pm 8	188 \pm 7†	188 \pm 7	193 \pm 8*‡	195 \pm 7*
O ₂ Pulse, ml/bt	24.0 \pm 2.9	25.3 \pm 3.5	24.1 \pm 2.9	24.8 \pm 3.5	21.1 \pm 3.0*†
Δ PV from rest, %		-10.5 \pm 4.7	-10.6 \pm 5.9	-12.3 \pm 2.7	-10.3 \pm 3.1
T _{re} , (°C)		38.45 \pm 0.23	38.6 \pm 0.3	38.60 \pm 0.39	39.4 \pm 0.3†
\bar{T}_{sk} , (°C)		34.08 \pm 0.50†	33.5 \pm 1.7	36.41 \pm 0.62‡	36.7 \pm 0.4
\bar{T}_b , (°C)§		37.88 \pm 0.22†	37.9 \pm 0.4	38.32 \pm 0.33‡	39.0 \pm 0.3†
Δ Body Mass, %		-0.2 \pm 0.4	-0.3 \pm 0.4	-0.2 \pm 0.2	-0.2 \pm 0.5
Test Duration, min	11.9 \pm 3.3	13.3 \pm 2.5†	12.4 \pm 1.3	11.2 \pm 1.6‡	8.1 \pm 2.2*†
Power Output, Watts	338 \pm 43	343 \pm 49*†	335 \pm 41	320 \pm 47*‡	283 \pm 46*†

\dot{V}_E = minute ventilation, $\dot{V}O_{2max}$ = oxygen uptake, RER = respiratory exchange ratio, RPE = rating of perceived exertion, HR = heart rate, Δ PV = plasma volume change from rest, 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 H value, ‡ = p < 0.05 from 15-min C value.

Relation of CV drift to $\dot{V}O_{2max}$. The mean changes in $\dot{V}O_{2max}$ were directly related to those of SV during the preceding exercise (Figure 1); both variables decreased nearly proportionately following exercise in the heat and neither changed during exercise in the cool environment. Individual changes in SV were correlated to individual changes in $\dot{V}O_{2max}$ in C ($r=0.66$, $p<0.05$), but not in H ($r=0.05$, $p>0.05$). Mean changes in $\dot{V}O_{2max}$ were inversely related to those of HR during the preceding exercise (Figure 2); $\dot{V}O_{2max}$ decreased and HR increased nearly proportionately in H, but $\dot{V}O_{2max}$ did not change significantly during C. Individual increases in HR were significantly correlated with decreases in $\dot{V}O_{2max}$ in both H ($r=0.99$, $p<0.005$) and C ($r=0.94$, $p<0.005$) (Figure 2). The different ($p<0.05$) slopes for the relation of $\Delta\dot{V}O_{2max}$ to ΔHR in C and H indicates that CV drift is associated with decreased $\dot{V}O_{2max}$ under both conditions, but a given change in HR is associated with a greater change in $\dot{V}O_{2max}$ in C than in H.

Consequences of altered $\dot{V}O_{2max}$ for exercise prescription. Consequences of CV drift and the decrease in $\dot{V}O_{2max}$ for variables used in prescription of exercise intensity are summarized in Table 3 and Figure 3. The greater increase in HR between 15 and 45 min in H than in C was reflected in greater increases in $\%HR_{max}$ and $\%HRR$ in H than in C. $\%HR_{max}$ increased 7 ± 2 points in H and 1 ± 1 point in C. $\%HRR$ increased 13 ± 3 points in H and 3 ± 1 points in C. Similarly, the greater decrease in $\dot{V}O_{2max}$ between 15 and 45 min in H than in C was reflected in greater increases in $\%\dot{V}O_{2max}$ and $\%\dot{V}O_{2R}$ in H than in C. $\%\dot{V}O_{2max}$ and $\%\dot{V}O_{2R}$ increased 10 ± 7 points in H and 3 ± 3 points in C. The change in $\%\dot{V}O_{2R}$ was significantly correlated with the change in $\%HRR$ in both H and C ($r = 0.92$, $p<0.005$) and ($r = 0.92$, $p<0.005$). The slope of the regressions of $\%\dot{V}O_{2R}$ on $\%HRR$ was greater ($p<0.05$) in C than in H.

Table 3: Consequences of CV drift and changes in $\dot{V}O_{2\max}$ for variables used in exercise prescription (mean \pm SD).

Variable	22°C		35°C	
	15-min	45-min	15-min	45-min
%HR _{max}	74 \pm 3	75 \pm 4	78 \pm 3	85 \pm 4
%HRR	60 \pm 4	63 \pm 4	65 \pm 6	78 \pm 7
% $\dot{V}O_{2\max}$	54 \pm 3	57 \pm 3	57 \pm 4	67 \pm 8
% $\dot{V}O_{2R}$	52 \pm 3	55 \pm 3	55 \pm 4	65 \pm 8

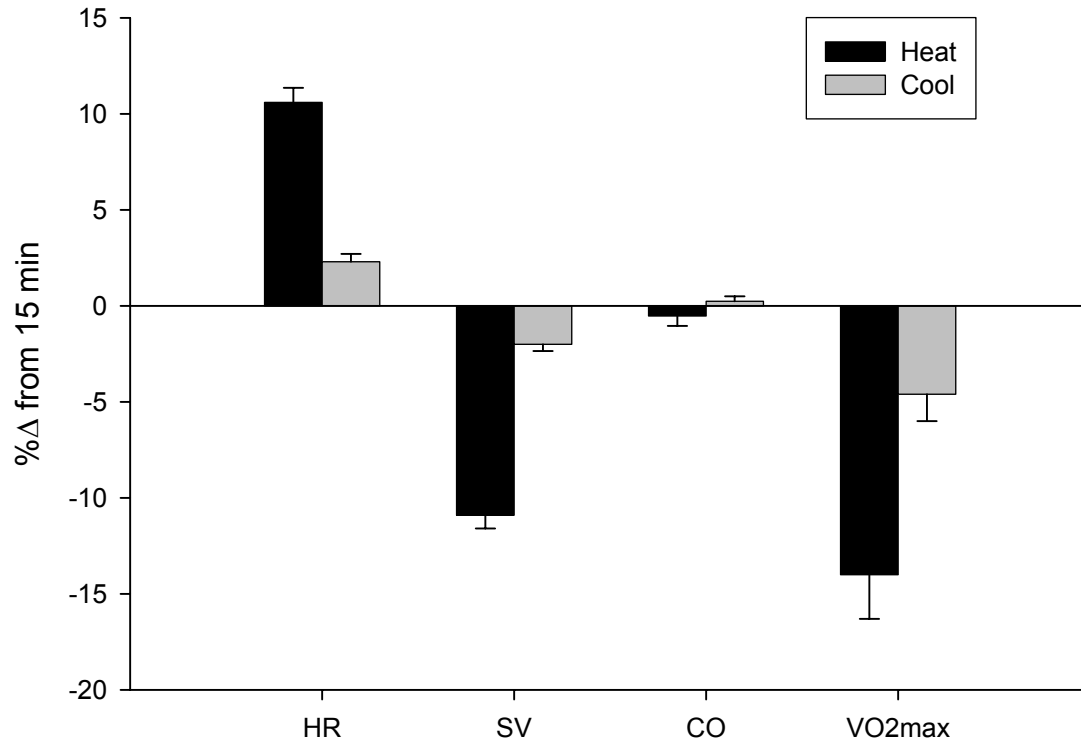


Figure 1: Changes in cardiovascular and metabolic variables during prolonged exercise in H and C. Data presented as % change from min 15 (mean \pm SEM).

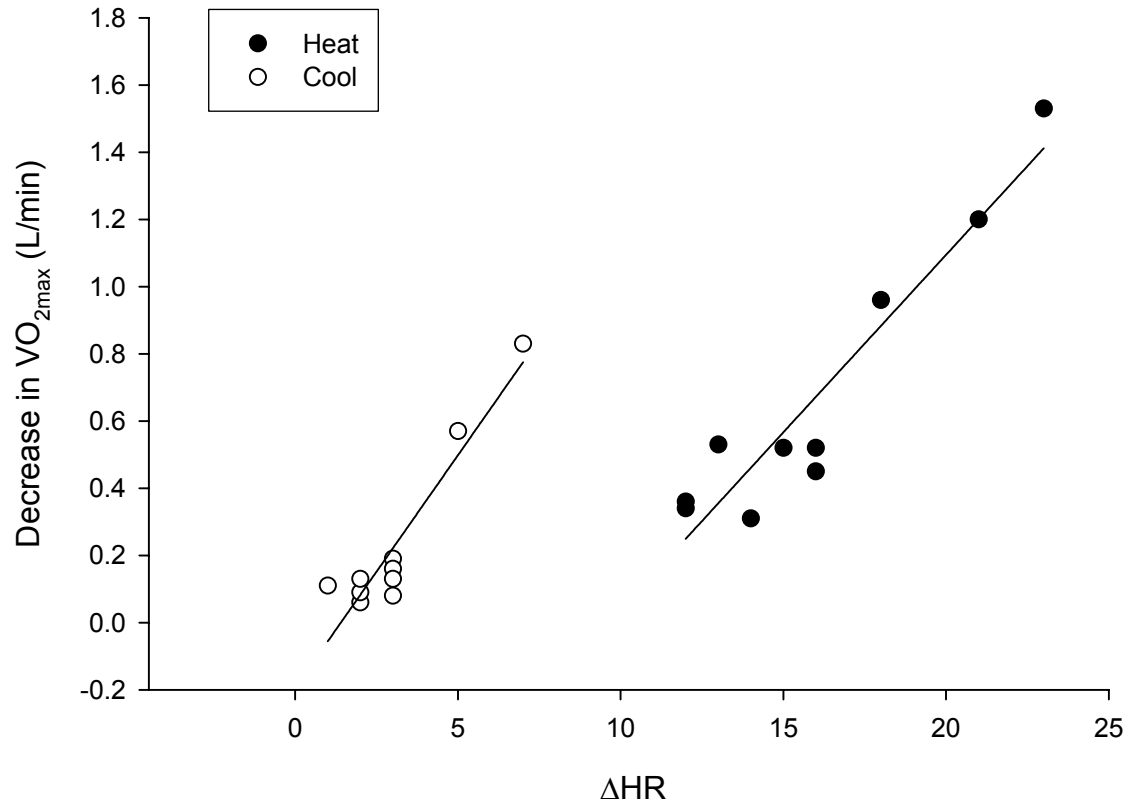


Figure 2: Relation between change in heart rate (HR) and change in maximal oxygen uptake ($\dot{V}O_{2max}$) in C ($\Delta\dot{V}O_{2max} = 0.138 \Delta HR - 0.194$, $r=0.94$, $SEE=0.095$, $p<0.05$) and H ($\Delta\dot{V}O_{2max} = 0.106 \Delta HR - 1.018$, $r=0.94$, $SEE=0.15$, $p<0.05$). Slopes of the regression equations are significantly different ($p<0.05$).

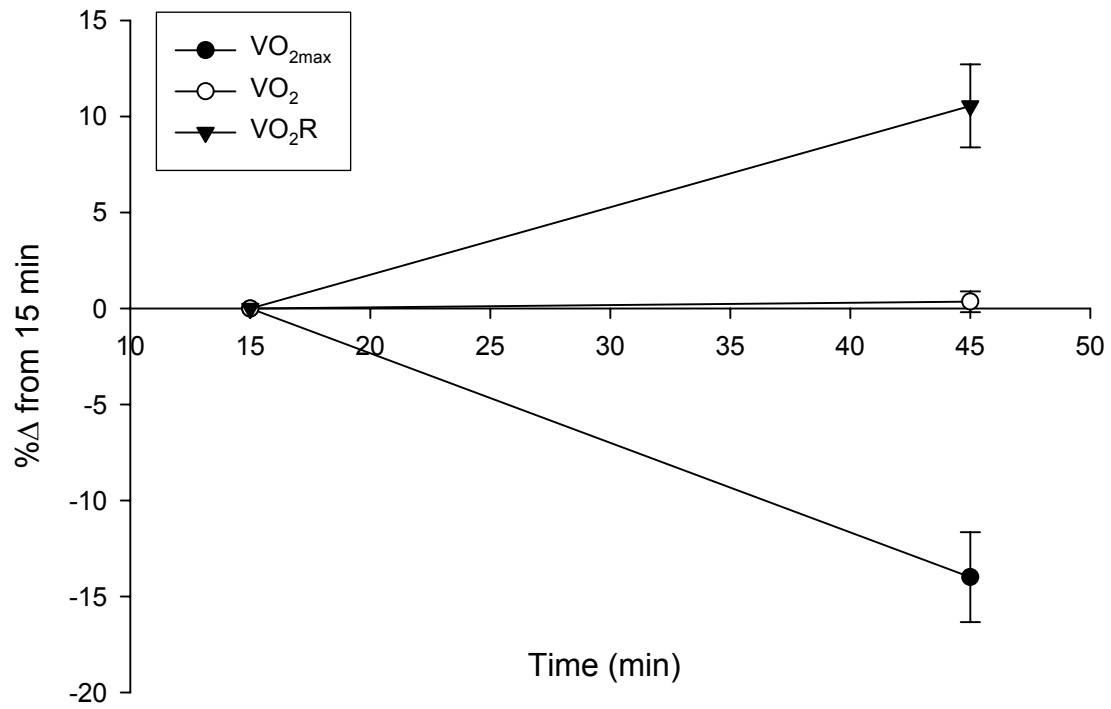


Figure 3: Changes in $\dot{V}O_2$, $\dot{V}O_{2max}$, and $\dot{V}O_{2R}$ after 45 min of moderately-intense upright cycling in hot conditions.

DISCUSSION

The main objective of this study was to manipulate the degree of CV drift by comparing responses during cool and hot ambient temperatures, and observe the associated changes in $\dot{V}O_{2\max}$ and work capacity. The primary finding was that CV drift and the reduction in $\dot{V}O_{2\max}$ between 15 and 45 min of cycling were greater in 35°C (H) than in 22°C (C). Individual increases in HR were strongly related to decreases in $\dot{V}O_{2\max}$ within H and C, but in H, the ΔHR corresponding to a given decrease in % $\dot{V}O_{2\max}$ was greater than in C.

CV drift between 15 and 45 min of cycling was much greater in H than C. In H, HR increased 17 ± 4 bpm (11%, $p < 0.05$) and SV decreased 12 ± 3 ml (11%, $p < 0.05$), but in C, HR only increased 3 ± 2 bpm (2%, $p < 0.05$) and SV decreased 2 ± 1 ml (2%, $p < 0.05$). This finding supports the first hypothesis of the study. The small CV drift in the cool condition in our study is in contrast to some studies that have reported increases in HR of 10-16% and decreases in SV of 12-15% in comfortable conditions (24-27°C) (12; 37; 39). The discrepancy appears to be related to the lower exercise intensity, shorter exercise duration, and trained state of the subjects used in this study. Studies that have reported CV drift under cool conditions have used higher intensity ($>70\%$ $\dot{V}O_{2\max}$) (12) or longer durations (>60 min) (37; 39), and untrained subjects. The magnitude of CV drift in this study is similar to that reported by Nassis et al. (28), however, who found an increase in HR of 5 bpm and a decrease in SV of 5 ml/bt in healthy but not highly-trained subjects after 45 min at the same relative intensity (60% $\dot{V}O_{2\max}$).

The magnitude of CV drift in the heat (35°C) measured in this study is similar to values reported in previous research. As in research on CV drift in thermoneutral conditions, greater increases in HR (10-17%) and decreases in SV (15-27%) in the heat found in previous studies have been associated with higher relative intensity ($>65\%$) (27) and longer duration exercise (>120 min) (18; 20; 27). When adjusted for the magnitude of CV drift at 45 min, previous

studies show increases in HR (7-10%) and decreases in SV (7-9%) (18; 20; 27), which agree with the increase in HR (11%) and the decrease in SV (11%) found in this study.

$\dot{V}O_{2\max}$ was significantly reduced in hot (15%, $p < 0.05$) but not cool (5%, $p > 0.05$) conditions following prolonged (45 min) exercise. $\dot{V}O_{2\max}$ following 15 min of submaximal exercise in a hot environment did not differ significantly from the control condition. The presence of little or no reduction in $\dot{V}O_{2\max}$ in cool conditions is similar to findings obtained in other studies reporting moderate decreases in $\dot{V}O_{2\max}$ (5-12%) (4; 7) following prolonged exercise.

The decrease in $\dot{V}O_{2\max}$ of 15% in hot conditions was also in agreement with the decreases in $\dot{V}O_{2\max}$ (16-25%) (3; 29; 31) reported by other studies that have measured $\dot{V}O_{2\max}$ in hot conditions (35-46°C) in which considerable core hyperthermia occurred. Pirnay et al. (31) reported a large decrease in $\dot{V}O_{2\max}$ (25%) in subjects at an ambient temperature of 46°C following 20 min of low-intensity exercise that elevated core temperature. Nybo et al. (29) reported a 16% decrease in $\dot{V}O_{2\max}$ following preheating that elevated core temperature to 39°C and skin temperature to 37°C. Arngrimsson et al. (3) reported a decrease in $\dot{V}O_{2\max}$ of 18% with core temperature elevated above 39°C and skin temperature elevated above 38°C in subjects who were preheated prior to performing a $\dot{V}O_{2\max}$ test.

Studies showing no or minimal change in $\dot{V}O_{2\max}$ during brief exposure to heat and little core hyperthermia (31; 35; 38) suggest that for short periods of maximal exercise, vasoconstriction of blood vessels in the skin and gut can shunt blood to active skeletal muscles and maintain muscle blood flow and oxygen delivery, increasing stroke volume to levels of those found in thermoneutral conditions. In contrast, it has been suggested that during prolonged work in the heat, vasodilation of skin blood vessels dominates and $\dot{V}O_{2\max}$ is reduced (34). Studies that have shown large reductions in $\dot{V}O_{2\max}$ in the heat usually have some combination of core and skin temperature that may prevent vasoconstriction of skin blood vessels during high

intensity exercise in the heat (2; 29; 31). The results of this study are consistent with the mechanism suggested by Rowell (33) in which a combination of core and skin temperature causes cutaneous vasodilation to prevail.

The decline in $\dot{V}O_{2\max}$ after prolonged moderate intensity exercise in hot but not cool conditions may be due to greater hyperthermia and cardiovascular changes associated with CV drift. The findings that $\dot{V}O_{2\max}$ did not decrease after 45 min of moderate exercise in cool conditions but did significantly decrease after 45 min of moderate exercise in hot conditions suggest that the decline in $\dot{V}O_{2\max}$ is not due to local muscular fatigue caused by the prolonged exercise per se (40). The attainment of equal maximum power output values after 45 min of exercise in the thermoneutral compared to the control condition also supports this argument. The greater reduction of $\dot{V}O_{2\max}$ following 45 min compared to 15 min in H also was not due to lack of effort or inability to reach $\dot{V}O_{2\max}$. Each subject attained a HR_{\max} within 5 bpm of control during the last completed stage of the GXT. In fact, HR_{\max} during the two experimental trials in H was slightly higher than in C. Attainment of higher HR_{\max} during exercise in the heat that elicits core hyperthermia has been reported previously (29; 31). Reaching a HR_{\max} equal to or higher than control suggests that chronotropic cardiac function was not inhibited. In addition, mean RPE at the end of the control and experimental GXTs was 19, indicating that near-maximal effort was given under all four conditions.

Because HR_{\max} was not lower after 45 min of cycling compared to 15 min of cycling in H, according to the Fick principle, the decrease in $\dot{V}O_{2\max}$ must be due to either a decrease in (a-v) O_2 difference or SV. Previous studies have shown that (a-v) O_2 difference is unaltered in the heat (16; 36; 42). Therefore, the lower $\dot{V}O_{2\max}$ was probably caused by reduced SV and CO, which would result in a lower maximal skeletal muscle blood flow and reduced oxygen delivery to active muscles, thus limiting $\dot{V}O_{2\max}$ (16).

Two studies suggest that CV drift, measured as a decrease in SV, persists during maximal exercise. Saltin and Stenberg (37) measured central circulatory responses to three young men during 195 min of cycling and running at 75% $\dot{V}O_{2\max}$, and, following 90 min of rest, during an additional 15 min of submaximal exercise proceeding directly into a $\dot{V}O_{2\max}$ test. HR increased 20% and SV decreased 8% from min 15 to min 195 of submaximal exercise. Cardiorespiratory values measured at the end of the subsequent 15 min of submaximal exercise remained the same as at the end of the 195 min of submaximal exercise. $\dot{V}O_{2\max}$ was decreased 5% compared to the control. The results suggest the reduction in SV observed following prolonged exercise persisted during maximal exercise and was related to slightly reduced $\dot{V}O_{2\max}$. While 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 by possibly allowing elevated core temperature to subside.

Eklom (10) measured cardiovascular changes and $\dot{V}O_{2\max}$ immediately after 1 h of cycling at 75% $\dot{V}O_{2\max}$ under thermoneutral conditions before and after aerobic training in 7 subjects. 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 was unchanged from that at 60 min, indicating that changes in SV persisted during maximal exercise. As a result, $\dot{V}O_{2\max}$ was reduced 6% following the 60 min of submaximal exercise prior to training and 12% after training.

The reduction in SV over time in a hot environment may be caused by 1) cutaneous vasodilation and increased venous volume, reducing central blood volume, ventricular filling pressure and end-diastolic volume (35) and 2) increased sympathetic nervous system activity increasing SVR and HR (25). The higher HR in the heat after 45 min of exercise would decrease ventricular filling time and therefore end-diastolic volume (14). This study showed that in H,

increasing exercise intensity to that which elicits $\dot{V}O_{2\max}$ after CV drift has occurred, SV cannot be restored to control levels, suggesting that factors are influencing SV other than just the increase in HR. Following 15 min of submaximal exercise in H and C, HR increases to maximum, and $\dot{V}O_{2\max}$ is not reduced, suggesting SV is not reduced.

One explanation for the reduced SV in H is that the higher T_{re} and \bar{T}_b seen after 45 min of moderate intensity exercise is associated with greater cutaneous venous volume, which reduces central blood volume, central venous pressure and SV (33). Pirnay et al. (31) were the first to observe a large decrease (27%) in $\dot{V}O_{2\max}$ in the heat. Their protocol consisted of measuring $\dot{V}O_{2\max}$ following 20 min of low-intensity exercise that elevated core body temperature. When subjects were not preheated, $\dot{V}O_{2\max}$ decreased only 7%. Nybo et al. (29) observed a 16% decrease in $\dot{V}O_{2\max}$ when core temperature was 39°C and skin temperature was 37°C. Arngrimsson et al. (3) performed a $\dot{V}O_{2\max}$ test in subjects with varying degrees of elevated skin and core body temperatures. They observed a decrease in $\dot{V}O_{2\max}$ of 18% when core body temperature was elevated above 39°C and skin temperature was elevated above 38°C, and mean body temperature was $\geq 39.2^\circ\text{C}$. The similar decrease (15%) in $\dot{V}O_{2\max}$ reported in this study corresponds with a similar elevated mean body temperature of 39°C.

Although the phenomenon of CV drift has been studied for years, its metabolic consequences have not been clear. We found that an increase in HR and decrease in SV over time is associated with a reduction in $\dot{V}O_{2\max}$. Because $\dot{V}O_2$ during submaximal cycling remained constant, the metabolic intensity, as reflected by $\% \dot{V}O_{2\max}$ or $\% \dot{V}O_{2R}$, increased between 15 and 45 min of exercise in the heat when substantial CV drift occurred (Figure 3). The elevation in HR during submaximal exercise performed in the heat is related in part to the increase in $\% \dot{V}O_{2\text{peak}}$ elicited in the heat. Arngrimsson et al. (2) showed that during sustained submaximal exercise in high ambient temperatures, the dissociation of HR from $\% \dot{V}O_{2\text{peak}}$ is less than that predicted by assuming $\dot{V}O_{2\max}$ is unchanged in the heat.

This is the first study that we are aware of to evaluate whether the strong link between HR and relative metabolic intensity in hot and comfortable conditions, demonstrated during short-term exercise, persists during prolonged, constant-rate exercise during which submaximal $\dot{V}O_2$ remains constant, but HR rises progressively, over time. In this study, CV drift was found to be associated with a reduced $\dot{V}O_{2max}$, indicating that an increase in HR over time during prolonged exercise in the heat is an important marker of changes in $\dot{V}O_{2max}$ and work capacity. The regression of change in $\dot{V}O_{2max}$ on change in HR in H and C had significantly different ($p < 0.05$) slopes, with a 1 bpm increase in HR from min 15 to min 45 accompanied by a decrease in $\dot{V}O_{2max}$ of 0.11 l/min in C and 0.14 l/min in H (Figure 3). Therefore, ambient temperature affected the relationship between CV drift and decrease in $\dot{V}O_{2max}$. This suggests that other factors may contribute to the increase in HR in H other than a decrease in SV.

The slopes of the $\% \dot{V}O_{2max}$ to $\%HR_{max}$ and $\% \dot{V}O_{2R}$ to $\%HRR$ relationships in cool conditions (22°C) were different ($p < 0.05$) in hot conditions (35°C). Because there was no, or little change in submaximal $\dot{V}O_2$, CV drift occurred independent of a change in submaximal $\dot{V}O_2$, suggesting that change in HR is an indicator of the changes in relative exercise intensity ($\dot{V}O_{2max}$).

CONCLUSION

In summary, we found that there is a strong but different relationship between CV drift and the reduction in $\dot{V}O_{2max}$, as reflected by the rise in HR over time, in C and H. No significant change in $\dot{V}O_{2max}$ was found when no or little CV drift occurs in cool (22°C) conditions, but a large change in $\dot{V}O_{2max}$ occurred in the heat, when CV drift was substantial. There were strong individual relationships between ΔHR and $\Delta \dot{V}O_{2max}$ in cool and hot environments. The increase in HR over time in hot and cool environments reflected an increase in relative metabolic intensity. We conclude that CV drift during prolonged exercise is (1) greater in a hot than in a

cool environment, (2) associated with a decrease in $\dot{V}O_{2\max}$ in the heat and (3) reflects an increase in relative metabolic intensity.

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CHAPTER IV

SUMMARY AND CONCLUSION

After about 10 minutes of prolonged moderate-intensity exercise (50-75% $\dot{V}O_{2max}$), cardiovascular drift occurs (39). CV drift is characterized by progressive decreases in stroke volume, pulmonary and systemic mean arterial pressures, and a progressive increase in HR (42). The cause of the reduced SV is multifactorial, with increased skin blood flow and cutaneous venous volume all being possible contributing factors. Whether the reduction in SV during prolonged submaximal exercise reflects reduced cardiovascular capacity and physical work capacity is unknown. CV drift appears to be greater in a hot than cool environment, but it is not known if this reflects a greater reduction in $\dot{V}O_{2max}$. The effect of CV drift on $\dot{V}O_{2max}$ during prolonged exercise at different ambient temperatures may alter the HR-relative metabolic intensity relationship used for exercise prescription.

This study determined whether ambient temperature affects the magnitude of CV drift and reduction in $\dot{V}O_{2max}$ over time during prolonged cycling and whether the relation of CV drift to reduction in $\dot{V}O_{2max}$ is the same in cool and hot ambient environments. CV drift was measured between 15 and 45 min of cycling in two ambient temperatures (22°C and 35°C). $\dot{V}O_{2max}$ was measured immediately after the completion of each experiment trial to determine the relationship between CV drift and the reduction in $\dot{V}O_{2max}$.

CV drift and the associated decrease in $\dot{V}O_{2max}$ was greater ($p<0.05$) in H than C. Between 15 and 45 min of cycling in H, an increase in HR of 17 ± 4 bpm (11%, $p<0.05$) and decrease in SV of 12 ± 3 ml (11%, $p<0.05$) was associated with a 15% decrease in $\dot{V}O_{2max}$ (4.7 ± 0.6 vs 4.0 ± 0.5 l/min, $p<0.05$). Between 15 and 45 min of cycling in C, HR increased (3 ± 2 bpm, 2%, $p<0.05$), and SV decreased (2 ± 1 , 2%, $p<0.05$) and there was no significant decrease in $\dot{V}O_{2max}$ (4.7 ± 0.6 vs 4.5 ± 0.5 l/min, 5%, $p>0.05$). Nevertheless, there was a significant relationship between ΔHR and $\Delta \dot{V}O_{2max}$ in H ($\Delta \dot{V}O_{2max} = 0.106 \Delta HR - 1.018$, $r=0.94$, $SEE=0.15$

l/min, $p < 0.05$) and C ($\Delta \dot{V}O_{2\max} = 0.138 \Delta HR - 0.194$, $r = 0.94$, $SEE = 0.09$ l/min, $p < 0.05$). The slopes of the regression equations of ΔHR on $\Delta \dot{V}O_{2\max}$ were significantly different ($p < 0.05$). Submaximal $\dot{V}O_2$ was unchanged over time ($p > 0.05$), but $\% \dot{V}O_{2\max}$ increased from $57 \pm 4\%$ at 15 min to $67 \pm 8\%$ at 45 min in H ($p < 0.05$), and from $54 \pm 3\%$ at 15 min to $57 \pm 3\%$ at 45 min in C ($p < 0.05$).

We conclude that CV drift during prolonged exercise is (1) greater in a hot than in a cool environment, (2) associated with a decrease in $\dot{V}O_{2\max}$ in the heat and (3) reflects an increase in relative metabolic intensity.

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