CARDIOVASCULAR DRIFT AND MAXIMAL OXYGEN UPTAKE DURING HEAT STRESS

by

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(Under the Direction of Kirk J. Cureton)

ABSTRACT

The mechanisms causing cardiovascular drift (CV drift), the temporal rise in heart rate (HR) and fall in stroke volume (SV) during prolonged, constant-rate exercise, are well studied. Whether or not CV drift has any consequences is unknown. The research for this dissertation was designed to determine if the CV drift that occurs during prolonged exercise in hot conditions is associated with decreased maximal oxygen uptake ($\dot{V}O_{2\text{max}}$), and further, whether this relationship is cause and effect. Three studies were completed. In each, CV drift was measured during 45 min of cycling at 60% $\dot{V}O_{2\text{max}}$ in 35°C, immediately followed by measurement of $\dot{V}O_{2\text{max}}$. $\dot{V}O_{2\text{max}}$ also was measured after 15 min of cycling in 35°C. The purpose of the 15- and 45-min trials was to measure $\dot{V}O_{2\text{max}}$ during the same time interval in which CV drift occurred. In Study 1, the 15- and 45-min trials were performed with and without fluid ingestion. Fluid ingestion did not affect CV drift or change in $\dot{V}O_{2\text{max}}$. A 19% $\dot{V}O_{2\text{max}}$ decrease accompanied a 12% HR increase and 16% SV decrease, while %$\dot{V}O_{2\text{max}}$ increased from 63% to 78% between 15 and 45 min. In Study 2, the 45-min trials occurred either with HR held constant by lowering exercise intensity by a magnitude sufficient to attenuate
CV drift or with unaltered exercise intensity. Power output decreased 37%, $\dot{V}O_{2\text{max}}$ decreased 7.5%, and $%\dot{V}O_{2\text{max}}$ decreased from ~60% to ~50% from 15 to 45 min when HR was held constant. When exercise intensity was unaltered, HR increased 13%, SV decreased 10%, and $\dot{V}O_{2\text{max}}$ decreased 15%. In Study 3, the 45-min trials were performed with and without body cooling via fan airflow. $\dot{V}O_{2\text{max}}$ fell 18%, HR rose 16%, and SV fell 12% from 15–45 min without cooling, whereas $\dot{V}O_{2\text{max}}$ declined 5.7% with fan cooling, and HR and SV remained more stable across time. In conclusion, CV drift that occurs during prolonged exercise in hot conditions is associated with decreased $\dot{V}O_{2\text{max}}$, and the relationship appears to be cause and effect. Additionally, the HR–$%\dot{V}O_{2\text{max}}$ relationship is not preserved over time during prolonged exercise in heat stress in the absence of CV drift.

INDEX WORDS: Heart rate, Stroke volume, Circulation, Thermoregulation, Exercise, Aerobic capacity, Exercise prescription
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DEDICATION

I dedicate this work to my wife, Kelly, for her love and support during my years of graduate school, and for her help in completing this dissertation. And, to Jeff and Dad…you are not forgotten.
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TABLE OF CONTENTS

ACKNOWLEDGEMENTS........................................................................................................v

CHAPTER

1 INTRODUCTION .............................................................................................................1
   Hypotheses ..................................................................................................................2
   Significance of the Study ..........................................................................................3
   Definition of Terms ....................................................................................................4

2 REVIEW OF THE RELATED LITERATURE ...............................................................6
   Hypotheses Regarding Causes of Cardiovascular Drift ...........................................7
   Other Factors Influencing Cardiovascular Drift .....................................................11
   Potential Consequences of Cardiovascular Drift ...................................................13
   Maximal Oxygen Uptake .........................................................................................16
   Body Cooling During Exercise ...............................................................................24
   Summary ..................................................................................................................26
   References ..............................................................................................................27

3 CARDIOVASCULAR DRIFT IS RELATED TO REDUCED MAXIMAL
   OXYGEN UPTAKE DURING HEAT STRESS ...........................................................40
   Abstract ....................................................................................................................41
   Introduction ..............................................................................................................43
   Methods ....................................................................................................................45
APPENDICES

A  STUDY 1 REPRINT PERMISSION.............................................................124
B  STUDY 2 REPRINT PERMISSION.............................................................126
CHAPTER 1
INTRODUCTION

Cardiovascular drift (CV drift) is a well-known phenomenon characterized by a rise in heart rate (HR) and fall in stroke volume (SV) over time during constant-rate, submaximal exercise. CV drift may be influenced by many factors, including ambient (33, 75) and core body temperatures (37); exercise duration (30), intensity (100), and posture (21, 38); and hydration status (37). While the causes of and factors affecting CV drift have been extensively studied, its consequences, like effects on maximal oxygen uptake (\(\dot{V}O_{2\text{max}}\)), remain unclear.

Whether CV drift affects \(\dot{V}O_{2\text{max}}\) or not needs elucidation because it has implications for exercise prescription. Since a target HR is often used to prescribe relative metabolic intensity [\(\%\dot{V}O_{2\text{max}}\)] (52), it is important to know whether the higher HR’s associated with CV drift that occurs during constant-rate, submaximal exercise in high ambient temperatures reflect a dissociation from a prescribed \(\%\dot{V}O_{2\text{max}}\) or not. Additionally, since \(\dot{V}O_{2\text{max}}\) sets the upper limit of the rate of aerobic metabolism, understanding whether CV drift is merely related to reduced \(\dot{V}O_{2\text{max}}\) or whether the relationship between these is cause and effect is important in: 1) understanding what limits work capacity in the heat and 2) optimizing training for, and performance in, endurance events held in hot temperatures.

This dissertation is comprised of 3 studies investigating the effect of CV drift on \(\dot{V}O_{2\text{max}}\). The overall aim was to determine if CV drift that occurs during prolonged,
moderate-intensity, constant-rate exercise in a hot environment is associated with reduced \( \dot{V}O_{2\text{max}} \), and furthermore whether this relationship is cause and effect. Study 1 (112) was intended to establish if CV drift is related to reduced \( \dot{V}O_{2\text{max}} \) during prolonged exercise in a hot environment as well as determine the extent to which dehydration accounted for any potential relationship between CV drift and \( \dot{V}O_{2\text{max}} \). Study 2 was intended to determine if \( \dot{V}O_{2\text{max}} \) is reduced when CV drift is manipulated by lowering exercise intensity by a magnitude sufficient to hold HR constant during prolonged exercise in the heat, and thereby examine if the HR–%\( \dot{V}O_{2\text{max}} \) relationship is preserved. Finally, Study 3 was proposed to discover whether \( \dot{V}O_{2\text{max}} \) is reduced when CV drift is manipulated by body cooling during prolonged exercise in a hot environment. Body cooling was initially brought about by 1) use of airflow from a floor fan in combination with a hand-cooling device purported to blunt the rise in core temperature during exercise and 2) fan airflow alone. Because the hand-cooling device had no effect beyond that of fan airflow alone, it was omitted from analysis and presentation.

**Hypotheses**

**Study 1.** The hypotheses for Study 1 were:

1) CV drift during 45 min of cycling at 60% \( \dot{V}O_{2\text{max}} \) in 35°C is associated with reduced \( \dot{V}O_{2\text{max}} \) and increased relative metabolic intensity (%\( \dot{V}O_{2\text{max}} \)).

2) Fluid ingestion decreases the magnitude of CV drift and accompanying reduction in \( \dot{V}O_{2\text{max}} \).
Study 2. The hypothesis for Study 2 was:

\( \dot{V}O_{2\text{max}} \) is not reduced when power output and absolute \( \dot{V}O_2 \) are decreased by a magnitude sufficient to reduce CV drift and maintain constant HR during 45 min of cycling at 60% \( \dot{V}O_{2\text{max}} \) in 35°C compared to when they are unaltered.

Study 3. The hypothesis for Study 3 was:

Body cooling via fan airflow at 4.5 m/s during 45 min of cycling at 60% \( \dot{V}O_{2\text{max}} \) in 35°C attenuates CV drift and reduces the accompanying decrement in \( \dot{V}O_{2\text{max}} \).

Significance of the Study

This series of studies contributes new information to the existing body of knowledge regarding what limits \( \dot{V}O_{2\text{max}} \) in the heat. Furthermore, determining that CV drift is related to reduced \( \dot{V}O_{2\text{max}} \) offers new insight into the relation between HR and relative metabolic intensity (\%\( \dot{V}O_{2\text{max}} \)) when CV drift occurs. Heart rate increases during submaximal exercise in hot conditions (57, 58, 112) because of direct effects of blood temperature on the intrinsic heart rate (8, 56) as well as effects of temperature on autonomic nervous system activity (42). Since most studies have reported little change in oxygen uptake (\( \dot{V}O_2 \)) over time during constant-rate exercise in the heat (87, 112), the rise in HR associated with CV drift has been considered a dissociation from \%\( \dot{V}O_{2\text{max}} \). Since it was found that CV drift is related to reduced \( \dot{V}O_{2\text{max}} \), however, the rise in HR with CV drift reflects increased relative metabolic intensity (\%\( \dot{V}O_{2\text{max}} \)). When CV drift does not occur and HR is held constant, though, the HR–\%\( \dot{V}O_{2\text{max}} \) relationship is not preserved since \%\( \dot{V}O_{2\text{max}} \) must be lowered for HR to be maintained in the heat. Whether CV drift is manipulated via exercise intensity or body cooling, the effect on \( \dot{V}O_{2\text{max}} \) is the same, which suggests the relationship between CV drift and reduced \( \dot{V}O_{2\text{max}} \) during
prolonged exercise in the heat is cause and effect. Furthermore, combined with findings from other studies (30, 111), the present findings suggest the link between CV drift and decreased \( \dot{V}O_{2\text{max}} \) is independent of the mode of manipulation of CV drift, but dependent on factors that influence the magnitude of CV drift, like dehydration and hyperthermia. This offers new insight into what limits work capacity in the heat and may offer a basis for strategies to optimize training for, and performance in, endurance events.

**Definition of Terms**

In this dissertation, “CV drift” is used as a global term to refer to a phenomenon that occurs during constant-rate, prolonged exercise [that lasting 30 min or more (20)] in which heart rate rises and stroke volume declines over time. Other studies and discussions of CV drift have included declines in pulmonary and mean systemic pressures as part of the definition (20, 85), but these aspects are not requisite for the definition of CV drift in the current study. Furthermore, “classic CV drift” refers to CV drift that occurs in moderate ambient temperatures (room temperature and humidity).

Maximal oxygen uptake (\( \dot{V}O_{2\text{max}} \)) is defined as the highest rate oxygen can be taken up by the body. For the purposes of this series of studies, \( \dot{V}O_{2\text{max}} \) refers to the maximum rate oxygen can be taken up by the body for a given exercise mode, which is the cycle ergometer in this case. A critical muscle mass is necessary to elicit a level of oxygen uptake that can be considered maximal, but no minimum fraction of total muscle mass necessary to elicit \( \dot{V}O_{2\text{max}} \) has been established (7, 85). During cycling exercise like that to be employed in this study, a large muscle mass will be utilized such that the physiological endpoints of \( \dot{V}O_{2\text{max}} \) as defined by the Fick principle (85):

\[
\dot{V}O_{2\text{max}} = HR_{\text{max}} \times SV_{\text{max}} \times (a-\bar{v})O_{2\text{max}},
\]
where $\dot{V}O_{2max}$ is maximal oxygen uptake, $HR_{max}$ is maximum heart rate, $SV_{max}$ is maximum stroke volume, and $(a-\tilde{v})O_{2max}$ is maximum arteriovenous oxygen difference, should be reached to an extent similar to that reached if uphill treadmill running [a large-muscle mass activity known to elicit $\dot{V}O_{2max}$ in healthy individuals (50, 59)] was used instead. A procedure to ensure a plateau in $\dot{V}O_2$ was reached at maximum was used in control tests in all three studies, but this same procedure could not be completed after experimental trials, so values obtained at maximum in those trials are referred to as $\dot{V}O_{2peak}$. 
CHAPTER 2

REVIEW OF THE RELATED LITERATURE

The literature for this review was primarily selected from relevant journal articles and accompanying reference lists. Additional sources were obtained from electronic searches of ProQuest Dissertation and Theses Database and PubMed/Medline (no date ranges were specified) using keywords pertinent to the topics in the studies featured herein. Sources were gathered via electronic retrieval, library holdings, and inter-library loan (in the case of one dissertation and one thesis).

Much research has focused on cardiovascular responses to prolonged exercise, including progressive increases in heart rate (HR) and decreases in stroke volume (SV) over time during constant-rate exercise, which characterize a phenomenon commonly known as cardiovascular drift [CV drift] (84). The different hypotheses describing the causes of CV drift that occurs in a temperate environment in which there is little dehydration and change in blood volume (i.e., classic CV drift) are reviewed here, in addition to other factors that influence CV drift when extensive dehydration occurs. While CV drift has been studied as far back as the 1960’s, its consequences are less known, so literature related to potential consequences of CV drift is also reviewed. One possible consequence of CV drift is a reduction in maximal oxygen uptake ($\dot{V}O_{2\text{max}}$). Thus, this review will present literature defining $\dot{V}O_{2\text{max}}$ as well as describing its determinants and limitations. Finally, body cooling administered during prolonged
exercise has been shown to attenuate CV drift (43, 48, 64, 100), so a brief review of studies investigating CV drift and body cooling is included.

**Hypotheses Regarding Causes of Cardiovascular Drift**

As mentioned above, CV drift is characterized by a continuous, time-dependent change in some cardiovascular responses. In addition to the progressive rise in HR and decline in SV, pulmonary and systemic mean arterial pressures may decline, while cardiac output (\(\dot{Q}\)) is held fairly constant (20, 22, 23, 84). There have been two prevailing schools of thought as to the causes of *classic CV drift*. The traditional hypothesis links peripheral displacement of the blood volume to a decline in SV (84), whereas a more contemporary hypothesis asserts that increased HR lowers ventricular filling time and thereby SV (13). Research on traditional and contemporary perspectives as to the causes of CV drift will be discussed in the following sections.

*Traditional Hypothesis.* Rowell (84) asserts that a progressive increase in skin blood flow accompanied by reductions in central venous pressure is what ultimately leads to the reduction in SV that occurs as part of *classic CV drift* during prolonged exercise. The increase in HR is most likely a baroreceptor-mediated reflex intended to maintain \(\dot{Q}\) and blood pressure in the wake of a falling SV (84). However, HR may continue to increase even when central venous pressure, and thereby SV, is maintained (74), suggesting the rise in HR may be influenced by other factors. Rising body temperature can increase the rate of sinoatrial node firing and intrinsic rate of heart contraction (8, 42, 56), as well as increase sympathetic nervous system activity (42, 62), thereby increasing HR. Additionally, recent evidence (30, 111, 112) demonstrating a link between increased HR (associated with CV drift) and decreased maximal oxygen uptake (\(\dot{V}O_{2\text{max}}\)) suggests
HR may be increased in conjunction with a higher relative metabolic intensity (if absolute work rate is unchanged and $\dot{V}O_{2\text{max}}$ is reduced). Regardless of what causes HR to increase, decreased SV is the principal component of classic CV drift around which debate regarding underlying mechanisms centers (26).

Since skin is a highly compliant vascular bed, based on Rowell’s logic, increases in blood flow to the skin increase the volume of blood there, which causes a transient drop in volume and pressure in the central veins that ultimately decreases ventricular filling and SV (103). Shaffrath (100) demonstrated that SV declined over the same time interval in which forearm blood flow [representative of skin blood flow (55)] increased, which suggests central blood volume was displaced peripherally. Furthermore, techniques like bandaging the legs compress veins in active limbs and can increase SV (albeit only slightly) during exercise in neutral temperatures (45), thus further supporting the notion that peripheral displacement of blood volume causes decreased SV.

If increased skin blood flow causes a reduction in SV during prolonged, constant-rate exercise, it is natural to wonder what causes the increased skin blood flow. More than likely, increased body temperature is responsible. Even though Rowell’s hypothesis is based on exercise of moderate intensity in cool conditions (84), body temperature nonetheless rises over time. This increase in core temperature initiates a thermoregulatory response that shunts blood to the compliant skin vasculature for heat dissipation (55). The notion that blood is shunted to the skin and not another vascular bed, like muscle, is evidenced by a relatively stable $\dot{Q}$ during prolonged exercise. Muscle blood flow should remain relatively constant during exercise if $\dot{Q}$ is constant (84). Furthermore, if blood flow did increase to a non-compliant region like skeletal muscle, it
would not alter central venous pressure because despite the increased flow, the volume of blood in the region would change little because of the stiffness of the vasculature there (103). Evidence that blood is not shunted to other non-compliant beds is provided by studies that have shown decreased renal (44) and hepatic (83) blood flows with increased heart rate (which is representative of CV drift occurring over time during prolonged exercise).

Contemporary Hypothesis. Opponents (13, 26) of Rowell’s hypothesis have argued that no measures of ventricular filling (central venous pressure or central blood volume) were obtained in the studies (20-23) Rowell uses to support his theory regarding causes of classic CV drift. Thus, while elimination of venous tone by extremely high skin temperature (38°C) has been shown to result in venous pooling to an extent that can reduce SV (89), Coyle (13) asserts that this is unlikely to occur under the conditions of classic CV drift described by Rowell (84). Furthermore, despite the evidence from the Shaffrath (100) study mentioned above that shows decreased SV during exercise in which forearm blood flow increases, in other studies, forearm blood flow appears to level off after about 20-30 min of moderate-intensity, constant-rate exercise in temperate conditions (27, 55, 72), even though SV continues to decline during this time (27, 72). This apparent dissociation between skin blood flow and SV decline has led Coyle (12, 13) and others (27) to suggest that an increase in HR lowers ventricular filling time (107), and thereby SV, under the conditions of classic CV drift described by Rowell (84).

Fritzsche et al. (27) provided evidence suggesting a strong link between the rise in HR and fall in SV over time during prolonged, moderate-intensity cycling. Under one condition, an 11% rise in HR was accompanied by a 13% decline in SV between 15 and
55 min of exercise. Conversely, β-adrenergic blockade prevented the rise in HR, and thereby the decline in SV, between 15 and 55 min. Skin blood flow, and other variables known to influence SV (which will be described later) were not different between conditions, thus suggesting peripheral displacement of the blood volume was not responsible for the decline in SV (27).

Likewise, Nassis (72) observed a decline in SV over time during treadmill running at 60% \( \dot{V}O_{2\text{max}} \) even though skin blood flow remained stable or even declined slightly over time. HR drifted 15 or more beats/min over the course of the exercise bout, again suggesting that peripheral displacement of blood volume did not cause the reduced SV (72), but instead, reduced ventricular filling time associated with tachycardia could have been responsible.

Other evidence which argues against cutaneous venous pooling as a mechanism causing decreased SV involves exercise in the supine position. Supine exercise diminishes the effect of gravity on distention of capacitance vessels in the peripheral circulation, and in so doing better maintains central blood volume and central venous pressure, which should better maintain SV. Nevertheless, Ekelund (21) found that SV still declined ~11.5% from 10-60 min in individuals who performed cycling exercise while supine. The decline in SV was accompanied by an ~13% increase in HR (21). Ekelund performed a follow-up study (22) with a similar protocol, except exercise was performed in the sitting position. Hemodynamic responses were similar, which led him to conclude that posture was not the main factor affecting the variables characterizing CV drift. No measures of forearm or cutaneous blood flow were taken in these studies, though, so it cannot be concluded that the increased HR was entirely responsible for the
decreased SV. Since rectal temperature increased ~1.2–1.9°C from 10-60 min in these studies (21, 22), it is possible that a reflex increase in skin blood flow contributed to peripheral displacement of central blood volume, which could lower SV and support Rowell’s (84) hypothesis mentioned earlier in this review.

From the available literature, it is difficult to pinpoint a single hypothesis explaining the cause of classic CV drift. In all probability, both the traditional and contemporary hypotheses are feasible explanations under a broad range of circumstances. It is beyond the scope of this review to endorse either hypothesis, but instead, the purpose has been to present what is currently known regarding the mechanisms responsible for CV drift. In addition to these hypotheses, other factors may act independently or interact with the mechanisms described earlier (i.e., peripheral displacement of or decrease in blood volume) to alter CV drift. Hence, literature pertaining to these factors will be reviewed in the following sections.

**Other Factors Influencing Cardiovascular Drift**

**Ambient Temperature.** While several studies have demonstrated a substantial CV drift during exercise in high ambient temperature [> 30°C] (3, 33, 37, 48, 70, 75, 111, 112), only a few of these (33, 75, 111) have directly compared CV drift in hot and cool temperatures in the same subjects. Results from all three of these studies indicate the magnitude of CV drift is greater in hot versus cool temperatures. Only HR was measured in one of these (75), however, so the magnitude of CV drift per se could not be determined, and hydration was not controlled in another of these (33), which confounded the ability to undoubtedly conclude whether it was hydration differences or ambient
temperature differences that influenced the magnitude of CV drift. The results of these studies, nonetheless, suggest CV drift is exacerbated in hot environments.

**Core Body Temperature.** Most likely, the disparities between the magnitude of CV drift observed in hot versus cool ambient temperatures can be linked to differences in core body ($T_c$) and mean skin ($T_{sk}$) temperatures between the two environments. Both Wingo et al. (111) and Gliner et al. (33) found a higher mean body temperature ($T_b$) during exercise in 35°C versus exercise in 22°C (111) and 25°C (33), respectively. High $T_c$ and $T_{sk}$ are known to increase skin blood flow (61, 87) as well as heart rate (96), both of which were described earlier in this review as potential mechanisms causing the reduced SV associated with CV drift. Additionally, Gonzalez-Alonso et al. (37) found that hyperthermia (esophageal temperature, $T_{es} = 39°C$) increased HR and lowered SV to an extent greater than that occurring during a control condition ($T_{es} = 38°C$). Finally, MacDougall et al. (64) induced hyperthermia via a water-perfused suit in subjects running at 70% $\dot{V}O_{2max}$ and observed greater CV drift compared to hypothermic or control conditions. These studies clearly indicate that high body temperature can have a substantial impact on the degree of CV drift.

**Hydration status.** In addition to hyperthermia, dehydration can influence CV drift (25, 30, 37, 47, 69, 70, 73). In fact, the effect appears to be graded in that the degree of CV drift is proportional to the level of dehydration (39, 70). Furthermore, the effects of dehydration and hyperthermia may be additive. In the Gonzalez-Alonso et al. study mentioned at the end of the previous section (37), dehydration of 4% body weight lowered SV and increased HR to the same extent as hyperthermia alone, and when dehydration was superimposed on hyperthermia, the effects on SV and HR were additive.
This, however, was not the case in one study in which hyperthermia and degrees of CV drift were similar between fluid and no fluid conditions despite different levels of dehydration [0.3% versus 2.5%, respectively] (112). This suggests there may be a threshold level of dehydration at which the effects of dehydration and hyperthermia become additive.

*Exercise duration.* A final factor that can influence CV drift is exercise duration (30). All things being equal, exercise of longer duration elicits a greater magnitude of CV drift than exercise of shorter duration. The author is not aware of any study that has systematically determined the minimum exercise duration necessary to elicit CV drift under a given set of conditions, but most reviews assert CV drift can begin to occur after as little as 10 min under conditions of *classic CV drift* (12, 13, 84).

While this review of factors that can influence CV drift is not all-inclusive, the ones presented are those theorized to be most pertinent and influential to CV drift under the conditions of the studies carried out in this dissertation. The main purpose of this project was not to determine the causes of CV drift, but rather to offer further insight into the potential consequences of this phenomenon. Thus, literature regarding these consequences will be reviewed next.

**Potential Consequences of Cardiovascular Drift**

Studies that have focused on the effects of prior exercise on subsequent measurements of $\dot{\text{VO}}_{2\text{max}}$ have suggested that $\dot{\text{VO}}_{2\text{max}}$ is reduced when measured after prior exercise during which CV drift occurs (17, 93, 94, 98), although these studies did not measure the effects of CV drift on $\dot{\text{VO}}_{2\text{max}}$ per se. This is evidenced by the fact that the variables characterizing CV drift (i.e., SV and HR) were not measured at the same
points in time as \(\dot{V}O_{2\text{max}}\). Moreover, \(\dot{V}O_{2\text{max}}\) was measured after a rest, and the way in which CV drift occurred (whether via dehydration, hyperthermia, etc.) was not controlled, thus potentially confounding interpretation of the effects of CV drift on \(\dot{V}O_{2\text{max}}\) (17, 93, 94, 98). For instance, Ekblom (17) had subjects cycle for 1 hr at 75% \(\dot{V}O_{2\text{max}}\) before and after 22 weeks of training. The ambient temperature was not provided, which suggests all exercise testing took place in room temperature and humidity. In the trained state, HR increased 10% and SV decreased \(\sim\)8% over the course of the 1-hr exercise bout. \(\dot{V}O_{2\text{max}}\) was measured after “a few minutes of rest” and then again after another 90-120 min of rest. From interpolation of the data, it appears that \(\dot{V}O_{2\text{max}}\) was reduced \(\sim\)11.5% during both \(\dot{V}O_{2\text{max}}\) tests after the 1-hr submaximal bout. A smaller decrement in \(\dot{V}O_{2\text{max}}\) (5%) was observed by Saltin and Stenberg (93), who had subjects exercise for 180 min at 75% \(\dot{V}O_{2\text{max}}\) in 19°C, followed by 90 min of rest before the measurement of \(\dot{V}O_{2\text{max}}\). They observed an increase in HR of \(\sim\)14% and decline in SV of \(\sim\)9%. The differences in exercise protocols and rest periods probably account for the discrepant results, and these studies illustrate the difficulty in precisely determining the effects of CV drift on \(\dot{V}O_{2\text{max}}\).

Because of the inability to directly assess the effects of CV drift on \(\dot{V}O_{2\text{max}}\) from previous research, further research was warranted. Four investigations have recently studied this. In these four studies, an association was found between CV drift and \(\dot{V}O_{2\text{max}}\) in cool \([\sim 22°C]\) (51, 111), warm \([30°C]\) (30), and hot \([35°C]\) (111, 112) temperatures with \(\sim\)40% relative humidity. Of particular importance is the fact that \(\dot{V}O_{2\text{max}}\) was measured over the same time interval as the variables comprising CV drift (change in HR and SV over time). Exercise duration in the studies employing the hot and cool
environments was 45 min, and CV drift and \( \dot{V}O_{2\text{max}} \) were measured at 15 and 45 min. Greater CV drift and decrements in \( \dot{V}O_{2\text{max}} \) occurred in the studies with the hot (111, 112) versus cool (51, 111) temperature. Exercise duration in the study employing the warm environment (30) was 120 min, and CV drift and \( \dot{V}O_{2\text{max}} \) were measured at 15, 60, and 120 min. Between 15 and 60 min, the magnitude of CV drift was small, and consequently the decline in \( \dot{V}O_{2\text{max}} \) was prevented. Fluid ingestion attenuated the rise in HR and prevented the decline in SV associated with CV drift between 15 and 120 min, so the decline in \( \dot{V}O_{2\text{max}} \) also was prevented (30). These studies illustrate that regardless of the primary means by which CV drift occurs, the effect on \( \dot{V}O_{2\text{max}} \) is similar.

Besides \( \dot{V}O_{2\text{max}} \), three of the four studies mentioned above have indicated CV drift may negatively influence performance (as indicated by maximum power output attainable and duration of \( \dot{V}O_{2\text{max}} \) test) in addition to \( \dot{V}O_{2\text{max}} \) (30, 111, 112). In two of these studies, CV drift was observed between 15 and 45 min of cycling at 60% \( \dot{V}O_{2\text{max}} \) in 35°C with subsequent measurement of \( \dot{V}O_{2\text{max}} \), and maximum power output declined by 11.6% and 15.7%, respectively, while \( \dot{V}O_{2\text{max}} \) test duration declined by ~3.1 min (28%) and ~3.5 min (35%), respectively (111, 112). In the other study, CV drift was observed between 15 and 120 min of cycling at 60% \( \dot{V}O_{2\text{max}} \) in 30°C with no fluid ingestion, followed by measurement of \( \dot{V}O_{2\text{max}} \) (30). Maximum power output declined 12.7% while \( \dot{V}O_{2\text{max}} \) test duration decreased ~3.3 min [27%] (30). While there are more representative and valid measures of performance available (54), these results nonetheless suggest that CV drift has the potential to negatively impact endurance performance.
Maximal Oxygen Uptake

Since \( \dot{V}O_{2\text{max}} \) is a physiological outcome potentially affected by CV drift and since it is the principal outcome measure in the studies comprising this dissertation, it is important to understand its meaning, significance, determinants, and limitations. These areas will be reviewed in the following sections.

**Definition and Significance.** As mentioned under “Definition of Terms,” \( \dot{V}O_{2\text{max}} \) is defined as the highest rate oxygen can be taken up and utilized by body tissues. It represents the upper limit of aerobic metabolism and is a highly reproducible measure (85), varying only 2-4 percent from day to day (82) with a reliability coefficient as high as 0.95 (106). The application and significance of \( \dot{V}O_{2\text{max}} \) can be seen in many recreational, occupational, and athletic settings that require work at a sustained, high rate of metabolism. Having a low \( \dot{V}O_{2\text{max}} \) may result in reduced physical performance and work capacity in these settings (95). While activities performed at a low percentage of \( \dot{V}O_{2\text{max}} \) are less demanding than those performed at a high percentage, a high \( \dot{V}O_{2\text{max}} \) (versus a low \( \dot{V}O_{2\text{max}} \)) permits easier performance of activities requiring a given absolute level of oxygen uptake (\( \dot{V}O_2 \)). Additionally, increases in \( \dot{V}O_{2\text{max}} \) permit greater ease (after training, as compared to before training) in performing an activity at a given absolute level of \( \dot{V}O_2 \) (95). If \( \dot{V}O_{2\text{max}} \) changes transiently during exercise, as has been seen during exercise in which CV drift occurs (30, 111, 112), then each absolute level of \( \dot{V}O_2 \) represents a higher percentage of \( \dot{V}O_{2\text{max}} \). This can have detrimental effects on physical work capacity and exacerbate physiological strain.

**Determinants.** The determinants of \( \dot{V}O_{2\text{max}} \) are the components defined by the Fick principle expressed at maximum:
\[ \dot{V}O_{2\text{max}} = \dot{Q}_{\text{max}} \times (a-v)O_{2\text{max}}, \]

where \( \dot{V}O_{2\text{max}} \) is maximal oxygen uptake, \( \dot{Q}_{\text{max}} \) is maximal cardiac output, and \((a-v)O_{2\text{max}}\) is maximal arteriovenous oxygen difference. While not a determinant itself, the active muscle mass can influence each of these components. Thus, the influence of muscle mass on \( \dot{V}O_{2\text{max}} \), as well as the determinants featured in the Fick equation, will be discussed in the following paragraphs.

A certain percentage of the total muscle mass should be engaged in order for attainment of a true maximum oxygen uptake (5, 85). While this percentage has not been established, it is estimated to be at least 50% (82). The influence of muscle mass on \( \dot{V}O_{2\text{max}} \) can be seen in studies in which uphill treadmill running has been shown to elicit higher \( \dot{V}O_{2\text{max}} \) values than cycling or arm exercise (7, 24, 49, 50, 59, 68, 101). Likewise, Reybrouck et al. (81) and Gleser et al. (32) found subjects who were not accustomed to cycling attained higher \( \dot{V}O_{2\text{max}} \) in cycling with the addition of arm cranking, which suggests the proportion of active muscle mass can influence \( \dot{V}O_{2\text{max}} \). Results in these studies are difficult to interpret, however, because other activities, like skiing and laddermill climbing, which presumably employ greater muscle mass than uphill running, did not elicit greater \( \dot{V}O_{2\text{max}} \) (7, 59). This may be related to the findings of Secher et al. (99) who observed that reduced driving pressure to the arms and increased resistance to flow in the legs can occur in combined arm and leg ergometry compared to that with each muscle group alone. Thus, the \( \dot{V}O_2 \) of combined arm and leg ergometry is not equal to the sum of the \( \dot{V}O_2 \) during arm cranking plus the \( \dot{V}O_2 \) during leg cycling (99).

Subject familiarity with the exercise mode also may influence whether \( \dot{V}O_{2\text{max}} \) measured with different exercise modes varies or not, as long as some critical muscle
mass has been activated. In subjects of high aerobic fitness accustomed to cycling, differences between \( \dot{\text{VO}}_{2\text{max}} \) measured while cycling or running, or while cycling or cycling plus arm cranking, may be only slight or even nonexistent (5, 81). Regardless of these complexities, the active muscle mass has at least some influence on \( \dot{\text{VO}}_{2\text{max}} \) by influencing both of the determinants of \( \dot{\text{VO}}_{2\text{max}} \) defined by the Fick principle, i.e. \( \dot{Q} \) and \( (a-v)\text{O}_2 \).

Cardiac output (\( \dot{Q} \)), the product of heart rate (HR) and stroke volume (SV), is one of two determinants of oxygen uptake. Cardiac output increases approximately linearly with oxygen uptake except when the range is considered up to maximal values, at which point \( \dot{Q} \) may increase less proportionately (4). For this reason, regression equations that have been developed to predict \( \dot{Q} \) from \( \dot{\text{VO}}_2 \) are best utilized at submaximal exercise intensities (4).

As mentioned above, \( \dot{Q} \) is the product of HR and SV. Like \( \dot{Q} \), HR increases linearly with \( \dot{\text{VO}}_2 \) up to maximum values, although in some individuals HR may increase relatively less than \( \dot{\text{VO}}_2 \) at heavy workloads (5). Proper interpretation and reproducibility of the HR–\( \dot{\text{VO}}_2 \) relationship depends on standardized conditions (106). For instance, high ambient temperature and/or prolonged exercise can result in changes in HR disproportionate to changes in \( \dot{\text{VO}}_2 \) (i.e., CV drift) (22, 23, 27, 112), and static exercise may increase the HR higher than expected by the metabolic rate (5). Maximal HR, however, is highly reproducible under a variety of dynamic exercise protocols [with an intraindividual standard deviation of 3 beats/min observed in one study (6)], but higher maximal HR’s may occur under stressful situations like high ambient temperatures (76, 85, 112) or dehydration (85).
The stroke volume relation to \( \dot{V}O_2 \) is less predictable than that of HR. SV increases with \( \dot{V}O_2 \) during dynamic exercise, but it reaches a peak around an intensity of 40% \( \dot{V}O_{2\text{max}} \), above which it no longer increases, or increases only slightly (5, 109). This effect, however, may be absent in highly trained individuals (18, 31). For instance, Gledhill et al. (31) measured SV in highly trained endurance athletes at various workloads leading up to \( \dot{V}O_{2\text{max}} \) and found that SV did not plateau but instead increased all the way up to \( \dot{V}O_{2\text{max}} \). In a similar study, Ekblom and Hermansen (18) found SV did not reach maximum until \( \dot{V}O_{2\text{max}} \) in 9 of 13 highly trained endurance athletes. Evidently, the SV response to graded exercise can be quite variable across individuals.

The final component of the Fick equation and determinant of \( \dot{V}O_{2\text{max}} \) is arteriovenous oxygen difference \( [(a-v)O_2] \). This variable represents the difference in oxygen concentration between the arterial and mixed venous circulations. This difference becomes larger with increasing \( \dot{V}O_2 \), from values at rest around 4.5 mL O\(_2\)/100 mL blood (with approximately 23% of the available oxygen extracted from the total blood volume) to values at \( \dot{V}O_{2\text{max}} \) around 16 or 17 mL O\(_2\)/100 mL blood (with approximately 80-85% of available oxygen extracted) (85).

Limitations. Logically, the determinants of \( \dot{V}O_{2\text{max}} \) have a direct bearing on what limits \( \dot{V}O_{2\text{max}} \). Thus, these determinants are involved in three areas in which \( \dot{V}O_{2\text{max}} \) can be limited: respiratory, metabolic, and cardiovascular responses to exercise. A review of literature pertaining to how these areas may limit \( \dot{V}O_{2\text{max}} \) is presented in the following sections.

Respiratory Limitations. Pulmonary gas exchange may limit \( \dot{V}O_{2\text{max}} \) in some highly trained endurance athletes (79, 80). Some elite athletes experience arterial
hypoxemia in which arterial blood is under-saturated with oxygen, thus limiting oxygen uptake. This is postulated to occur because of 1) the presence of a venoarterial shunt, 2) $V_A/Q$ mismatch, 3) hypoventilation, and 4) inadequate gas exchange between alveoli and pulmonary capillaries, possibly because of short pulmonary capillary transit times associated with high cardiac outputs (16, 80, 90). Some investigators suggest that mean transit time is only reduced at the onset of exercise, with no further reductions thereafter, even up to $VO_{2\text{max}}$, so some other mechanism (like perhaps differences in interregional capillary transit time) could cause an exercise-induced hypoxemia in highly trained athletes (108). Regardless, $VO_{2\text{max}}$ may be reached prematurely in athletes with exercise-induced hypoxemia. Exposure to hyperoxic conditions enables endurance athletes with exercise-induced hypoxemia to experience higher $VO_{2\text{max}}$ than when exposed to normoxic conditions, thus further suggesting that pulmonary gas exchange contributes to the limitation of $VO_{2\text{max}}$ in these individuals (79).

Despite the potential limitations to $VO_{2\text{max}}$ imposed by pulmonary function in highly trained endurance athletes, the respiratory system does not normally limit $VO_{2\text{max}}$ at altitudes below 1200–1500 m in healthy sedentary or active individuals (at higher altitudes the decrease in barometric pressure can lower the alveolar–arterial $O_2$ gradient such that diffusion of oxygen into the blood is compromised) (28, 85, 104). Arterial oxygen tension is maintained at heavy work loads [thus preventing arterial hypoxemia] (67), and ventilation ($V_E$) continues to rise after $VO_{2\text{max}}$ has been attained (5, 6, 77). In addition, it is estimated that respiratory muscles deprive the working muscles of only 7% of the total $VO_2$ at maximal exercise intensities (66). Furthermore, Rowell (85) asserts that increasing the cost of $V_E$ could only alter variables in the Fick equation, and thus
reduce $\dot{V}O_{2\text{max}}$, if oxygen extraction by respiratory muscle was somehow less efficient than that by skeletal muscle, which is unlikely.

Metabolic Limitations. It is unlikely that metabolic limitations, such as mitochondrial uptake and use of oxygen, limit $\dot{V}O_{2\text{max}}$ in healthy individuals (34). When arterial oxygen content is increased via exposure to hyperoxic conditions or via increased hemoglobin content, $\dot{V}O_{2\text{max}}$ is increased, suggesting skeletal muscle has a capacity to extract “excess” oxygen (60, 79). Ekblom et al. (19) had healthy males cycle to $\dot{V}O_{2\text{max}}$ under one condition in which they were exposed to room air and another condition in which they breathed 50% oxygen (hyperoxia). Despite similar maximal cardiac outputs, $\dot{V}O_{2\text{max}}$ was higher in the hyperoxic condition, suggesting that the working muscle was able to utilize the additional oxygen provided by the 50% oxygen air mixture. Skeletal muscles’ capacity to extract and use oxygen provided to it is apparently not at full capacity at $\dot{V}O_{2\text{max}}$.

Cardiovascular Limitations. More than likely, the circulatory delivery of oxygen limits $\dot{V}O_{2\text{max}}$ in most healthy individuals during exercise involving large muscle groups (34, 35, 41, 71, 82, 102). Cardiac output plateaus as $\dot{V}O_{2\text{max}}$ is reached (35, 109) [unless exercise involves a small muscle mass, in which case $\dot{Q}$ does not plateau (81)]. As mentioned above, in individuals untrained or only moderately trained, SV tends to plateau at ~40% $\dot{V}O_{2\text{max}}$ (4, 5), and HR normally peaks at or near work loads that elicit peak oxygen uptake (77, 113). Gonzalez-Alonso et al. (35, 41) and Mortensen et al. (71) eloquently showed a plateau in $\dot{Q}$ and SV at $\dot{V}O_{2\text{max}}$ during cycling exercise, but as the exercise period extended briefly beyond $\dot{V}O_{2\text{max}}$ to exhaustion, HR and (a-\text{a})O$_2$ continued to increase slightly while $\dot{Q}$ and SV decreased. These results strongly support the notion
that a central limitation (i.e., plateaued or reduced \( \dot{Q} \) via reduced SV) is responsible for limiting systemic and skeletal muscle blood flow and thereby \( \dot{V}O_{2\text{max}} \). Furthermore, when restraints on central circulatory capacity (i.e., pericardium impediments to stroke volume) were removed in dogs, \( \dot{V}O_{2\text{max}} \) increased 8%, suggesting that \( \dot{V}O_{2\text{max}} \) was limited by blood flow (105). Studies that do not show an increase in \( \dot{V}O_{2\text{max}} \) (or only a small increase disproportionate to what would be expected) with addition of muscle groups to those already eliciting \( \dot{V}O_{2\text{max}} \) provide further evidence that \( \dot{V}O_{2\text{max}} \) is blood-flow dependent and not dependent on the amount of muscle mass activated once a critical mass has been engaged (7, 59).

Since circulatory transport of oxygen is the probable limitation of \( \dot{V}O_{2\text{max}} \) in most healthy athletes and non-athletes, conditions that compromise circulatory capacity also compromise \( \dot{V}O_{2\text{max}} \). Two such conditions are hyperthermia and dehydration. Just as each one of these can influence the magnitude of CV drift, so each one can also negatively influence \( \dot{V}O_{2\text{max}} \).

Hyperthermia has been shown to reduce \( \dot{V}O_{2\text{max}} \) (2, 35, 76), and the effect appears to be graded in proportion to the extent of thermal strain reflected in high skin and core temperatures (2). In other words, the higher the combination of skin and core temperatures, the greater the decrement in \( \dot{V}O_{2\text{max}} \) (2). The exact mechanism by which \( \dot{V}O_{2\text{max}} \) is reduced during hyperthermia is uncertain, but Gonzalez-Alonso and Calbet (35) assert that heat stress accelerates decreases in \( \dot{Q} \) (via decreased SV) and mean arterial pressure that reduce skeletal muscle blood flow and thereby \( O_2 \) delivery. Maximal HR is unchanged or even higher in the heat (2, 112), so the decrease in \( \dot{V}O_{2\text{max}} \) is most likely because of reduced SV because maximal (a-v)\( O_2 \) is probably not reduced in the heat (88,
Reduced SV may be caused by 1) increased cutaneous vasodilation and peripheral displacement of blood volume such that central blood volume, and thereby ventricular filling/end-diastolic volume, is reduced (88) or 2) tachycardia such that ventricular filling time, and thereby end-diastolic volume, is reduced (27).

It is important to point out that exercise in high ambient temperatures does not limit \( \dot{V}O_2\text{max} \) per se (78). Results are mixed, as some reports have indicated a reduced \( \dot{V}O_2\text{max} \) (63, 87, 97, 106) in acute exposure to hot versus comfortable temperatures, while others have demonstrated no difference (78, 86, 88, 109). Reasons for the discrepancies are uncertain, but may hinge on differences in protocol used and acclimation state of subjects. For instance, Taylor et al. (106) found that \( \dot{V}O_2\text{max} \) was reduced 4% in a 90°F environment compared to a 78°F one, but Williams et al. (109) found no difference in \( \dot{V}O_2\text{max} \) measured in 97°F versus 70°F. The unacclimated subjects in the Taylor et al. study (106) were exposed to the heat for as much as 60 min before measurement of \( \dot{V}O_2\text{max} \) (thus allowing time for core temperature to rise), whereas the subjects in the Williams et al. study (109) were acclimated and only acutely exposed to heat before measurement of \( \dot{V}O_2\text{max} \). During acute heat exposure, as in the Williams et al. study (109), vasoconstriction of skin at maximal exercise intensities enabled maintenance of blood flow to working muscle such that \( \dot{V}O_2\text{max} \) was not compromised. Conversely, if duration of heat exposure (either active or passive) is long enough to elevate skin and core temperatures, \( \dot{V}O_2\text{max} \) is reduced (2, 10, 78) because apparently \( \dot{Q} \) is not sufficient to both adequately dissipate heat (via skin blood flow) and perfuse working skeletal muscle.

Dehydration also may negatively affect \( \dot{V}O_2\text{max} \) because of decreases in active muscle blood flow and \( \dot{Q} \) (36), but the effects appear to be mild (11, 76) or negligible (91,
92), unless the dehydration is diuretically-induced (15) or associated with CV drift during prolonged exercise (30), in which cases the reduction in \( \dot{V}O_{2max} \) can be substantial (9–11%). Moreover, when dehydration is combined with hyperthermia, the effects are additive (36) and \( \dot{V}O_{2max} \) is reduced (14, 76), although most of the decline is associated with hyperthermia (76).

In summary, \( \dot{V}O_{2max} \) is a physiological phenomenon with two determinants [\( \dot{Q} \) and (a-v-)O\(_2\)] and several potential limitations. In most healthy individuals, \( \dot{V}O_{2max} \) is limited primarily by the capacity of the circulatory system to deliver oxygen to working skeletal muscle. This capacity is compromised in conditions of hyperthermia and (possibly) dehydration, which are known to be associated with CV drift. Thus, attenuating hyperthermia, and thereby CV drift, is important for maintenance of \( \dot{V}O_{2max} \).

Body cooling during exercise has been shown to attenuate CV drift (43, 100), which might influence alterations in \( \dot{V}O_{2max} \). Therefore, literature regarding body cooling will be reviewed in the final section of this chapter.

**Body Cooling During Exercise**

Many studies investigating the ergogenic effects of body cooling have focused on precooling (1, 40, 110) or exercise in cool ambient temperatures (29). Regardless of the cooling protocol, the beneficial effect is a reduction in cardiovascular and thermal strain. Despite these benefits, two problems exist with the cooling methods mentioned above: 1) the effects of some precooling protocols are short-lived (1, 9, 46, 65), and 2) exercise in cool ambient temperatures is not always feasible. Therefore, cooling during exercise has the potential to be more effective in attenuating CV drift and thereby preserving \( \dot{V}O_{2max} \).
Research investigating the effects of body cooling on CV drift during prolonged exercise is limited, and results are mixed. Hsu et al. (53) found that HR responses during 1 hr of cycling at 60% $\dot{V}O_2\text{peak}$ in ~32°C were the same with cooling or no cooling, but other studies have shown positive effects (43, 48, 64, 100). HR rose less during treadmill walking at 5.63 km/h at a grade 60-65% of that known to elicit 90% maximum HR in 40°C when individuals used a cooling device compared to when they did not (43). Similarly, subjects cycling at 60% $\dot{V}O_2\text{max}$ in 32°C, 70-80% RH for 60 min had lower heart rates throughout exercise while wearing a cooling jacket than during a control trial without the cooling jacket (48). Subjects wearing a water-perfused suit while running at 70% $\dot{V}O_2\text{max}$ experienced lower heart rates when the suit was perfused with water at 18°C and fan airflow was directed on them (hypothermic condition) as compared to when they completed the same exercise with no water perfused through the suit and with no fan airflow (control condition) or with water equal to rectal temperature perfused through the suit [hyperthermic condition] (64). Additionally, the decline in SV was more gradual in the hypothermic compared to the control and hyperthermic conditions (64). Use of fan airflow also attenuated CV drift in a study by Shaffrath (100) in which the increase in HR and decrease in SV over time during cycling exercise performed in ~24°C at 60% $\dot{V}O_2\text{max}$ was less when the exercise was performed in front of a fan generating airflow at 4.3 m/s compared to when the exercise was performed with no fan airflow. Greater effects would be expected if the ambient temperature was higher, like that in the studies comprising this dissertation. Thus, since body cooling can attenuate CV drift, it has the potential to also attenuate the decrease in $\dot{V}O_2\text{max}$ concomitant with CV drift during prolonged exercise in hot conditions.
Summary

The causes of cardiovascular drift are multifactorial, with two prevailing hypotheses. Peripheral displacement of blood volume associated with hyperthermia may contribute to reduced SV and increased HR, and/or increased HR may reduce ventricular filling time and thereby SV. Regardless of the cause, CV drift can be modified by several factors, including ambient and core body temperatures, hydration status, and exercise duration. The consequences of CV drift are less known than the causes, but include reduced $\dot{V}O_{2\text{max}}$ and potentially reduced performance. Maximal oxygen uptake can be limited by several factors, most probable of which is circulatory capacity and oxygen delivery in healthy individuals. Under conditions in which CV drift occurs prior to measurement of $\dot{V}O_{2\text{max}}$, maximal circulatory capacity may be reached prematurely and/or reductions in SV observed during submaximal exercise may persist during maximal exercise such that $\dot{V}O_{2\text{max}}$ is reduced. Cooling modalities administered during exercise can reduce the magnitude of CV drift and thereby possibly attenuate a decrement in $\dot{V}O_{2\text{max}}$. 
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CHAPTER 3

CARDIOVASCULAR DRIFT IS RELATED TO REDUCED MAXIMAL OXYGEN UPTAKE DURING HEAT STRESS\(^1\)

Abstract

Introduction/Purpose: This study investigated whether the progressive rise in heart rate (HR) and fall in stroke volume (SV) during prolonged, constant-rate, moderate-intensity exercise (cardiovascular drift, CV drift) in a hot environment is associated with a reduction in $\dot{V}O_{2\text{max}}$. Methods: CV drift was measured in nine male cyclists between 15 and 45 min of cycling at 60% $\dot{V}O_{2\text{max}}$ in 35°C that was immediately followed by measurement of $\dot{V}O_{2\text{max}}$. $\dot{V}O_{2\text{max}}$ also was measured after 15 min of cycling on a separate day, so that any change in $\dot{V}O_{2\text{max}}$ between 15 and 45 min could be associated with the CV drift that occurred during that time interval. This protocol was performed under one condition in which fluid was ingested and there was no significant body weight change (0.3±0.4%) and under another in which no fluid was ingested and dehydration occurred (2.5±1%, p<0.05). Results: Fluid ingestion did not affect CV drift or change in $\dot{V}O_{2\text{max}}$. A 12% increase in HR (151±9 vs. 169±10 beats·min$^{-1}$, p<0.05) and 16% decrease in SV (120±12 vs. 101±10 mL·beat$^{-1}$, p<0.05) between 15 and 45 min was accompanied by a 19% decrease in $\dot{V}O_{2\text{max}}$ (4.4±0.6 vs. 3.6±0.4 L·min$^{-1}$, p<0.05) despite attainment of a higher maximal HR (p<0.05) at 45 min (194±5 bpm) vs. 15 min (191±5 bpm). Submaximal $\dot{V}O_2$ increased only slightly over time, but %$\dot{V}O_{2\text{max}}$ increased from 63±5% at 15 min to 78±8% at 45 min (p<0.05). Conclusion: We conclude CV drift during 45 min of exercise in the heat is associated with decreased $\dot{V}O_{2\text{max}}$ and increased relative metabolic intensity. The results support the validity of using changes in HR to reflect changes in relative metabolic intensity during prolonged exercise in a hot environment in which CV drift occurs.
Keywords: Circulation, Heart rate, Oxygen consumption, Stroke volume,
Thermoregulation
Introduction

After about 10 minutes of prolonged, constant-rate, moderate-intensity (50-75% of maximal oxygen uptake, \(\dot{VO}_{2\text{max}}\)) exercise, a slow, progressive change over time, or drift, occurs in several cardiovascular measures. Heart rate (HR) increases progressively; stroke volume (SV), mean arterial and pulmonary pressures decrease progressively; while cardiac output (\(\dot{Q}\)) remains relatively constant (11,25). Cardiovascular drift (CV drift) is observed in thermoneutral (11) and warm (20) environments, but greater changes occur in a warm environment (13).

The mechanism underlying CV drift is controversial (6). One hypothesis is that CV drift is caused by peripheral displacement of the blood volume with progressive increases in cutaneous blood flow and venous volume causing the progressive fall in central venous pressure, SV and arterial pressure (11,16,25). An alternate hypothesis is that the progressive increase in HR caused by hyperthermia and increased sympathetic nervous system activity decreases ventricular filling time, end-diastolic volume and SV (6).

Regardless of the cause, the consequence of CV drift for physical work capacity is not fully understood. Studies that have measured \(\dot{VO}_{2\text{max}}\) following prolonged exercise suggest that under thermoneutral conditions, CV drift is associated with a modest (5 – 12%) reduction in \(\dot{VO}_{2\text{max}}\) (10,29,30). However, in these studies, \(\dot{VO}_{2\text{max}}\) was not measured at the same points in time as the variables characterizing CV drift. Thus, it is uncertain whether the altered \(\dot{VO}_{2\text{max}}\) accurately reflected the effect of CV drift. Furthermore, the effect on \(\dot{VO}_{2\text{max}}\) of CV drift that occurs during prolonged exercise in a hot environment is unresolved (13).
Whether CV drift is associated with a reduction in $\dot{V}O_{2\text{max}}$ or not is an important issue, with practical implications for the use of HR for prescription of exercise intensity. Intensity of exercise is commonly prescribed using HR, based on the strong relation between percent of maximal HR or percent of HR reserve (%HRR), and relative metabolic intensity as reflected by %$\dot{V}O_{2\text{max}}$ or percent of oxygen uptake reserve (%$\dot{V}O_2$R) (1). However, the use of HR as an indicator of relative metabolic intensity is based on validation studies that employed short-term exercise of progressively increasing intensity (8,35). Whether change in HR is a valid indicator of change in relative metabolic intensity during more prolonged exercise is uncertain.

The primary purpose of our study was to determine whether CV drift was associated with a reduction in $\dot{V}O_{2\text{max}}$ during prolonged exercise in a hot environment. This was accomplished by measuring CV drift between 15 and 45 min of cycling and then immediately measuring $\dot{V}O_{2\text{max}}$. $\dot{V}O_{2\text{max}}$ also was measured immediately after 15 min of cycling on a separate day so that any change in $\dot{V}O_{2\text{max}}$ between 15 and 45 min could be associated with the CV drift that occurred in the same time interval. Forty-five min of exercise was selected because this is a duration during which considerable CV drift occurs (11) and which is typical of aerobic exercise used for conditioning. To determine the extent to which any relation of CV drift to $\dot{V}O_{2\text{max}}$ was accounted for by dehydration, the protocol above was completed under one condition in which no fluid was ingested (NF) and during a second condition in which fluid was ingested to prevent dehydration (F). Because studies on the effect of fluid ingestion on the magnitude of CV drift when the duration of exercise is less than 1 hr have been equivocal (20,22), we were uncertain whether any effect would be evident. We hypothesized that the increase in HR and
decrease in SV between 15 and 45 min of submaximal exercise would be associated with reduced \( \dot{V}O_{2\text{max}} \) and increased relative metabolic intensity, and that fluid ingestion would reduce the magnitude of CV drift and reduction in \( \dot{V}O_{2\text{max}} \).

**Methods**

**Subjects.** Nine healthy men volunteered as subjects. This sample size is sufficient to detect a five percent decrease in \( \dot{V}O_{2\text{max}} \) using a two-tailed t test for dependent samples at \( \alpha = 0.05 \) and statistical power of 0.8, assuming individuals have a mean \( \dot{V}O_{2\text{max}} \) of 55 mL·kg\(^{-1}\)·min\(^{-1}\) with a SD of 7 mL·kg\(^{-1}\)·min\(^{-1}\) and that the test-retest correlation for \( \dot{V}O_{2\text{max}} \) is 0.95 (18). Subject physical characteristics (means ± SD) were: age = 25 ± 4 yr, mass = 71.8 ± 5.1 kg, height = 179.0 ± 5.0 cm, and percent body fat [using the Jackson-Pollock equations for predicting body density from seven skinfolds in men (15) and the Siri equation to determine percent fat from body density (34)] = 8.1 ± 3.7%. The subjects were trained cyclists averaging 116 ± 98 km·week\(^{-1}\) and runners averaging 34 ± 19 km·week\(^{-1}\), or subjects who trained cycling and running for a combined 6 h·week\(^{-1}\) during the previous 6 mo. 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 design was used in which subjects were tested under all conditions. Following acclimation rides and a control \( \dot{V}O_{2\text{max}} \) test, four experimental trials in which subjects cycled for either 15 or 45 minutes at 60% \( \dot{V}O_{2\text{max}} \) were completed on separate days. Subjects completed two trials with no fluid ingestion (15NF and 45NF) and two trials with fluid ingestion (15F and 45F), followed immediately by measurement of \( \dot{V}O_{2\text{max}} \). For the fluid trials, equal volumes of tap water at 35°C were ingested just prior to beginning exercise and at 10 min (15F), or just prior to
beginning exercise and at 10 min, 25 min, and 35 min (45F), respectively. The volume of water ingested was designed to prevent dehydration and was based on the sweat rate measured during the last acclimation ride plus the estimated additional sweat loss that would occur during the VO_{2max} test portion of the protocol. The order of conditions and trials within each condition was randomized, but the two trials for each condition were performed in succession. All trials occurred in an environmental chamber at 35°C, 40% relative humidity (RH) without fan airflow. Each subject was tested at the same time of day to minimize the effects of circadian variation in HR and core temperature, and trials were separated by one day.

**Protocol and Procedures.** Prior to testing, subjects completed a 4-d protocol designed to acclimate them to the environmental conditions under which the experimental trials were performed and to provide practice with procedures. The protocol consisted of cycling 1 h per day at 60% HRR in an environmental chamber at 35°C, 40% RH without fan airflow. Rides occurred on successive days and at the same time each day. Nude body weight was measured to the nearest 10 g with an electronic scale (A&D Co., Ltd., Tokyo, model FW-150KA1) before and after each ride for determination of sweat rate. During the last two trials, the CO_{2}-rebreathing procedure for measuring cardiac output was practiced. Two days after the fourth acclimation ride, subjects completed a control graded exercise test (GXT) to measure VO_{2max}. Subjects reported to the laboratory following a 3-h fast, but they were well hydrated. 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, subjects completed a 24-h history questionnaire designed 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% RH. Following a warm-up, the GXT began with subjects cycling at 200 W, with power output increased 25 W every 2 min until subjects could no longer continue pedaling. \( \dot{V}O_2 \) 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). HR and ratings of perceived exertion (RPE) were measured each minute. HR was measured with a Polar® Vantage XL heart rate monitor (Polar Electro, Inc. Woodbury, NY, model 145900). RPE was measured by the Borg 6-20 scale using standardized instructions (4). Three minutes after completion of the test, a finger-stick blood sample was obtained for determination of blood lactate. Blood lactate concentration was measured using a YSI 2300 Stat Plus Analyzer (Yellow Springs Instruments, Inc., Yellow Springs, OH).

To ensure that a plateau in \( \dot{V}O_2 \) was attained, subjects completed an additional bout of cycling following 20 min of rest. Subjects cycled to exhaustion at a power output 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 power output 25 W higher than the last workload performed during the graded test (if ≥ 1 min was completed during the last stage of the graded test).

Attainment of \( \dot{V}O_{2\text{max}} \) (average of the two highest consecutive 30-s values) was determined by attainment of a plateau as evidenced by an increase in \( \dot{V}O_2 \) between the last two stages of < 135 mL·min\(^{-1}\), which is half the expected increase in \( \dot{V}O_2 \) of 270 mL·min\(^{-1}\) based on the American College of Sports Medicine metabolic equation (1):

\[
\dot{V}O_2 = (10.8 \cdot W \cdot M^{-1}) + 7,
\]
where \( \dot{V}O_2 \) is gross oxygen consumption in \( \text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \), \( W \) is power in watts, and \( M \) is body mass in kg. Using this protocol, all subjects demonstrated a plateau in \( \dot{V}O_2_{\text{max}} \), either during the GXT (3 subjects) or during the subsequent bout (6 subjects).

For the experimental trials, subjects arrived at the laboratory after following the same pre-test instructions provided for the control GXT. Adherence to instructions was verified using the 24-h history questionnaire. Tympanic temperature (\( T_t \)) and urine specific gravity (USG) were measured to verify subjects did not have a fever (\( T_t < 37.8^\circ\text{C} \)) and were adequately hydrated (USG < 1.030). Subjects then measured their nude body weight and inserted a rectal temperature probe. Next, a Teflon® venous catheter was inserted into an antecubital vein and was kept patent with 0.5 mL of 10 USP units·mL\(^{-1}\) heparin lock flush. Subjects then sat upright for 20 min in order for plasma volume to stabilize while skin temperature probes were attached, resting HR was measured, and blood samples were drawn. Subjects then began cycling on the Lode ergometer at a power output estimated to elicit 60% \( \dot{V}O_2_{\text{max}} \). In 45F and 45NF, metabolic, cardiovascular and perceptual measures were taken between min 8 and min 15 and between min 38 and 45. Systolic (SBP) and fourth-phase diastolic (DBP) blood pressures were measured by auscultation, RPE was obtained, blood samples were drawn, expired air was analyzed for 2 min for measurement of \( \dot{V}O_2 \) and \( \dot{V}CO_2 \) using the Parvo Medics system, HR was measured using the Polar® HR monitor, and two trials of CO\(_2\)-rebreathing were performed for measurement of \( \dot{Q} \), in that order.

At the end of either 15 or 45 min of cycling during all four experimental trials, subjects immediately began a GXT with no cessation of cycling. Power output was initially increased 25 W above the power output maintained during the submaximal
exercise (60% VO_{2max}), with additional 25-W increases in power output every 2 min until the subject could not continue. VO_{2} and other metabolic measures were measured over 30-s intervals, HR was measured each min, and RPE was obtained every 2 min and at the point of exhaustion. Blood samples were drawn 3 min following the termination of the test and nude body weight was re-measured. The measure of VO_{2max} on the experimental trials was considered valid if 1) the plateauing criterion used for the control test was met (4 subjects for 45F, 4 for 15 NF and 3 for 45 NF) or 2) if a HR within 5 bpm of that on the control test was obtained (remainder of tests).

\dot{Q} was measured using the indirect-Fick CO_{2}-rebreathing method, as described by Jones (17), using the Parvo Medics metabolic system and software. This involved measuring the \dot{V}CO_{2}, end-tidal CO_{2} concentration, and the equilibrium CO_{2} concentration following rebreathing in succession. Two rebreathing trials, separated by approximately one min, were always performed and averaged. The reliability of values from the two trials was high (intraclass 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 \dot{Q}.

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 temperature probes (Ellab, Inc., Arvada, CO, model MHF-18058-A) attached on the back, forearm and thigh. Rectal and skin temperature probes were connected to a temperature data acquisition system (Ellab, Inc., model TM9608 with Eval 2.1 software), which collects and stores temperatures continuously. Mean skin temperature (T_{sk}) was calculated according to the formula of Burton (5):
\[ \bar{T}_{sk} = 0.5 \cdot T_1 + 0.36 \cdot T_2 + 0.14 \cdot T_3, \]

where \( T_1, T_2, \) and \( T_3 \) are back, thigh, and forearm skin temperatures, respectively. Mean body temperature (\( \bar{T}_b \)) was calculated from \( T_{re} \) and \( \bar{T}_{sk} \) with the formula of Baum et al. (3):

\[ \bar{T}_b = 0.87 \cdot T_{re} + 0.13 \cdot \bar{T}_{sk}. \]

Blood samples were drawn into 2- and 4-mL vacutainers containing EDTA. Samples used for catecholamine analysis (4 mL) were immediately refrigerated, centrifuged at 4°C within 1 h and the plasma stored at -80°C until analyzed. The other samples (2 mL) were used to measure hemoglobin in duplicate with a HemoCue B-Hemoglobin photometer and hematocrit in triplicate with the microhematocrit method. Plasma volume (PV) change during cycling relative to pre-exercise rest was estimated from measures of hemoglobin and hematocrit using the Dill-Costill equation (9).

Plasma norepinephrine (NE) and epinephrine (E) were quantified by high performance liquid chromatography with electrochemical detection after alumina extraction of 250 \( \mu \)L plasma. A refrigerated autosampler (Waters 717-plus, Milford, MA) and a pump (Waters 510, Milford, MA) were used in conjunction with a glassy carbon working electrode set at +650 mV and a range of 0.5 nA with respect to an Ag/AgCl pulsed electrochemical detector (Waters 464 pulsed Electrochemical Detector, Milford, MA). A C12, 4 \( \mu \)m, reverse-phase column (Phenomenex Synergi, 4 \( \mu \), Max-RP, 150. 0 \( \times \) 4. 6 mm) was used. Chromatograms were monitored, recorded, and analyzed with Millennium 32 software (Version 3, 1999; Millipore, Milford, MA). Peaks were quantified by height and compared with daily standard lines fitted by regression analysis to a series of 4-5 standards that were analyzed throughout the day. Correlation coefficients for these daily standard lines always exceeded 0.98. The detection limit of the assay was 1 pg per sample.
Standards (Sigma Chemical, St. Louis, MO) were solubilized in mobile phase and stored at -70°C until used (approximately 2 weeks after preparation). The mobile phase was a 0.1 M phosphate buffer with 0.1 M EDTA, 0.25 M octanesulfonic acid, and 4.5% acetonitrile in ultrapure water (18 megohms·cm\(^{-1}\) resistance; pH 3.10). The mobile phase was filtered through a C18 cartridge (Alltech, Deerfield, IL), degassed by helium sparging, and delivered at a flow rate of 1.0 mL·min\(^{-1}\).

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 \(\dot{V}O_2\)\(_{max}\), %HRR and %\(V\)\(_2\)R utilized during submaximal exercise were calculated. %HRR was calculated as: \([\frac{HR_{ex} - HR_{rest}}{HR_{max} - HR_{rest}}]\) \times 100. %\(\dot{V}O_2\)R was calculated as: \([\frac{\dot{V}O_{2ex} - 3.5}{\dot{V}O_{2max} - 3.5}]\) \times 100. Resting HR was measured using the Polar\(^\circ\) HR monitor in the environmental chamber with the subject upright on the cycle ergometer prior to beginning the submaximal exercise.

Statistical Analysis. Statistical analyses were performed using SPSS v. 11 for Windows (SPSS, Inc., Chicago, IL). Data are reported as means ± SD unless specified otherwise. A 2 \times 2 (Condition \times Time) ANOVA with repeated measures on both factors was used to test the significance of mean differences between fluid conditions and between 15 and 45 min in time, and their interaction. Repeated t tests were used to test for simple effects. A one-way ANOVA with repeated measures, with follow-up repeated-measures t-tests and the Bonferroni \(\alpha\) correction was used to compare means from the four experimental tests and the control \(\dot{V}O_2\)\(_{max}\) test. An \(\alpha\) level of 0.05 was used for all significance tests.
Results

_Hydration status and fluid ingestion._ There was no difference (p > 0.05) in mean body mass (71.8 ± 5.1 – 72.2 ± 4.8 kg) or urine specific gravity (1.007 ± 0.006 – 1.012 ± 0.007) prior to the control or experimental exercise tests, suggesting hydration status was similar at the beginning of all tests. Fluid ingested during 15F (700 ± 103 mL) and 45F (1778 ± 249 mL) was successful in preventing dehydration during the bouts of submaximal exercise and the subsequent GXT to measure \( \dot{V}O_{2\text{max}} \). The body mass percent change from rest in 15F and 45F were each -0.3%, which was smaller (p < 0.05) than the body mass percent change from rest in 15NF (-1.4%) and 45NF (-2.5%). Despite this difference in percent change in body mass between fluid and no fluid conditions, two-way ANOVAs indicated that fluid ingestion did not significantly affect CV drift (\( \Delta SV \) or \( \Delta HR \)), change in \( \dot{V}O_{2\text{max}} \) from control, or other measures that affected the tests of our hypotheses. Therefore, data from the two conditions were combined to simplify presentation.

_Responses to submaximal exercise._ Changes in responses between min 15 and min 45 of submaximal exercise are contained in Table 3.1 and Figure 3.1. \( \dot{V}O_{2} \) and blood lactate concentration increased only slightly (p < 0.05) over time, with the relative metabolic intensity averaging 63% of the control \( \dot{V}O_{2\text{max}} \) and blood lactate concentration ~2.5 mmol·L\(^{-1}\). A substantial CV drift occurred as evidenced by an 18 beat·min\(^{-1}\) (12%) increase in HR (p < 0.05) and a 19 mL (16%) decrease in SV (p < 0.05) (Figure 3.1). \( \dot{Q} \) decreased (p < 0.05) by 1 L·min\(^{-1}\) (6%) and \( O_{2} \) pulse decreased (p < 0.05) by 10%. MAP was unchanged, but SVR increased (p < 0.05) by about 5%. PV change from rest increased (p < 0.05) about 2 percentage points. NE increased (p < 0.05) 64% but E did not change significantly (9.8%, p > 0.05). \( T_{re} \), \( T_{sk} \) and \( T_{b} \) increased (p < 0.05) by 1.0, 0.3, and 1.0\(^\circ\)C,
respectively. RPE increased (p < 0.05) by about 2 points.

Table 3.1: Responses during submaximal exercise with data from fluid and no fluid conditions combined (mean ± SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>15-min</th>
<th>45-min</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \dot{V}O_2 ) (L·min(^{-1}))</td>
<td>2.69 ± 0.19</td>
<td>2.74 ± 0.19*</td>
</tr>
<tr>
<td>( \dot{V}O_2 ) (% control ( VO_{2\text{max}} ))</td>
<td>62.7 ± 4.0</td>
<td>63.7 ± 3.9*</td>
</tr>
<tr>
<td>( \dot{Q} ) (L·min(^{-1}))</td>
<td>18.1 ± 1.3</td>
<td>17.1 ± 1.6*</td>
</tr>
<tr>
<td>SV (mL·beat(^{-1}))</td>
<td>120.5 ± 12.2</td>
<td>101.0 ± 10.3*</td>
</tr>
<tr>
<td>HR (beats·min(^{-1}))</td>
<td>151.1 ± 8.5</td>
<td>169.3 ± 9.7*</td>
</tr>
<tr>
<td>( O_2 ) pulse (mL·beat(^{-1}))</td>
<td>17.9 ± 1.9</td>
<td>16.2 ± 1.7*</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>100.3 ± 8.1</td>
<td>100.3 ± 10.5</td>
</tr>
<tr>
<td>SVR (dyn·cm·s(^{-5}))</td>
<td>5.6 ± 0.6</td>
<td>5.9 ± 0.9*</td>
</tr>
<tr>
<td>Blood lactate (mmol·L(^{-1}))</td>
<td>2.4 ± 1.0</td>
<td>2.6 ± 1.1*</td>
</tr>
<tr>
<td>( \Delta PV ) from rest (%)</td>
<td>-6.1 ± 2.1</td>
<td>-8.2 ± 3.2*</td>
</tr>
<tr>
<td>Epinephrine (ng·mL(^{-1}))</td>
<td>6.1 ± 3.4</td>
<td>6.7 ± 3.8</td>
</tr>
<tr>
<td>Norepinephrine (ng·mL(^{-1}))</td>
<td>1.4 ± 0.9</td>
<td>2.3 ± 0.7*</td>
</tr>
<tr>
<td>( T_r ) (°C)</td>
<td>37.9 ± 0.3</td>
<td>38.9 ± 0.3*</td>
</tr>
<tr>
<td>( T_{sk} ) (°C)</td>
<td>35.9 ± 0.4</td>
<td>36.2 ± 0.5*</td>
</tr>
<tr>
<td>( T_b ) (°C)</td>
<td>37.6 ± 0.3</td>
<td>38.6 ± 0.5*</td>
</tr>
<tr>
<td>RPE</td>
<td>12.2 ± 0.5</td>
<td>14.3 ± 1.6*</td>
</tr>
</tbody>
</table>

\( \dot{V}O_2 \) = oxygen uptake, \( \dot{Q} \) = cardiac output, SV = stroke volume, HR = heart rate, MAP = mean arterial pressure, SVR = systemic vascular resistance, \( T_r \) = rectal temperature, \( T_{sk} \) = mean skin temperature, \( T_b \) = mean body temperature, RPE = rating of perceived exertion, * Significantly different from 15-min value at p < 0.05.
Figure 3.1: Changes in mean (±SEM) heart rate (HR) and stroke volume (SV) between 15 and 45 min of cycling at ~63% of the control $\dot{V}O_{2max}$. * Change significant at p < 0.05.

Responses to maximal exercise. Data on $\dot{V}O_{2max}$ and related measures from the control and experimental tests are presented in Table 3.2. $\dot{V}O_{2max}$, HR$_{max}$, $O_2$ pulse and RPE measured after 15 min of submaximal exercise were not different (p > 0.05) from the control test, although these values were achieved at lower power output and in less time as well as being accompanied by lower (p < 0.05) levels of $\dot{V}_E$, RER, and blood lactate. $\dot{V}O_{2max}$ measured after 45 min of submaximal exercise was decreased (p < 0.05) by 19% compared to the tests following 15 min of submaximal exercise. A similar pattern was observed for maximal power output, test duration, $\dot{V}_E$, $O_2$ pulse, blood lactate, and NE. There were no differences in maximal RER, ΔPV, RPE, and $T_{sk}$ between the tests following 15 and 45 minutes of submaximal exercise. Maximum HR following 45 min of submaximal exercise was higher than control (p < 0.05) and values measured following 15 min of submaximal exercise (p < 0.05), by 3-4 beats·min$^{-1}$. $T_{rc}$ and $T_b$ also were higher by ~1.0°C, and E was higher by 37%.
Table 3.2: Responses to maximal exercise during a control graded exercise test and following 15 min or 45 min of submaximal exercise with data from fluid and no fluid conditions combined.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>15-min</th>
<th>45-min</th>
</tr>
</thead>
<tbody>
<tr>
<td>(\dot{V}_E) (STPD, L·min(^{-1}))</td>
<td>137.2 ± 10.8</td>
<td>129.5 ± 9.6(^{†})</td>
<td>111.8 ± 14.5(^{∗†})</td>
</tr>
<tr>
<td>(\dot{V}O_2) (L·min(^{-1}))</td>
<td>4.3 ± 0.5</td>
<td>4.4 ± 0.6</td>
<td>3.6 ± 0.4(^∗)</td>
</tr>
<tr>
<td>(\dot{V}O_2) (mL·kg(^{-1})·min(^{-1}))</td>
<td>60.4 ± 8.6</td>
<td>61.2 ± 8.5</td>
<td>49.4 ± 6.0(^∗)</td>
</tr>
<tr>
<td>RER</td>
<td>1.14 ± 0.04</td>
<td>1.04 ± 0.03(^{†})</td>
<td>1.03 ± 0.04(^{∗†})</td>
</tr>
<tr>
<td>RPE</td>
<td>19.1 ± 0.8</td>
<td>18.9 ± 1.0</td>
<td>19.0 ± 0.9</td>
</tr>
<tr>
<td>HR (beats·min(^{-1}))</td>
<td>189 ± 5.7</td>
<td>191 ± 5</td>
<td>194 ± 5(^∗)</td>
</tr>
<tr>
<td>(O_2) pulse (mL·beat(^{-1}))</td>
<td>22.9 ± 2.8</td>
<td>23.1 ± 2.9</td>
<td>18.4 ± 2.1(^∗)</td>
</tr>
<tr>
<td>Blood lactate (mmol·L(^{-1}))</td>
<td>8.5 ± 1.4</td>
<td>7.0 ± 1.0(^{†})</td>
<td>5.2 ± 1.7(^{∗†})</td>
</tr>
<tr>
<td>ΔPV from rest (%)</td>
<td>—</td>
<td>-13.7 ± 3.3</td>
<td>-11.8 ± 3.1</td>
</tr>
<tr>
<td>Epinephrine (ng·mL(^{-1}))</td>
<td>—</td>
<td>5.7 ± 3.8</td>
<td>7.8 ± 3.7(^∗)</td>
</tr>
<tr>
<td>Norepinephrine (ng·mL(^{-1}))</td>
<td>—</td>
<td>5.1 ± 1.8</td>
<td>4.3 ± 1.1(^∗)</td>
</tr>
<tr>
<td>(T_{re}) (°C)</td>
<td>—</td>
<td>38.2 ± 0.4</td>
<td>39.2 ± 0.3(^∗)</td>
</tr>
<tr>
<td>(\bar{T}_{sk}) (°C)</td>
<td>—</td>
<td>36.0 ± 0.7</td>
<td>36.1 ± 0.8</td>
</tr>
<tr>
<td>(\bar{T}_b) (°C)</td>
<td>—</td>
<td>37.9 ± 0.4</td>
<td>38.8 ± 0.3(^∗)</td>
</tr>
<tr>
<td>Δ Body Mass (%)</td>
<td>—</td>
<td>-0.9 ± 0.7</td>
<td>-1.4 ± 1.3(^∗)</td>
</tr>
<tr>
<td>Test Duration (min)</td>
<td>11.9 ± 3.5</td>
<td>10.0 ± 2.0</td>
<td>6.5 ± 2.3(^{†,#})</td>
</tr>
<tr>
<td>Power Output (watts)</td>
<td>333 ± 45</td>
<td>300 ± 44(^{†,#})</td>
<td>253 ± 41(^{∗,#})</td>
</tr>
</tbody>
</table>

\(\dot{V}_E\) = minute ventilation, \(\dot{V}O_{2\max}\) = maximal oxygen uptake, RER = respiratory exchange ratio, RPE = rating of perceived exertion, HR = heart rate, ΔPV = plasma volume change from rest, \(T_{re}\) = rectal temperature, \(\bar{T}_{sk}\) = mean skin temperature, \(\bar{T}_b\) = mean body temperature, \(^∗\) Significantly different from 15-min value at \(p < 0.05\). \(^{†}\) Significantly different from control value at \(p < 0.05\).

*Measures of exercise intensity.* Data on measures of exercise intensity based on the \(\dot{V}O_{2\max}\) values obtained during the control graded exercise test (GXT) and at the respective time points are provided in Table 3.3. Because \(\dot{V}O_{2\max}\) was reduced after 45 min of submaximal exercise, \(\%\dot{V}O_{2\max}\) increased accordingly. Likewise, the drift in HR resulted in a higher \(\%HR_{\max}\) at 45 min compared with 15 min. The change in \(\%\dot{V}O_2R\) over time
(16.5 ± 9.1) was greater than the change in %HRR (13.0 ± 5.2) over time, but 45-min values were similar. Figure 3.2 shows that the individual changes in %HRR and %\(\hat{V}O_2R\) were significantly related. The regression coefficient indicated each 1 unit increase in %HRR was accompanied by a 1.24 unit increase in %\(\hat{V}O_2R\).

**Table 3.3:** Measures of exercise intensity during submaximal exercise based on maximal values obtained during the control graded exercise test (GXT) and during the GXTs following each experimental trial.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Maximal values based on control GXT</th>
<th>Maximal values based on GXTs following experimental trials</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>15-min</td>
<td>45-min*</td>
</tr>
<tr>
<td>% (\hat{V}O_2)max</td>
<td>62.7 ± 4.0</td>
<td>63.7 ± 3.9*</td>
</tr>
<tr>
<td>% HR(_{\text{max}})</td>
<td>80.0 ± 3.7</td>
<td>89.6 ± 3.9*</td>
</tr>
<tr>
<td>% (\hat{V}O_2)R</td>
<td>60.2 ± 4.1</td>
<td>61.2 ± 3.8</td>
</tr>
<tr>
<td>% HRR</td>
<td>69.0 ± 5.5</td>
<td>84.0 ± 6.0*</td>
</tr>
</tbody>
</table>

\(\hat{V}O_2\)max = maximal oxygen uptake, HR\(_{\text{max}}\) = maximal heart rate, %\(\hat{V}O_2\)R = percent of maximal oxygen uptake reserve, %HRR = percent of maximal heart rate reserve. * Significantly different from 15-min value at p < 0.05.
Figure 3.2: Relation of change in percent oxygen uptake reserve (%\(\dot{V}O_2R\)) and change in percent heart rate reserve (%HRR) [\(\%\Delta\dot{V}O_2R = 1.24 \Delta\%HRR + 0.46, r = 0.71, \text{SEE} = 6.7\%, p < 0.05\)] between 15 and 45 min of cycling at \(~63\%\) of the control \(\dot{V}O_2\text{max}\).

Discussion

Cardiovascular drift is a well established phenomenon (6,25). However, it is not clear whether the progressive increase in HR and decrease in SV over time are benign with little implication for performance, or whether they reflect altered \(\dot{V}O_2\text{max}\) and thus have implications for performance and exercise prescription. Our primary purpose was to determine whether or not CV drift is associated with a change in \(\dot{V}O_2\text{max}\), and to determine the implications for prescription of exercise intensity for people exercising to develop aerobic fitness. A limitation of the study was that subjects were a homogeneous population of young, fit males who were tested under specific environmental conditions. Thus, applying the current findings to a population and/or environment different from the one employed in this study warrants caution.

The main finding of this study is that CV drift, as reflected by the fall in SV and rise in HR over time, is accompanied by a decrease in \(\dot{V}O_2\text{max}\) during 45 min of submaximal,
constant-rate exercise in a hot environment. Because submaximal $\dot{V}O_2$ increased only slightly, the decrease in $\dot{V}O_{2\text{max}}$ increased the relative metabolic intensity during submaximal cycling. The data provide support for the validity of using changes in HR to reflect changes in relative metabolic intensity during prolonged exercise in a hot environment in which CV drift occurs.

We designed a protocol that previous studies (11,21,33) have shown should cause considerable CV drift using an intensity and duration of exercise typically utilized for conditioning. In accordance with our expectation, considerable CV drift occurred; HR increased 18 beats·min$^{-1}$ (12%) and SV decreased 19 mL (16%) between 15 and 45 min of exercise. In comparison to other studies measuring CV drift under similar ambient temperatures, the magnitude of CV drift that occurred was greater in the current study; however, these studies employed the use of a fan (19,20), which would presumably lower the amount of thermal strain and accompanying CV drift. A study by Fritzsche et al. (12) utilized a lower ambient temperature (27°C), but changes in HR (11% increase) and SV (13% decrease) were similar to those of the current study.

Because studies on the effect of fluid ingestion on the magnitude of CV drift when the duration of exercise is less than 1 hr have been equivocal (20,22), we wanted to determine whether or not dehydration might account for any change in CV drift under the conditions of our study. Dehydration by 2.5% during the 45 min of submaximal exercise combined with the subsequent $\dot{V}O_{2\text{max}}$ test in NF had no effect on the primary measures of CV drift, namely HR and SV, or on the change in $\dot{V}O_{2\text{max}}$ between 15 and 45 min, compared to the condition in which dehydration was prevented. The lack of a differential effect on $\dot{V}O_{2\text{max}}$ is not surprising in light of the work of Nybo et al. (23). In that study,
investigators determined that marked skin and core body hyperthermia alone or combined with dehydration by 4% was associated with a 16% reduction in \( \dot{V}O_{2\text{max}} \). Since \( T_{re} \) was similar between F and NF conditions in the present study, \( \dot{V}O_{2\text{max}} \) was equally affected. Furthermore, Montain et al. (20) demonstrated under similar conditions but with fan cooling that differences in \( T_{re} \) between F and NF conditions did not manifest until 80 min into exercise. Thus, despite no fan cooling (which would presumably cause differences in \( T_{re} \) between fluid conditions to manifest sooner) in the current study, it is possible that the 45 min duration of exercise was insufficient to elicit the consequences of dehydration necessary to differentially influence the magnitude of CV drift or reduction in \( \dot{V}O_{2\text{max}} \).

As mentioned, the lack of an effect of dehydration on \( \dot{V}O_{2\text{max}} \) is not surprising based on the findings of Nybo et al. (23). However, these findings were contrary to that of Craig and Cummings (7), who found that dehydration by 1.9% and 4.3% caused by sweating at rest for 6 hr in the heat with and without fluid ingestion reduced \( \dot{V}O_{2\text{max}} \) measured in 46°C heat by 10% and 27%, respectively. Differences in heat stress and dehydration protocols probably account for the discrepant results. Additionally, \( \dot{V}O_{2\text{max}} \) measured in more temperate environments such as ours is not reduced with dehydration of less than 3% body weight (31).

Our primary finding was that \( \dot{V}O_{2\text{max}} \) was reduced 19% when measured following 45 min, compared to 15 min, of submaximal cycling in the heat during which substantial CV drift occurred. The reduction following 45 min was not the result of lack of effort or failure to achieve \( \dot{V}O_{2\text{max}} \), or a \( \dot{V}O_{2\text{peak}} \) for these conditions. Individual subjects attained a \( HR_{\text{max}} \) within 5 bpm of the control \( HR_{\text{max}} \) (189 ± 6 bpm) on all experimental tests. Furthermore, mean \( HR_{\text{max}} \) during the experimental tests was not different from control
HR_{max} for the tests following 15 min (191± 5 bpm), and was significantly greater than control HR_{max} in the tests following 45 min (194 ± 5 bpm) of submaximal exercise. The same or higher HR_{max} during the experimental trials strongly suggests cardiovascular capacity had been attained. Mean RPE at the point of exhaustion during the graded exercise tests was near 19, indicating a similar, near-maximal effort was given under all conditions. Blood lactate accumulation, ventilation and RER were lower in the experimental tests than control values, but this more likely reflects reduced absolute power output at VO_{2max} rather than lack of effort.

A limitation to the current design is that there was no baseline condition without CV drift present. Thus, ascertaining whether CV drift caused the reduction in VO_{2max} is not possible from these findings. However, the markedly lower VO_{2max} following 45 min compared to 15 min of submaximal exercise indicates that cardiovascular alterations or some other changes that develop over time are at least associated with reduced VO_{2max}. Although we are not the first to report reduced VO_{2max} following prolonged exercise, we are the first that we are aware of to show that VO_{2max} is reduced over the same time interval that CV drift occurs. The change in VO_{2max} in our study was greater than that in several previous studies in which decreases in VO_{2max} of 5-12% following prolonged strenuous exercise in thermoneutral conditions were reported (10,29,30). However, in these studies, VO_{2max} was not measured at the same points in time as the variables characterizing CV drift. The initial VO_{2max} was measured on a different day and the VO_{2max} after exercise was usually measured following a rest period. A period of rest would alter some of the conditions that appear to contribute to CV drift, such as hyperthermia. Thus, it is uncertain whether the altered VO_{2max} accurately reflected the effect of CV drift. In addition, during
prolonged, continuous exercise in a hot environment, in which there is greater hyperthermia and CV drift (13) than in a thermoneutral environment, the metabolic consequences of cardiovascular drift may be greater. The greater change in \( \dot{V}O_2_{\text{max}} \) following prolonged exercise in our study than in previous studies in thermoneutral conditions may reflect the effects of greater heat stress and hyperthermia.

Greater hyperthermia at \( \dot{V}O_2_{\text{max}} \) following 45 min compared to 15 min of submaximal exercise could have contributed to the reduction in \( \dot{V}O_2_{\text{max}} \). \( T_{\text{re}} \) and \( T_b \) at \( \dot{V}O_2_{\text{max}} \) were higher by \( \sim 1^\circ \text{C} \), and \( T_{sk} \) was higher by 0.3 \( \circ \text{C} \), following 45 compared to 15 min of submaximal exercise. The 19% reduction in \( \dot{V}O_2_{\text{max}} \) following 45 min of exercise in the heat in our study is consistent with the results of other studies in which large (16-27%) decreases in \( \dot{V}O_2_{\text{max}} \) have been observed following prior exercise in severe heat stress that markedly elevated both core and skin temperatures (2,23,24). Likewise, the lack of reduction in \( \dot{V}O_2_{\text{max}} \) following 15 min of submaximal exercise at 35 \( \circ \text{C} \) in which the increase in \( T_{\text{re}} \) was much smaller than following 45 min is consistent with studies that have found only small (3-8%) (24,27,32) or no change (26,36) in \( \dot{V}O_2_{\text{max}} \) after brief heat exposure without prior exercise or preheating and with only modest increases in core body temperature.

The cardiovascular and/or metabolic alterations through which \( \dot{V}O_2_{\text{max}} \) was reduced following 45 min compared to 15 min of submaximal exercise in the heat cannot be determined from the data collected in this study. \( HR_{\text{max}} \) was not reduced and, in fact, was significantly higher by an average of 3 beats·min\(^{-1}\). Therefore, the decrease in \( \dot{V}O_2_{\text{max}} \) following 45 min was due to lower arteriovenous oxygen difference [(a-\( \bar{v} \))\( O_2 \)] or SV, as reflected by the reduction in \( O_2 \) pulse. Assuming the (a-\( \bar{v} \))\( O_2 \) diff was not reduced
(14,28,36), the lower O\textsubscript{2} pulse accompanying reduced \(\dot{V}O_{2\text{max}}\) would reflect reduced maximal SV and \(\dot{Q}\). This deduction is consistent with the findings of González-Alonso and Calbet (14) who found the reduction in \(\dot{V}O_{2\text{max}}\) with heat stress was due to a reduction in MAP, SV and \(\dot{Q}\). Lower \(\dot{Q}_{\text{max}}\) was associated with lower leg blood flow and reduced oxygen delivery to the active muscles, limiting \(\dot{V}O_{2\text{max}}\). The cause of the reduced SV was uncertain. Reduced SV could be caused by 1) greater cutaneous vasodilation and increased venous volume, which reduces central blood volume, ventricular filling pressure and end-diastolic volume (28); 2) a slightly greater degree of dehydration and reduced plasma volume, which would contribute to lowered filling pressure and end-diastolic volume; and, 3) slightly greater tachycardia, which would reduce ventricular filling time and may contribute to reduced end-diastolic volume (12).

Our data do not provide new insight into the mechanism underlying CV drift, but suggest that if exercise intensity is increased up to the intensity eliciting HR\textsubscript{max} after CV drift has occurred, the reduced SV present at the end of 45 min of submaximal exercise in the heat apparently cannot be overcome and restored to the control level. This could mean 1) that factors other than increased HR contributed to the reduction in SV during prolonged exercise and that these factors or conditions persisted during maximal exercise or 2) that as intensity was increased to \(\dot{V}O_{2\text{max}}\), the expected increases in myocardial contractility and vasoconstriction in the splanchnic, non-active muscle and skin vascular beds that maintain or increase SV were inadequate to increase the reduced SV to the control level.

Our data have implications for the use of HR to prescribe exercise intensity. This study is the first that we are aware of to evaluate whether the strong link between HR and relative metabolic intensity, demonstrated during short-term exercise (8,35), persists
during prolonged, constant-rate exercise during which submaximal \( \dot{V}O_2 \) remains nearly constant, but HR rises progressively, over time. We found that the increase in HR did reflect an increase in relative metabolic intensity, because \( \dot{V}O_2_{\text{max}} \) decreased over time. While not exactly proportional, the changes in %HRR and %\( \dot{V}O_2 \)R were quite similar (Table 3.3 and Figure 3.2). Thus, these data provide support for the practice of using HR as a marker of change in relative metabolic intensity during prolonged exercise in the heat in which a progressive rise in HR occurs over time.

We conclude CV drift during 45 min of continuous exercise in a hot environment is associated with decreased \( \dot{V}O_2_{\text{max}} \) and increased relative metabolic intensity. The results support the validity of using changes in HR to reflect changes in relative metabolic intensity under conditions like those imposed in this study in which CV drift occurs.
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CHAPTER 4

MAXIMAL OXYGEN UPTAKE AFTER ATTENUATION OF
CARDIOVASCULAR DRIFT DURING HEAT STRESS\(^1\)

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Abstract

Introduction: Exercise intensity is often regulated in hot conditions by maintaining constant target heart rate (HR) to counteract increased physiological strain and thereby avoid premature fatigue. It is unknown, however, whether the HR–percent maximal oxygen uptake (%VO$_{2\text{max}}$) relationship is maintained during prolonged exercise in the heat when the rise in HR concomitant with cardiovascular drift (CV drift) is eliminated by lowering exercise intensity. The purpose of this study was to determine if VO$_{2\text{max}}$ is reduced when exercise intensity and absolute VO$_{2}$ are lowered by a magnitude sufficient to reduce CV drift and maintain constant HR during prolonged exercise in the heat, and thereby examine if the HR–%VO$_{2\text{max}}$ relationship is preserved. Methods: Seven men cycled at 60% VO$_{2\text{max}}$ in 35°C for 15 min (1 trial) and 45 min (2 trials) while HR rose over time (HRvar) or remained constant (HRcon). VO$_{2\text{max}}$ was measured immediately after the 15 and 45 min trials to correspond with the same time interval in which CV drift occurred. Results: Power output decreased 37%, VO$_{2}$ decreased 24%, and VO$_{2\text{max}}$ decreased 7.5% from 15 to 45 min in HRcon, while HR remained the same. In HRvar, HR increased 13%, SV decreased 10%, and VO$_{2\text{max}}$ decreased 15%. Discussion: %VO$_{2\text{max}}$ was decreased from ~60% to 50% to hold HR constant in these conditions, so the HR–%VO$_{2\text{max}}$ relationship was not preserved in the absence of CV drift. Attenuating CV drift by lowering exercise intensity only partially eliminated the reduction in VO$_{2\text{max}}$ after prolonged exercise in the heat.

Key Words: Heart rate, Stroke volume, Circulation, Exercise prescription, Thermoregulation
Introduction

Heart rate (HR) rises disproportionately in relation to oxygen uptake (\(\dot{V}O_2\)) over time during prolonged, constant-rate, moderate-intensity exercise in hot conditions (3, 22). This rise in HR, in combination with other, time-dependent cardiovascular changes, characterizes a phenomenon commonly referred to as cardiovascular drift (CV drift). CV drift can occur in temperate conditions, but the magnitude is greater in hot conditions (14). To compensate for CV drift, prediction equations have been developed to assist in estimating heart rate (HR) at different intensities performed under varying environmental conditions with assorted clothing ensembles (11, 12) for use in individually adjusting exercise prescription target heart rate (THR). Instead of adjusting THR, however, maintaining a constant THR is often recommended as a way to regulate exercise intensity during exercise in the heat in order to compensate for increased physiological strain and thereby avoid heat injury (7, 19). Avoiding CV drift by maintaining a constant HR to maintain THR requires decreasing exercise intensity (speed, power output, etc.) during submaximal exercise, but the impact of this practice on the relative metabolic intensity has not been quantified.

Recent studies by our laboratory have demonstrated that CV drift during 45 min of exercise in a hot environment was associated with a reduction in maximal oxygen uptake (\(\dot{V}O_{2\text{max}}\)), whereas exercise in a cool environment in which CV drift did not occur was not associated with a decrease in \(\dot{V}O_{2\text{max}}\) (30, 31). These findings showed a reduction in \(\dot{V}O_{2\text{max}}\) in the heat concomitant with CV drift results in an increase in \(%\dot{V}O_{2\text{max}}\), independent of changes in absolute metabolic intensity [\(\dot{V}O_2\)] (31). Simultaneous increases in HR and \(%\dot{V}O_{2\text{max}}\), and near maintenance of the HR–\(%\dot{V}O_{2\text{max}}\)
relationship, supports the use of HR to reflect changes in relative metabolic intensity during prolonged exercise in the heat in which CV drift occurs. An extension of this conclusion is that exercising at a constant HR in the heat should result in constant relative intensity (\%\dot{VO}_{2\max}), thereby preserving the HR–\%\dot{VO}_{2\max} relationship, but this could neither be determined from the data in our previous study nor has it been demonstrated elsewhere. Whether or not \dot{VO}_{2\max} is still reduced when CV drift is attenuated to maintain constant HR during prolonged exercise in the heat needs to be established to know whether the HR–\%\dot{VO}_{2\max} relationship is preserved under these conditions.

The purpose of the present study was to determine if \dot{VO}_{2\max} is reduced when exercise intensity and absolute \dot{VO}_2 are lowered by a magnitude sufficient to reduce CV drift and maintain constant HR during prolonged, submaximal exercise in the heat. Based on our previous research (31) that suggests an inverse relationship between CV drift and \dot{VO}_{2\max}, we hypothesized \dot{VO}_{2\max} would not be reduced when power output and absolute \dot{VO}_2 were decreased to eliminate CV drift and hold HR constant compared to when they were unaltered. Reduced submaximal \dot{VO}_2 accompanied by constant HR and unaltered \dot{VO}_{2\max} would reduce \%\dot{VO}_{2\max} independent of a change in HR, so the HR–\%\dot{VO}_{2\max} relationship would not be preserved during prolonged exercise in the heat when CV drift does not occur.

Methods

A repeated measures research design was used in which all subjects were tested under all conditions. After a control maximal oxygen uptake test (\dot{VO}_{2\max}), subjects completed the following trials (in random order) in 35°C, 40% relative humidity (RH) with equal airflow (no fan used) and without fluid provision: 1) 15 min of cycling at 60%
\( \dot{V}O_{2\text{max}} \) followed by a graded exercise test (GXT) to measure \( \dot{V}O_{2\text{peak}} \) (15max), 2) 45 min of cycling at 60\% \( \dot{V}O_{2\text{max}} \) followed by a GXT to measure \( \dot{V}O_{2\text{peak}} \) (HRvar), and 3) condition 2 with the use of computer software to automatically adjust power output so that HR was kept constant at the rate present at 8 min into exercise (HRcon). The purpose of the 15- and 45-min trials was to measure \( \dot{V}O_{2\text{max}} \) during the same time interval in which CV drift occurred. Each subject was tested at the same time of day to minimize the effects of circadian rhythm on HR and core temperature, and trials were separated by at least 1 d.

Seven active men (means ± SD) [age = 25 ± 4 yr, height = 180.9 ± 5.0 cm, mass = 73.2 ± 4.7 kg, percent body fat estimated from skinfolds = 8.2 ± 2.6\%] were recruited to participate in this study. Subjects were considered active if they were engaging in some form of dynamic exercise (running, cycling, etc.) 3-5 d/week for 30 min or more per session. Only male subjects were recruited because the time constraints during which data had to be collected were not adequate to allow proper control of core temperature fluctuations concomitant with the female menstrual cycle. The sample size of \( N = 7 \) is sufficient to detect a \( \dot{V}O_{2\text{max}} \) effect size of \( d = 0.5 \) SD [where \( d \) equals the difference between the expected largest and smallest means within a factor divided by \( \sigma \), the expected mean within-cell standard deviation (24)] across the 4 measures of \( \dot{V}O_{2\text{max}} \) (control, 15-min, 45-min with no HR control, and 45-min with HR control) analyzed in a one-way repeated measures ANOVA followed by pairwise comparisons with paired t tests, assuming that the test-retest correlation for \( \dot{V}O_{2\text{max}} \) is 0.95 (29) and power \( \approx 0.71 \) (23). This calculation is based on an alpha level of 0.01 because to detect differences among treatments, a Type I error rate adjustment must be made, which approximates \( \alpha/6 \).
= 0.008 \approx 0.01. The study protocol was approved in advance by the Institutional Review Board at the University of Georgia, and each subject provided written informed consent before participating.

On visit one, subjects reported to the laboratory after a 2-h fast, but well hydrated. Subjects were instructed to avoid consumption of alcohol, caffeine, or nonprescription drugs the day before and the day of testing. Upon arrival, subjects completed a 24-h history questionnaire designed to determine adherence to pre-test instructions, and they completed medical history and physical activity questionnaires. After signing consent forms, subjects had body fat percentage estimated from skinfolds. Subjects then determined their body mass (to the nearest 10 g) on an electronic scale while wearing shorts and socks. All exercise testing took place on an electronically-braked cycle ergometer (Lode Excalibur Sport, Lode B.V., Groningen, The Netherlands). After a 10-min warm-up in an environmental chamber maintained at 22°C, 40% RH, subjects completed a GXT to measure \( \dot{V}O_2^{\text{max}} \) (determined as the highest 2 consecutive 30-s averages). Power output began at 200 W and increased 25 W every 2 min until subjects could no longer continue. Oxygen uptake, ventilation, and respiratory quotient were determined by indirect calorimetry using a Parvo Medics TrueOne 2400 Metabolic Measurement System (Parvo Medics, Inc., Salt Lake City, UT) and averaged every 30 s. HR was monitored continuously using a Polar® Vantage XL heart rate monitor. Rating of perceived exertion (RPE) was obtained at the end of every 2-min stage during the GXT using the 6–20 scale developed by Borg (6). Three minutes after the end of the GXT, a 2-ml blood sample was obtained from a forearm vein for determination of blood
lactate concentration using a YSI 2300 Stat Plus Analyzer (Yellow Springs Instruments, Inc., Yellow Springs, OH).

The follow-up procedure that was utilized to ensure a plateau in \( \dot{V}_O_2 \) had been attained has been described elsewhere (31). The plateauing criterion utilized was a modified version of that described by Taylor et al. (29) in which the increase in \( \dot{V}_O_2 \) between the final two stages increased < 135 ml/min, which is half of the expected increase based on the American College of Sports Medicine metabolic equation for gross \( \dot{V}_O_2 \) during leg ergometry (1). While no subjects showed a plateau during the initial GXT, a plateau was demonstrated in all subjects by applying this criterion to the initial GXT combined with the follow-up procedure.

Twenty minutes after the follow-up procedure to verify presence of a plateau in \( \dot{V}_O_2 \), subjects cycled for 10 min while the prescribed intensity (60% \( \dot{V}_O_{2max} \)) for the experimental trials was verified. \( \dot{V}_O_2 \) was measured, and minor adjustments to the work load were made accordingly in order to elicit 60% \( \dot{V}_O_{2max} \). While still cycling, subjects then practiced the CO\(_2\) rebreathing maneuver that was used to estimate cardiac output during the experimental trials. Prior to the experimental trials, subjects followed the same pretest instructions given for the control GXT. Upon arrival at the laboratory, the 24-h history questionnaire was administered to verify adherence to pretest instructions. Additionally, subjects provided a urine sample for determination of urine specific gravity (USG). USG was measured to ensure subjects were adequately hydrated (USG ≤ 1.021). In addition to USG, tympanic temperature (\( T_t \)) was measured to ensure subjects were apyretic (\( T_t < 37.8^\circ C \)).
After USG and T<sub>1</sub> were measured, subjects measured nude body weight and inserted a rectal temperature probe (Ellab, Inc., Arvada, CO, model MOV-5504-A) 10 cm past the anal sphincter. Then, a flexible venous catheter was inserted into a forearm vein and kept patent with 0.5 ml of 10 USP units/ml heparin lock flush. Next, subjects entered an environmental chamber maintained at 35°C, 40% RH and sat upright on the cycle ergometer for ~ 20 min while skin temperature probes (Ellab, Inc., Arvada, CO, model MHF-18058-A) were attached to the right pectoralis major, right lateral sub-deltoid, right anterior quadriceps mid-way between the inguinal crease and the patella, and right lateral gastrocnemius. Additionally, a skin blood flow probe (Transonic Systems, Ithaca, NY, model ALF 21D) was attached to the posterior surface of the right forearm. After measurement of resting \( \dot{V}O_2 \), HR, skin blood flow (Biopac Systems, Inc., Goleta, CA, model MP100), systolic (SBP) and fourth phase diastolic blood pressure (DBP) by auscultation, and rectal and skin temperatures (Ellab, Inc., Arvada, CO, model TM9608 with Eval 2.1 software), a 2-ml blood sample was drawn into a Vacutainer containing EDTA. Blood samples were used to measure lactate levels, hemoglobin (Hb) in duplicate with a HemoCue B-Hb photometer (HemoCue, Inc., Lake Forest, CA), and hematocrit (Hct) in triplicate using the microhematocrit method. The resting and subsequent exercise Hb and Hct values were used to estimate plasma volume (PV) change using the Dill-Costill equation (8).

After all resting measures were obtained, subjects began cycling at a power output corresponding to 60% \( \dot{V}O_2 \max \) for either 15 or 45 min followed immediately by a GXT to measure \( \dot{V}O_2 \max \). During the submaximal exercise, HR and skin temperatures were measured continuously and skin temperatures at the various sites were used to calculate
mean skin temperature as described by Ramanathan (25). Rectal temperature ($T_{re}$) also was measured continuously, and from $\bar{T}_{sk}$ and $T_{re}$ mean body temperature ($\bar{T}_{b}$) was calculated using the equation by Baum et al. (5). During 15max, subjects cycled for 15 min before the GXT. During HRvar and HRcon, subjects cycled for 45 min before the GXT, and oxygen uptake ($\dot{V}O_2$), blood pressure, and cardiac output ($\dot{Q}$) were measured, in that order, between 8-18 and 35-45 min. Blood samples also were obtained during these time intervals (immediately before measurement of $\dot{Q}$) for measurement of PV change and blood lactate.

$\dot{Q}$ was measured using the indirect Fick CO$_2$-rebreathing method, as described by Jones et al. (20), using the Parvo Medics system. This procedure was the same as that performed during the practice session. Two to three rebreathing trials, separated by 1-2 min, were performed in succession. The heart rate recorded during the measurement of $\dot{V}CO_2$ was used to calculate SV from $\dot{Q}$, and $\dot{Q}$ and SV obtained during the collection periods were averaged.

During the HRcon trial, a computer program (Lode Ergometer Manager, Lode B.V., Groningen, The Netherlands) was used to keep HR constant beginning at min 15 and continuing until the end of submaximal exercise at 45 min. The HR chosen to be held constant was that recorded at 8 min (during measurement of $\dot{V}CO_2$ before the CO$_2$-rebreathing procedure). After 45 min in both HRcon and HRvar, subjects immediately began a GXT (with no cessation of cycling) in order to determine $\dot{V}O_2_{peak}$. Values obtained at maximum in these trials are referred to as $\dot{V}O_{2peak}$, because in the experimental trials, we could not perform the procedure that was used in the control trial to ensure a plateau in $\dot{V}O_{2max}$. Power output was instantly increased 25 W above the
power output used during the submaximal exercise, and additional 25-W increases occurred every 2 minutes until subjects could no longer continue. \( \dot{V}O_2 \) and HR were measured continuously, and RPE was obtained at the end of each 2-min stage and at exhaustion. A final blood sample was drawn approximately 3 min after the end of the GXT. Once skin temperature probes and the skin blood flow probe were removed, the subject exited the chamber, toweled off, and re-measured nude body weight. A \( \dot{V}O_2\text{peak} \) value was considered valid if 1) the plateaui ng criterion used for the control test was met (2 subjects during HRcon, none during HRvar, none during 15max), or 2) if a HR within 5 bpm of that obtained on the control GXT was obtained (all subjects during all experimental trials).

Measures of exercise intensity during the submaximal bouts (Table 4.3) were expressed as percentage of HR\(_\text{max} \) (%HR\(_\text{max} \)), percentage of \( \dot{V}O_2\text{max} \) (%\( \dot{V}O_2\text{max} \)), percentage of HR reserve (%HRR), and percentage of \( \dot{V}O_2 \) reserve (%\( \dot{V}O_2\text{R} \)). %HRR was calculated as: \( [(HR_{\text{ex}} - HR_{\text{rest}})/(HR_{\text{max}} - HR_{\text{rest}})] \times 100 \). Similarly, %\( \dot{V}O_2\text{R} \) was calculated as: \( [(\dot{V}O_{2\text{ex}} - 3.5)/\dot{V}O_{2\text{max}} - 3.5)] \times 100 \).

Statistical analyses were performed with SPSS v. 13.0 for Windows (SPSS, Inc., Chicago, IL). A paired samples t-test was used to compare mean differences in power output between min 15 and min 45 during HRcon. A univariate, one-way repeated measures analysis of variance (ANOVA) was used to test the significance of mean differences in \( \dot{V}O_2\text{max} \) among treatment conditions (control, 15max, HRvar, HRcon). For other variables, such as cardiovascular, thermoregulatory, and metabolic measures, two-way (treatment \( \times \) time) repeated measures ANOVAs were conducted to test the significance of mean differences. The Greenhouse-Geisser adjustment to degrees of
freedom was utilized for all ANOVA tests, and when appropriate, paired samples t-tests [with the Hochberg $\alpha$ adjustment (18)] were used to perform pairwise comparisons. The Hochberg $\alpha$ adjustment is a powerful statistical technique that maintains family-wise $\alpha$ level at 0.05 for multiple comparisons, but the critical $\alpha$ level, $\alpha'$, for each individual pairwise comparison in a given set of comparisons is adjusted. After running pairwise comparisons for a given set of comparisons, P-values are rank ordered and compared sequentially to the following $\alpha'$ values: 0.05, 0.025, 0.0167, 0.0125, 0.01, 0.0083, and so on (if there are more than 6 pairwise comparisons). Once a P-value is deemed statistically significant, all smaller P-values in a set of comparisons are considered statistically significant as well. Precise P-values are provided throughout the paper, but when a P-value is less than 0.05 but greater than $\alpha'$, $\alpha'$ is provided for clarity. Additionally, in cases in which the P-value decimal place extended beyond 0.001, $P < 0.001$ is given. All significance tests used an $\alpha$ level of 0.05.

Results

Pre-exercise USG was not different for 15max (1.008 ± 0.006), HRvar (1.011 ± 0.005), and HRcon (1.009 ± 0.006) [$P = 0.276$]. Likewise, body mass before the control and experimental trials was not different ($P = 0.556$), which, together with USG, indicated that subjects began each trial in a similar state of hydration. Body mass change from rest was greater in the HRvar (-2.2 ± 0.5%) and HRcon (-2.3 ± 0.6%) conditions than in 15max (-1.3 ± 0.4%) [$P < 0.001$], but there was no difference between the two 45-min trials ($P = 0.570$). Plasma volume decreased from rest during the submaximal exercise (Table 4.1) and after the GXT (Table 4.2) in both 45-min trials, but it was not different between conditions ($P = 0.146$).
Table 4.1: Responses to submaximal exercise with HR variable or HR held constant (mean ± SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>HR variable</th>
<th></th>
<th>HR constant</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>15-min</td>
<td>45-min</td>
<td>15-min</td>
<td>45-min</td>
</tr>
<tr>
<td>( \dot{V}O_2 ) (L·min(^{-1}))</td>
<td>2.46 ± 0.30</td>
<td>2.53 ± 0.28</td>
<td>2.50 ± 0.27</td>
<td>1.91 ± 0.25</td>
</tr>
<tr>
<td>( \dot{V}O_2 ) (% control ( \dot{V}O_2_{max} ))</td>
<td>58.7 ± 3.7</td>
<td>60.6 ± 3.1</td>
<td>59.9 ± 2.6</td>
<td>45.7 ± 4.0</td>
</tr>
<tr>
<td>SV (mL·beat(^{-1}))</td>
<td>106.6 ± 11.0</td>
<td>96.0 ± 6.7</td>
<td>111.7 ± 12.5</td>
<td>89.1 ± 12.4</td>
</tr>
<tr>
<td>HR (beats·min(^{-1}))</td>
<td>149.3 ± 11.6</td>
<td>169.0 ± 14.2</td>
<td>148.3 ± 14.2</td>
<td>148.6 ± 16.5</td>
</tr>
<tr>
<td>Blood lactate (mmol·L(^{-1}))</td>
<td>2.4 ± 0.8</td>
<td>2.2 ± 1.0</td>
<td>2.5 ± 1.0*</td>
<td>1.4 ± 0.4</td>
</tr>
<tr>
<td>RPE</td>
<td>11.7 ± 0.8*</td>
<td>14.1 ± 0.9†</td>
<td>11.9 ± 0.7</td>
<td>12.4 ± 1.0</td>
</tr>
<tr>
<td>Power output (W)</td>
<td>157.1 ± 25.3</td>
<td>157.1 ± 25.3</td>
<td>98.4 ± 12.4</td>
<td></td>
</tr>
<tr>
<td>Average power output from 15 to 45 min (W)</td>
<td>—</td>
<td>—</td>
<td>112 ± 21</td>
<td></td>
</tr>
<tr>
<td>Change in power output from 15 to 45 min (%)</td>
<td>—</td>
<td>—</td>
<td>-36.8 ± 6.7</td>
<td></td>
</tr>
</tbody>
</table>

\( \dot{V}O_2 \), oxygen uptake; SV, stroke volume; HR, heart rate; RPE, rating of perceived exertion; * P < 0.05 comparing 15-min and 45-min values within the same condition; † P < 0.05 versus the value at the same time point in the HRcon condition.

Cardiovascular, Metabolic, and Perceptual Responses. As shown in Table 4.1, between 15 and 45 min, HR increased 13% (P = 0.001) and SV decreased 10% (P = 0.004) concomitant with CV drift during HRvar, whereas HR remained stable during HRcon. Because maintenance of HR at a constant rate during HRcon required a reduction in power output of nearly 37% from 15 to 45 min (P < 0.001), absolute \( \dot{V}O_2 \) and blood lactate concentration were lower at min 45 than min 15 (P < 0.001 and P = 0.007, respectively). Like HR, RPE rose over time in HRvar (P = 0.001), and, as a result,
the value at 45 min was higher in HRvar than in HRcon (P = 0.007) since RPE did not change over time in HRcon.

Table 4.2 shows the maximal and peak cardiovascular and metabolic responses during the control and experimental trials, respectively. $\dot{V}O_{2\text{peak}}$ decreased 14.8% ($P < 0.001$) between that measured after 15 min of submaximal exercise (15max) and that measured after 45 min in HRvar, whereas it fell less, 7.5% ($P = 0.003$), in HRcon. In HRvar, the decrease in $\dot{V}O_{2\text{peak}}$ was accompanied by a decrease in peak power output of 14.7% ($P = 0.001$) while peak power output was not different between 15max and HRcon ($P = 0.180$). Despite a longer GXT duration in HRcon than in HRvar ($P = 0.002$), the peak power outputs achieved under these two conditions were not statistically significantly different ($P = 0.028$, $\alpha' = 0.0167$).
Table 4.2: Maximal responses during a control graded exercise test (GXT) and peak responses during a GXT following 15 min of submaximal exercise and following 45 min of submaximal exercise with HR variable or HR held constant (mean ± SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
</tr>
<tr>
<td>$\dot{V}_E$ (STPD, L·min$^{-1}$)</td>
<td>127.1 ± 18.1</td>
</tr>
<tr>
<td>$\dot{V}O_2$ (L·min$^{-1}$)</td>
<td>4.2 ± 0.4</td>
</tr>
<tr>
<td>$\dot{V}O_2$ (mL·kg$^{-1}$·min$^{-1}$)</td>
<td>57.1 ± 5.3</td>
</tr>
<tr>
<td>RER</td>
<td>1.10 ± 0.04</td>
</tr>
<tr>
<td>RPE</td>
<td>18.6 ± 0.8</td>
</tr>
<tr>
<td>HR (beats·min$^{-1}$)</td>
<td>187.0 ± 4.3</td>
</tr>
<tr>
<td>$O_2$ pulse (mL·beat$^{-1}$)</td>
<td>22.3 ± 1.8</td>
</tr>
<tr>
<td>$\Delta$SkBF from rest (%)</td>
<td>—</td>
</tr>
<tr>
<td>Blood lactate (mmol·L$^{-1}$)</td>
<td>8.0 ± 1.2</td>
</tr>
<tr>
<td>$\Delta$PV from rest (%)</td>
<td>—</td>
</tr>
<tr>
<td>$T_{re}$ (°C)</td>
<td>—</td>
</tr>
<tr>
<td>$\overline{T}_{sk}$ (°C)</td>
<td>—</td>
</tr>
<tr>
<td>$\overline{T}_b$ (°C)</td>
<td>—</td>
</tr>
<tr>
<td>Test Duration (min)</td>
<td>10.0 ± 2.3</td>
</tr>
<tr>
<td>Power Output (watts)</td>
<td>303.6 ± 30.4</td>
</tr>
</tbody>
</table>

$\dot{V}_E$, minute ventilation; $\dot{V}O_2$, oxygen uptake; RER, respiratory exchange ratio; RPE, rating of perceived exertion; HR, heart rate; $\Delta$SkBF, change in skin blood flow; $\Delta$PV, change in plasma volume; $T_{re}$, rectal temperature; $\overline{T}_{sk}$, mean skin temperature; $\overline{T}_b$, mean body temperature; * P < 0.05 compared with control; † P < 0.05 compared with 15-min value; ‡ P < 0.05 compared with HRcon.
Thermoregulatory Responses. While submaximal $T_{re}$ and $\bar{T}_b$ increased over time in both HRvar ($P < 0.001$ for $T_{re}$ and $\bar{T}_b$) and HRcon ($P = 0.001$ for $T_{re}$ and $P = 0.005$ for $\bar{T}_b$), $\bar{T}_{sk}$ was neither different over time within each condition nor different between conditions ($P = 0.085$). Skin blood flow increased from resting values in response to the rise in core body temperature, but it leveled off after 15 min and remained stable for the remainder of submaximal and maximal exercise in both conditions ($P = 0.143$).

On average, by the time individuals reached maximum during the GXT following 45 min of submaximal exercise in HRvar, $T_{re}$ increased an additional 0.3°C from the 45-min value (Table 4.2). This maximal core temperature was higher than that in 15max ($P = 0.001$) but not different from that in HRcon ($P = 0.047$, $\alpha' = 0.025$). Despite no differences in $\bar{T}_{sk}$ at maximum between HRvar and HRcon, $\bar{T}_b$ was about 0.6°C higher in HRvar ($P = 0.047$).
Maximal values are based on: $^1$ the control graded exercise test (GXT), $^2$ the GXT after 15 min (15max), and $^3$ the GXT following 45 min of exercise in the HRvar and HRcon conditions, respectively. \%$\dot{V}O_2$\textsubscript{max}, percent of maximal oxygen uptake; \%HR\textsubscript{max}, percent of maximal heart rate; \%$\dot{V}O_2$R, percent of oxygen uptake reserve; \%HRR, percent of heart rate reserve; * P < 0.05 compared with 15-min$^1$ within same condition; † P < 0.05 compared with 45-min$^1$ within same condition; ‡ P < 0.05 compared with 15-min$^2$ within same condition.

Table 4.3: Measures of exercise intensity at 15 and 45 min under each condition based on maximal values (mean ± SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>HRvar</th>
<th></th>
<th></th>
<th></th>
<th>HRcon</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>15-min$^1$</td>
<td>45-min$^1$</td>
<td>15-min$^2$</td>
<td>45-min$^3$</td>
<td>15-min$^1$</td>
<td>45-min$^1$</td>
<td>15-min$^2$</td>
</tr>
<tr>
<td>%$\dot{V}O_2$\textsubscript{max}</td>
<td>58.7 ± 3.7</td>
<td>60.6 ± 3.1*</td>
<td>59.3 ± 5.5</td>
<td>72.1 ± 7.9*‡</td>
<td>59.9 ± 2.6</td>
<td>45.7 ± 4.0*</td>
<td>60.5 ± 4.4†</td>
</tr>
<tr>
<td>%HR\textsubscript{max}</td>
<td>79.7 ± 4.5</td>
<td>90.3 ± 6.0*</td>
<td>78.5 ± 4.1†</td>
<td>87.2 ± 4.5*‡</td>
<td>79.2 ± 6.1</td>
<td>79.3 ± 7.3</td>
<td>78.0 ± 5.2</td>
</tr>
<tr>
<td>%$\dot{V}O_2$R</td>
<td>56.5 ± 4.2</td>
<td>58.4 ± 3.5*</td>
<td>56.8 ± 5.4</td>
<td>70.1 ± 8.2*‡</td>
<td>57.7 ± 2.8</td>
<td>42.4 ± 4.1*</td>
<td>58.0 ± 4.1†</td>
</tr>
<tr>
<td>%HRR</td>
<td>67.9 ± 6.2</td>
<td>84.5 ± 9.7*</td>
<td>66.3 ± 5.1†</td>
<td>80.0 ± 7.3*‡</td>
<td>66.4 ± 9.6</td>
<td>66.6 ± 11.5</td>
<td>64.7 ± 8.5</td>
</tr>
</tbody>
</table>
Measures of Exercise Intensity. Because HR drifted up over time in HRvar, HR expressed as %HR_{max} and %HRR relative to control HR_{max} drifted up as well (Table 4.3). Likewise, because HR_{max} after the GXTs in 15max and HRvar was similar to control (P = 0.129; and P = 0.026, α′ = 0.0083, respectively), %HR_{max} and %HRR expressed relative to those maximums also drifted up over time in conjunction with CV drift (P = 0.001 and P < 0.001, respectively). Since HR was kept constant in HRcon, there were no differences across time in expressions of exercise intensity based on HR in that condition (all P > 0.05).

In HRvar, %\dot{V}O_2_{max} and %\dot{V}O_2R increased significantly over time (P = 0.001, α′ = 0.0083; and P = 0.001, α′ = 0.01, respectively) when expressed relative to \dot{V}O_2peak measured after the GXT in HRvar (Table 4.3). The increase in %\dot{V}O_2R (13.3 ± 4.9 percentage points) from min 15 (expressed relative to 15max) to min 45 (expressed relative to \dot{V}O_2peak measured in HRvar) was nearly identical to that in %HRR (13.7 ± 5.1 percentage points).

In HRcon, power output had to be substantially lowered in order to keep HR constant, so absolute submaximal \dot{V}O_2 was lowered, which resulted in significant decreases in %\dot{V}O_2_{max} and %\dot{V}O_2R over time (both P < 0.001) of ~10.6 and 11.6 percentage points, respectively (Table 4.3).

Discussion

The purpose of the present study was to determine if \dot{V}O_{2max} is reduced when exercise intensity and absolute \dot{V}O_2 are lowered by a magnitude sufficient to reduce CV drift and maintain constant HR during prolonged, submaximal exercise in the heat, and thereby resolve whether or not the HR–%\dot{V}O_{2max} relationship is preserved under these
conditions. Keeping HR constant with computer software enabled us to accurately quantify the amounts power output and absolute exercise intensity were lowered in order to keep HR constant. The study design utilized was intended to measure \( \dot{V}O_{2\text{max}} \) during the same time interval in which CV drift occurred because previous research investigating the relationship between these two variables failed to temporally link them \((9, 27)\). Additionally, our design was intended to eliminate a rise in HR while attempting to hold other variables constant that could influence \( \dot{V}O_{2\text{max}} \). Core body, skin, and mean body temperatures were similar across the HRvar and HRcon conditions at the 45-min time point and at \( \dot{V}O_{2\text{max}} \), respectively (Table 4.2). Besides temperatures, indices of hydration (change in body mass and change in plasma volume from rest) showed that subjects became dehydrated to the same extent in each condition, and sweat rates were similar between HRvar and HRcon as well.

In a previous study by our laboratory \((30)\), we found when power output stayed constant during prolonged exercise in a cool environment, CV drift did not occur and there was subsequently no significant change in \( \dot{V}O_{2\text{max}} \), so relative metabolic intensity was unchanged as well. We were unsure, however, how much exercise intensity would have to be changed to attenuate CV drift and hold HR constant in a hot environment, and additionally whether attenuating CV drift in a hot environment would preserve \( \dot{V}O_{2\text{max}} \) to the same extent as in a cool environment. For the HR–%\( \dot{V}O_{2\text{max}} \) relationship to be preserved over time in a hot environment like in a cool environment, the rise in HR concomitant with CV drift would have to be accompanied by a decrease in \( \dot{V}O_{2\text{max}} \), if submaximal \( \dot{V}O_2 \) remains constant. Our earlier work \((30, 31)\) showed this, but we could not determine from those data what would happen to the HR–%\( \dot{V}O_{2\text{max}} \) relation if
absolute exercise intensity was lowered by a magnitude sufficient to attenuate CV drift by holding HR constant. Would the reduction in absolute \( \dot{V}O_2 \) be proportional to a decrease in \( \dot{V}O_2_{\text{max}} \) over time such that a given HR would continue to reflect a similar \%\( \dot{V}O_2_{\text{max}} \), regardless of ambient temperature? We hypothesized \( \dot{V}O_2_{\text{max}} \) would not be reduced when power output and absolute \( \dot{V}O_2 \) were decreased to maintain constant HR compared to when they were unaltered. Therefore, the HR–%\( \dot{V}O_2_{\text{max}} \) relationship would not be preserved during prolonged exercise in the heat when CV drift does not occur since reduced submaximal \( \dot{V}O_2 \) accompanied by constant HR and unaltered \( \dot{V}O_2_{\text{max}} \) would reduce %\( \dot{V}O_2_{\text{max}} \) independent of a change in HR.

The main finding is that the HR–%\( \dot{V}O_2_{\text{max}} \) relationship was not preserved during prolonged exercise in a hot environment when exercise intensity was lowered by a magnitude sufficient to attenuate CV drift and hold HR constant. The relative metabolic intensity (%\( \dot{V}O_2_{\text{max}} \)) was reduced from the ~60% at min 15 as originally prescribed to ~50% at min 45 (Table 4.3) in order to attenuate CV drift and hold HR constant. Power output was lowered 37%, and this resulted in a 24% reduction in absolute \( \dot{V}O_2 \) (Table 4.1). \( \dot{V}O_2_{\text{max}} \) decreased 7.5%, so the change in absolute \( \dot{V}O_2 \) and \( \dot{V}O_2_{\text{max}} \) were not proportional, and the HR–%\( \dot{V}O_2_{\text{max}} \) relationship was not preserved. The 7.5% decrease in \( \dot{V}O_2_{\text{max}} \) in HRcon when power output and absolute \( \dot{V}O_2 \) were lowered to maintain HR was greater than hypothesized, but less than the 15% decrease in HRvar when power output and \( \dot{V}O_2 \) were unchanged.

While the conclusion that exercising at a constant HR resulted in decreased relative metabolic intensity (and dissociation of HR from %\( \dot{V}O_2_{\text{max}} \)) seems to contradict the finding of our previous study [that change in HR reflects change in relative metabolic
intensity during prolonged exercise in the heat in which CV drift occurs], we could not extrapolate the data from the previous study to determine the metabolic consequence of exercising at constant HR. Further, while it is known that HR is dissociated from %\(\dot{V}O_{2\text{max}}\) in the heat (3), no one that we are aware of has determined whether relative metabolic intensity is altered when HR is held constant during exercise in hot conditions. Hence, those who prescribe exercise at a constant HR should consider the effect this has on the absolute metabolic stimulus during exercise in hot temperatures. The intensity at 45 min in HRcon in the present study borders on the minimum intensity recommended for improving cardiorespiratory fitness (28). Thus, individuals attempting to exercise in the heat at a constant HR in conjunction with an exercise prescription may substantially reduce the metabolic and cardiovascular stimulus for improved fitness. As mentioned earlier, prediction equations are available that may enable adjustment of HR at different work rates under various environmental conditions (11, 12) so that relative metabolic intensity is less compromised, but these equations require ancillary information (clothing insulation factor, metabolic heat production, etc.) that may not be available. Hence, prescribing maintenance of THR, even in heat stress, is more likely to be utilized than these equations. Practitioners should at least be aware of the magnitude relative metabolic intensity must be lowered in order to maintain HR during conditions like those imposed in this study. Further research should focus on how much relative metabolic intensity must change in order to maintain constant HR under different conditions of ambient temperature and relative humidity.

Despite holding HR constant in the HRcon condition, SV still fell by \(~20\%\) (Table 4.1). It cannot be ascertained from these data whether this decrease was
exclusively associated with the fall in $\dot{Q}$ because of lower metabolic demand
(concomitant with the lower work rate necessary to keep HR constant), or whether this
fall in SV was in some way related to CV drift. SV would not be expected to decrease
when HR is held constant based on the findings of Fritzsche et al. (10), although in that
study they used a pharmacologic agent instead of work rate adjustment to prevent the rise
in HR. Because the mechanism responsible for the fall in SV in the HRcon condition
cannot be determined from these data, we cannot be certain whether CV drift was related
to the decrease in $\dot{V}O_{2\text{max}}$ in that trial or not. Apparently, the increase in HR concomitant
with CV drift is only partially related to a decrease in $\dot{V}O_{2\text{max}}$. Otherwise, holding HR
constant should have completely eliminated the reduction in $\dot{V}O_{2\text{max}}$

It also is possible that the increase in HR concomitant with CV drift is only
partially related to a decrease in SV. Fluid ingestion studies that include measures of CV
drift (HR and SV) indicate that SV is maintained during prolonged exercise with
dehydration when the rise in HR is attenuated (but not completely prevented) with fluid
ingestion (16, 17). If part of the rise in HR over time during constant-rate exercise in the
heat is not linked to a decrease in SV, then completely eliminating the rise in HR during
prolonged exercise by reducing power output and metabolic demand allowed SV to
decrease instead of being maintained. Perhaps attenuating—but not completely
preventing—the rise in HR would have better maintained SV. Under the conditions of this
study, once SV was decreased, it may not have been able to be increased to an extent
necessary to achieve $\dot{V}O_{2\text{max}}$ because of the combination of high skin and core
temperatures.
Decreased maximal SV ($SV_{\text{max}}$) most likely was responsible for the reduction in $\dot{V}O_{2\text{max}}$ under both the HRvar and HRcon conditions because $(a-v)O_2$ was unlikely to be reduced at maximum (15, 21) in either condition. The $O_2$ pulse [$SV \times (a-v)O_2$] data (Table 4.2) support this assertion. Furthermore, similar, near-maximal RPE after the GXT in each of the experimental trials suggests subjects gave a similar maximal effort under each condition. Finally, despite different peak power outputs among experimental trials (Table 4.2), HR was no different at maximum. So, while peak power output could have been influenced by leg fatigue, this apparently did not occur before achievement of maximal cardiovascular capacity, and thereby, $V_{O2\text{peak}}$.

If reduced $SV_{\text{max}}$ caused reduced $\dot{V}O_{2\text{max}}$ in both conditions, what caused $\dot{V}O_{2\text{peak}}$ to be different between HRvar and HRcon? This question cannot be directly answered from the data collected in this study, but it is likely that the SV at maximum was higher in the HRcon condition even though it was most likely reduced from that during 15max. Despite a shorter GXT duration, the $T_b$ was higher at maximum in the HRvar trial compared to HRcon (Table 4.2). Furthermore, even though $T_{re}$ at maximum was not statistically different between HRvar and HRcon, the $\sim$0.6°C higher $T_{re}$ at maximum (Table 4.2) in HRvar may have been physiologically meaningful. Combined with the high skin temperatures encountered at maximum in that trial, it may have contributed to a decrease in $\dot{V}O_{2\text{max}}$ since the combination of high skin and core temperatures has been shown to reduce $\dot{V}O_{2\text{max}}$ (2). $\dot{Q}$ has been shown to be lower at maximum during heat stress compared to temperate conditions (15), so perhaps the greater hyperthermia encountered in HRvar differentially affected $\dot{Q}_{\text{max}}$ in the current study. If lower $\dot{Q}_{\text{max}}$ in HRvar accounted for the lower $\dot{V}O_{2\text{max}}$, $SV_{\text{max}}$ must have been lower since $HR_{\text{max}}$ was not
different between these conditions. SV could have been lower because of severe
tachycardia limiting ventricular filling time and end-diastolic volume (10, 15) or because
of decreased central blood volume, central venous pressure, and end-diastolic volume
associated with high skin blood flow at maximum (26).

Previous research has shown that in untrained or moderately trained individuals,
SV plateaus at about 40–58% $\dot{V}O_{2\text{max}}$ (4, 13), whereas SV in highly-trained individuals
may continue to rise until $\dot{V}O_{2\text{max}}$ is reached (13). While the subjects in the current study
were active, they were not highly-trained cyclists. Thus, it is likely that SV in these
individuals had already reached its maximum at 15 min in HRvar. Since SV declined
substantially by 45 min in HRvar, and the relative intensity was still at 60% $\dot{V}O_{2\text{max}}$
(based on the control GXT; Table 4.3) or even higher (~72% $\dot{V}O_{2\text{max}}$ based on HRvar
GXT), then the decrement in SV present at 45 min could have persisted during the GXT
such that only a $SV_{\text{peak}}$, not a $SV_{\text{max}}$, could be attained. The reduction in SV at maximum
in HRcon was likely related to something other than CV drift, which suggests while
phenomena coinciding with CV drift may in part cause a reduction in $\dot{V}O_{2\text{max}}$, other
factors present in the absence of CV drift also contribute to a decrement, albeit a smaller
one, in $\dot{V}O_{2\text{max}}$.

A potential limitation of the study was that subjects were in various stages of
acclimation. Trials took place in Georgia during the late fall and winter months, so it is
likely that subjects were unacclimatized to heat and therefore could potentially become
acclimated during repeated trials in the heat. Randomization of trials prevented an order
effect on change in $\dot{V}O_{2\text{max}}$ even though there was an order effect on change in HR for
HRvar. While different states of acclimation among subjects could have added to the
variability of responses, the individuals who performed the HRvar trial after performing HRcon and 15max (n = 4) actually had a greater change in HR over time than those who performed the HRvar trial first (n = 3), which is an effect opposite that which would be expected if acclimation had occurred. As a result, we feel confident acclimation did not occur and there was no impact of treatment order on our main outcomes.

In summary, reducing exercise intensity by a magnitude sufficient to attenuate CV drift and hold HR constant only partially eliminated the reduction in $\dot{V}O_{2\text{max}}$ after prolonged exercise in the heat. %$\dot{V}O_{2\text{max}}$ was decreased from ~60% to 50% to hold HR constant in the hot conditions of this study, so the HR–%$\dot{V}O_{2\text{max}}$ relationship was not preserved in the absence of CV drift. Individuals prescribing exercise at a constant THR in a hot environment should consider that doing so reduces the absolute metabolic stimulus and, as a result, training adaptations for improved cardiorespiratory fitness may be compromised. Future training studies should determine if this is indeed the case.
Acknowledgements

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References


CHAPTER 5

BODY COOLING ATTENUATES THE DECREASE IN MAXIMAL OXYGEN UPTAKE ASSOCIATED WITH CARDIOVASCULAR DRIFT DURING HEAT STRESS

\[\text{\textsuperscript{1}}\]

\[\text{\textsuperscript{1} Wingo, J.E. and K.J. Cureton. Submitted to European Journal of Applied Physiology, 3/6/2006.}\]
Abstract

Previous research suggests cardiovascular drift (CV drift) is associated with decreased maximal oxygen uptake (\(\dot{V}O_{2\text{max}}\)) during heat stress, but more research manipulating CV drift with subsequent measurement of \(\dot{V}O_{2\text{max}}\) is needed to assess whether this relationship is cause and effect. To investigate this issue, after a control graded exercise test (GXT) to measure \(\dot{V}O_{2\text{max}}\), 10 men cycled in 35°C at 60% \(\dot{V}O_{2\text{max}}\) for 15 min (15max), 45 min with no cooling (NC), and 45 min with fan airflow (FAN) beginning at ~18 min into exercise. The 15- and 45-min bouts were immediately followed by a GXT to measure \(\dot{V}O_{2\text{max}}\) so that \(\dot{V}O_{2\text{max}}\) could be measured during the same time interval in which CV drift occurred. \(\dot{V}O_{2\text{max}}\) decreased 18%, HR increased 16%, and SV fell 12% (P<0.05) from min 15 to min 45 in NC, whereas \(\dot{V}O_{2\text{max}}\) fell 5.7% (P<0.05) during FAN. HR (152±7 vs. 158±9 beats min\(^{-1}\) at 15 and 45 min, respectively, P<0.05) and SV (137±11.5 vs. 133±11 ml beat\(^{-1}\) at 15 and 45 min, respectively, P<0.05) remained more stable across time in FAN. Rectal temperature was lower during FAN at min 45 and at max (P<0.05). The fall in \(\dot{V}O_{2\text{max}}\) associated with CV drift during exercise in a hot environment is attenuated with body cooling via fan airflow. The findings support the hypothesis that a causal link exists between CV drift that occurs during prolonged exercise in a hot environment and a decrease in \(\dot{V}O_{2\text{max}}\).

**Keywords** Thermoregulation, Heart rate, Stroke volume, Circulation, \(\dot{V}O_{2\text{max}}\)
Introduction

The term, “cardiovascular drift” (CV drift) refers to the phenomenon in which heart rate (HR) rises and stroke volume (SV) decreases progressively over time during constant-rate, prolonged, moderate-intensity exercise [50–75% maximal oxygen uptake, \( \dot{V}_{O_{2\text{max}}} \)] (Rowell 1974). This phenomenon also may involve progressive decreases in mean arterial (MAP) and pulmonary pressures, while cardiac output (\( \dot{Q} \)) is maintained. Much research has focused on the mechanisms evoking CV drift (Coyle 2002; Fritzsche et al. 1999; Rowell 1974), but little has been done to determine if this phenomenon has metabolic consequences.

The few manipulations of CV drift that have been done to determine its implications have attempted to elucidate whether this phenomenon has a detrimental effect on \( \dot{V}_{O_{2\text{max}}} \), and furthermore, whether this effect is causal. Ganio et al. (2006) used time and fluid ingestion to influence the magnitude of CV drift. They found that dehydration (3.7% body mass decline) in mild heat (30°C) with fan airflow elicited CV drift and a reduction in \( \dot{V}_{O_{2\text{max}}} \) after 120 min (9% decline) but not after 60 min (1.2% decline) of moderate intensity (60% \( \dot{V}_{O_{2\text{max}}} \)) exercise, and provision of fluid attenuated CV drift and reduced the decrement in \( \dot{V}_{O_{2\text{max}}} \) observed after 120 min (1.9% decline). In other investigations, greater heat stress (35°C) coupled with a shorter exercise duration (45 min) resulted in substantial CV drift and reduced \( \dot{V}_{O_{2\text{max}}} \) (15-19%), whereas exercise in a cool environment (22°C) in which CV drift did not occur was not associated with a decrease in \( \dot{V}_{O_{2\text{max}}} \) (Wingo et al. 2005a; Wingo et al. 2005b).

While these studies have shown a relation and even established a plausible causal link between CV drift and decrements in \( \dot{V}_{O_{2\text{max}}} \), they have not demonstrated what would
happen to \( \dot{V}O_{2\text{max}} \) if CV drift was manipulated during exercise in a hot environment (e.g., 35°C). To address that issue, another study (unpublished observations) was conducted in which power output was manipulated in order to keep HR constant over time during sub maximal exercise in a hot environment. \( \dot{V}O_{2\text{max}} \) still declined, albeit to a smaller extent (7.5%), despite attenuation of a rise in HR. SV fell in conjunction with the decrease in power output, and therefore absolute intensity (\( \dot{V}O_2 \)), necessary to hold HR constant in the heat, but investigators could not determine whether this was related to CV drift or to decreased \( \dot{Q} \) concomitant with decreased metabolic demand. Hence, manipulation of CV drift in the heat in which both HR and SV are maintained is needed to further elucidate the relationship between CV drift and \( \dot{V}O_{2\text{max}} \) and provide additional insight into factors limiting work capacity in the heat.

Use of fan airflow at 4.3 m s\(^{-1}\) in 24°C was successful in attenuating the rise in HR and fall in SV characterizing CV drift in a study by Shaffrath and Adams (1984), but they did not measure \( \dot{V}O_{2\text{max}} \), and they did not employ a hot environment. The purpose of the current study was to determine whether body cooling (via fan airflow) during exercise in a hot environment diminishes the magnitude of CV drift and whether this in turn diminishes a decline in \( \dot{V}O_{2\text{max}} \). We hypothesized that body cooling would reduce the magnitude of CV drift, and thereby the reduction in \( \dot{V}O_{2\text{max}} \), observed following prolonged exercise in the heat.

**Methods**

**Research Design.** A repeated measures research design was used in which all subjects were tested under all experimental conditions. After a control maximal oxygen uptake test (\( \dot{V}O_{2\text{max}} \)) to determine aerobic capacity, subjects completed the following (in
random order): 1) 15 min of cycling at 60% \( \dot{V}O_{2\max} \) in 35°C, 40% relative humidity (RH) followed by a graded exercise test (GXT) to measure \( \dot{V}O_{2\max} \) (15max), 2) 45 min of cycling at 60% \( \dot{V}O_{2\max} \) in 35°C, 40% RH followed by a GXT to measure \( \dot{V}O_{2\max} \) (No Cooling, NC), and 3) condition 2 with the use of fan airflow at 4.5 m s\(^{-1}\) beginning after measurements taken from 8-18 min (FAN). The purpose of the 15- and 45-min trials was to measure \( \dot{V}O_{2\max} \) during the same time interval in which CV drift occurred. Each subject was tested at the same time of day to minimize the effects of circadian rhythm on HR and core temperature, and trials were separated by at least 1 d.

Subjects. Ten endurance-trained men (means ± SD) [age = 24 ± 4 yrs, height = 180.4 ± 6.4 cm, mass = 72.0 ± 3.1 kg, percent body fat estimated from skinfolds = 7.0 ± 2.1%] volunteered to participate in this study. Subjects were cyclists and triathletes averaging 184 ± 82 km/week cycling and/or 16 ± 14 km/week running. Only male subjects were recruited because the time constraints during which data were collected were not adequate to allow proper control of core temperature fluctuations concomitant with the female menstrual cycle. This sample size is sufficient to detect a \( \dot{V}O_{2\max} \) effect size of \( d = 0.5 \) SD [where \( d \) equals the difference between the expected largest and smallest means within a factor divided by \( \sigma \), the expected mean within-cell standard deviation (Potvin and Schutz 2000)] across the 4 measures of \( \dot{V}O_{2\max} \) (control, 15-min, 45-min without cooling, and 45-min with fan) analyzed in a one-way repeated measures ANOVA followed by pairwise comparisons with paired t tests, assuming that the test-retest correlation for \( \dot{V}O_{2\max} \) is 0.95 (Taylor et al. 1955) and power ≈ 0.95 (Park and Schutz 1999). This calculation is based on an alpha level of 0.01 because to detect differences among treatments, an \( \alpha \) correction must be made, which approximates \( \alpha/6 = \)
0.008 = 0.01. The study was approved by the university’s institutional review board in advance, and subjects gave written informed consent prior to testing.

Protocol and Procedures. On visit one, subjects reported to the laboratory after a 2-h fast, but well hydrated. Subjects were instructed to avoid consumption of alcohol, caffeine, or nonprescription drugs the day before and the day of testing. Subjects completed a 24-h history questionnaire intended to confirm adherence to pre-test instructions, a medical history, and a physical activity questionnaire upon arrival. After signing consent forms, subjects had body fat percentage estimated from skinfolds, and then they measured their body mass (to the nearest 10 g) on an electronic scale (A&D Co., Ltd., Tokyo, model FW-150KA1) while wearing shorts and socks. All exercise testing took place on an electronically-braked cycle ergometer (Lode Excalibur Sport, Lode B.V., Groningen, The Netherlands). After a 10-min warm-up at a self-selected intensity in a 22°C, 40% RH environment, subjects completed a GXT to measure $\dot{V}O_{2\text{max}}$ (determined as the highest 2 consecutive 30-s averages). The GXT began at 200 W and increased 25 W every 2 min until subjects could no longer maintain 40 rpm. A Parvo Medics TrueOne 2400 Metabolic Measurement System (Parvo Medics, Inc., Salt Lake City, UT) was used to measure oxygen uptake and other gas exchange measures via open-circuit spirometry, and values were averaged every 30 s. HR was recorded continuously using a Polar® Vantage XL heart rate monitor (Polar Electro, Inc., Woodbury, NY, model 145900), and rating of perceived exertion (RPE) was obtained at the end of every 2-min stage during the GXT using the 6–20 scale developed by Borg (1974).
Twenty minutes after the end of the GXT, subjects completed a follow-up procedure to ensure a plateau in $\dot{V}O_2$ had been attained, as has been described elsewhere (Wingo et al. 2005b). A modification of the criterion asserted by Taylor (1955) was used to define a plateau in $\dot{V}O_{2\text{max}}$ between two successive stages as an increase in $\dot{V}O_2$ of less than half (135 mL/min) of the expected increase (270 mL/min) based on the American College of Sports Medicine metabolic equation for gross $\dot{V}O_2$ during leg ergometry (American College of Sports Medicine 2000):

$$\dot{V}O_2 = (10.8 \times W \times M^{-1}) + 7,$$

where $\dot{V}O_2$ is gross oxygen consumption in milliliters per kilogram per minute, $W$ is power in watts, and $M$ is body mass in kilograms. Three subjects demonstrated a plateau on the initial GXT while all others showed a plateau on the follow-up test.

Twenty minutes after the follow-up procedure, subjects cycled for 10 min while the prescribed intensity (60% $\dot{V}O_{2\text{max}}$) for the experimental trials was verified. $\dot{V}O_2$ was measured, and if necessary, minor adjustments were made to the power output in order to elicit 60% $\dot{V}O_{2\text{max}}$. Subjects continued cycling and then practiced the CO$_2$ rebreathing maneuver that was used to estimate cardiac output during the experimental trials.

Prior to the experimental trials, subjects followed the same pretest instructions given for the control GXT. Upon arrival at the laboratory, the 24-h history questionnaire was administered to verify adherence to pretest instructions. Additionally, subjects provided a urine sample for determination of urine specific gravity (USG), which was measured with a refractometer (Atago Co., Ltd., Bellevue, WA, model URC-PN). USG was used to ensure subjects were adequately hydrated (USG ≤ 1.020). In addition to
USG, tympanic temperature ($T_t$) was measured with a digital ear thermometer to ensure subjects were apyretic ($T_t < 37.8^\circ$C).

After USG and $T_t$ were measured, subjects measured nude body weight and inserted a rectal temperature probe (Ellab, Inc., Arvada, CO, model MOV-5504-A) 10 cm past the anal sphincter. Then, a flexible venous catheter (Angiocath™, Becton Dickinson, Franklin Lakes, NJ, product 381137) was inserted into a forearm vein and kept patent with 0.5 ml of 10 USP units m$^{-1}$ heparin lock flush. Next, subjects entered an environmental chamber maintained at 35$^\circ$C, 40% RH and sat upright on the cycle ergometer for ~ 20 min while skin temperature probes (Ellab, Inc., Arvada, CO, model MHF-18058-A) were attached to 4 sites as illustrated by Mitchell and Wyndham (1969) for use with the formula for mean skin temperature ($\overline{T_{sk}}$) described by Ramanathan (1964):

$$\overline{T_{sk}} = 0.3 \cdot (T_1 + T_2) + 0.2 \cdot (T_3 + T_4),$$

where $T_1$, $T_2$, $T_3$, and $T_4$ are chest, lateral sub-deltoid, quadriceps, and gastrocnemius skin temperatures, respectively. In addition to skin temperature probes, a skin blood flow probe (Biopac Systems, Inc., Goleta, CA, model TSD 142) was attached to the posterior surface of the right forearm. After measurement of resting $\dot{V}O_2$, skin blood flow (Biopac Systems, Inc., Goleta, CA, model MP100), systolic (SBP) and fourth phase diastolic blood pressure (DBP) by auscultation, and rectal and skin temperatures (Ellab, Inc., Arvada, CO, model TM9608 with Eval 2.1 software), a 2-ml blood sample was drawn into a Vacutainer containing EDTA. Blood samples were used to measure hemoglobin (Hb) in duplicate with a HemoCue B-Hb photometer (HemoCue, Inc., Lake Forest, CA), and hematocrit (Hct) in triplicate using the microhematocrit method. The resting and
subsequent exercise Hb and Hct values were used to estimate plasma volume (PV) change using the Dill-Costill equation (1974).

After all resting measures were obtained, subjects began cycling at a power output corresponding to 60% \( \dot{V}O_{2\max} \) for either 15 or 45 min followed immediately by a GXT to measure \( \dot{V}O_{2\max} \). During the submaximal exercise, skin temperature, rectal temperature \( (T_{re}) \), and HR were recorded continuously. Mean body temperature \( (T_b) \) was calculated from \( T_{sk} \) and \( T_{re} \) using the equation by Baum et al. (1976). Subjects cycled for 15 min before the GXT on one visit. During the other experimental trials, subjects cycled for 45 min before the GXT, and oxygen uptake \( (\dot{V}O_2) \) and blood pressure were measured, 2 ml of blood was sampled, and cardiac output \( (\dot{Q}) \) was measured, in that order, between 8-18 and 35-45 min. SBP and DBP obtained during exercise were used to estimate MAP as \( MAP = (0.33 \times SBP) + (0.66 \times DBP). \)

\( \dot{Q} \) was measured using the indirect Fick CO\(_2\)-rebreathing method, as described by Jones et al. (1975), using the Parvo Medics system. Two to three rebreathing trials, separated by 1-2 min, were performed in succession. The heart rate recorded during the measurement of \( \dot{V}CO_2 \) was used to calculate SV from \( \dot{Q} \), and \( \dot{Q} \) and SV obtained during the collection periods were averaged. Systemic vascular resistance (SVR) was calculated by dividing MAP by \( Q \), and a correction factor of 80 [converting mm Hg min l\(^{-1}\) to dyne s cm\(^{-5}\)] (Mier et al. 1996) was utilized.

During the trials involving fan airflow at 4.5 m s\(^{-1}\), the fan (Home Depot, Atlanta, GA, model Hampton Bay HBS 20) was turned on at \( \sim 18 \) min (after the first data collection period from 8-18 min). The fan was directed at each subject’s back and side,
and it was calibrated before and periodically during the study using an anemometer (Control Co., Friendswood, TX, model 01-241).

After the 45 min of submaximal exercise, subjects immediately began a GXT (with no cessation of cycling) in order to determine $\dot{V}O_{2\text{peak}}$. Values obtained at maximum in the experimental trials are referred to as $\dot{V}O_{2\text{peak}}$ because we could not perform the procedure that was used in the control trial to ensure a plateau in $\dot{V}O_{2\text{max}}$. Power output was instantly increased 25 W above the power output incorporated during the submaximal exercise, and additional 25-W increases occurred every two minutes until subjects could no longer continue. $\dot{V}O_2$ and HR were measured and recorded continuously, and RPE was obtained at the end of each 2-min stage and at exhaustion. A final blood sample was drawn approximately 3 min after the end of the GXT. Once skin temperature probes and the skin blood flow probe were removed, the subject exited the chamber, toweled off, and re-measured nude body weight. A $\dot{V}O_{2\text{peak}}$ value was considered valid if 1) the plateauing criterion used for the control test was met (no subjects during 15max, 2 during NC, and 4 during FAN), or 2) if a HR within 5 beats min$^{-1}$ of that obtained on the control GXT was obtained (all other trials).

**Statistical Analysis.** Statistical analyses were performed with SPSS v. 13.0 for Windows (SPSS, Inc., Chicago, IL). A univariate, one-way repeated measures analysis of variance (ANOVA) was used to test the significance of mean differences in $\dot{V}O_{2\text{max}}$ among treatment conditions (control, 15max, NC, and FAN). For other variables, such as cardiovascular, temperature, and metabolic measures, two-way (treatment × time) repeated measures ANOVAs were conducted to test the significance of mean differences. The Greenhouse-Geisser adjustment to degrees of freedom was utilized for all ANOVA
tests, and when appropriate, paired samples t-tests [with the Hochberg α adjustment (Hochberg 1988)] were used to test for individual differences between treatments and time points. All significance tests used an alpha level of 0.05.

Results

Indicators of hydration status were not different between NC and FAN (pre-exercise USG = 1.005 ± 0.002 and 1.008 ± 0.005 for NC and FAN, respectively). Moreover, pre-exercise body mass was similar before the control and experimental trials (P > 0.05). Taken together, similar pre-exercise USG and body mass indicated that subjects began each trial in a similar state of hydration. Body mass decreased significantly from pre- to post-exercise, but neither the change in body mass (-2.18 ± 0.33% vs. -2.24 ± 0.40%, P > 0.05) nor the sweat rate (1817 ± 280 ml vs. 1751 ± 329 ml, P > 0.05) was different between NC and FAN. Plasma volume (Table 5.1) decreased from rest during the 45-min submaximal exercise and after the GXT (Table 5.2) in NC and FAN, but values were not different between conditions (P > 0.05).

Cardiovascular and Metabolic Responses. Figure 5.1 and Table 5.1 show that HR and SV changed significantly across time in both conditions (P < 0.05), but the effects were larger in NC (16% increase in HR and 12% decrease in SV in NC vs. 4% increase in HR and 3% decrease in SV in FAN). As a result, HR at 45 min was higher in NC (P < 0.001), while SV was lower (P = 0.003). The virtual lack of change in HR (4%, P = 0.007) and SV in FAN resulted in maintenance of Q̇ over time (P > 0.05), while the rise in HR offset the fall in SV in NC so that Q̇ also was maintained in that condition (P > 0.05). Furthermore, maintenance of Q̇ and SVR (Table 5.1) resulted in a constant MAP.
across time in each condition (P > 0.05). Constant $\dot{Q}$ corresponded to a steady $\dot{V}O_2$ as indicated by similar values at 15 and 45 min under both NC and FAN conditions.

*Figure 5.1: Mean (± SD) heart rate response during cycling at 60% $\dot{V}O_2_{\text{max}}$ and at maximum after a graded exercise test in No Cooling (NC) and FAN. * 15-min vs. 45-min value in NC, P < 0.05; † 15-min vs. 45-min value in FAN, P < 0.05; ‡ Significantly different from FAN at 45 min, P < 0.05; § Significantly different from FAN at maximum, P < 0.05.
Table 5.1: Responses to submaximal exercise under different conditions of body cooling (mean ± SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>No Cooling</th>
<th>Fan</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>15-min</td>
<td>45-min</td>
</tr>
<tr>
<td>( \dot{V}\text{O}_2 ) \text{(l min}^{-1})</td>
<td>2.71 ± 0.30</td>
<td>2.73 ± 0.25</td>
</tr>
<tr>
<td>( \dot{V}\text{O}_2 ) (% control ( \dot{V}\text{O}_2 \text{max})</td>
<td>59.4 ± 2.7</td>
<td>59.9 ± 2.9</td>
</tr>
<tr>
<td>( \dot{Q} ) \text{(l min}^{-1})</td>
<td>20.4 ± 1.8</td>
<td>20.8 ± 1.4</td>
</tr>
<tr>
<td>SV \text{(ml beat}^{-1})</td>
<td>137.0 ± 12.7</td>
<td>120.5 ± 8.8</td>
</tr>
<tr>
<td>O\text{2 pulse} \text{(ml beat}^{-1})</td>
<td>18.2 ± 2.0</td>
<td>15.8 ± 1.2</td>
</tr>
<tr>
<td>MAP \text{(mm Hg)}</td>
<td>109.9 ± 10.8</td>
<td>103.2 ± 12.7</td>
</tr>
<tr>
<td>SVR \text{(dyn cm s}^{-5})</td>
<td>433.7 ± 49.5</td>
<td>399.0 ± 54.0</td>
</tr>
<tr>
<td>( \Delta \text{SkBF from rest} ) (%)</td>
<td>331.5 ± 207.6</td>
<td>359.9 ± 228.2</td>
</tr>
<tr>
<td>( \Delta \text{PV from rest} ) (%)</td>
<td>-5.8 ± 2.1</td>
<td>-8.9 ± 2.7</td>
</tr>
<tr>
<td>( \overline{T}_{sk} ) \text{(°C)}</td>
<td>36.3 ± 0.3</td>
<td>36.0 ± 0.7</td>
</tr>
<tr>
<td>( \overline{T}_{b} ) \text{(°C)}</td>
<td>37.4 ± 0.3</td>
<td>38.3 ± 0.4</td>
</tr>
<tr>
<td>RPE</td>
<td>11.9 ± 1.0</td>
<td>13.9 ± 1.5</td>
</tr>
</tbody>
</table>

\( \dot{V}\text{O}_2 \), oxygen uptake; \( \dot{Q} \), cardiac output; SV, stroke volume; O\text{2 pulse}, oxygen pulse; MAP, mean arterial pressure; SVR, systemic vascular resistance; \( \Delta \text{SkBF} \), change in skin blood flow; \( \Delta \text{PV} \), change in plasma volume; \( \overline{T}_{sk} \), mean skin temperature; \( \overline{T}_{b} \), mean body temperature; RPE, rating of perceived exertion; * \( P < 0.05 \) comparing 15-min and 45-min values within the same condition; † \( P < 0.05 \) versus the value at the same time point in the Fan condition.
HR at max was higher in NC than FAN (P = 0.008), but no different compared to the other conditions (Table 5.2). HRmax during FAN followed a similar pattern and also was no different from control or 15max (P > 0.05). Despite reaching a higher HRmax in NC, \( \dot{\text{VO}}_2\text{peak} \) after 45 min was lower than that in FAN (P < 0.001), and this value represented an 18% decrease from 15max (P < 0.001). \( \dot{\text{VO}}_2\text{peak} \) also decreased significantly from 15max in FAN (P = 0.027), but the effect was smaller (-5.7%). A decrease in maximal power output of 17% accompanied the reduction in \( \dot{\text{VO}}_2\text{peak} \) during NC (P < 0.001), but maximal values were not different between 15max and FAN (P > 0.05).

*Thermoregulatory Responses.* \( T_{\text{re}} \) rose between 15 and 45 min during both NC and FAN (Figure 5.2, P < 0.001), but fan airflow blunted the rise during FAN such that \( T_{\text{re}} \) was lower after 45 min in that condition compared to NC (P = 0.006). During the GXT, \( T_{\text{re}} \) continued to rise in both conditions, but the final \( T_{\text{re}} \) was higher in NC (Figure 5.2, P < 0.001), and maximal core temperature in both conditions was higher than that in 15max (38.1 ± 0.4°C, P < 0.05). \( T_{\text{sk}} \) remained stable over time during NC (Table 5.1), while commencement of fan airflow lowered \( T_{\text{sk}} \) between min 15 and min 45 during FAN (P < 0.001) such that the values at 45 min (P = 0.006) and at maximum (P = 0.002) were lower in that condition than in NC. Skin blood flow (Tables 5.1 and 5.2) increased from rest over time in response to the rise in core body temperature, but after 15 min it remained stable for the rest of the submaximal and maximal exercise periods in both conditions. Because of the attenuation of the rise in \( T_{\text{re}} \) and the decrease in \( T_{\text{sk}} \) over time during FAN, \( T_{\text{b}} \) was about 0.5°C lower both after 45 min of submaximal exercise and after the maximal exercise bout in FAN compared to NC (P < 0.05).
Table 5.2: Responses to maximal exercise during a control graded exercise test (GXT) and peak responses during a GXT following 15 min of submaximal exercise and following 45 min of submaximal exercise under different conditions of body cooling (mean ± SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>15max</th>
<th>No Cooling</th>
<th>Fan</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\dot{V}_E$ (STPD, l min$^{-1}$)</td>
<td>124.9 ±</td>
<td>112.8 ±</td>
<td>94.4 ±</td>
<td>111.1 ±</td>
</tr>
<tr>
<td></td>
<td>10.8</td>
<td>14.3*</td>
<td>14.6*†‡</td>
<td>15.6*</td>
</tr>
<tr>
<td>$\dot{V}O_2$ (l min$^{-1}$)</td>
<td>4.55 ±</td>
<td>4.34 ±</td>
<td>3.56 ±</td>
<td>4.09 ±</td>
</tr>
<tr>
<td></td>
<td>0.40</td>
<td>0.42*</td>
<td>0.37*†‡</td>
<td>0.44*†</td>
</tr>
<tr>
<td>$\dot{V}O_2$ (ml kg$^{-1}$ min$^{-1}$)</td>
<td>63.3 ±</td>
<td>60.5 ±</td>
<td>49.4 ±</td>
<td>56.6 ±</td>
</tr>
<tr>
<td></td>
<td>5.5</td>
<td>5.8*</td>
<td>5.9*†‡</td>
<td>6.0*†</td>
</tr>
<tr>
<td>RER</td>
<td>1.08 ±</td>
<td>0.99 ±</td>
<td>0.96 ±</td>
<td>1.01 ±</td>
</tr>
<tr>
<td></td>
<td>0.05</td>
<td>0.04*</td>
<td>0.04*†‡</td>
<td>0.03*</td>
</tr>
<tr>
<td>RPE</td>
<td>18.6 ±</td>
<td>18.5 ±</td>
<td>18.6 ±</td>
<td>18.6 ±</td>
</tr>
<tr>
<td></td>
<td>0.7</td>
<td>0.8</td>
<td>1.1</td>
<td>0.7</td>
</tr>
<tr>
<td>$O_2$ pulse (ml beat$^{-1}$)</td>
<td>24.2 ±</td>
<td>23.0 ±</td>
<td>18.6 ±</td>
<td>21.8 ±</td>
</tr>
<tr>
<td></td>
<td>2.3</td>
<td>2.0*</td>
<td>1.7*†‡</td>
<td>2.1*†</td>
</tr>
<tr>
<td>$\Delta$ SkBF from rest (%)</td>
<td>—</td>
<td>—</td>
<td>363.9 ±</td>
<td>373.0 ±</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>263.9</td>
<td>196.1</td>
</tr>
<tr>
<td>$\Delta$ PV from rest (%)</td>
<td>—</td>
<td>—</td>
<td>-10.6 ±</td>
<td>-10.9 ±</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.6</td>
<td>3.2</td>
</tr>
<tr>
<td>$T_{sk}$ (°C)</td>
<td>—</td>
<td>—</td>
<td>36.2 ±</td>
<td>35.4 ±</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.5‡</td>
<td>0.7</td>
</tr>
<tr>
<td>$T_b$ (°C)</td>
<td>—</td>
<td>—</td>
<td>38.5 ±</td>
<td>38.0 ±</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.4‡</td>
<td>0.2</td>
</tr>
<tr>
<td>Test Duration (min)</td>
<td>13.1 ±</td>
<td>10.8 ±</td>
<td>6.8 ±</td>
<td>10.5 ±</td>
</tr>
<tr>
<td></td>
<td>2.4</td>
<td>1.3*</td>
<td>2.0*†‡</td>
<td>2.8*</td>
</tr>
<tr>
<td>Power Output (watts)</td>
<td>337.5 ±</td>
<td>308.0 ±</td>
<td>255.5 ±</td>
<td>305.5 ±</td>
</tr>
<tr>
<td></td>
<td>31.7</td>
<td>26.9*</td>
<td>29.8*†‡</td>
<td>36.1*</td>
</tr>
</tbody>
</table>

$\dot{V}_E$, minute ventilation; $\dot{V}O_2$, oxygen uptake; RER, respiratory exchange ratio; RPE, rating of perceived exertion; $O_2$ pulse, oxygen pulse; $\Delta$ SkBF, change in skin blood flow; $\Delta$ PV, change in plasma volume; $T_{sk}$, mean skin temperature; $T_b$, mean body temperature; * P < 0.05 compared with control; † P < 0.05 compared with 15max; ‡ P < 0.05 compared with Fan.
Figure 5.2: Mean (± SD) rectal temperature response during cycling at 60% $\dot{V}O_{2\text{max}}$ and at maximum after a graded exercise test in No Cooling (NC) and FAN. * 15-min vs. 45-min value in NC, $P < 0.05$; † 15-min vs. 45-min value in FAN, $P < 0.05$; ‡ Significantly different from FAN at 45 min, $P < 0.05$; § Significantly different from FAN at maximum, $P < 0.05$.

**Discussion**

The main objective of the current study was to determine if fan airflow directed at subjects during 45 min of constant-rate, moderate intensity exercise, would diminish the decline in $\dot{V}O_{2\text{max}}$ associated with CV drift in the heat. Body cooling via fan airflow was successful in mitigating CV drift to a similar extent as that seen in another study (Shaffrath and Adams 1984) incorporating fan airflow with a similar exercise protocol but in a cooler environment (24°C). When CV drift occurred to a greater extent (16% increase in HR and 12% decrease in SV) as in NC, $\dot{V}O_{2\text{max}}$ was reduced to a greater extent (18%), compared to when CV drift occurred to a lesser extent (4% increase in HR and 3% decrease in SV), as in FAN, and $\dot{V}O_{2\text{max}}$ was reduced to a lesser extent (5.7%). These findings are consistent with data from other studies manipulating the degree of CV
drift during prolonged exercise with fluid ingestion (Ganio et al. 2006), ambient temperature (Wingo et al. 2005a), and exercise intensity (unpublished observations). In all cases, the greater the magnitude of CV drift, the greater the decrement in \( \dot{V}O_{2\text{max}} \), and vice versa. Manipulation of CV drift under varying conditions of hyperthermia and hydration with similar effects on \( \dot{V}O_{2\text{max}} \) supports the hypothesis that a causal link exists between CV drift that occurs during prolonged exercise in a hot environment and a decrease in \( \dot{V}O_{2\text{max}} \), regardless of the primary factor contributing to CV drift.

While these data support a causal link between CV drift and decreased \( \dot{V}O_{2\text{max}} \), CV drift that occurs during prolonged, submaximal exercise in the heat may not be exclusively responsible for the decrease in \( \dot{V}O_{2\text{max}} \) observed in this and other studies with similar conditions (Ganio et al. 2006; Wingo et al. 2005a; Wingo et al. 2005b). For instance, data from other studies indicate physiological responses associated with factors, like hyperthermia and dehydration, occurring concurrently with and contributing to CV drift, are related to decrements in \( \dot{V}O_{2\text{max}} \) during heat stress (Gonzalez-Alonso and Calbet 2003; Mortensen et al. 2005; Nybo et al. 2001). These studies, however, employed short exercise protocols in which hyperthermia and/or dehydration were present from the start of exercise, not resultant from the exercise itself. The aforementioned studies of Ganio et al. (2006) and Wingo et al. (2005a; 2005b) have explored if and how these same factors limit \( \dot{V}O_{2\text{max}} \) after prolonged exercise in conjunction with CV drift. If the cardiovascular changes occurring with CV drift during prolonged exercise in the heat, viz., a decrement in SV, persist during the maximal exercise period, as some research suggests (Ekblom 1970; Saltin and Stenberg 1964), then CV drift appears to be one way the effects of
hyperthermia and dehydration on $\dot{VO}_{2\text{max}}$ are mediated during prolonged exercise in the heat.

While CV drift may be exacerbated with thermal strain and/or dehydration, greater effects on $\dot{VO}_{2\text{max}}$ seem to occur when the CV drift is more related to thermal strain than to dehydration. Compared to the current study, after two hours of cycling in 30°C with a similar magnitude of CV drift, similar $T_{re}$ at the end of submaximal exercise before the GXT, but with greater dehydration (3.7%), Ganio et al. (2006) found a 9% decrease in $\dot{VO}_{2\text{max}}$. Despite the shorter submaximal exercise duration in the current study, thermal strain, as indicated by $T_b$, was higher (because of higher $T_{sk}$), and the decrement in $\dot{VO}_{2\text{max}}$ was twice as large (18%). Similarly, thermal strain was greater in NC vs. FAN (higher $T_b$ at 45 min and at maximum) in the current study, and the decrement in $\dot{VO}_{2\text{max}}$ was three times as large. These deductions support the findings by Nybo et al. (2001) that during maximal exercise of short duration (without a prior submaximal exercise period involving CV drift), most of the decline in $\dot{VO}_{2\text{max}}$ with combined hyperthermia and dehydration is associated with hyperthermia. Furthermore, the differences in $\dot{VO}_{2\text{peak}}$ between NC and FAN in the present study support the conclusions of Arngrimsson et al. (2004) that the combination of high skin and core temperatures reduces $\dot{VO}_{2\text{max}}$.

The mechanism through which $\dot{VO}_{2\text{max}}$ was reduced in the NC and FAN conditions cannot be determined from the data collected in this study. A limitation is that we were unable to measure all components of the Fick equation at maximum in either condition. Since the decrement in $\dot{VO}_{2\text{max}}$ was different between NC and FAN, either 1) different mechanisms were operating in the respective conditions to lower $\dot{VO}_{2\text{max}}$, or 2)
the same mechanisms were operating, albeit at a different magnitude in FAN. We speculate #2 is the more likely explanation. $HR_{\text{max}}$ was similar between $15_{\text{max}}$ and each condition, and it was even higher in NC vs. FAN, despite a lower $\dot{VO}_{2\text{peak}}$ in NC. Therefore, HR was not the limiting factor, and attainment of maximal HR indicates cardiovascular capacity had been attained, which is improbable if leg fatigue had been a limiting factor. Maximal $(a-v)O_2$ would not likely be reduced at maximum under these conditions (Gonzalez-Alonso and Calbet 2003; Mortensen et al. 2005; Rowell et al. 1966; Williams et al. 1962). Decreased $SV_{\text{max}}$, consequently, was probably responsible for the reductions in $\dot{VO}_{2\text{max}}$ observed.

If decreased $SV_{\text{max}}$ was responsible, why was $\dot{VO}_{2\text{peak}}$ different between NC and FAN? We cannot be certain of the reason for this difference based on the data collected in this study, but as mentioned earlier in this discussion, we believe the CV drift that occurred during submaximal exercise persisted during the GXT such that SV at the end of submaximal exercise remained the same during the subsequent GXT, or if it increased it did so proportionally the same under each condition. $SV_{\text{peak}}$, therefore, would have been higher in the FAN condition, although this value would have been lower than that in $15_{\text{max}}$. Submaximal SV could have remained higher in FAN because of less blood shunted to skin for heat dissipation due to the cooling effect of the fan airflow, therefore leaving more blood available to the central circulation. Similar percent change in skin blood flow ($\%\Delta SkBF$) from rest in both NC and FAN does not support this conclusion, but it is possible that despite the similar $\%\Delta SkBF$ from rest in each condition, the absolute volume of blood shunted to skin could have been lower in FAN, so more blood would have been available to the central circulation. $SV_{\text{peak}}$ could have been lower in
either NC or FAN compared to 15max because of reduced central blood volume, central venous pressure, and end-diastolic volume associated with elevated skin blood flow at maximum (Rowell et al. 1966) or because of tachycardia limiting ventricular filling time and end-diastolic volume (Fritzsche et al. 1999; Gonzalez-Alonso and Calbet 2003). Additionally, the increases in myocardial contractility and vasoconstriction in nonactive muscle, splanchnic, and cutaneous vascular beds that sustain or increase SV could have been compromised at maximum, although to a greater extent in NC.

In conclusion, CV drift and the associated decrease in $\dot{V}O_{2\text{max}}$ after prolonged exercise in a hot environment are attenuated with body cooling via fan airflow. These findings, added to that of others showing similar effects on $\dot{V}O_{2\text{max}}$ with different manipulations of CV drift, support the hypothesis that a causal link exists between CV drift that occurs during prolonged exercise in a hot environment and a decrease in $\dot{V}O_{2\text{max}}$. Future studies exploring this relationship should determine if the decrement in SV concomitant with CV drift present at the end of submaximal exercise indeed persists during a subsequent GXT to exhaustion.
Acknowledgements

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References


CHAPTER 6
SUMMARY AND CONCLUSIONS

Cardiovascular drift (CV drift) is characterized by a progressive rise in heart rate (HR) and fall in stroke volume (SV) over time during prolonged, constant-rate, moderate-intensity (50–75% of maximal oxygen uptake, \( \dot{V}O_{2\text{max}} \)) exercise. The mechanisms underlying the cause of CV drift, while controversial, have been well-studied, but whether or not it has any consequences is unclear. Data from studies in which \( \dot{V}O_{2\text{max}} \) was measured following prolonged exercise in temperate environments suggests CV drift is associated with a decrease, albeit modest, in \( \dot{V}O_{2\text{max}} \). \( \dot{V}O_{2\text{max}} \) was not measured at the same points in time as the variables characterizing CV drift in these studies, however, so the effects on \( \dot{V}O_{2\text{max}} \) could not be temporally linked to CV drift. Furthermore, while CV drift occurs in both temperate and hot environments, greater effects occur in hot environments, which suggests any effect of CV drift on \( \dot{V}O_{2\text{max}} \) might be exacerbated in the heat.

The effect of CV drift on \( \dot{V}O_{2\text{max}} \) during prolonged exercise in the heat has implications for physical work capacity in the heat as well as implications for exercise prescription. If \( \dot{V}O_{2\text{max}} \) is lowered in conjunction with CV drift, any given absolute work level represents a higher relative metabolic intensity (%\( \dot{V}O_{2\text{max}} \)), and thereby physiologic strain. Additionally, reduced \( \dot{V}O_{2\text{max}} \) concomitant with CV drift suggests the rise in HR associated with CV drift during heat stress is representative of a given %\( \dot{V}O_{2\text{max}} \), and
therefore the HR–%\(\dot{V}O_{2\text{max}}\) relationship remains similar to that obtained in a cool environment.

The primary purpose of this research was to determine if CV drift that occurs during prolonged exercise in a hot environment results in a decrease in \(\dot{V}O_{2\text{max}}\), and furthermore, whether this relationship is cause and effect. A secondary aim was to establish whether the HR–%\(\dot{V}O_{2\text{max}}\) relationship was upheld under conditions when CV drift was prevented via alteration of exercise intensity.

Three individual studies were completed, and during each, CV drift was measured in active men during 45 min of cycling at 60% \(\dot{V}O_{2\text{max}}\) in 35°C, immediately followed by measurement of \(\dot{V}O_{2\text{max}}\). \(\dot{V}O_{2\text{max}}\) also was measured after 15 min of cycling in 35°C so that \(\dot{V}O_{2\text{max}}\) was measured during the same time interval (15 to 45 min) in which CV drift occurred. CV drift was manipulated with exercise intensity and body cooling to elucidate whether the effect on \(\dot{V}O_{2\text{max}}\) was causal.

CV drift of a substantial magnitude (12–16% increase in HR, 10–16% decrease in SV) occurred during 45 min of moderate-intensity, constant-rate cycling exercise in a hot environment, and this consistently resulted in a substantial decrease in \(\dot{V}O_{2\text{max}}\) (15–19%). When the magnitude of CV drift was smaller (0–4% increase in HR, 3% decrease in SV) because of reduced exercise intensity or body cooling, the decrease in \(\dot{V}O_{2\text{max}}\) was concomitantly smaller (5.7–7.5%). When CV drift occurred and \(\dot{V}O_{2\text{max}}\) was reduced, %\(\dot{V}O_{2\text{max}}\) increased from ~60% to ~80% between 15 and 45 min. When CV drift was attenuated and HR was held constant, %\(\dot{V}O_{2\text{max}}\) decreased from ~60% to ~50% over time.

The results of this research support the following conclusions:

1) Mean changes in HR and SV (CV drift) followed by proportional mean
changes in $\dot{VO}_{2\text{max}}$ across studies suggests CV drift may cause the reduction in $\dot{VO}_{2\text{max}}$ observed after prolonged, submaximal exercise in hot conditions.

2) The increase in $\%\dot{VO}_{2\text{max}}$ concurrent with CV drift suggests the rise in HR concomitant with CV drift is representative of increased relative metabolic intensity during prolonged exercise in a hot environment, so the HR–$\%\dot{VO}_{2\text{max}}$ relationship is preserved in the heat when CV drift occurs.

3) The decrease in $\%\dot{VO}_{2\text{max}}$ concurrent with attenuation of CV drift and maintenance of constant HR suggests the HR–$\%\dot{VO}_{2\text{max}}$ relationship is not preserved during exercise in the heat when CV drift does not occur.
APPENDIX A

STUDY 1 REPRINT PERMISSION

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Pam

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> Thanks
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Thanks for the clarification. That really does help. You have permission to do just as you describe below. No problem.
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>
> Thanks for your help
>
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