FLUID INGESTION ATTENUATES THE DECLINE IN $\dot{V}O_{2\text{max}}$

ASSOCIATED WITH CARDIOVASCULAR DRIFT

by

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(Under the Direction of Kirk J. Cureton)

ABSTRACT

To determine whether manipulation of cardiovascular drift (CV drift) by changing exercise duration or by fluid ingestion is associated with altered $\dot{V}O_{2\text{max}}$ during prolonged exercise, $\dot{V}O_{2\text{max}}$ was measured in 11 trained men immediately after they cycled at 60% control $\dot{V}O_{2\text{max}}$ for 15, 60 and 120 min with no fluid (15NF, 60NF, 120NF), or 120 min with fluid (120F). Markers of CV drift, stroke volume (SV) and heart rate (HR), were measured in 120NF and 120F at 15-, 60-, and 120-min time points. Subjects became 2.3%, 3.7%, and 0.7% dehydrated in 60NF, 120NF, and 120F. Compared to 15-min values, SV did not decline significantly after 60 min (6 ml, 4.6%, $P = 0.128$) but was reduced after 120 min (16 ml, 13.8%, $P < 0.001$) in 120NF. After 120 min, the decline in SV (3 ml, 2.1%) in 120F was significantly less than during 120NF ($P = 0.001$). Compared to 15NF, $\dot{V}O_{2\text{max}}$ was not decreased in 60NF (-1.2%, $P = 1.00$), but was lower in 120NF (-8.7%, $P = 0.016$). Fluid ingestion significantly reduced the decline in $\dot{V}O_{2\text{max}}$ (-1.9%, $P = 0.035$). Changes in $\dot{V}O_{2\text{max}}$ were correlated with changes in SV and HR during the preceding submaximal exercise ($r = 0.47$ and 0.64, SEE = 6.6 and 5.7). Manipulation of CV drift by altering exercise duration and fluid ingestion is associated with corresponding changes in $\dot{V}O_{2\text{max}}$. The results provide support for the hypothesis that, under these conditions, CV drift causes reduced $\dot{V}O_{2\text{max}}$. 
INDEX WORDS: Cardiovascular Drift, Maximal Oxygen Uptake, Heat, Dehydration, Stroke Volume, Cardiac Output, Skin Blood Flow, Heart Rate, Prolonged Exercise, Cycling, Fluid Ingestion
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CHAPTER 1
INTRODUCTION

Cardiovascular drift (CV drift) is a physiological phenomenon that has been described since the early 1960’s (15, 52). Rowell (44) defined CV drift as the progressive decline in stroke volume (SV), mean arterial pressure, and central venous pressure, and progressive rise in heart rate (HR) sufficient to maintain cardiac output (Q̇) at a constant level during prolonged exercise. He hypothesized that CV drift was due to pooling blood in the cutaneous veins, displacing central vascular volume peripherally, reducing central blood volume and venous pressure, resulting in reduced ventricular filling pressure and a fall in SV (44). In 1999, Coyle (17) provided an alternative explanation, proposing that the reduction in SV and increase in HR that occurs with prolonged exercise is caused by increased hyperthermia and sympathetic nervous system activity, which increase HR. The increase in HR reduces ventricular filling time and SV. Because of these opposing explanations, the mechanism underlying CV drift remains a topic of debate (10).

Regardless of the cause of CV drift, it is affected by many factors, including ambient temperature (19, 22, 24), blood volume (16, 23, 25, 36, 40), hydration level (21-24, 26, 37, 39), mode of exercise (38), training status (34), exercise intensity (56), air velocity (56), and previous exercise (54). Exercise in high ambient temperature causes a greater degree of CV drift than in a thermoneutral environment, apparently because of the greater increase in core temperature (Tcore) over time, which reduces central blood volume and increases HR (19, 22, 24, 40). When Tcore is controlled, change in blood volume affects the degree of CV drift (22). In plasma-volume expansion studies in which blood volume is maintained during exercise in both thermoneutral (40) and hot conditions (36, 40), CV drift is reduced. Reducing blood volume prior to exercise causes a larger degree of CV drift during exercise (16). Plasma-volume expansion prior to exercise does not prevent CV drift, but Q̇ and SV are higher and HR is lower than without plasma-volume expansion (23, 25). Thus, the degree of CV drift that occurs in thermoneutral
(26, 39) and hot conditions (21, 37) is directly related to the degree of dehydration and associated hyperthermia that occurs during prolonged exercise. A similar pattern of findings is observed when exercise is started in a dehydrated state with varying ambient (22-24) and core (22) temperatures. In long duration exercise (2 h) when dehydration is completely prevented by fluid ingestion, the increase in HR is attenuated and decrease in SV is eliminated over time in thermoneutral (26) and in hot conditions (21, 36).

Despite the many factors affecting CV drift, its implications for physical performance are unclear. It is not known whether or not the progressive rise in HR and fall in SV over time are benign and can be overcame if exercise intensity is increased, or whether they reflect altered circulatory capacity and are associated with a reduced maximal oxygen uptake ($\dot{V}O_{2\text{max}}$). A number of studies have reported that prolonged exercise (3, 7, 13, 52, 54) has modest detrimental effects on $\dot{V}O_{2\text{max}}$, but there have been few studies examining the relationship between $\dot{V}O_{2\text{max}}$ and the reduced SV that occurs with CV drift following prolonged exercise. Most studies observing modest reductions in $\dot{V}O_{2\text{max}}$ after prolonged exercise did not measure $\dot{V}O_{2\text{max}}$ at the same points in time as the variables characterizing CV drift (13, 52, 54). Wingo et al. (61), however, observed large reductions in $VO_{2\text{max}}$ measured immediately after 45 min of submaximal exercise during heat stress in which there was substantial CV drift.

Although studies have suggested the reduction in SV that occurs with CV drift is associated with reduced $\dot{V}O_{2\text{max}}$, it is not known if the relation is cause and effect. While it is known that CV drift occurs in a progressive fashion over time (56), it is not known if the reduction in $\dot{V}O_{2\text{max}}$ follows a similar tendency. Experimentation is needed in which the degree of CV drift during prolonged exercise is manipulated and the effect on $\dot{V}O_{2\text{max}}$ is measured. Fluid ingestion manipulations of SV, and thereby CV drift, that elicit corresponding and proportional changes in $\dot{V}O_{2\text{max}}$ would imply a causal link between CV drift and changes in $\dot{V}O_{2\text{max}}$. Likewise, establishing whether or not the time course of changes in SV and changes in $\dot{V}O_{2\text{max}}$ during prolonged exercise are similar or not would help determine if there is a causal link between the two.
Purpose

The primary purpose of this study was to determine whether CV drift is associated with a reduction in \( \dot{V}O_{2\text{max}} \) after prolonged exercise under conditions in which the magnitude of CV drift is manipulated. CV drift was manipulated in two ways. First, the association between CV drift and \( \dot{V}O_{2\text{max}} \) following two different durations of exercise in which different degrees of CV drift occurred was studied. This was accomplished by measuring CV drift between min 15 and min 60, and between min 15 and min 120 of cycling, and assessing the change in \( \dot{V}O_{2\text{max}} \) after 15, 60, and 120 min of exercise. Second, the magnitude of CV drift was manipulated with fluid ingestion. This was accomplished by performing another 120-min trial while ingesting fluid to replace sweat losses and measuring \( \dot{V}O_{2\text{max}} \) immediately after the 120 min.

Hypotheses

The research hypotheses for this study were:

1. The increase in HR and decline in SV are progressive between 15 min and 120 min, and these changes are associated with a progressive decrease in \( \dot{V}O_{2\text{max}} \).
2. Ingestion of fluid during 2 h of exercise attenuates CV drift and the reduction in \( \dot{V}O_{2\text{max}} \).

Significance of the Study

By manipulating the degree of CV drift and observing the resulting effects on \( \dot{V}O_{2\text{max}} \), this study provides insight into whether the reduction in SV during prolonged submaximal exercise causes a reduction in \( \dot{V}O_{2\text{max}} \). The relationship of CV drift and \( \dot{V}O_{2\text{max}} \) is important to help understand how cardiovascular adjustments during prolonged exercise affect the relative metabolic intensity. If the progressive rise in HR and fall in SV reduce \( \dot{V}O_{2\text{max}} \), but submaximal \( \dot{V}O_2 \) is unchanged, this means that the relative metabolic intensity (\%\( \dot{V}O_{2\text{max}} \)) increases with CV drift. The study also will provide new information on the metabolic consequences of fluid ingestion during prolonged exercise. If fluid ingestion that prevents CV drift prevents a reduction \( \dot{V}O_{2\text{max}} \), it would help explain a possible ergogenic effect. Fluid ingestion is common practice and recommended during exercise of similar durations and conditions as this study (1).
Limitations of the Study

The sample of this study was drawn from local cyclists who train in the surrounding areas. Their relatively-high fitness level makes the results difficult to generalize to less-fit, recreational athletes. The exercise mode used in this study was cycling, so the results may not be generalized to other exercise modes such as running. Also, the results may not be applicable for populations other than those tested, including women and unacclimated individuals.
In this chapter, literature of CV drift and its association with altered \( \dot{V}O_{2\text{max}} \) during prolonged exercise is reviewed.

**Cardiovascular Drift**

CV drift is the progressive decline in SV, mean arterial pressure, and central venous pressure, and progressive rise in HR sufficient to maintain \( \dot{Q} \) at a constant level during prolonged exercise (44). It was first described and studied in the 1960’s, and there was speculation about its cause. Later, experimental investigations assessed the effects of various factors on CV drift, which provided much greater insight into its cause.

In one of the first studies measuring variables defining CV drift, Saltin and Stenberg (52), in 1964, had subjects cycle for 195 min at 75\% \( \dot{V}O_{2\text{max}} \). During the submaximal exercise in 19°C, the subjects became 3-5\% dehydrated. HR increased 23 bpm, SV decreased 9.5\%, and \( \dot{Q} \) increased slightly. At the end of submaximal exercise, rectal temperature (\( T_r \)) ranged from 38.0-38.6°C. The proposed reason for declines in SV over time was “impaired performance of the heart.”

Also in 1964, Ekelund and Holmgren (15) observed an increase in HR of ~20 bpm, a SV decline of 17 ml (13.8\%), and a stable \( \dot{Q} \) during 1 h of cycling at ~60\% \( \dot{V}O_{2\text{max}} \) in thermoneutral conditions. The reductions in SV were hypothesized to be a direct result of reductions in end-diastolic volume or a “defect” in the emptying of the heart. The cause of these changes was hypothesized to be redistribution of central blood volume for reasons of heat regulation, but they had no experimental evidence to support these hypotheses.

In order to investigate the effect of posture on the central blood volume during long-duration exercise as a possible reason for reductions in SV, Ekelund (14) in 1966, studied CV drift in subjects cycling for 1 h in a supine position. Over the exercise bout, HR increased 14 bpm, SV declined ~10 ml, and \( \dot{Q} \) remained stable. It was concluded that cardiovascular changes...
were still a result of the redistribution of central blood volume during exercise, but the degree of change was less in the supine position than upright (15) because initial blood distribution is dependent on posture. Regardless of differences, the same phenomenon was observed with no experimental evidence to help understand the mechanism underlying cardiovascular changes during prolonged exercise.

It was not until 1975 that support for a mechanism underlying CV drift was provided. Johnson and Rowell (29) measured forearm blood flow over 1 h of exercise as subjects cycled in 24°C. CV drift, evidenced by a drift in HR of 28 bpm, occurred during the exercise bout. Forearm arm blood flow continually increased over time and provided evidence that central blood volume decreased as blood was diverted to the periphery. It was proposed that this shift of central blood volume reduced central venous return, which decreased end-diastolic volume and, in turn, decreased SV and resulted in CV drift. It was also observed that the initial vasoconstriction of blood flow to inactive muscle was maintained throughout 1 h and that esophageal temperature (T_{es}) rose throughout the exercise bout.

For the 25 years after this evidence was presented, it was well accepted that CV drift, the decline in SV during prolonged exercise, was caused by a progressive increase in cutaneous blood flow (8). In 1999, Fritzsche et al. (17) provided an alternative hypothesis for the mechanism underlying CV drift. Subjects cycled for 1 h at 57% \dot{VO}_{2\text{max}} while ingesting a \beta-blocker to prevent the increase in HR or a placebo. From 15-55 min of exercise with a placebo, HR increased 11% while SV decreased 13%. Ingesting a \beta-blocker resulted in HR and SV not changing over the same time period. Because both conditions resulted in the same increase of cutaneous and forearm blood flow, it was hypothesized that reductions in SV with CV drift are not a result of a redistribution of central blood volume to the periphery, but instead, a direct consequence of the increase in HR.

Despite conflicting hypotheses on the causes of CV drift, a number of studies over the years have observed CV drift under different conditions and manipulations. It has been shown that the increase in T_{core} (hyperthermia) is closely linked to CV drift (17, 29). Blood volume
reduction resulting from dehydration also is linked closely to CV drift (22). Blood volume can be manipulated before or during exercise, and with fluid ingestion or plasma-volume expansion to result in different degrees of CV drift. Dehydration often results in hyperthermia and few studies tease these two factors apart (24). Because dehydration and hyperthermia occurred in the present study, it is the goal of this review to understand their affects. It is important to know how CV drift is affected by different levels of blood volume and hyperthermia regardless if changes in these factors occur before or during prolonged exercise.

**Blood volume and hyperthermia prior to exercise.** In order to understand how blood volume affects the cardiovascular system, studies have used plasma-volume expansion as a direct way of increasing blood volume. Like fluid ingestion, plasma-volume expansion has been used to study the affect of varying blood volumes prior to and during exercise on CV drift.

One of the first studies to examine the effects of plasma-volume expansion prior to exercise in the heat was conducted by Fortney et al. (16) in 1983. Subjects exercised for 30 min at 65-70% \( \dot{V}O_{2\text{max}} \) in 30°C after either having blood volume reduced 8.7% with diuretics (blood volume reduced) or expanded 8.5% with saline infusion (plasma-volume expansion). Cardiovascular measures at the beginning of exercise provided evidence that compared to control conditions, SV and \( \dot{Q} \) were 14% and 13% lower, respectively, with reduced blood volume, and 11% and 8% higher, respectively, in plasma-volume expansion. Initial HR did not differ between conditions. Although initial SV and \( \dot{Q} \) differed, CV drift occurred to the same degree in each condition. HR increased about 15% and SV and \( \dot{Q} \) decreased about 16% and 5%, respectively. Reduced blood volume resulted in higher \( T_{re} \) (38.9°C) than control (38.6°C), while plasma-volume expansion resulted in lower \( T_{re} \) (38.3°C).

In 1997 in thermoneutral conditions (23°C), Grant et al. (25) found that plasma-volume expansion of different degrees (14% or 21%) prior to 2 h of light exercise (46% \( \dot{V}O_{2\text{max}} \)), reduced the initial increase in HR and decrease in SV, but did not prevent CV drift. In control conditions SV decreased ~13%, HR increased 18%, and \( \dot{Q} \) remained constant. After 15 min, plasma-volume expansion in both conditions (14% and 21%) had increased SV 14%, reduced HR by 4%, and
increased \( \dot{Q} \) 10% compared to control. From 15 min to 2 h, these cardiovascular differences persisted while the same degree of CV drift occurred as in the control. \( T_{re} \) increased to same degree (37.6°C to 38.7°C) and did not differ between conditions at any time point.

In 1997, Gonzalez-Alonso et al. (22) was the first to separate the individual effects of hyperthermia and dehydration on CV drift. Exercise was used to manipulate blood volume. Heat-acclimated subjects exercised for up to 2 h at 60% \( \dot{V}O_{2\text{max}} \) while remaining euhydrated with fluid ingestion or becoming dehydrated by 4%. Subjects then exercised for 30 min at 71% \( \dot{V}O_{2\text{max}} \) in 23°C while euhydrated, 35°C while euhuydrated, 35°C while dehydrated, 2°C while dehydrated, or 2°C while given plasma-volume expansion. Trials conducted in 35°C resulted in hyperthermia over the 30 min. Hyperthermia alone and dehydration alone equally reduced SV by \( \sim \)8% and increased HR by \( \sim \)5%. The combination of dehydration and hyperthermia reduced SV by 20% and increased HR by 9% which resulted in \( \dot{Q} \) being reduced 13%. Trials with plasma-volume expansion in the cold provided evidence that when dehydration does not result in hyperthermia, the reduced SV is due solely to loss of blood.

In 1999, Gonzalez-Alonso et al. (23) used supine exercise as means of plasma-volume expansion. Subjects cycled for 30 min at 62% \( \dot{V}O_{2\text{max}} \) in 35°C while upright or supine. Cardiovascular measures were made in the last 10 min of exercise. Prior to testing, subjects exercised for 2 h at 62% \( \dot{V}O_{2\text{max}} \) in 35°C while remaining euhydrated or becoming 5% dehydrated. In upright exercise, dehydration resulted in SV being 21% lower, HR 9% higher, and \( \dot{Q} \) 14% lower than euhuydrated. Dehydration had less of an affect on supine exercise and resulted in SV being 13% lower, HR 7% higher, and \( \dot{Q} \) 8% lower than euhydrated. While euhydrated, supine exercise restored two-thirds the decline in SV and prevented one-third the increase in HR that occurred in upright exercise. While dehydrated, supine exercise attenuated the increase in HR by 2%, the decrease in SV and \( \dot{Q} \) by 8% and 6%, respectively, that occurred in upright exercise. Supine exercise as a means of plasma-volume expansion did not affect \( T_{re} \), but regardless of exercise position, dehydration resulted in higher temperatures (38.6°C) than
euhydration (37.9°C) and may have contributed to higher degrees of CV drift in dehydrated conditions.

Using the same dehydration protocol (22), Gonzalez-Alonso et al. (24) in 2000, kept subjects euhydrated or dehydrated them by 1.5%, 3.0% or 4.2% and then had them cycle at 72% \( \dot{V}O_{2\text{max}} \) for 30 min in 35°C or 8°C. Cardiovascular measures were made during the last 10 min of the exercise bout. In 35°C, the average \( T_{es} \) was 0.2°C higher for each 1% increase in dehydration and showed that hyperthermia and dehydration are correlated. SV was the same when euhydrated regardless of environmental temperature even though skin blood flow (SkBF) was greater in 35°C. In 35°C and 1°C, each 1% dehydration resulted in SV being reduced 6.4 ml and 3.4 ml, respectively. HR was about 12 bpm higher in 35°C compared to 8°C at each level of dehydration, and each 1% increase in dehydration resulted in an increase of 3.5 bpm independent of temperature. When euhydrated, \( \dot{Q} \) was higher in 35°C than in 8°C and increased SkBF without a reduction in SV. \( \dot{Q} \) was lowered in proportion to dehydration level when exercising in 35°C but not in 8°C.

In conclusion, plasma-volume expansion prior to exercise initially decreases HR and increases SV and \( \dot{Q} \), but as these differences persist, the same degree of CV drift occurs as without plasma-volume expansion. This seems to occur in both thermoneutral (25) and hot (16, 23) environments and short (16, 23) and long-duration exercise (25). Plasma-volume expansion does not result in different degrees of hyperthermia in thermoneutral conditions (25), but decreases hyperthermia when exercising in the heat (16), except when supine exercise is used for plasma-volume expansion (23). It can also be concluded that at least one-half of the decline in SV with dehydration prior to moderate-intensity exercise in the heat is not due solely to elevated SkBF. One-half of the reduction in SV in the heat is a result of increased HR and hyperthermia (which may result in increases of SkBF), while the other half is a result of a reduction in blood volume. In the cold, in which hyperthermia during exercise does not occur, reduced blood volume is the main factor responsible for reduction in SV (22, 24). Also, the degree of increase
in HR and hyperthermia and reductions in SV and \( \dot{Q} \) are related to the degree of dehydration (23, 24).

**Blood volume and hyperthermia during exercise.** The present study uses conditions that result in changes in blood volume and hyperthermia during exercise, so it is important to understand how these factors, when manipulated to different degrees, affect CV drift. One of the first studies investigating the effects of dehydration during exercise on CV drift was conducted by Gliner et al. in 1975 (19). By having subjects cycle 3.5 h in either 25°C or 35°C at a low intensity (35% \( \dot{VO}_{2\text{max}} \)), different degrees of dehydration occurred (1.9% and 3.2% in 25°C and 35°C, respectively). Because there were no differences in \( T_{\text{re}} \) between conditions, the degree of CV drift that occurred was a result of dehydration and not hyperthermia. SV decreased 7% more (15% vs 22%) and HR increased 14% more (11% vs 25%) when larger degrees of dehydration occurred. Despite the varying levels of CV drift, \( \dot{Q} \) remained relatively stable in both temperatures over time and did not differ from one another.

In 1979, Sawka et al. (53) observed that accumulating dehydration and hyperthermia during subsequent exercise bouts increased the degree of CV drift. In ~24°C, subjects ran twice for 80 min at 70% \( \dot{VO}_{2\text{max}} \) with 90 min rest between runs. The subjects became dehydrated 2% during each run and thus accumulated 4% dehydration by the end of the second run. \( T_{\text{re}} \) was 39.7°C and 40.3°C at the end of each run, respectively. During the first run, HR increased 4%, SV decreased 10%, and \( \dot{Q} \) decreased 8%. After 20 min of the second run, HR, SV, and \( \dot{Q} \) were at the same values as they were at the end of the first run, but during the remainder of the run, HR increased 6%, SV decreased 11% and \( \dot{Q} \) decreased 6% further.

In a study examining the effects of airflow and work intensity on CV measures, Shaffrath and Adams (56) had subjects cycle for 70 min in 24°C with either a fan directed on the subject (4.3 m/s) or no fan and either at ~ 40% or ~ 60% \( \dot{VO}_{2\text{max}} \). At 60% \( \dot{VO}_{2\text{max}} \) the fan and no fan resulted in HR increasing 6% and 16%, respectively, SV decreasing 2% and 12%, respectively, and \( \dot{Q} \) being maintained in all trials but consistently lower with no fan. Hyperthermia was greater without a fan, as evidenced by ending \( T_{\text{re}} \) being 0.3°C higher (38.6°C vs 38.3°C). Because of the
very low work load at 40% $\text{VO}_2\text{max}$, cardiovascular measures and $T_{\text{re}}$ were largely unaffected over time regardless of fan or not. SkBF increased in 60% no fan, while 40% no fan and 60% fan resulted in similarly stable SkBF over time. It was concluded that SkBF was higher with no fan, and this caused a decrease in central blood volume and thus a higher degree of CV drift.

In 1990, Nose et al. (40) studied the effects of temperature on plasma-volume expansion by having subjects exercise in either 22°C or 30°C for 50 min at 60% $\text{VO}_2\text{max}$. From 20-50 min of exercise, saline was either infused at a constant rate to maintain blood volume or not. In 22°C, HR increased ~10% regardless of infusion. In 30°C, HR increased 18% with no infusion and only 13% with infusion. Other cardiovascular measures were not measured. After 20 min SkBF was higher in 30°C than in 22°C and remained this way throughout the exercise bout regardless of infusion. In 30°C, SkBF reached a plateau without infusion and increased with infusion. In 22°C, SkBF increased regardless of infusion but increased to a greater extent with infusion. $T_{\text{es}}$ was higher in 30°C throughout exercise regardless of infusion, but within each environment infusion did not alter $T_{\text{es}}$.

In the early 1990’s, a series of studies investigated CV drift over 2 h of exercise when performed in thermoneutral and hot conditions, with varying degrees of dehydration, and when varying volumes of fluid where ingested when exercising.

In 1991, Hamilton et al. (26) investigated the effects of fluid ingestion and hyperthermia on CV drift in thermoneutral conditions. Subjects cycled in 22°C for 2 h at 70% $\text{VO}_2\text{max}$ while either receiving no fluid or enough fluid to replace sweat losses. This resulted in 2.9% dehydration in the no-fluid condition and no body weight change with fluid replacement. $\dot{Q}$ and SV decreased 7% and 15%, respectively while HR increased 10% with no fluid. With fluid replacement, $\dot{Q}$ increased 7%, SV did not change, and HR increased 5%. Fluid replacement attenuated the rise in $T_{\text{re}}$ in the 2nd h of exercise and resulted in $T_{\text{re}}$ of 38.3°C and 38.9°C after 2 h of cycling with fluid and no-fluid replacement, respectively. It was hypothesized that fluid replacement prevented the decline in SV because it prevents hyperthermia by means of increasing SkBF, but direct evidence supporting the hypothesis was not available until 1992.
In 1992, Montain and Coyle (37) investigated the effects of different degrees of dehydration and hyperthermia in hot conditions on CV drift. Subjects cycled at 65% \( \dot{\text{VO}}_2\text{max} \) for 2 h in 33°C while receiving different amounts of fluid that resulted in 4.2% (no fluid), 3.4%, 2.3%, or 1.1% (fluid) weight loss. Fluid attenuated the decline in SV, and increase in HR and \( T_{re} \) in proportion to the volume of fluid ingested during exercise. After 2 h with no fluid, SV had declined 27%, HR increased 22%, and \( T_{re} \) reached 39.1°C. Fluid resulted in SV decreasing ~9%, HR increasing ~8%, and \( T_{re} \) reaching 38.4°C. \( \dot{Q} \) decreased 11% with no fluid, but was maintained with fluid. It was concluded that the magnitude of increase in \( T_{re} \) and HR and decline in SV and \( \dot{Q} \) were directly related to levels of dehydration. Increased SkBF with fluid attenuated hyperthermia and thus contributed to the attenuation of the decline in SV.

Plasma-volume expansion during exercise in the heat was studied by Montain and Coyle (36) in 1992. Subjects cycled for 2 h at 62% \( \dot{\text{VO}}_2\text{max} \) in 33°C while either ingesting no fluid, fluid to replace 80% of sweat losses, or receiving equivalent amounts of Dextran® to expand plasma volume. HR increased 24%, 18%, and 9% with no fluid, plasma-volume expansion, and fluid, respectively. SV declined 27%, 17% and 9% with no fluid, plasma-volume expansion, and fluid, respectively. Therefore, the 11% decline in \( \dot{Q} \) with no fluid was attenuated with fluid and plasma-volume expansion. Despite preventing CV drift, plasma-volume expansion did not attenuate the increase in \( T_{re} \), whereas fluid did, compared to control. This difference could be attributed to a 16% increase in SkBF with fluid, while SkBF with plasma-volume expansion and no fluid did not increase nor differ from each other over time. Therefore, fluid ingestion during exercise increased SkBF independent of increases in blood volume.

Another study observing the effects of hyperthermia and dehydration on CV drift was conducted by Gonzalez-Alonso et al. (21) in 1995. Subjects cycled for 2 h in 35°C at 62% \( \dot{\text{VO}}_2\text{max} \) while becoming dehydrated 4.9% or remaining euhydrated with fluid ingestion. After 2 h, \( \dot{Q} \) was reduced 14% when dehydrated but was increased 3% when euhydrated. These changes with dehydration were a result of SV declining to a greater degree (28%) than HR increasing (19%). When euhydrated, SV was maintained while HR increased 6%. Ending \( T_{re} \)
was 39.2°C and 38.0°C when dehydrated and euhydrated, respectively, so hyperthermia also contributed to differences in CV drift. SkBF was maintained throughout exercise when euhydrated and dropped 20% when dehydrated. The changes in SkBF were a result of mean arterial pressure (MAP) and systemic vascular resistance (SVR) being maintained when euhydrated. When dehydrated, increases in SVR maintained MAP and resulted in reduced SkBF. It can be concluded that in this study, large reductions in SV with severe dehydration and hyperthermia were due to factors other than elevated SkBF.

More recently (2002), Nassis and Geladas (39) had subjects cycle or run and ingest fluid (60% of sweat lost) or not for 90 min at 60% \( \dot{V}O_{2\text{max}} \) in 23°C. With no fluid, subjects became dehydrated 2.8% and 3.3% when cycling and running, respectively. \( \dot{Q} \) was maintained in fluid trials regardless of exercise mode, but was reduced 12% and 3.5% while cycling and running with no fluid, respectively. SV decreased 9% regardless of exercise mode with fluid and decreased 21% and 13% while cycling and running with no fluid, respectively. HR increased 11% with no fluid, regardless of exercise mode, but with fluid, HR increased 9% and 8% while cycling and running, respectively.

The degree of CV drift that occurs during long-duration exercise is directly related to the degree of dehydration and hyperthermia that occurs. This is true in thermoneutral conditions (19, 26, 53) and in the heat (21, 36, 37). SV decline is prevented and the increase in HR is attenuated if fluid replacement is sufficient to prevent dehydration and hyperthermia in thermoneutral conditions (26) and in the heat (21). It appears that plasma-volume expansion while exercising in a thermoneutral environment has little effect on CV drift (40), but in the heat CV drift is attenuated (36, 40). Hyperthermia is not attenuated to the same degree with plasma-volume expansion as it is with fluid replacement (36, 40), and the reason may be because plasma-volume expansion does not increase SkBF in some conditions (36).

**Maximal Oxygen Uptake**

\( \dot{V}O_{2\text{max}} \) is the highest rate of oxygen that can be taken up by the blood during physical work and is a measure of the maximal capacity to transport oxygen to the tissues of the body
(27). It is a very stable and highly reproducible measure that is unique to an individual, but is altered by conditions that affect cardiovascular or respiratory capacity on the active muscle’s ability to carry out oxidative phosphorylation (45). Because the present study involves hyperthermia, dehydration, and prolonged exercise, it is important to review how $\dot{V}O_2\text{max}$ is affected by these factors.

**Hyperthermia.** As discussed above, hyperthermia is closely linked to CV drift, and thus it is important to understand how hyperthermia effects $\dot{V}O_2\text{max}$. In 1955, Taylor et al. (58) found a reduction in $\dot{V}O_2\text{max}$ in the heat when subjects walked for 60 min at a light intensity, and then performed a $\dot{V}O_2\text{max}$ test in 25.5°C and 32°C. Although degrees of hyperthermia were not measured, it can be assumed that hyperthermia occurred to a greater extent in 32°C. There was a small (4%) but significant reduction in $\dot{V}O_2\text{max}$ at 32°C.

In 1962, Williams, et. al (60), after measuring $\dot{V}O_2\text{max}$ in control conditions (21°C), had subjects become heat acclimatized and then retested in the heat (36°C). A $\dot{V}O_2\text{max}$ protocol was used in which cardiovascular measures were taken at different stages over a testing period that lasted about 1 h. Despite the long protocol and differences in $T_r$ between the two trials, there was no reduction in $\dot{V}O_2\text{max}$ in 36°C compared to 21°C.

In a study by Rowell et al. (48) in 1966, to test cardiovascular changes at different intensities up to $\dot{V}O_2\text{max}$ and different temperatures, unacclimated subjects were tested in 43°C and 26°C. $T_r$ increased at the same rate regardless of ambient temperature (0.03°C per min), but ending $T_r$ was 1.2°C higher in 43°C (39.4°C vs 38.2°C). Despite these differences and a mean decrease in $\dot{V}O_2\text{max}$ of 2.6%, there was not a statistical significance between $\dot{V}O_2\text{max}$ in the two temperatures. Around this same time, Klausen et al. (31) also observed a significant reduction of 3.4% (compared to 25°C) when subjects performed a $\dot{V}O_2\text{max}$ test in 39°C, but degrees of hyperthermia were not measured.

In 1969, Rowell et al. (47) had 3 subjects preheat by walking for 15 min at 10% grade, 3.5 mph in 49°C or 26°C, and then perform a $\dot{V}O_2\text{max}$ test. $\dot{V}O_2\text{max}$ was not reduced in one subject but was reduced 10% and 14% in the other subjects at 49°C. In the same study, Rowell had 27
subjects perform a \( \dot{\text{V}}\text{O}_2\text{max} \) test at 43°C and 26°C without preheating. \( \dot{\text{V}}\text{O}_2\text{max} \) at 43°C was reduced significantly by 3% compared to 26°C. Hyperthermia (> 39°C) occurred in the high ambient temperatures, and it was found that \( T_{\text{re}} \) at \( \dot{\text{V}}\text{O}_2\text{max} \) depended on the duration of the subject’s warm-up prior to the heat and thus could have contributed to the reductions in \( \dot{\text{V}}\text{O}_2\text{max} \).

Around the same time, Pirnay et al. (42) also systematically examined the effects of preheating on \( \dot{\text{V}}\text{O}_2\text{max} \). Subjects performed a \( \dot{\text{V}}\text{O}_2\text{max} \) test in thermoneutral conditions (23°C), immediately after entering an environment of 37°C, or after a 20 min warm up (41% of control \( \dot{\text{V}}\text{O}_2\text{max} \)) in 37°C. Compared to thermoneutral conditions, \( \dot{\text{V}}\text{O}_2\text{max} \) in an environment of 37°C was reduced 25% only when preceded by a warm up. The condition involving warm up resulted in \( T_{\text{core}} \) being 39.2°C at \( \dot{\text{V}}\text{O}_2\text{max} \).

In 1978 Sakate (49), in order to observe the effects of ambient temperature on \( \dot{\text{V}}\text{O}_2\text{max} \), had subjects perform a graded exercise test without preheating. Upon entering an environment of either 5°C, 20°C, 35°C, or 50°C. The brief exposure to these temperatures resulted in \( \dot{\text{V}}\text{O}_2\text{max} \) being highest in 35°C and 6% lower than that in 50°C. So, despite lack of preheating, very high ambient temperatures lowered \( \dot{\text{V}}\text{O}_2\text{max} \), but unfortunately \( T_{\text{core}} \) was not measured, so it is not known the degree to which subjects became hyperthermic in each condition.

In 1985, in a controlled heat-acclimation study, Sawka et al. (55) measured a 7-8% reduction in \( \dot{\text{V}}\text{O}_2\text{max} \) in the heat regardless of acclimation state. Subjects performed a \( \dot{\text{V}}\text{O}_2\text{max} \) test in a moderate (21°C) and hot (49°C) environment before and after a 9-day acclimation protocol. Even though the subjects sat in the respective environment for 20 min before performing the \( \dot{\text{V}}\text{O}_2\text{max} \) test, elevation in \( T_{\text{re}} \), was not correlated to a reduction in \( \dot{\text{V}}\text{O}_2\text{max} \). Because of the short duration of the \( \dot{\text{V}}\text{O}_2\text{max} \) test, dehydration also was not probably the cause of reduction.

In 2003, in one of the few studies with cardiovascular measures at \( \dot{\text{V}}\text{O}_2\text{max} \), Gonzalez-Alonso and Calbet (20) had subjects cycle to \( \dot{\text{V}}\text{O}_2\text{max} \) either starting with normal mean skin temperature (\( T_{\text{sk}} \)) and \( T_{\text{es}} \) or \( T_{\text{sk}} \) and \( T_{\text{es}} \) elevated 10°C. Despite starting hyperthermic, cycling resulted in similar ending \( T_{\text{es}} \) between conditions. It was shown that \( \dot{\text{V}}\text{O}_2\text{max} \) was reduced 8% in the heat because there was a more rapid decline in \( \dot{Q} \) and MAP, which reduced exercising muscle
blood flow, O\textsubscript{2} delivery and O\textsubscript{2} uptake. This provided evidence that the cardiovascular system reaches capacity before exercising muscles. The decline in SV caused the drop in $\dot{Q}$, but it is important to note that both conditions had a drop of $\dot{Q}$ at $\dot{VO}_{\text{2max}}$. The heat brought about the drop quicker. This study provides evidence that in the heat, SV declines quicker and results in a reduced $\dot{VO}_{\text{2max}}$. So, it appears that changes in SV affect $\dot{VO}_{\text{2max}}$, but it is not known how a reduction in SV as a result of CV drift, affects $\dot{VO}_{\text{2max}}$.

More recently, in 2004, Arngrimsson et al. (2) tested the effects of different ambient temperatures and preheating protocols on $\dot{VO}_{\text{2max}}$ in men and women. Subject’s control $\dot{VO}_{\text{2max}}$ was compared to $\dot{VO}_{\text{2max}}$ after walking at a light intensity (~33\% control $\dot{VO}_{\text{2max}}$) and short duration (20 min) in either 25°C, 35°C, 40°C, or 45°C. There were also conditions in which the subject performed a $\dot{VO}_{\text{2max}}$ test in 45°C with no preheating and one in which passive preheating occurred at 45°C until $T_{es}$ and $T_{sk}$ were increased to the same degree as at the end of the 20-min walk in 45°C. Compared to control, $\dot{VO}_{\text{2max}}$ was reduced by a small amount in 35°C after preheating (3-5\%), by a modest amount in 40°C after preheating and 45°C without preheating (~9\%), and by a large amount in 45°C with preheating (17-19\%). Large reductions in $\dot{VO}_{\text{2max}}$ occurred when there were large degrees of hyperthermia.

In studies that observe a reduction in $\dot{VO}_{\text{2max}}$ in the heat, it has been suggested that the reduction in $\dot{VO}_{\text{2max}}$ was related to lower SV at $\dot{VO}_{\text{2max}}$ (2, 20, 42, 49). It is hypothesized that the need to thermoregulate overwhelms the body and causes large degrees of cutaneous vasodilation. This reduces central blood volume, lowering maximal $\dot{Q}$, and thus lowering maximal skeletal blood flow and reduced oxygen delivery to the active muscles, limiting $\dot{VO}_{\text{2max}}$ (31, 42, 47, 49, 55). Because maximum HR was the same in each condition, and arteriovenous oxygen difference [$\text{(a-v)O}_2$ difference] has been observed to not be reduced at $\dot{VO}_{\text{2max}}$ (20, 48, 60), reduced SV is probably the cause of reduction in $\dot{VO}_{\text{2max}}$.

Dehydration. Because dehydration frequently occurs during exercise, and contributes to CV drift, it is important to know whether a reduction in blood volume by dehydration affects $\dot{VO}_{\text{2max}}$. Many of the previous studies reviewed observing a reduction in $\dot{VO}_{\text{2max}}$ in the heat either
controlled for dehydration (20), or used protocols that were too short to result in a significant amount of dehydration (2).

In 1964, in order to assess $\dot{V}O_2$\textsubscript{max} after controlled dehydration, Saltin (50) dehydrated subjects 2.4-6.5% by means of exercise in thermoneutral conditions, exercise in a hot environment (36°C-38.5 °C), or passive heating in a hot environment (80°C). Despite the significant dehydration in each condition, $\dot{V}O_2$\textsubscript{max} was not altered when measured 1.5 h after the dehydration protocol.

In the same year, in a study by Saltin (51), cardiovascular measures were made before and 1.5 h after subjects were dehydrated up to 5.2% body weight in a hot (80°C) sauna bath. Despite a 25% reduction in plasma volume, $\dot{V}O_2$\textsubscript{max} was not influenced by dehydration. SV was lower in submaximal exercise after dehydration, but then was apparently restored to pre-dehydration values as subjects approached $\dot{V}O_2$\textsubscript{max}.

The results of these two studies are hard to interpret because of the long period of time between the dehydration phase and the $\dot{V}O_2$\textsubscript{max} testing. Fluid shifts and body temperature changes would have occurred in this amount of time, compensating for lost central circulating blood volume. Regardless, it seems that dehydration alone does not cause reductions in $\dot{V}O_2$\textsubscript{max}.

Hyperthermia and Dehydration. A combination of hyperthermia and dehydration are known to affect CV drift, and thus it is important to know whether these same factors affect $\dot{V}O_2$\textsubscript{max}. In 1966, Craig and Cummings (11) found that $\dot{V}O_2$\textsubscript{max} was greatly reduced when measured in the heat after being dehydrated. Subjects performed a $\dot{V}O_2$\textsubscript{max} test in 46°C, immediately after sweating for 6 h (in 46°C) while either replacing sweat losses or restricting water intake. Dehydration of ~4% and 2% in the two conditions resulted in reductions in $\dot{V}O_2$\textsubscript{max} of 27% and 10%, respectively, and were related to the fact that subjects were more hyperthermic when dehydrated.

In 2001, in an attempt to separate out the independent effects of hyperthermia and dehydration on $\dot{V}O_2$\textsubscript{max}, Nybo et al. (41) had subjects perform light exercise (50% control $\dot{V}O_2$\textsubscript{max}) for 2 h while either becoming 4% dehydrated or remaining euhydrated by ingesting
fluid. After 1 h of rest, the subjects performed a $\dot{V}O_{2\max}$ test while wearing a water-perfused vest with circulating water at 14°C or 44°C. $T_{sk}$ was ~30.5°C and 36.5°C with 14°C and 44°C water, respectively. $T_{es}$ at $\dot{V}O_{2\max}$ was 38.5-39.0 regardless of condition. Compared to being euhydrated with 14°C water, $\dot{V}O_{2\max}$ was equally reduced by 16% when euhydrated and dehydrated with 44°C water. Preventing hyperthermia in dehydrated subjects resulted in a decline in $\dot{V}O_{2\max}$ of only 5%. The results of this study provide evidence that most of the decline in $\dot{V}O_{2\max}$ is because of hyperthermia ($T_{sk}$ and $T_{es}$ are elevated). It was calculated that in the conditions in which $\dot{V}O_{2\max}$ was reduced 16%, SV and $\dot{Q}$ were reduced 26 ml and 4 l/min, respectively.

**Previous exercise.** It is important to understand how prolonged exercise, in which CV drift can occur, may affect $\dot{V}O_{2\max}$. In an early attempt to link CV drift and $\dot{V}O_{2\max}$, in 1964, Saltin and Stenberg (52) had subjects cycle for 195 min at 75% $\dot{V}O_{2\max}$ and then measure $\dot{V}O_{2\max}$ after 90-min rest. During the submaximal exercise in 19°C, the subjects became 3.2-5.2% dehydrated. Over the course of the 195 min, HR increased 23 bpm, SV decreased 9.5%, and $\dot{Q}$ increased slightly. Ending submaximal exercise $T_{re}$ ranged from 38.0-38.6°C. Despite 90 min of rest before measuring $\dot{V}O_{2\max}$, it was still reduced 5% compared to control values.

Around this same time, Astrand et al. (3) found reductions when measuring $\dot{V}O_{2\max}$ in 5 cross-country skiers before and after an 85 km race that took anywhere from 5-8.5 h to complete. $\dot{V}O_{2\max}$ was tested before the race and then 1.5-2 h after the race. Despite this large amount of time between the race and testing, there was still an average 11% reduction in $\dot{V}O_{2\max}$. The cause of the lower $\dot{V}O_{2\max}$ was hypothesized as being a result of low glycogen levels, but this hypothesis was not tested until 1971.

In 1971, Costill et al. (7) found $\dot{V}O_{2\max}$ was reduced following prolonged exercise despite adequate muscle glycogen. A 16.1-km run was performed at 80% $\dot{V}O_{2\max}$, and 45 min later a $\dot{V}O_{2\max}$ test was performed. The 8% reduction in $\dot{V}O_{2\max}$ was not a result of a decrease in maximal HR. The reduced lactate levels after prolonged exercise were probably because of a lower maximal work load and not reduced glycogen levels.
Around this same time, a training study by Ekblom (13) examined \( \dot{V}O_{2\text{max}} \) after prolonged exercise. Subjects, before and after training, cycled at 75% \( \dot{V}O_{2\text{max}} \) for 1 h. A \( \dot{V}O_{2\text{max}} \) test was performed immediately after the exercise bout and 90-120 min later. Regardless of training status, \( \dot{V}O_{2\text{max}} \) immediately post exercise and 90-120 min after exercise did not differ, but was 6-11% lower from the \( \dot{V}O_{2\text{max}} \) performed prior to the experimental protocol. Training did not change the degree of CV drift that occurred during the 1 h of cycling. HR increased 4-5% and SV declined 3-6%. The changes that occurred during the exercise bout persisted after resting (90-120 min). The results from this study are difficult to interpret because \( \dot{V}O_{2\text{max}} \) was only measured after CV drift had occurred and not at the same time points when cardiovascular measures were first taken (prior to CV drift).

In 1979, Sawka et al. (54) had subjects perform a \( \dot{V}O_{2\text{max}} \) test in thermoneutral conditions (22-25°C) several days before and immediately after a 21-mile run at 70% \( \dot{V}O_{2\text{max}} \) (160 min at 8 mph with a grade that elicited 70% \( \dot{V}O_{2\text{max}} \)). Subjects became 4% dehydrated during the run, which could have contributed to the 6% reduction of \( \dot{V}O_{2\text{max}} \) observed after exercise.

Only one study to the author’s knowledge has systematically measured \( \dot{V}O_{2\text{max}} \) at the same time points in which markers for CV drift were measured. Wingo et al. (61) directly investigated the consequences of CV drift on \( \dot{V}O_{2\text{max}} \) by having subjects cycle for either 15 or 45 min at 60% \( \dot{V}O_{2\text{max}} \) in the heat (35°C) with or without fluid ingestion. \( \dot{V}O_{2\text{max}} \) was measured directly after each submaximal ride, and just prior to the \( \dot{V}O_{2\text{max}} \) test, cardiovascular measures were made. Probably, because of the short duration of exercise, fluid had no affect on any measures, thus data from the two conditions were combined. From 15 to 45 min, SV decreased 16% and HR increased 12%. \( \dot{V}O_{2\text{max}} \) was reduced 18% after 45 min of exercise compared to 15 min.

\( \dot{V}O_{2\text{max}} \) seems to be lowered when performed after previous exercise of long-duration and moderate-intensity (3, 7, 13, 47, 52, 54). Unfortunately, most studies observing modest reductions in \( \dot{V}O_{2\text{max}} \) after prolonged exercise either did not have cardiovascular measures to explain reductions, or did not measure \( \dot{V}O_{2\text{max}} \) at the same points in time as the variables.
characterizing CV drift (13, 52, 54). In the one study that does measure \( \dot{V}O_{2\text{max}} \) at the same time points before and after CV drift (61), a relationship between CV drift and reductions in \( \dot{V}O_{2\text{max}} \) is observed, but no evidence is provided to indicate whether the relationship is coincidental or causal.
CHAPTER 3

FLUID INGESTION ATTENUATES THE DECLINE IN $\dot{V}O_{2\text{max}}$
ASSOCIATED WITH CARDIOVASCULAR DRIFT$^1$

$^1$Ganio, M.S., Wingo, J.E., Carroll, C., Thomas, M.K., Cureton, K.J. To be submitted to the *Journal of Applied Physiology*. 
Abstract

To determine whether manipulation of cardiovascular drift (CV drift) by changing exercise duration or by fluid ingestion is associated with altered \( \dot{VO}_2\text{max} \) during prolonged exercise, \( \dot{VO}_2\text{max} \) was measured in 11 trained men immediately after they cycled at 60% control \( \dot{VO}_2\text{max} \) for 15, 60 and 120 min with no fluid (15NF, 60NF, 120NF), or 120 min with fluid (120F). Markers of CV drift, stroke volume (SV) and heart rate (HR), were measured in 120NF and 120F at 15-, 60-, and 120-min time points. Subjects became 2.3%, 3.7%, and 0.7% dehydrated in 60NF, 120NF, and 120F. Compared to 15-min values, SV did not decline significantly after 60 min (6 ml, 4.6%, \( P = 0.128 \)) but was reduced after 120 min (16 ml, 13.8%, \( P < 0.001 \)) in 120NF. After 120 min, the decline in SV (3 ml, 2.1%) in 120F was significantly less than during 120NF (\( P = 0.001 \)). Compared to 15NF, \( \dot{VO}_2\text{max} \) was not decreased in 60NF (-1.2%, \( P = 1.00 \)), but was lower in 120NF (-8.7%, \( P = 0.016 \)). Fluid ingestion significantly reduced the decline in \( \dot{VO}_2\text{max} \) (-1.9%, \( P = 0.035 \)). Changes in \( \dot{VO}_2\text{max} \) were correlated with changes in SV and HR during the preceding submaximal exercise (\( r = 0.47 \) and 0.64, SEE = 6.6 and 5.7). Manipulation of CV drift by altering exercise duration and fluid ingestion is associated with corresponding changes in \( \dot{VO}_2\text{max} \). The results provide support for the hypothesis that, under these conditions, CV drift causes reduced \( \dot{VO}_2\text{max} \).
Introduction

Cardiovascular drift (CV drift) is a physiological phenomenon that has been described since the early 1960’s (15, 52). Rowell (44) defined CV drift as the progressive decline in stroke volume (SV), mean arterial pressure, and central venous pressure, and progressive rise in heart rate (HR) sufficient to maintain cardiac output ($\dot{Q}$) at a constant level during prolonged exercise. He hypothesized that CV drift was due to pooling blood in the cutaneous veins, displacing central vascular volume peripherally, reducing central blood volume and venous pressure, resulting in reduced ventricular filling pressure and a fall in SV (44). In 1999, Coyle (17) provided an alternative explanation, proposing that the reduction in SV and increase in HR that occurs with prolonged exercise is caused by increased hyperthermia and sympathetic nervous system activity, which increase HR. The increase in HR reduces ventricular filling time and SV. Because of these opposing explanations, the mechanism underlying CV drift remains a topic of debate (10).

Regardless of the cause of CV drift, it is affected by many factors, including ambient temperature (19, 22, 24), blood volume (16, 23, 25, 36, 40), hydration level (21-24, 26, 37, 39), mode of exercise (38), training status (34), exercise intensity (56), air velocity (56), and previous exercise (54). Exercise in high ambient temperature causes a greater degree of CV drift than in a thermoneutral environment, apparently because of the greater increase in core temperature ($T_{core}$) over time, which reduces central blood volume and increases HR (19, 22, 24, 40). When $T_{core}$ is controlled, change in blood volume affects the degree of CV drift (22). In plasma-volume expansion studies in which blood volume is maintained during exercise in both thermoneutral (40) and hot conditions (36, 40), CV drift is reduced. Reducing blood volume prior to exercise causes a larger degree of CV drift during exercise (16). Plasma-volume expansion prior to exercise does not prevent CV drift, but $\dot{Q}$ and SV are higher and HR is lower than without plasma-volume expansion (23, 25). Thus, the degree of CV drift that occurs in thermoneutral (26, 39) and hot conditions (21, 37) is directly related to the degree of dehydration and associated hyperthermia that occurs during prolonged exercise. A similar pattern of findings is observed
when exercise is started in a dehydrated state with varying ambient (22-24) and core (22) temperatures. In long duration exercise (2 h) when dehydration is completely prevented by fluid ingestion, the increase in HR is attenuated and decrease in SV is eliminated over time in thermoneutral (26) and in hot conditions (21, 36).

Despite the many factors affecting CV drift, its implications for physical performance are unclear. It is not known whether or not the progressive rise in HR and fall in SV over time are benign and can be overcame if exercise intensity is increased, or whether they reflect altered circulatory capacity and are associated with a reduced maximal oxygen uptake (\(\dot{V}O_{2max}\)). A number of studies have reported that prolonged exercise (3, 7, 13, 52, 54) has a modest detrimental effect on \(\dot{V}O_{2max}\), but few have examined the relationship between \(\dot{V}O_{2max}\) and the reduced SV that occurs with CV drift following prolonged exercise. Most studies that have observed a modest reduction in \(\dot{V}O_{2max}\) after prolonged exercise did not measure \(\dot{V}O_{2max}\) at the same points in time as the variables characterizing CV drift (13, 52, 54). Wingo et al. (61), however, observed a large reduction in \(\dot{V}O_{2max}\) measured immediately after 45 min of submaximal exercise during heat stress in which there was substantial CV drift.

Although studies have suggested the reduction in SV that occurs with CV drift is associated with reduced \(\dot{V}O_{2max}\), it is not known if the relation is cause and effect. While it is known that CV drift occurs in a progressive fashion over time (56), it is not known if the reduction in \(\dot{V}O_{2max}\) follows a similar trend. Experimentation is needed in which the degree of CV drift during prolonged exercise is manipulated and the effect on \(\dot{V}O_{2max}\) is measured. Fluid ingestion manipulations of SV, and thereby CV drift, that elicit corresponding and proportional changes in \(\dot{V}O_{2max}\) would imply a causal link between CV drift and changes in \(\dot{V}O_{2max}\). Likewise, establishing whether or not the time course of changes in SV and changes in \(\dot{V}O_{2max}\) during prolonged exercise are similar or not would help determine if there is a causal link between the two.

The primary purpose of this study was to determine whether CV drift is associated with a reduction in \(\dot{V}O_{2max}\) after prolonged exercise under conditions in which the magnitude of CV
drift is manipulated. CV drift was manipulated in two ways. First, the association between CV drift and \( \dot{V}O_{2\text{max}} \) following two different durations of exercise in which different degrees of CV drift occurred was studied. This was accomplished by measuring CV drift between min 15 and min 60, and min 15 and min 120 of cycling and assessing the change in \( \dot{V}O_{2\text{max}} \) after 15, 60, and 120 min of exercise. Second, the magnitude of CV drift was manipulated with fluid ingestion. This was accomplished by performing a second 120-min trial during which fluid was ingested to replace sweat losses, and measuring \( \dot{V}O_{2\text{max}} \) immediately after the 120 min.

**Methods**

*Subjects.* Eleven male, healthy, competitive cyclists were recruited to participate in this study. They had been involved in training for the last 6 (3) yr and averaged 178 (97) km/wk of cycling for the 6 mo prior to the study. Their mean (SD) age, mass, height, and % body fat were 26 (5) yr, 75.63 (10.49) kg, 179.2 (5.6) cm, and 13.5 (4.8) %, respectively. This sample size is sufficient to detect a 5% 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/min with a SD of 7 ml/kg/min and that the test-retest correlation for \( \dot{V}O_{2\text{max}} \) is 0.95 (33). This study was approved by the Institutional Review Board, and subjects gave written informed consent prior to testing.

*Research Design.* A repeated-measures experimental design in which each subject served as his own control was used. Following a control \( \dot{V}O_{2\text{max}} \) test and practice ride, each subject was tested in four experimental sessions, presented in a randomized order separated by 7.4 (3.2) days. During each trial, the subject cycled at a power output eliciting 60% \( \dot{V}O_{2\text{max}} \) [169 (16) watts (W)] under the following conditions: (1) 15 min without fluid ingestion (15NF), (2) 60 min without fluid ingestion (60NF), (3) 120 min without fluid ingestion (120NF), and (4) 120 min during which a volume of fluid estimated to replace anticipated sweat losses was ingested (120F). Immediately following each ride, without cessation of cycling, subjects began a step-wise \( \dot{V}O_{2\text{max}} \) protocol to volitional fatigue. The main dependent variables for all trials were the degree of CV
drift and accompanying change in $\dot{VO}_{2\text{max}}$. These changes were assessed by comparing SV, HR, and $\dot{VO}_{2\text{max}}$ after 15 min with the same measures in the 60- and 120-min trials.

**Protocol**

For the six days prior to each test session, subjects were instructed to maintain training volume and not to exercise vigorously the day before testing. For the three days prior to each test session, subjects were instructed to consume similar diets, and compliance was facilitated by recording food and beverages ingested. For each test, subjects reported to the laboratory following a 3-h fast, but they were asked to remain hydrated by drinking 16oz of water 1-hr before arrival. 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. Urine specific gravity (USG) and tympanic body temperature ($T_t$) was measured. Subjects were not tested if they were dehydrated (USG > 1.030 g/ml), if they had a fever ($T_t > 37.8^\circ\text{C}$) or if they were not feeling well enough to be tested that day.

All testing was conducted in an environmental chamber at 30°C, 40% relative humidity (RH) and with a wind speed of ~2.5 m/s. Each subject was tested at the same time of day to minimize the affects of the circadian rhythms in HR and $T_{\text{core}}$.

**Preliminary Session.** During a preliminary test session, seven skinfolds were measured to estimate body composition. Following a warm-up for approximately 10 min at a self-selected power output, a control $\dot{VO}_{2\text{max}}$ test was conducted. Approximately 45 min after the test, each subject measured his nude body weight, and then completed a practice ride consisting of cycling for 1 h on a cycle ergometer. Oxygen uptake ($\dot{VO}_2$) was measured for the first 10 minutes and the power output was adjusted to elicit 60% $\dot{VO}_{2\text{max}}$. The CO$_2$-rebreathing procedure for measuring $\dot{Q}$ was practiced at the 15- and 60-min time points. Finally, nude body weight was re-measured for determination of sweat rate.

**Experimental sessions.** During each experimental test session, following 15 min of seated rest in the chamber, subjects cycled at 60% $\dot{VO}_{2\text{max}}$ for 15, 60, or 120 min and then immediately...
performed a graded exercise test to measure \( \dot{V}O_{2\text{max}} \). Rectal (\( T_{re} \)) and skin temperatures and HR were measured continuously during each experimental trial. During the 2-h rides (120NF and 120F), the procedure to measure \( \dot{Q} \) by CO\(_2\) rebreathing was started at min 7, 52, and 112. CV drift has typically been described as starting after 15 min (8), therefore the CO\(_2\) rebreathing started at min 7 was identified as a baseline measure (before CV drift had occurred). \( \dot{V}O_2 \), rating of perceived exertion (RPE), forearm skin blood flow (SkBF), blood pressure, and blood samples were obtained, in that order, at the same time points as \( \dot{Q} \). RPE, SkBF, and blood samples were obtained at the termination of the \( \dot{V}O_{2\text{max}} \) test.

During 120F, equal volumes of a commercially available 7% carbohydrate-electrolyte sport drink at 30°C were ingested at min 15 and at 15-min intervals thereafter until min 105. The volume of fluid ingested was that estimated to be enough to prevent dehydration and was based on the sweat rate measured during the practice ride and the estimated sweat loss that would occur during the \( \dot{V}O_{2\text{max}} \) test portion of the ride.

**Procedures**

*Preliminary \( \dot{V}O_{2\text{max}} \).* The control \( \dot{V}O_{2\text{max}} \) test consisted of cycling on an electronically-braked ergometer while progressively increasing the power output until subjects could no longer continue. Starting at 200 W, the power output was increased 25 W every 2 min. \( \dot{V}O_2 \) and related metabolic measures were obtained using open-circuit spirometry using a PARVO Medics TrueOne 2400 Metabolic Measurement System (Parvo Medics, Inc., Salt Lake City, UT). HR and RPE were measured at rest, every 2 min, and at the end of the \( \dot{V}O_{2\text{max}} \) test. Three min after completion of the test, a finger-stick blood sample was obtained for determination of blood lactate concentration. In order to ensure \( \dot{V}O_{2\text{max}} \) was attained, subjects rested for 20 min, after which they 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 \( \geq 1 \) 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 using a modification of the plateauing criterion described by Taylor et al. (58). The criterion for determining a plateau was an increase in $\dot{V}O_2$ (l/min) between the last two stages of less than half of the expected increase (0.135 l/min) based on the American College of Sports Medicine metabolic equation (1):

$$\dot{V}O_2 (\text{ml/min}) = \text{work rate (kpm/min)} \cdot 2 (\text{ml/kpm}) + [3.5 (\text{ml/kg/min}) \cdot \text{body weight (kg)}].$$

Using this protocol, all subjects demonstrated a plateau in $\dot{V}O_{2\text{max}}$, either during the graded test or during the subsequent bout.

$\dot{V}O_{2\text{max}}$ after submaximal exercise. At the end of the rides at 60% $\dot{V}O_{2\text{max}}$, subjects immediately began a $\dot{V}O_{2\text{max}}$ test protocol consisting of an initial 25 W increase in work rate, with an additional 25 W increase in work rate every 2 min thereafter until they could not continue. $\dot{V}O_2$ was measured continuously throughout the test, while HR and RPE were measured every 2 min and at the end of the test. Three min following the test, a blood sample was taken. The measurement of $\dot{V}O_{2\text{max}}$ was only considered valid if the $\dot{V}O_2$ reached a plateau according to the criteria described above, if the $\dot{V}O_{2\text{max}}$ reached the same value as in the control test, or if the highest HR was within 5 bpm of the control maximum HR. Using these criteria, all subjects’ $\dot{V}O_{2\text{max}}$ tests were considered valid.

Stroke volume and cardiac output. During 120NF and 120F, from min 7-15, 52-60 and 112-120, $\dot{Q}$ was measured using the indirect-Fick CO2-rebreathing method, as described by Jones (30), using the Parvo Medics metabolic system and software. This involved measuring the $\dot{V}CO_2$, end-tidal CO2 concentration, and the equilibrium CO2 concentration following rebreathing in succession. Two rebreathing trials, separated by approximately one min, were always performed and averaged. The reliability of values from two trials in our lab is high (intraclclass correlation = 0.93) (61). SV was calculated by dividing $\dot{Q}$ by HR. Mean arterial pressure (MAP) was estimated using systolic and fourth-phase diastolic blood pressures (SBP and DBP) and the formula:

$$\text{MAP} = \text{DBP} + 0.33 (\text{SBP} - \text{DBP}) \text{.}$$

Systemic vascular resistance (SVR) was calculated by dividing MAP by $\dot{Q}$. 
**Thermal Measurements.** Rectal temperature \((T_{re})\) was measured by having the subject insert a temperature probe (Ellab, Inc., Arvada, CO, model MOV-55044-A) 10 cm past the anal sphincter. Skin temperature was measured with probes (Ellab, Inc., Arvada, CO, model M HF-18058-A) attached on the right lateral sub-deltoid, chest, thigh, and lateral calf as described by Ramanathan (43). The 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 Ramanathan (43)

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

where \(T_1, T_2, T_3,\) and \(T_4\) are chest, lateral sub-deltoid, thigh and calf skin temperatures, respectively. Mean body temperature \((\bar{T}_b)\) was calculated from \(T_{re}\) and \(T_{sk}\) with the formula of Baum et al. (4):

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

This equation was developed on highly trained athletes and has been used for describing thermoregulatory responses during strenuous exercise (32).

**Hematological measures.** Throughout the 2-h rides, 2-ml blood samples were drawn from a Teflon® venous catheter inserted into an antecubital vein and kept patent with 0.5 ml of 10 USP units/ml heparin lock flush. The sample was collected into a tube containing EDTA to prevent clotting. Blood lactate and glucose concentration were measured using a YSI 2300 Stat Plus Analyzer (Yellow Springs Instruments, Inc., Yellow Springs, OH). Plasma osmolality was measured with the Fiske® ONE-TEN™ Osmometer (Fiske Associates, Norwood, MA). Plasma volume change (%∆PV) during cycling relative to pre-exercise rest was estimated from measures of hemoglobin and hematocrit using the Dill-Costill equation (12). Hematocrit was measured in triplicate via the microhematocrit method. Hemoglobin was measured in duplicate using a HemoCue B-Hemoglobin photometer (Angelhom, Sweden).
**Other Measures.** SkBF was measured with laser Doppler flowmetry (ALF 21D, Transonic Systems, Ithaca, NY). HR was measured with a Polar® Vantage XL heart rate monitor (Polar Electro, Inc. Woodbury, NY, model 145900) set to record and store HR every 5 seconds. HR at min 15, 60, and 120 was determined by using a regression equation formed from the 5-s data from min 10 to the end of submaximal exercise (15, 60, or 120 min) for each trial. Systolic and fourth-phase diastolic blood pressures were taken via auscultation. All exercise was conducted on an electronically-braked cycle ergometer (Lode Excalibur Sport, Lode B.V., Groningen, NL). Body weight was measured on an electronic scale with an error of ± 20 g (A&D Co., Ltd., Tokyo, model FW-150KA1). RPE were measured by the Borg 15-point category scale (5). Body density was estimated using the Jackson and Pollock equation (28) from the sum of 7 skinfolds measured with Lange skinfold calipers, and percent fat was estimated using the Siri equation (57).

**Statistical analysis.** Statistical analyses were performed using SPSS v.11 for Windows (SPSS, Inc., Chicago, IL). Data are reported as means (SD). To determine the effects of exercise duration on CV drift and $\dot{V}O_2_{max}$ from data collected during 15NF, 60NF and 120NF, a one-way repeated-measures analysis of variance (ANOVA) was used to test the significance of mean differences. To examine the effects of fluid ingestion on CV drift and $\dot{V}O_2_{max}$ from data collected during 15NF, 120NF and 120F, a two-way repeated-measures ANOVA (condition × time) was used to test the significance of mean differences. Data from 15NF was used as the baseline for 120F because fluid was not ingested prior to 15 min. The significance of differences in other variables was tested with either a one-way ANOVA (for the effect of duration) or a two-way ANOVA (for the effect of fluid ingestion). Greenhouse-Geisser corrections were made when the assumption of sphericity was violated. Follow-up repeated-measures t-tests and the Bonferroni alpha correction were used when appropriate.
Simple correlation and regression analysis were used to describe the relation between variables. HR and SV change during the submaximal exercise bout were used as the measures of CV drift. An alpha level of 0.05 was used for all significance tests.

**Results**

**Hydration Status**

Hydration status at the beginning of 15NF, 60NF, 120NF, and 120F was not statistically different, as indicated by similar body weights [75.43 (10.53), 75.08 (10.81), 75.19 (10.43), and 75.44 (10.86) kg], USG [1.010 (0.006), 1.010 (0.006), 1.011 (0.005), and 1.010 (0.006)], and plasma osmolality [307 (4), 303 (5), 306 (8), and 306 (12) mosmol/kg H₂O].

**Effect of Exercise Duration During No-Fluid Trials**

*Responses to submaximal exercise (Table 1, Figure 1).* Average \( \dot{VO}_2 \) drifted from 57.4 (3.9) % of control \( \dot{VO}_{2\text{max}} \) at 15 min to 61.0 (4.3) % at 120 min in 120NF (\( P < 0.001 \)). Between 15 and 60 min of submaximal exercise, SV did not change significantly (-6 ml, 4.6%, \( P = 0.128 \)), HR increased significantly by 8 bpm (6%, \( P < 0.001 \)), and \( \dot{Q} \) did not change significantly (\( P = 0.196 \)) (Figure 1). In the 2\(^{nd} \) h of exercise, there was an additional decrease in SV of 9.2% and an additional increase in HR of 7.8%. After 2 h with no-fluid ingestion, SV was reduced 16 ml (13.8%, \( P < 0.001 \)), HR was increased 19 bpm (13.8%, \( P < 0.001 \)), and \( \dot{Q} \) was unchanged (\( P = 0.196 \)) compared to 15-min values.

Plasma volume, decreased ~6% from rest during the first 15 min, but did not change significantly (\( P = 0.579 \)) between 15 and 60 min. An additional significant decline of 1.5 percentage points occurred during the 2\(^{nd} \) h of exercise (\( P = 0.003 \)). Other cardiovascular measures (SkBF, MAP, and SVR) did not change with time (\( P = 0.355, 0.781, 0.400 \), respectively). Blood glucose increased significantly by 0.6 mmol/l from 15-60 min (\( P < 0.001 \)), but from 60-120 significantly declined 0.3 mmol/l (\( P < 0.001 \)). This resulted in blood glucose at 120 min being not significantly different from the 15-min value (\( P = 0.097 \)). Blood lactate did
not change over time. A decrease from the 15-min value in $\bar{T}_{sk}$ of 0.6°C ($P = 0.027$) and an increase in $T_{re}$ of 1.1°C ($P < 0.001$) resulted in $\bar{T}_b$ increasing 0.9°C over time ($P < 0.001$).

Responses to maximal exercise (Table 2, Figure 2). $\dot{V}O_{2\text{max}}$ after 15 min (15NF) was not different from control ($P = 1.00$). Compared to 15NF, there was a not a statistically-significant change in $\dot{V}O_{2\text{max}}$ after 1 h (-1.2%, $P = 1.00$), but after 2 h with no fluid, $\dot{V}O_{2\text{max}}$ was significantly reduced 8.7% ($P = 0.016$). Maximal HR, $\%\Delta PV$, SkBF, and RPE did not differ between conditions ($P = 0.067, 0.546, 0.641, 0.326$, respectively), but maximal ventilation ($\dot{V}_E$), respiratory exchange ratio (RER), oxygen pulse (O$_2$ pulse) and blood lactate were all lower at $\dot{V}O_{2\text{max}}$ in 120NF compared to 15NF ($P = 0.025, 0.026, 0.016$ and $< 0.001$, respectively) and 60NF ($P = 0.035, 0.036, 0.020$, respectively).

Maximal $T_{re}$ and $\bar{T}_b$ were higher in 60NF and 120NF compared to 15NF ($P < 0.001$ and $P < 0.001$, respectively). Maximal $T_{re}$ also was higher by 0.3°C in 120NF compared to 60NF ($P < 0.001$). Maximal $\bar{T}_{sk}$ did not differ between conditions ($P = 0.055$). The duration of the graded exercise test to achieve $\dot{V}O_{2\text{max}}$ was shorter in 60NF ($P = 0.002$) and 120NF ($P < 0.001$) compared to 15NF, and shorter in 120NF compared to 60NF ($P = 0.007$). This resulted in a maximal power output ($W_{\text{max}}$) that was lower in 60NF ($P < 0.001$) and 120NF ($P < 0.001$) compared to 15NF, and lower in 120NF compared to 60NF ($P = 0.037$). Body weight loss was significantly greater with increased exercise duration ($P < 0.001$).

Effect of Fluid Ingestion

The ingestion of an average of 2.35 (0.28) L of fluid in 120F prevented a significant change in body weight. Net weight loss was 0.5 (0.4) kg [0.7 (0.5) %], which was significantly less than for 120NF ($P < 0.001$).

Responses to submaximal exercise (Table 1, Figure 3). Mean $\dot{V}O_2$ drifted from 58.6% (3.4) of control $\dot{V}O_{2\text{max}}$ at 15 min to 61.7% (2.2) at 120 min in 120F ($P = 0.031$). These values did not significantly differ from 120NF ($P = 0.441$).

By ingesting fluid, the decline in SV from 15-120 min [3 (10) ml] was significantly less than the decline in 120NF [(16 (5) ml) ($P = 0.002$) (Figure 3). SV did not significantly change
over time in 120F (P = 0.268), but declined significantly over 2 h in 120NF (P < 0.001). SV differences between 120NF and 120F occurred in the 2nd h of exercise and were evidenced by differences in SV at 2 h (P < 0.001) but not at 1 h (P = 0.081). The 10 (5) bpm increase in HR from 15-120 min (P < 0.001) was significantly less with fluid ingestion than without [19 (7) bpm] (P = 0.002). But, despite these differences, HR did not differ between conditions at any time point (P = 0.149, 0.698, 0.080 at 15, 60, and 120 min, respectively). Q̇ did not change significantly over time regardless of condition (P = 0.196, 0.169 in 120NF and 120F, respectively), but in 120F, Q̇ was higher at 60 and 120 min compared to the same time points in 120NF (P = 0.045 and P = 0.002).

In 120F, PV did not decrease over time (P = 0.063) as in 120NF (P = 0.008). After 60 and 120 min, %ΔPV tended to be lower in 120NF compared to the same time points in 120F (P = 0.059 and P = 0.076). In 120F, SkBF tended to rise from 15 min to 60 min (P = 0.103) and was significantly higher than the 15-min value after 120 min (P = 0.045). SkBF did not change over time in 120NF (P = 0.355).

Ingestion of a carbohydrate-electrolyte sport drink during 120F resulted in blood glucose increasing in the 1st h of exercise (P < 0.001), and then leveling off in the 2nd h. This increase in blood glucose over time in 120F resulted in higher values than in 120NF after 2 h (P = 0.027). Blood lactate, MAP, and SVR did not change with condition or time (P = 0.103, 0.953, 0.392, respectively). RPE was not different between conditions (P = 0.777), but increased over time in both conditions (P < 0.001).

Tsk decreased over time in both conditions (P = 0.016), but did not differ between conditions. Tre, on the other hand, increased significantly over time in both trials (P < 0.001), but in the 2nd h of exercise, the increase in Tre was less in 120F, and resulted in the 120-min value being significantly lower in 120F than in 120NF (P = 0.005). Tb increased over 2 h in 120NF (P < 0.001), but only increased in the 1st h in 120F (P < 0.001). Fluid ingested resulted in Tb being lower after 120 min in 120F compared to 120NF (P = 0.016).
Responses to maximal exercise (Table 2, Figure 4). A significant condition × time interaction (P = 0.035) revealed that the change in \( \dot{V}O_{2\text{max}} \) (\( \Delta \dot{V}O_{2\text{max}} \)) from 15NF to 120F was significantly less than the \( \Delta \dot{V}O_{2\text{max}} \) from 15NF to 120NF (-1.3 and -5.1 ml/kg/min, respectively). \( \dot{V}O_{2\text{max}} \) in 120NF was significantly less than in 15NF (P = 0.005) and 120F (P = 0.035), but \( \dot{V}O_{2\text{max}} \) in 120F was not different from that in 15NF (P = 0.282).

Maximal RER, RPE, %ΔPV, SkBF and HR did not differ between conditions (P = 0.223, 1.00, 0.056, 0.144, 0.192, respectively), but these values were reached at lower maximal \( \dot{V}E \) (P = 0.045), blood lactate (P = 0.003), and \( O_2 \) pulse (P = 0.046) in 120NF. A shorter graded exercise test (P = 0.001) in 120NF resulted in a lower \( W_{\text{max}} \) (P = 0.001). Fluid ingestion (120F) resulted in a lower maximal \( T_r\text{e} \) (P = 0.002), but higher \( T_s\text{k} \) (P = 0.023), which resulted in no differences in \( T_b \) between conditions (P = 0.072).

Relation of CV Drift and \( \dot{V}O_{2\text{max}} \)

Changes in \( \dot{V}O_{2\text{max}} \) were correlated to changes in SV and HR after 15 min (r = 0.47 and 0.64, respectively; SEE = 6.6 and 5.7, respectively) (Figure 5). In 120NF and 120F, for every 2 ml decline in SV from 15-120 min, \( \dot{V}O_{2\text{max}} \) decreased 1.1%.
Table 1: Responses to submaximal exercise of different durations with and without fluid ingestion. Values are means (SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>No Fluid</th>
<th>Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>15-min</td>
<td>60-min</td>
</tr>
<tr>
<td>( \dot{V}O_2 ), l/min</td>
<td>2.48 (0.24)</td>
<td>2.56 (0.24)</td>
</tr>
<tr>
<td>( \dot{Q} ), l/min</td>
<td>16.0 (1.5)</td>
<td>16.1 (1.6)</td>
</tr>
<tr>
<td>SV, ml</td>
<td>115 (15)</td>
<td>110 (14)</td>
</tr>
<tr>
<td>HR, bpm</td>
<td>140 (10)</td>
<td>148 (12)‡</td>
</tr>
<tr>
<td>PV, % Δ from rest</td>
<td>-6.1 (2.6)</td>
<td>-7.3 (3.8)</td>
</tr>
<tr>
<td>SkBF, % of 15 min</td>
<td>—</td>
<td>104.2 (9.7)</td>
</tr>
<tr>
<td>MAP</td>
<td>94.5 (8.1)</td>
<td>95.0 (8.2)</td>
</tr>
<tr>
<td>SVR</td>
<td>6.0 (0.8)</td>
<td>6.0 (0.9)</td>
</tr>
<tr>
<td>Blood Lactate, mmol/l</td>
<td>1.3 (0.5)</td>
<td>1.2 (0.4)</td>
</tr>
<tr>
<td>Blood Glucose, mmol/l</td>
<td>4.4 (0.6)</td>
<td>5.0 (0.5)‡</td>
</tr>
<tr>
<td>( \bar{T}_{sk} ) (°C)</td>
<td>34.1 (0.7)</td>
<td>34.0 (1.1)</td>
</tr>
<tr>
<td>( T_{res} ) (°C)</td>
<td>37.7 (0.3)</td>
<td>38.4 (0.2)†</td>
</tr>
<tr>
<td>( \bar{T}_{b} ) (°C)</td>
<td>37.2 (0.2)</td>
<td>37.8 (0.2)†</td>
</tr>
<tr>
<td>RPE</td>
<td>10.0 (2.4)</td>
<td>11.5 (2.3)†</td>
</tr>
</tbody>
</table>
\( \dot{V}O_2 \), oxygen uptake; \( \dot{Q} \), cardiac output; SV, stroke volume; HR, heart rate; PV, plasma volume; SkBF, skin blood flow; MAP, mean arterial pressure; SVR, systemic vascular resistance; \( T_{sk} \), mean skin temperature; \( T_{re} \), rectal temperature; \( T_b \), mean body temperature; RPE, rating of perceived exertion. †Significantly different from 15-min value, P < 0.05; *Significantly different from 60-min value, P < 0.05; ‡Significantly different from No-Fluid Condition at the same time point, P < 0.05.
Table 2: Responses to maximal exercise following submaximal exercise of different durations with and without fluid ingestion. Values are means (SD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>15NF</th>
<th>60NF</th>
<th>120NF</th>
<th>120F</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\dot{V}O_2$, l/min</td>
<td>4.32 (0.31)</td>
<td>4.33 (0.43)</td>
<td>4.28 (0.47)</td>
<td>3.94 (0.41)*</td>
<td>4.24 (0.44)*</td>
</tr>
<tr>
<td>$\dot{V}O_2$, ml/kg/min</td>
<td>57.78 (6.67)</td>
<td>58.08 (7.74)</td>
<td>57.76 (8.81)</td>
<td>52.95 (6.59)*</td>
<td>56.75 (5.97)*</td>
</tr>
<tr>
<td>$\dot{V}E$ (STPD), l/min</td>
<td>133.7 (14.4)</td>
<td>131.1 (18.1)</td>
<td>132.6 (18.9)</td>
<td>120.9 (24.1)*</td>
<td>131.2 (15.0)*</td>
</tr>
<tr>
<td>RER</td>
<td>1.15 (0.04)</td>
<td>1.13 (0.04)</td>
<td>1.11 (0.03)</td>
<td>1.04 (0.09)*</td>
<td>1.07 (0.04)</td>
</tr>
<tr>
<td>HR, bpm</td>
<td>187 (10)</td>
<td>185 (10)</td>
<td>188 (11)</td>
<td>185 (10)</td>
<td>187 (9)</td>
</tr>
<tr>
<td>$O_2$ pulse, ml/bt</td>
<td>23.7 (2.1)</td>
<td>24.1 (2.8)</td>
<td>23.7 (3.5)</td>
<td>21.6 (2.8)*</td>
<td>23.1 (2.8)*</td>
</tr>
<tr>
<td>Blood Lactate, mmol/l</td>
<td>8.9 (1.0)</td>
<td>7.9 (1.2)</td>
<td>6.8 (1.3)</td>
<td>5.4 (1.5)*</td>
<td>6.5 (0.9)*</td>
</tr>
<tr>
<td>PV, % Δ from rest</td>
<td>-14.0 (2.4)</td>
<td>-13.6 (3.0)</td>
<td>-14.8 (3.6)*</td>
<td>-11.9 (3.4)</td>
<td></td>
</tr>
<tr>
<td>SkBF, % of 15 min</td>
<td>103.9 (6.7)</td>
<td>102.5 (17.1)</td>
<td>96.2 (12.9)</td>
<td>102.2 (12.7)</td>
<td></td>
</tr>
<tr>
<td>$\bar{T}_sk$ (°C)</td>
<td>34.2 (0.5)</td>
<td>33.8 (1.0)</td>
<td>33.1 (1.3)</td>
<td>33.8 (1.3)*</td>
<td></td>
</tr>
<tr>
<td>$T_{re}$, (°C)</td>
<td>38.1 (0.2)</td>
<td>38.6 (0.2)*</td>
<td>38.9 (0.2)*</td>
<td>38.6 (0.2)*</td>
<td></td>
</tr>
<tr>
<td>$\bar{T}_b$ (°C)</td>
<td>37.7 (0.3)</td>
<td>38.0 (0.3)*</td>
<td>38.2 (0.3)*</td>
<td>38.1 (0.3)</td>
<td></td>
</tr>
<tr>
<td>Test Duration, min</td>
<td>11.4 (2.2)</td>
<td>12.1 (1.3)</td>
<td>10.7 (1.6)*</td>
<td>8.8 (2.2)*</td>
<td>10.7 (1.9)*</td>
</tr>
<tr>
<td>Power Output, Watts</td>
<td>330 (25)</td>
<td>332 (27)</td>
<td>307 (37)*</td>
<td>290 (34)*</td>
<td>317 (38)*</td>
</tr>
<tr>
<td>Decrease in Body Mass, %</td>
<td>1.3 (0.3)</td>
<td>2.3 (0.4)*</td>
<td>3.7 (0.6)*</td>
<td>0.7 (0.5)*</td>
<td></td>
</tr>
<tr>
<td>RPE</td>
<td>18.7 (0.9)</td>
<td>19.2 (0.6)</td>
<td>19.0 (0.4)</td>
<td>19.3 (0.5)</td>
<td>19.3 (0.6)</td>
</tr>
</tbody>
</table>

* indicates a significant difference from the Control group, † indicates a significant difference from 15NF, ‡ indicates a significant difference from 60NF.
\( \dot{V}O_2 \), oxygen uptake; \( \dot{V}E \), minute ventilation; RER, respiratory exchange ratio; HR, heart rate; PV, plasma volume; SkBF, skin blood flow; \( \bar{T}_{sk} \), mean skin temperature; \( T_{re} \), rectal temperature; \( \bar{T}_b \), mean body temperature; RPE, rating of perceived exertion; †Significantly different from 15NF, \( P < 0.05 \); ‡Significantly different from 60NF, \( P < 0.05 \); ‡‡Significantly different from 120NF, \( P < 0.05 \).
Figure 1. Percent change in mean (SD) stroke volume (SV) and heart rate (HR) from 15 min to 60 and 120 min of exercise without fluid ingestion. †Significantly different from 15-min value, P < 0.05; *Significantly different from 60-min value, P < 0.05.
Figure 2: Percent change in mean (SD) \( \dot{V}O_2_{\text{max}} \) from control, measured after either 15 min, 60 min, or 120 min without fluid ingestion (15NF, 60NF, 120NF). †Significantly different from 15NF, \( P < 0.05 \).
Figure 3. Percent change in mean (SD) stroke volume (SV) and heart rate (HR) from 15 min to 120 min of exercise with and without fluid ingestion (120NF and 120F). 120-min time points are shifted for clarity. †Significantly different from 15-min value, P < 0.05; ‡Significantly different from No-Fluid Condition at the same time point, P < 0.05.
Figure 4. Percent change in mean (SD) $\dot{V}O_2_{\text{max}}$ from control, measured after either 15 min, 120 min without fluid ingestion, or 120 min with fluid ingestion (15NF, 120NF, 120F).

†Significantly different from 15NF, $P < 0.05$; ‡Significantly different from 120NF, $P < 0.05$. 
Figure 5. Relation of the change in stroke volume (SV; top graph) and heart rate (HR; bottom graph) to change in VO$_{2\text{max}}$ measured after exercise of either 60 min or 120 min with (F) and without (NF) fluid ingestion. SV and HR in 60NF were measured after 60 min in 120NF.
Discussion

The primary finding of this study was that \( \dot{V}O_{2\text{max}} \) was reduced only when a significant reduction in SV occurred as a result of CV drift. Declines in SV and \( \dot{V}O_{2\text{max}} \) were non-significant after 1 h of exercise. The decline in SV was significant after 2 h of exercise without fluid ingestion (14%) and was associated with a significant decrease in \( \dot{V}O_{2\text{max}} \) (9%). The declines in SV and \( \dot{V}O_{2\text{max}} \) were prevented during 2 h of exercise in which fluid was ingested. The results suggest there is a cause-and-effect relation between reduction in SV and reduction in \( \dot{V}O_{2\text{max}} \) during prolonged exercise performed under the conditions of this study.

We used a protocol similar to that of previous studies (21, 26, 37) in which considerable CV drift occurred using an intensity and duration of exercise typically utilized for conditioning. By studying responses during and immediately following 1 and 2 h of cycling at \( \sim60\% \dot{V}O_{2\text{max}} \) without fluid ingestion, we obtained two degrees of CV drift. After 1 h of exercise, HR had increased 6% and SV had decreased 5%. Despite these changes, there were no significant differences between SV at min 15 and min 60. In the last hour of the 2 h exercise bout, HR increased an additional 8% and SV decreased an additional 9%. This resulted in a total CV drift of 14% in HR and SV and significant differences in HR and SV from min 15. This pattern of findings was expected under these ambient conditions (26). Other changes influencing CV drift, such as rise T_{re} and dehydration, also were similar to those observed in other studies (21, 26, 37).

In order to determine whether or not the different magnitude of CV drift was associated with a corresponding change in \( \dot{V}O_{2\text{max}} \), subjects performed a graded exercise test immediately after submaximal exercise. After 60 min, when there was a non-significant decline in SV, there was also a non-significant decline in \( \dot{V}O_{2\text{max}} \) (1.2%). However after 2 h, when SV had declined significantly (14%), the \( \dot{V}O_{2\text{max}} \) was also reduced significantly (9%). This magnitude of reduction in \( \dot{V}O_{2\text{max}} \) is similar to that observed in other studies in which \( \dot{V}O_{2\text{max}} \) was measured following prolonged exercise performed in similar ambient conditions (2, 3, 11, 13, 47).

In order to provide additional insight into whether the association between the decline in SV and \( \dot{V}O_{2\text{max}} \) was causal, we used fluid ingestion to prevent dehydration and CV drift over the
2-h time period. After 2 h, fluid ingestion had lowered HR by 7% and increased SV by 12% compared to 120NF. SV was restored to 15-min values, preventing the drift in SV observed in 120NF. Fluid ingestion also resulted in lower T_{re} after 2 h and eliminated the decrease in PV. The elimination of CV drift with fluid ingestion has been observed in other studies in which body weight change is prevented (21, 37).

The reduction in CV drift with the fluid ingestion was paralleled by a corresponding reduction in the magnitude of decrease in \( \dot{V}O_{2\text{max}} \) (from 9% to 2%). Over the same time period in which SV declined 16 ml, \( \dot{V}O_{2\text{max}} \) declined 5.1 ml/kg/min in 120NF. Ingesting fluid significantly reduced these declines, resulting in SV being reduced 3 ml and \( \dot{V}O_{2\text{max}} \) 1.3 ml/kg/min. These findings support the hypothesis that attenuation of the decline in SV with fluid ingestion attenuates the decline in \( \dot{V}O_{2\text{max}} \). The similar direction and magnitude of the changes suggest that the reduction in \( \dot{V}O_{2\text{max}} \) may be a result of a reduction in SV associated with CV drift.

The possibility of a casual link between CV drift and changes in \( \dot{V}O_{2\text{max}} \) was supported by moderately-strong correlations (\( r = 0.47-0.64 \)) between changes in HR or SV and changes in \( \dot{V}O_{2\text{max}} \) during prolonged exercise. Regardless of fluid ingestion, a 2 ml decrease in SV from 15-120 min of exercise results in a 1.1 percentage point decline in \( \dot{V}O_{2\text{max}} \) over 2 h. The similar slopes of the regression of \( \Delta \dot{V}O_{2\text{max}} \) on \( \Delta SV \) reinforce the conclusion that a decline in SV with CV drift causes a reduction in \( \dot{V}O_{2\text{max}} \).

There is strong evidence that \( \dot{V}O_{2\text{max}} \) was attained in all subjects in all experimental trials. \( \dot{V}O_{2} \) either reached a plateau with an increase in workload (18 occurrences), was the same as control condition (12 occurrences), or maximum HR was within 5 bpm of control (14 occurrences). There were no statistical differences among trials in maximum RPE or maximum HR, and mean values of 19 and ~190 bpm, respectively, indicate that a near-maximal effort was given. Maximal blood lactate and \( V_{E} \) were only lower in 120NF, probably because a lower \( W_{\text{max}} \) was achieved and not because of a lack of degree of effort (2). Lower blood lactate at \( \dot{V}O_{2\text{max}} \) in 120NF, in which \( \dot{V}O_{2\text{max}} \) was reduced, also provides evidence that hydrogen-ion accumulation was not a factor inhibiting the ability to reach maximal power output.
During the 120F trial, subjects ingested a carbohydrate-electrolyte sport drink. By ingesting carbohydrate, effects other than prevention of a decline in blood volume could have occurred. Muscle glycogen level is one factor that could have been affected by ingestion of carbohydrate. Some studies show that there is not a difference in muscle glycogen levels following ingestion of carbohydrate compared to placebo even after 3 h of exercise (9), while others show that muscle glycogen can be spared with a carbohydrate-electrolyte fluid ingestion in as little as 1 h (59). Regardless of these mixed results, higher glycogen levels would be expected only to result in a better performance time (i.e., time trial) (35) and not a higher \( \dot{V}O_{2\text{max}} \), assuming a true \( \dot{V}O_{2\text{max}} \) was measured.

The mechanism underlying the reduction in \( \dot{V}O_{2\text{max}} \) in 120NF cannot be determined from the data collected in this study. There was no statistical difference in maximum HR among conditions. If arteriovenous oxygen difference [(a-v)\( O_2 \)] is not different under the conditions imposed in this study (20, 48, 60) than, according to the Fick equation, differences in \( \dot{V}O_{2\text{max}} \) would be due to differences in SV. If SV in trained individuals does not change with increasing \( \dot{V}O_2 \) after ~50% \( \dot{V}O_{2\text{max}} \) (18, 44), the SV at the end of each submaximal exercise bout measured in this study would be representative of the SV at the corresponding \( \dot{V}O_{2\text{max}} \). Thus, the decline in SV with CV drift could cause the reduction in \( \dot{V}O_{2\text{max}} \).

The cause of the increased HR and decreased SV observed during CV drift is controversial (10). One hypothesis is that increasing hyperthermia and SkBF over time cause CV drift by reducing central blood volume, central venous pressure, venous return and SV (29). Experimental evidence for this hypothesis is conflicting, and may be a result of different degrees of hyperthermia and dehydration (21, 29, 36, 37). We did not observe an increase in SkBF over time during 120NF, during which substantial CV drift occurred. Other studies have reported similar findings (36, 37). On the other hand, with fluid replacement, we observed an increase in SkBF during the first hour of exercise, which may have contributed to the attenuation of hyperthermia in 120F (26, 36, 37).
Hyperthermia also has been hypothesized to reduce $\dot{V}O_{2\text{max}}$ via the same mechanism described above through which it could cause CV drift, resulting in premature failure of the cardiovascular system (45). When hyperthermia has not reduced $\dot{V}O_{2\text{max}}$, it has been hypothesized that as power output is increased and blood flow to the active muscles is increased, thermoregulation is sacrificed, and vasoconstriction of blood vessels in the skin occurs, reducing SkBF (6, 46, 51). Vasoconstriction restores central blood volume and a decline in $\dot{V}O_{2\text{max}}$ is prevented. We observed a progressive rise in $T_\text{re}$, but no increase in SkBF during submaximal exercise in 120NF. $T_\text{re}$ increased ($P = 0.033$) and SkBF was decreased at $\dot{V}O_{2\text{max}}$ compared to the 120-min time point ($P = 0.005$). Despite this apparent vasoconstriction, $\dot{V}O_{2\text{max}}$ was substantially reduced. The reduction in $\dot{V}O_{2\text{max}}$ probably was due to the reduction in blood volume associated with dehydration coupled with hyperthermia (11). With fluid ingestion, we also observed vasoconstriction at $\dot{V}O_{2\text{max}}$ ($P = 0.005$), but because blood volume was not reduced, and $T_\text{re}$ at $\dot{V}O_{2\text{max}}$ was lower, vasoconstriction could have prevented a decline in $\dot{V}O_{2\text{max}}$.

The change in $\dot{V}O_{2\text{max}}$ associated with CV drift during prolonged exercise has implications for performance. Since $\dot{V}O_2$ during the submaximal exercise increased only slightly, the decline in $\dot{V}O_{2\text{max}}$ that occurred following 2 h of exercise in 120NF means that the relative metabolic cost of exercise ($\%\dot{V}O_{2\text{max}}$) was increased compared to 120F, even though the same work rate was being performed (61). Compared to 15NF, there was 13% and 27% decline in $W_{\text{max}}$ and time to exhaustion, respectively in 120NF. Fluid ingestion throughout exercise (120F) reduced the decline in $W_{\text{max}}$ and time to exhaustion that occurred in 120NF to ~5% and ~12%, respectively (8% and 16% less decline in $W_{\text{max}}$ and time to exhaustion in 120F compared to 15NF). Thus, the improvement in performance associated with carbohydrate-electrolyte ingestion during prolonged exercise appears to be mediated, at least in part, via effects of dehydration and hyperthermia on blood volume and $\dot{V}O_{2\text{max}}$.

We conclude that a decline in SV during prolonged submaximal exercise as a result of CV drift has a detrimental effect on $\dot{V}O_{2\text{max}}$. Manipulation of CV drift by altering exercise
duration and fluid ingestion is associated with proportional changes in $\dot{V}O_{2\text{max}}$. The results provide support for the hypothesis that, under these conditions, CV drift causes reduced $\dot{V}O_{2\text{max}}$. 
CHAPTER 4
SUMMARY AND CONCLUSION

CV drift is a physiological phenomenon that has been described since the early 1960’s (15, 52). Rowell (44) defined CV drift as the progressive decline in SV, mean arterial pressure, and central venous pressure, and progressive rise in HR sufficient to maintain $\dot{Q}$ at a constant level during prolonged exercise. He hypothesized that CV drift was due to pooling blood in the cutaneous veins, displacing central vascular volume peripherally, reducing central blood volume and venous pressure, resulting in reduced ventricular filling pressure and a fall in SV (44). In 1999, Coyle (17) provided an alternative explanation, that the reduction in SV and increase in HR that occurs with prolonged exercise is caused by increased hyperthermia and sympathetic nervous system activity, which increases HR. The increase in HR reduces ventricular filling time and SV.

Regardless of the mechanism underlying CV drift, it is affected by many factors, including ambient temperature (19, 22, 24), blood volume (16, 23, 25, 36, 40), hydration level (21-24, 26, 37, 39), mode of exercise (38), training status (34), exercise intensity (56), air velocity (56), and previous exercise (54). Knowledge of the many factors that can affect CV drift has not shed light on its implications for physical performance. Thus, until recently, it was not known whether the progressive rise in HR and fall in SV over time are associated with an altered $\dot{V}O_{2\max}$ or not. Wingo et al. (61), with short-duration exercise in heat, found that with large reductions in SV, $\dot{V}O_{2\max}$ was reduced.

Although studies have suggested the reduction in SV that occurs with CV drift is associated with reduced $\dot{V}O_{2\max}$, it is not known if the relation is cause and effect. Experimental evidence is needed manipulating the degree of CV drift during prolonged exercise and observing the effect on $\dot{V}O_{2\max}$. Therefore, the primary purpose of this study was to determine whether CV drift is associated with a reduction in $\dot{V}O_{2\max}$ after prolonged exercise under conditions in which the magnitude of CV drift was manipulated.
Eleven trained, male subjects each performed a graded exercise test to measure \( \dot{VO}_{2\text{max}} \) under control conditions and four experimental trials. The experimental trials consisted of the subjects riding at 60% of control \( \dot{VO}_{2\text{max}} \) for 15, 60, 120 min without fluid, or 120 min while ingesting a 7% carbohydrate-electrolyte drink. Immediately after the submaximal exercise, a test to measure \( \dot{VO}_{2\text{max}} \) was conducted. In order to assess CV drift, SV and HR were measured at min 60 and 120 in 120NF and 120NF and compared to 15-min values in the same trials. \( \dot{VO}_{2\text{max}} \) in 15NF, before CV drift had occurred, was compared to \( \dot{VO}_{2\text{max}} \) in other trials.

CV drift occurred progressively over time with greatest declines in SV being observed in the 2nd h of exercise (6 ml and 16 ml after 60 and 120 min, respectively). The decrease in SV was prevented when fluid was ingested in exercise of 120 min (3 ml). \( \dot{VO}_{2\text{max}} \) was reduced significantly only in conditions in which there was a significant reduction in SV. SV decreased significantly in the 2nd h of exercise (-1.2% and -8.7% after 60 and 120 min, respectively) without fluid. Fluid ingestion over 2 h prevented the decline in \( \dot{VO}_{2\text{max}} \) that occurred over the same time period with no fluid (-1.9% and -8.7% declines after 2 h with and without fluid, respectively). Changes in \( \dot{VO}_{2\text{max}} \) were correlated with changes in SV and HR after 15 min during submaximal exercise \((r = 0.47 \text{ and } 0.64, \text{ SEE } = 6.6 \text{ and } 5.7, \text{ respectively})\). Independent of fluid ingestion, there was a proportional decline in SV and \( \dot{VO}_{2\text{max}} \). For every 2 ml decline in SV between 15-120 min there was 1.1% decrease in \( \dot{VO}_{2\text{max}} \).

In conclusion, CV drift has a detrimental effect on \( \dot{VO}_{2\text{max}} \). This effect is observed under warm ambient conditions, following 2 h, but not 1 h of cycling. Fluid ingestion attenuates the declines in SV and also \( \dot{VO}_{2\text{max}} \). The results of this study support the hypothesis that CV drift causes reduction in \( \dot{VO}_{2\text{max}} \).
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