ARTICLE

Cardiovascular Drift During Heat Stress: Implications for Exercise Prescription

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WINGO, J.E., M.S. GANIO, and K.J. CURETON. Cardiovascular drift during heat stress: implications for exercise prescription. Exerc. Sport Sci. Rev., Vol. 40, No. 2, pp. 88–94, 2012. Cardiovascular drift, the progressive increase in heart rate and decrease in stroke volume that begins after approximately 10 min of prolonged moderate-intensity exercise, is associated with decreased maximal oxygen uptake, particularly during heat stress. Consequently, the increased heart rate reflects an increased relative metabolic intensity during prolonged exercise in the heat when cardiovascular drift occurs, which has implications for exercise prescription. Key Words: heart rate, stroke volume, hyperthermia, thermoregulation, maximal oxygen uptake.

INTRODUCTION

Cardiovascular (CV) drift is a well-known phenomenon characterized by a rise in heart rate (HR) and a fall in stroke volume (SV) over time during constant-rate submaximal exercise. CV drift may be influenced by many factors. For example, the magnitude of CV drift is greater in hot versus cool temperatures (12,18). Most likely, the disparities between the magnitudes of CV drift observed in hot versus cool ambient temperatures can be linked to differences in core body (T_c) and mean skin (T_sk) temperatures between the two environments. Higher T_c and T_sk observed during exercise in 35°C versus 22°C–25°C (12,18) lead to greater relative increases in skin blood flow (23) and HR (18), both of which are potential mechanisms causing the reduced SV associated with CV drift (see next section).

In addition to hyperthermia, dehydration influences CV drift in a graded fashion, such that the degree of CV drift is proportional to the extent of dehydration (10,19). Furthermore, the effects of dehydration and hyperthermia may be additive. Gonzalez-Alonso et al. (14) found that 4% dehydration lowered SV and increased HR to the same extent as hyperthermia alone, and when dehydration was superimposed on hyperthermia, the effects on SV and HR were additive. There may be a threshold level of dehydration at which the effects of dehydration and hyperthermia become additive, however. In one study, exercise with and without fluid to offset sweat losses resulted in dehydration of 0.3% and 2.5%, respectively, but the magnitudes of hyperthermia and CV drift were similar between fluid conditions (34). In the presence of hyperthermia, dehydration greater than 2.5% may be necessary to add to the magnitude of CV drift.

Exercise duration also influences CV drift. However, it is likely that factors, such as hyperthermia and dehydration that occur concomitantly with long-duration exercise and become more pronounced as exercise is prolonged, are responsible for the effect of exercise duration on CV drift (10).

Whereas the causes of and factors affecting CV drift have been extensively studied, its consequences, like effects on maximal oxygen uptake (VO_2max), only recently have gained attention. This review integrates what is known regarding CV drift and associated mechanisms with a series of studies by the authors (10,18,32–34) that test the hypothesis that CV drift is related to decreased VO_2max. The connection between CV drift and VO_2max has implications for exercise prescription and work capacity. Because a target HR often is used to prescribe relative metabolic intensity (percent maximal oxygen uptake (%VO_2max) or percent oxygen uptake reserve (%VO_2R)), it is important to know whether the higher HR associated with CV drift that occurs during constant-rate submaximal exercise in high ambient temperatures reflects a dissociation from a prescribed relative metabolic intensity or not. In addition, because VO_2max sets the upper limit of the rate of aerobic metabolism, understanding the nature of the relationship between CV drift and reduced VO_2max is important in (i)
understanding what limits work capacity in the heat and (ii) optimizing training for, and performance in, endurance events held in hot temperatures.

**POTENTIAL CAUSES OF CV DRIFT**

There have been two prevailing hypotheses regarding the causes of CV drift. The traditional hypothesis links peripheral displacement of the blood volume to a decline in SV (22), whereas a more contemporary hypothesis asserts that increased HR lowers ventricular filling time and, thereby, SV (2).

**Traditional Hypothesis**

Rowell (22) asserts that a progressive increase in skin blood flow, associated with thermoregulation and accompanied by reductions in central venous pressure, is what ultimately leads to the reduction in SV that occurs as part of CV drift during prolonged exercise. The increase in HR is most likely a baroreceptor-mediated reflex intended to maintain cardiac output (Q) and blood pressure in the wake of a falling SV (22). However, HR may continue to increase even when central venous pressure, and thereby SV, is maintained (21), suggesting the rise in HR may be influenced by other factors. For example, rising body temperature can increase the rate of sinoatrial node firing and intrinsic rate of heart contraction, as well as increase sympathetic nervous system activity, thereby increasing HR (15).

Because skin is a highly compliant vascular bed, increases in blood flow to the skin increase cutaneous blood volume, which causes a transient drop in volume and pressure in the central veins that ultimately decreases ventricular filling and SV (28). Shaffrath and Adams (27) demonstrated that SV declined over the same time interval in which forearm blood flow (representative of skin blood flow) increased, which suggests that central blood volume was displaced peripherally. Furthermore, techniques like bandaging the legs compress veins in active limbs and can increase SV (albeit only slightly) during exercise in neutral temperatures (16), thus further supporting the notion that peripheral displacement of blood volume causes decreased SV.

**Contemporary Hypothesis**

Opponents (2) of the traditional hypothesis have argued that no measures of ventricular filling (central venous pressure or central blood volume) were obtained in the studies (5–8) used to support the traditional hypothesis regarding the causes of CV drift. Thus, whereas elimination of venous tone by extremely high skin temperature (38°C) has been shown to result in venous pooling to an extent that can reduce SV (24), Coyle (2) asserts that this is unlikely to occur under the conditions of CV drift described in studies used to support the traditional hypothesis (22). Furthermore, despite the evidence from the study by Shaffrath and Adams (27) mentioned previously that shows decreased SV during exercise in which forearm blood flow increases, in other studies, forearm blood flow appears to level off after about 20–30 min of moderate-intensity constant-rate exercise in temperate conditions (9,20). This occurs at a time when SV continues to decline, thereby implying a disconnection between SV and skin blood flow (9,22). Paradoxically, SV even may be augmented by hyperthermia when the hyperthermia-related increase in HR is prevented (30). This apparent dissociation between skin blood flow and SV decline has led some (2,9) to suggest that an increase in HR lowers ventricular filling time (31) and, thereby, SV under the conditions of CV drift featured in the traditional hypothesis.

Fritzsche et al. (9) provided evidence suggesting a strong link between the rise in HR and fall in SV over time during prolonged moderate-intensity cycling. Under one condition, an 11% rise in HR was accompanied by a 13% decline in SV between 15 and 55 min of exercise. Conversely, β-adrenergic blockade prevented the rise in HR and, thereby, the decline in SV, between 15 and 55 min. Skin blood flow and other variables known to influence SV (which will be described later) were not different between conditions, thus suggesting that peripheral displacement of the blood volume was not responsible for the decline in SV (9).

Likewise, Nassis and Geladas (20) observed a decline in SV over time during treadmill running at 60% \( \dot{V}O_{2\text{max}} \) even though skin blood flow remained stable or even declined slightly over time. HR drifted 15 or more beats/min over the course of the exercise bout, again suggesting that peripheral displacement of blood volume did not cause the reduced SV, but, instead, reduced ventricular filling time associated with tachycardia could have been responsible.

From the available literature, it is difficult to pinpoint a single hypothesis explaining the cause of CV drift. In all probability, both the traditional and contemporary hypotheses are feasible explanations under a broad range of circumstances, and furthermore, mechanisms asserted by both hypotheses may interact to cause CV drift. In addition to the mechanisms asserted by these hypotheses, other factors may act independently or interact with the mechanisms described earlier (i.e., peripheral displacement of or decrease in blood volume) to alter CV drift.

**CV DRIFT IS ASSOCIATED WITH REDUCED \( \dot{V}O_{2\text{max}} \) DURING HEAT STRESS**

CV drift should not be interpreted as a benign response with no consequences. Because SV is a determinant of \( \dot{V}O_{2\text{max}} \) and is negatively influenced by hyperthermia and dehydration, it is logical to surmise that CV drift may reflect a condition in which \( \dot{V}O_{2\text{max}} \) is compromised.

Early studies demonstrated that \( \dot{V}O_{2\text{max}} \) is reduced when measured after previous exercise during which CV drift occurs (4,25,26). However, investigators in these studies did not measure the effects of CV drift on \( \dot{V}O_{2\text{max}} \) per se because the primary variables characterizing CV drift (i.e., SV and HR) were not measured at the same points in time as \( \dot{V}O_{2\text{max}} \). Moreover, \( \dot{V}O_{2\text{max}} \) was measured after a rest, and the way in which CV drift occurred (whether via dehydration, hyperthermia, etc.) was not controlled, thus potentially confounding interpretation of the effects of CV drift on \( \dot{V}O_{2\text{max}} \) (4,25,26).

To resolve the uncertainties from previous research regarding the effects of CV drift on \( \dot{V}O_{2\text{max}} \) we conducted a series of studies in which CV drift was manipulated using hyperthermia (34), exercise intensity (33), body cooling (32), hydration status (10), and ambient temperature (18). Outcomes from these
studies are summarized in the Table. \( \dot{Q} \) was measured using the indirect Fick \( \dot{CO}_2 \)-rebreathing method, and SV was calculated by dividing \( \dot{Q} \) by HR. In all of the studies except the one regarding hydration status (10), CV drift was assessed between 15 and 45 min of moderate-intensity (60% \( \dot{VO}_{2\text{max}} \)) constant-rate exercise in ambient temperatures ranging from 22°C to 35°C. To more directly measure the effect of CV drift on \( \dot{VO}_{2\text{max}} \) compared with previous studies, we measured \( \dot{VO}_{2\text{max}} \) at the same points in time as CV drift. This was accomplished by measuring \( \dot{VO}_{2\text{max}} \) after separate 15- and 45-min bouts of exercise. A graded exercise test (GXT) to assess \( \dot{VO}_{2\text{max}} \) was completed immediately after the 45-min exercise bout. On a separate day, participants completed 15 min of submaximal exercise under the same conditions, followed by a GXT to measure \( \dot{VO}_{2\text{max}} \). CV drift was assessed between the 15- and 45-min time points within the 45-min trial.

### TABLE. Summary of studies investigating the effect of CV drift on \( \dot{VO}_{2\text{max}} \) during heat stress.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>( T_a ) °C</td>
<td>Wingo et al. (34) 35</td>
</tr>
<tr>
<td></td>
<td>Wingo and Cureton (33) 35</td>
</tr>
<tr>
<td></td>
<td>Wingo and Cureton (32) 35</td>
</tr>
<tr>
<td></td>
<td>Ganio et al. (10) 30</td>
</tr>
<tr>
<td></td>
<td>Lafrenz et al. (18) 35 heat</td>
</tr>
<tr>
<td></td>
<td>22 cool</td>
</tr>
<tr>
<td>Submaximal exercise duration, min</td>
<td>45 45 45 120 45</td>
</tr>
<tr>
<td>HR, % change</td>
<td>( \uparrow ) 12 ( \uparrow ) 13 ( \uparrow ) 16 ( \uparrow ) 14 ( \uparrow ) 11 heat</td>
</tr>
<tr>
<td>HR at 15 min, beats( \text{min}^{-1} )</td>
<td>151 ± 9 150 ± 12 150 ± 8 140 ± 10 151 ± 12 heat</td>
</tr>
<tr>
<td></td>
<td>140 ± 6 cool</td>
</tr>
<tr>
<td>HR at the end of exercise, beats( \text{min}^{-1} )</td>
<td>169 ± 10 169 ± 14 173 ± 9 159 ± 15 168 ± 11 heat</td>
</tr>
<tr>
<td></td>
<td>164 ± 6 cool</td>
</tr>
<tr>
<td>HR at maximum, beats( \text{min}^{-1} )</td>
<td>194 ± 5 194 ± 8 191 ± 5 185 ± 10 195 ± 7 heat</td>
</tr>
<tr>
<td></td>
<td>188 ± 7 cool</td>
</tr>
<tr>
<td>SV, % change</td>
<td>( \downarrow ) 16 ( \downarrow ) 10 ( \downarrow ) 12 ( \downarrow ) 14 ( \downarrow ) 11 heat</td>
</tr>
<tr>
<td>SV at 15 min, mL</td>
<td>121 ± 12 107 ± 11 137 ± 13 115 ± 15 110 ± 12 heat</td>
</tr>
<tr>
<td></td>
<td>117 ± 9 cool</td>
</tr>
<tr>
<td>SV at the end of exercise, mL</td>
<td>101 ± 12 96 ± 7 121 ± 9 99 ± 13 98 ± 9 heat</td>
</tr>
<tr>
<td></td>
<td>117 ± 9 cool</td>
</tr>
<tr>
<td>Q, % change</td>
<td>( \downarrow ) 6 -- ( \uparrow ) 5 -- -- ( \downarrow ) 5 heat</td>
</tr>
<tr>
<td>Q at 15 min, L.( \text{min}^{-1} )</td>
<td>18 ± 1 16 ± 2 20 ± 2 16 ± 2 16 ± 1 heat</td>
</tr>
<tr>
<td></td>
<td>16 ± 1 cool</td>
</tr>
<tr>
<td>Q at the end of exercise, L.( \text{min}^{-1} )</td>
<td>17 ± 2 16 ± 2 21 ± 1 16 ± 2 16 ± 1 heat</td>
</tr>
<tr>
<td></td>
<td>16 ± 1 cool</td>
</tr>
<tr>
<td>( \dot{VO}_{2\text{max}} ), % change</td>
<td>( \downarrow ) 19 ( \downarrow ) 15 ( \downarrow ) 18 ( \downarrow ) 19 ( \downarrow ) 15 heat</td>
</tr>
<tr>
<td></td>
<td>( \downarrow ) 5 cool</td>
</tr>
<tr>
<td>( T_e ) at the end of exercise, °C</td>
<td>38.9 38.7 38.5 38.8 39.1 heat</td>
</tr>
<tr>
<td></td>
<td>38.3 cool</td>
</tr>
<tr>
<td>( T_d ) at the end of exercise, °C</td>
<td>36.2 36.2 36.0 33.5 36.5 heat</td>
</tr>
<tr>
<td></td>
<td>33.5 cool</td>
</tr>
<tr>
<td>( T_b ) at the end of exercise, °C</td>
<td>38.6 38.4 38.3 38.1 38.7 heat</td>
</tr>
<tr>
<td></td>
<td>37.7 cool</td>
</tr>
<tr>
<td>Body mass loss during exercise, %</td>
<td>2.5 2.2 2.2 3.7 N/A; fluid provided</td>
</tr>
<tr>
<td>Peak power output, % change</td>
<td>( \uparrow ) 16 ( \uparrow ) 15 ( \uparrow ) 17 ( \uparrow ) 13 ( \uparrow ) 12</td>
</tr>
<tr>
<td>Graded exercise test duration, % change</td>
<td>( \uparrow ) 35 ( \uparrow ) 33 ( \uparrow ) 37 ( \uparrow ) 27 ( \uparrow ) 28</td>
</tr>
</tbody>
</table>

Where applicable, values represent percentage changes at the end of exercise from the value at 15 min. HR and SV data are means ± SD. In the Lafrenz et al. study (18), heat = 35°C and cool = 22°C; \( \uparrow \), increase; \( \downarrow \), decrease; --, no change; \( T_e \), ambient temperature; HR, heart rate; SV, stroke volume; Q, cardiac output; \( \dot{VO}_{2\text{max}} \), maximal oxygen uptake; \( T_d \), rectal temperature; \( T_b \), mean skin temperature; \( T_a \), mean body temperature.
Effect of CV Drift on \( VO_{2\text{max}} \)

We initially identified a relationship between CV drift that occurs during exercise in a hot environment and a decrease in \( VO_{2\text{max}} \). In a study involving cycling at 60% \( VO_{2\text{max}} \) for 45 min in 35°C and 40% relative humidity (33). A 12% increase in HR and a 16% decrease in SV during the course of the exercise bout between 15 and 45 min were associated with a 19% reduction in \( VO_{2\text{max}} \). This occurred despite achievement of a slightly higher HR at \( VO_{2\text{max}} \), measured after 45 min compared with 15 min. Mean skin temperature was similar between 15 and 45 min of submaximal exercise, but rectal and mean body temperatures increased approximately 1°C on average (to 38.9°C and 38.6°C, respectively) between 15 and 45 min (34) (Table).

\( VO_{2\text{max}} \) After Attenuation of CV Drift via Decreased Exercise Intensity

We conducted another study to determine if \( VO_{2\text{max}} \) was reduced when exercise intensity and absolute \( VO_{2} \) were lowered by a magnitude sufficient to reduce CV drift and sustain constant HR during prolonged submaximal exercise in 35°C and 45% relative humidity (33). This type of intervention mimics the practice of lowering exercise intensity to keep HR at a prescribed target level, which often is done as part of exercise prescription. In the control condition, HR increased 13%, SV decreased 10%, and \( VO_{2\text{max}} \) decreased 15%, similar to the results in the study described in the previous section (34). In the experimental condition, power output had to be lowered approximately 37% (on average from 157 W to 98 W) to keep HR constant. SV declined approximately 21%, although this likely was caused by the decreased \( Q \) in response to reduced metabolic demand from the lower work load and not an indication of CV drift (33). Because only HR was manipulated in this study, a research design in which both HR and SV were maintained was necessary to manipulate CV drift and further elucidate the nature of the relation between CV drift and \( VO_{2\text{max}} \).

Effect of Body Cooling on CV Drift and \( VO_{2\text{max}} \) During Heat Stress

To manipulate CV drift by maintaining both HR and SV and then determine the subsequent effect on \( VO_{2\text{max}} \), we conducted a study with a similar design as those mentioned previously except the intervention involved body cooling (fan airflow at 4.5 m s\(^{-1}\) directed at participants) commencing at the same time in which CV drift began (~15 min). In the experimental treatment with body cooling, HR only increased 4% and SV only decreased 3%, which was associated with a 6% decrease in \( VO_{2\text{max}} \). These results demonstrate the relationship between CV drift and a decrease in \( VO_{2\text{max}} \) in that when CV drift is mitigated, effects on \( VO_{2\text{max}} \) are mitigated as well.

Effect of Dehydration on CV Drift and \( VO_{2\text{max}} \) During Heat Stress

To examine the influence of dehydration on CV drift and \( VO_{2\text{max}} \), we carried out a study (10) with a similar design as the aforementioned studies (32–34) except that exercise was more prolonged (2 h), took place in a cooler environment (30°C), and elicited a greater level of dehydration (3.7%). The magnitude of CV drift between 15 and 120 min of moderate-intensity exercise was similar to those of the aforementioned studies (14% increase in HR and 14% decrease in SV), but the decrease in \( VO_{2\text{max}} \) was only about half as large (9%; Table). Rectal temperatures at the end of exercise were similar to those in the studies using a 45-min submaximal exercise period. However, thermal strain, as indicated by \( T_b \), was lower (because of lower \( T_d \)) (10). These findings suggest that the CV drift accompanying thermal strain has a greater effect on \( VO_{2\text{max}} \) than the CV drift associated with dehydration. Furthermore, these findings support the notion that the ergogenic effects of fluid ingestion may in part be mediated through a reduction in the detrimental effect of CV drift on \( VO_{2\text{max}} \).

Effect of Ambient Temperature on CV Drift and \( VO_{2\text{max}} \)

The preceding studies (10,32–34) established a plausible causal link between the CV drift that occurs in a warm-hot environment and decreases in \( VO_{2\text{max}} \). However, these studies did not evaluate whether the CV drift that occurs in temperate conditions (9,25), when thermal strain is less, is associated with reduced \( VO_{2\text{max}} \). Given that thermal strain may be an important modulator of the relationship between CV drift and \( VO_{2\text{max}} \) (see previous section), it was important to more precisely examine the CV drift–\( VO_{2\text{max}} \) relationship when thermal strain was manipulated using varied environmental conditions. Using the same design (separate 15- and 45-min trials to assess CV drift and \( VO_{2\text{max}} \)) as the studies manipulating CV drift using hyperthermia (34), exercise intensity (33), and body cooling (32), we performed an additional study using a cool (22°C) environment as an experimental condition. The control condition was carried out in 35°C as before. Fluid was provided to offset sweat losses so that effects of ambient temperature and accompanying thermal strain could be evaluated independently of effects of dehydration. The magnitude of CV drift and accompanying decrement in \( VO_{2\text{max}} \) in the control condition were comparable to our other studies (11% increase in HR, 11% decrease in SV, and 15% decrease in \( VO_{2\text{max}} \)). However, CV drift was mitigated in the cool condition (2% increase in HR and 2% decrease in SV) such that the decrease in \( VO_{2\text{max}} \) was mitigated as well (nonsignificant 5% decrease). Manipulation of CV drift in this study with a consequent directionally similar effect on the reduction in \( VO_{2\text{max}} \) adds further support to a cause-and-effect nature of the relation between CV drift and reduced \( VO_{2\text{max}} \). In other words, a larger CV drift corresponds to a larger decrement in \( VO_{2\text{max}} \), and a smaller CV drift corresponds to a smaller decrement in \( VO_{2\text{max}} \).

Mechanism Explaining Decreased \( VO_{2\text{max}} \) Associated With CV Drift

We cannot be certain of the precise mechanism explaining the decrement in \( VO_{2\text{max}} \) associated with CV drift. However, we speculate that the decreased SV associated with CV drift that was present during submaximal exercise persisted during the GXT to determine \( VO_{2\text{max}} \) (4,25). This relationship is illustrated in Figure 1. The decrease in SV observed in studies using a variety of methods manipulating CV drift corresponded to a directionally similar decrease in \( VO_{2\text{max}} \). Measures of maximal effort, like ratings of perceived exertion,
Implications for Exercise Performance

Besides negatively influencing \( VO_{2\text{max}} \), our studies also have demonstrated that CV drift negatively influences performance (as indicated by maximum power output attainable and duration of GXT to measure \( VO_{2\text{max}} \)) (10,18,32–34). In our studies in which CV drift and \( VO_{2\text{max}} \) were evaluated between 15 and 45 min of exercise in 35 °C, maximum power output declined 12%–17% and GXT duration declined 28%–37% (18,32–34). In our other study in which CV drift and \( VO_{2\text{max}} \) were evaluated between 15 and 120 min of exercise in 30°C with no fluid ingestion, maximum power output declined 13% whereas \( VO_{2\text{max}} \) test duration decreased 27% (10). Although there are more representative and valid measures of performance available (17), these results nonetheless suggest that CV drift has the potential to negatively impact endurance performance. In addition, because \( VO_{2\text{max}} \) is lowered in conjunction with CV drift, any given absolute work level represents a higher relative metabolic intensity (%\( VO_{2\text{max}} \) or %\( VO_{2R} \)) and, thereby, physiological strain (1), which may impair performance.

Implications for Exercise Prescription

“The scientific evidence demonstrating the beneficial effects of exercise is indisputable, and the benefits far outweigh the risks in most adults” (11). The power of this statement is realized fully only when exercise is prescribed appropriately. Exercise can be prescribed many ways, but because of its linear relation to relative metabolic intensity (%\( VO_{2\text{max}} \) or %\( VO_{2R} \), ease of measurement, and relative low cost for monitoring equipment that can be worn during a variety of types of exercise, HR frequently is used to gauge exercise intensity. However, studies that validated the relation between HR and relative metabolic intensity used brief exercise of progressively increasing intensity (3,29), thereby leaving the question as to what extent HR reflects relative metabolic intensity during prolonged exercise unanswered. Our research demonstrates that \( VO_{2\text{max}} \) is decreased concurrently with CV drift, so a given absolute work level reflects a higher relative metabolic intensity. As such, the upward drift in HR during heat stress coincides with exercise requiring a higher percent of \( VO_{2\text{max}} \). Importantly, data from our studies suggest that the HR-%\( VO_{2\text{max}} \) relationship is preserved (Fig. 2). Consequently, HR remains a reliable indicator of the relative CV or exercise intensity. Individuals prescribing exercise at a constant target HR under conditions in which CV drift is likely to occur should consider that doing so reduces the absolute metabolic stimulus of the exercise, and therefore, cardiorespiratory training adaptations may be compromised. That said, maintaining absolute intensity and allowing HR to rise over time in a hot environment will result in increased thermal and CV strain, which may render some individuals more susceptible to complications associated with exercise in a hot environment, such as development of a heat illness. Practitioners must balance maintaining a target training stimulus with ensuring participant safety.

SUMMARY

A schematic diagram summarizing the major factors affecting CV drift and subsequent effects of CV drift on \( VO_{2\text{max}} \) is presented in Figure 3. The causes of CV drift are multifactorial, with two prevailing hypotheses: Peripheral displacement of blood volume associated with hyperthermia may

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**Figure 1.** Relationship between the change in stroke volume (SV) that occurs over time during submaximal, moderate-intensity (~60% maximal oxygen uptake, \( VO_{2\text{max}} \)) constant-rate exercise in a warm-hot environment and the change in \( VO_{2\text{max}} \), that occurs over the same time interval. The decrease in SV observed during submaximal exercise may persist during a maximal effort such that \( VO_{2\text{max}} \) is decreased. Data from (10,18,32–34). Because some data points represent multiple trials for the same subjects, a correlation adjusted for repeated measures is presented.

**Figure 2.** Relationship between the change in percent heart rate reserve (%HRR) and percent oxygen uptake reserve (%\( VO_{2R} \)) during submaximal, moderate-intensity (60% maximal oxygen uptake), constant-rate exercise in a hot environment. The increase in %HRR is associated with a proportional directionally similar increase in %\( VO_{2R} \), thereby implying that the relation between HR and relative metabolic intensity is preserved during exercise in the heat when CV drift occurs. Data from (18,32–34). Because some data points represent multiple trials for the same subjects, a correlation adjusted for repeated measures is presented.
Figure 3. Schematic of the interrelationships among mediators of CV drift, CV drift, and decreased maximal oxygen uptake (VO2max) over time during prolonged submaximal exercise, with implications for exercise prescription. The decrease in VO2max associated with CV drift, coupled with maintained submaximal VO2 over time, results in increased percent oxygen uptake reserve (%VO2R) over time. This supports the use of HR as a marker of change in relative metabolic intensity during prolonged exercise in the heat in which HR rises progressively over time in conjunction with a CV drift. HR, heart rate; SV, stroke volume; HRR, heart rate reserve; +, accentuates CV drift; †, increase; ††, decrease; ---, no change.

Contribute to reduced SV and increased HR; and/or increased HR may reduce ventricular filling time and, thereby, SV. Regardless of the cause, CV drift can be modified by several factors, including ambient and core body temperatures, hydration status, and exercise duration. The consequences of CV drift are less known than the causes but include reduced VO2max and potentially reduced performance. Under conditions in which CV drift occurs, a maximal circulatory capacity may be reached prematurely and/or reductions in SV observed during submaximal exercise may persist during maximal exercise such that VO2max is reduced.

We have demonstrated that the CV drift (11%–16% increase in HR, 10%–16% decrease in SV) that occurs during 45–120 min of moderate-intensity constant-rate exercise in a warm-hot environment consistently results in a substantial decrease in VO2max (9%–19%) (10,18,32–34). When the magnitude of CV drift is smaller (0%–4% increase in HR, 2% decrease in SV), the decrease in VO2max is concomitantly smaller (5.0%–7.5%). Mean changes in HR and SV (CV drift) followed by proportional mean changes in VO2max across studies suggest that the decrease in SV associated with CV drift may cause the reduction in VO2max. The link between CV drift and decreased VO2max appears to be independent of the mode of manipulation of CV drift but dependent on factors that influence the magnitude of CV drift, such as hyperthermia and dehydration. Accompanying CV drift and decrements in VO2max is reduced performance, indicated by a decreased maximal power output. In addition, the upward drift in HR is proportional to the greater relative metabolic intensity resulting from the decrease in VO2max so HR remains useful for gauging exercise intensity. Lowering speed or power output to preserve HR results in a decreased exercise intensity, so aerobic training adaptations may be compromised.

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