

Cardiovascular Drift During Prolonged Exercise: New Perspectives

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COYLE, E. F., and J. GONZÁLEZ-ALONSO. Cardiovascular drift during prolonged exercise: New perspectives. *Exerc. Sports Sci. Rev.* Vol. 29, No. 2, pp. 88–92, 2001. *We propose that cardiovascular drift, characterized by a progressive decline in stroke volume after 10–20 min of exercise, is primarily due to increased heart rate rather than a progressive increase in cutaneous blood flow as body temperature rises.* **Keywords:** stroke volume, heart rate, cutaneous blood flow, hyperthermia, endurance, classic cardiovascular drift

INTRODUCTION

“Cardiovascular drift” (CV drift) is a phenomenon whereby some CV responses begin a continuous time-dependent change, or “drift,” after ~ 10 min of prolonged moderate-intensity exercise (e.g., 50–75% $\dot{V}O_{2\max}$) in a neutral or warm environment (2,8). As displayed in Figure 1A, it is characterized by a progressive decline in stroke volume (SV) and pulmonary and systemic mean arterial pressures (MAPs) and a parallel increase in heart rate (HR), whereas cardiac output is maintained nearly constant (2,13). In the classic study of CV drift (2), hydration was maintained with saline infusion, and blood volume did not change after 10 min of exercise. Although CV drift can occur without dehydration, the latter portion of this review discusses the mechanism by which dehydration greatly exacerbates CV drift. Therefore, CV drift is a phenomenon, classically observed in non-endurance-trained persons working in thermoneutral environments, that is associated with progressive but small increases in core temperature. SV reductions after the first 10- to 15-min period of exercise probably provide the single best index of CV drift and are generally mirrored by increased HR. However, during low-intensity exercise in the heat or exercise performed in the semirecumbent position, SV can be maintained as HR increases progressively throughout exer-

cise, leading to increased cardiac output (12–14). This review focuses on CV drift that is characterized by declining SV.

TRADITIONAL HYPOTHESES AS TO THE CAUSE OF CARDIOVASCULAR DRIFT

The cause of CV drift can be due to alterations in cardiac and/or vascular function. The long-held and presently prevailing concept, summarized by Rowell (13), is that CV drift is due to a progressive increase in cutaneous blood flow, as body temperature rises. The rise in cutaneous blood flow is thought to lead to an increase in cutaneous venous volume, thus reducing ventricular filling pressure, end-diastolic volume, and SV during moderate-intensity exercise (13). However, direct data for these hypotheses are lacking. First, the increase in cutaneous blood flow is not temporally related to the progressive reduction in SV. On average, cutaneous blood flow actually remains fairly stable after 20–30 min, when core temperature reaches a plateau (3,8,12). It seems there is little evidence linking the progressive decline in SV to the cutaneous circulation during prolonged exercise in most environmental conditions (Figure 2). It should be kept in mind that there are conditions promoting drastically different levels of skin circulation that do not compromise SV. In this light, we recently observed the same SV during moderately intense exercise in the heat (35°C) and cold (8°C) in trained subjects, despite a four-fold difference in cutaneous blood flow (7), reinforcing the dissociation between SV and cutaneous blood flow.

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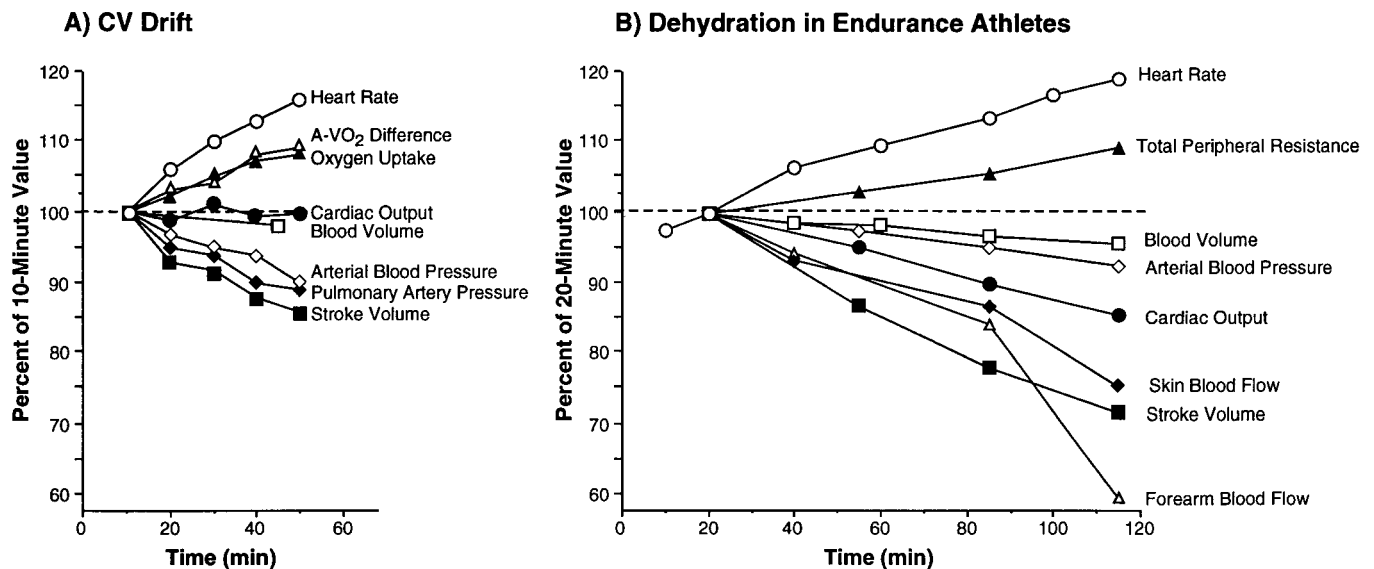


Figure 1. A. Classic CV drift without dehydration. (Redrawn from Ekelund et al. [2].) B. Summary of the effects of dehydration and concomitant hyperthermia, displaying the responses of endurance-trained cyclists during 120 min of exercise at 62–65% $\dot{V}O_{2\max}$ in a 35°C environment when they begin exercise while euhydrated and become dehydrated by 4.9% of body weight after 120 min of exercise. Data are not shown for another trial during which fluid was ingested to offset dehydration and all responses were remarkably stable. (Redrawn from González-Alonso et al. [4].)

THE DECLINE IN STROKE VOLUME IS RELATED TO INCREASED HEART RATE

An alternate hypothesis to the “cutaneous circulation” hypothesis is that the decline in SV during prolonged exercise is due to increased HR (Figures 2 and 3). This would decrease ventricular filling time and end-diastolic volume (1,15). To determine whether prevention of the increase in HR that occurs during the 15- to 55-min exercise period under conditions of classic CV drift would prevent the decline in SV during the same period, a recent study “clamped HR” by having subjects ingest a small dose of a β_1 -adrenoceptor blocker (i.e., Atenolol) immediately before exercise (3). Remarkably, this prevented the increase in HR after ~ 15 min but not the initial increase in HR (i.e., from 0 to ~ 15 min of exercise) (Figure 2).

As shown in Figure 2, during normal exercise under control conditions, the decline in SV after 15 min of exercise was temporally related to an increase in HR and unrelated to an increase in cutaneous blood flow. This is demonstrated first by the observation that β -blockade prevented the increase in HR after 15 min of exercise, and accordingly it prevented the decline in SV, despite a normal cutaneous blood flow response. Second, during the normal control situation, a stable cutaneous and forearm blood flow after 15–20 min of exercise did not prevent the decline in SV. During the 15- to 55-min period of control, SV declined 13%, HR increased 11%, and the final core temperature was 37.8°C; all of which are typical of CV drift as reported in classic studies (2,8).

Most important, the decline in SV from 15 to 55 min was totally eliminated with prevention of the increase in HR through the use of mild β -blockade (Figure 2). This indicates that the increase in HR is largely responsible for the decline in SV during prolonged exercise. Other variables that could have caused the decline in SV (i.e., blood volume, cutaneous blood flow, forearm blood flow, forearm venous volume, esophageal core temperature, and skin temperature) were

similar during β -blockade compared with control. HR pacing that increases HR at rest and during exercise can indeed reduce SV by reducing diastolic filling time (1,15).

VENOUS POOLING MIGHT DECREASE STROKE VOLUME

The observation that treatments that do indeed raise skin temperature to 38°C to manipulate venous pooling and the cutaneous circulation can alter SV does not invalidate the lack of relationship between cutaneous blood flow and SV during normal exercise. This is because under normal environmental conditions that display CV drift, skin temperature is typically 31–32°C (3,8). Skin temperatures that are elevated via a water-perfused suit to very high levels (i.e., ~ 38°C), and thus abolish cutaneous venous tone during exercise, can have potentially powerful effects on reducing SV during moderate-intensity exercise (14). Clearly, the observation that reductions in SV induced by heating the skin to 38°C can be reversed by cooling the skin (14) indicate involvement of the cutaneous circulation, probably by inducing venous pooling. Yet, there is no evidence to indicate that progressive venous pooling occurs during prolonged moderate-intensity exercise in environmental conditions that elicit skin temperatures of 31–34°C, which are the conditions that characterize classic CV drift (3,8).

RELATIONSHIP OF CORE TEMPERATURE TO CARDIOVASCULAR DRIFT

If indeed the decline in SV is due to increased HR, the next question is, What causes the increase in HR with the progression of exercise duration? Figure 3 hypothesizes that increased core temperature and sympathetic nervous activity

are two likely possibilities. Increases in core temperature and HR during CV drift are strongly correlated ($r^2 = 0.95$; see Fritzsche et al. [3]). A lack of increase in core temperature is also associated with a lack of decline in SV (4,5). González-Alonso et al. (5) observed that the elevation of core temperature from 38.3°C to 39.3°C, by itself (i.e., without dehydration), elicited a lower SV and an increased HR. Thus, hyperthermia seems to accentuate CV drift by increasing HR. This could occur from the direct effect of temperature on intrinsic HR or from the indirect effect of hyperthermia on increasing sympathetic outflow (5,9). Progressive increases in

HR might also be derived from increasing sympathetic nervous system activation in response to other factors, such as fatigue of the neuromuscular system.

CARDIOVASCULAR DRIFT INDUCED BY DEHYDRATION

Figure 1B displays the responses of endurance-trained cyclists exercising at 62% $\dot{V}O_{2\max}$ for 120 min in a hot environment (i.e., 35°C with fan) that produces a 4.9% loss of body weight (4). The prevention of dehydration through fluid ingestion results in remarkably stable CV responses as reflected in both SV and core temperature not changing during the 20- to 120-min period (not shown in figures). Clearly, CV drift under these experimental conditions is due to dehydration and not to other effects of prolonged exercise (e.g., fatigue). The most salient effects of dehydration are the 28% reduction in SV, an 18% decline in cardiac output, and the elevation in core temperature from 38.0°C to 39.3°C (4) (Figure 1B). This remarkable fall in cardiac output is due to the larger decline in SV compared with the increased HR. To cope with this severe and unique challenge, the CV system increases systemic vascular resistance (i.e., 17%), thus limiting reductions in MAP. The cutaneous circulation participates in this general vasoconstrictor response, thus reducing heat dissipation and promoting hyperthermia (4). The next two questions become, By what mechanisms do dehydration and hyperthermia reduce SV? By what mechanism does dehydration increase cutaneous vascular resistance, thus reducing cutaneous blood flow and promoting hyperthermia?

DEHYDRATION-INDUCED REDUCTIONS IN BLOOD VOLUME

Dehydration by 3–5% of body weight during exercise also reduces blood volume by ~ 3–5% during the 5- to 120-min period of exercise (3,4,10,11). Intravenous infusion of a plasma volume expander, to prevent dehydration induced

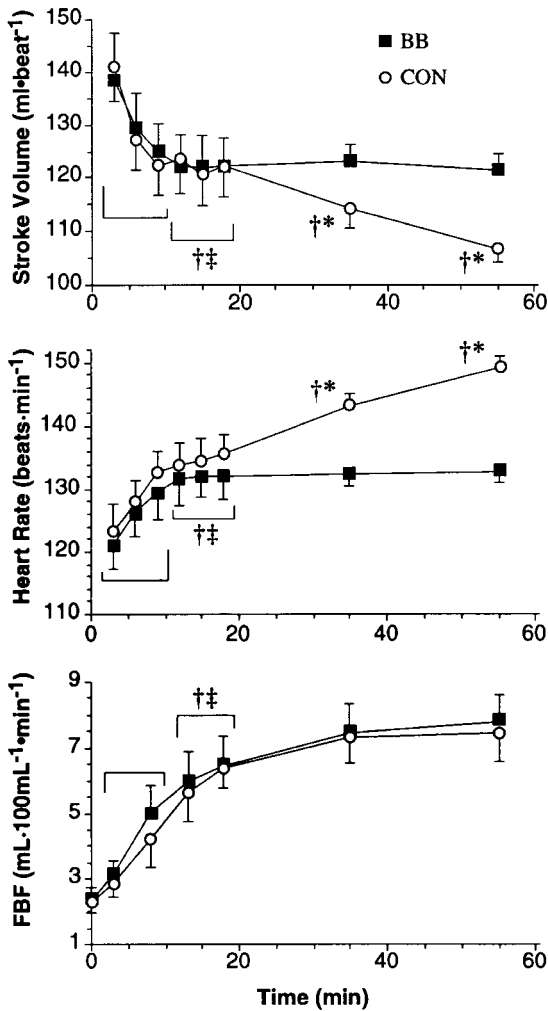


Figure 2. Mean \pm SEM ($n = 7$) values for SV, HR, and forearm blood flow (FBF) for 60 min of exercise during the β_1 -adrenoceptor blockade (BB, ■) and control (CON, ◇) treatments. During the 0- to 20-min exercise period (values connected by solid lines), there was no significant difference between BB and CON, and thus these treatments were pooled to analyze changes over time. * $P < 0.05$, different from the previous time point during the 0- to 20-min period; BB and CON treatments were pooled. To analyze the responses to prolonged exercise in both BB and CON, values during the 10- to 20-min period (marked with brackets) were averaged to derive a 15-min value and, together with the 35- and 55-min values (connected by dotted lines), were analyzed for treatment and time effects. § $P < 0.05$, BB different from CON. † $P < 0.05$, during CON, values at 55 min different from those at 35 min and/or values at 35 min different from those at 15 min. ‡ $P < 0.05$, during BB, values at 55 min different from those at 35 min and/or values at 35 min different from those at 15 min. (From Fritzsche et al. [3] with permission.)

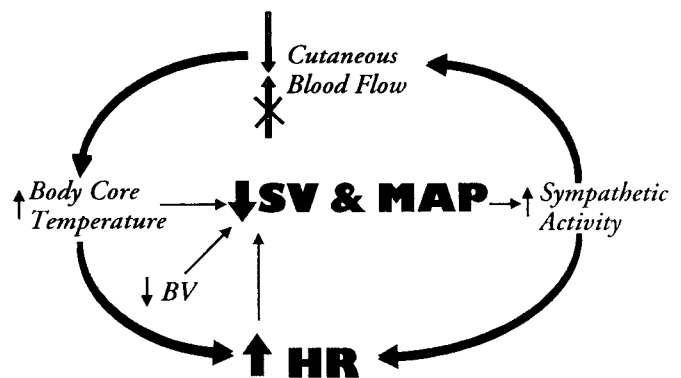


Figure 3. New perspective regarding mechanisms for CV drift during prolonged exercise under conditions of maintained cardiac output and how it is exacerbated by dehydration, which acts primarily by causing hyperthermia (i.e., increased body core temperature) and hypovolemia (i.e., decreased blood volume [BV]). CBF, cutaneous blood flow, calf blood flow.

reductions in blood volume, has no effect on attenuating the reduction in cutaneous blood flow or hyperthermia that is characteristic of dehydration (10). Keep in mind that only 200–300 mL of blood volume expansion is required to offset dehydration of the blood volume, and this does not offset dehydration of the extravascular space, which amounts to several liters of fluid. Therefore, dehydration-induced hyperthermia results from some other aspect of whole body dehydration rather than simply reductions in blood volume (i.e., dehydration of the blood).

However, reductions in blood volume from dehydration do account for approximately one half of the reduction in SV during hyperthermia, as depicted in Figure 3, based on blood volume expansion studies (10). What then accounts for the remaining reduction in SV? In that blood volume expansion did not attenuate hyperthermia and it only slightly reduced HR, it is possible that the maintained HR elevation contributed to reduced SV. Remarkably, the hyperthermia derived, in large part, from cutaneous vasoconstriction during moderate-intensity exercise can be prevented only by a cold environment that sufficiently lowers skin temperature and thus increases the core to skin temperature gradient (5,7). Under conditions of dehydration without hyperthermia, created by exercising dehydrated subjects in a cold environment, all of the reduction in SV (i.e., 7%) appears to be due to reduced blood volume (5). This is based on the observation that blood volume restoration fully restores SV to euhydrated levels despite the persistence of extravascular dehydration (5). Thus, hypovolemia from dehydration by itself can reduce SV by 7%, but when it is combined with hyperthermia, the declines in SV are much greater and synergistic (i.e., 20–28%) (4,5,7,10) (Figure 3).

DEHYDRATION CAUSES HYPERTHERMIA

It is quite possible that the large elevation in plasma norepinephrine experienced during exercise when dehydrated and hyperthermic is related to cutaneous vasoconstriction (4,6). The fact that the cutaneous circulation experiences vasoconstriction during hyperthermia suggests that the CV system is responding to nonthermal control, probably from baroreceptors (6). It is possible that both low- and high-pressure baroreceptors were unloaded, because dehydration reduced both SV and MAP (6). Cutaneous vasoconstriction with dehydration could result from the individual or combined effects of two efferent pathways: (a) imposition of cutaneous vasoconstriction on top of the thermally induced active vasodilatory process and/or (b) withdrawal of cutaneous active vasodilation. During exercise in the upright position, the plasma norepinephrine increase with dehydration is very large and is associated with hypotension and cutaneous vasoconstriction (4,6). However, when these same subjects who were equally dehydrated and hyperthermic exercised in the supine position, MAP was maintained at euhydrated levels, as was plasma norepinephrine and cutaneous blood flow (6). These observations support our hypothesis that reductions in MAP with dehydration elicit increased sympathetic nervous activity and decreased cutaneous blood flow from vasoconstriction, which in turn promotes greater hyperthermia (Figure 3).

Acute elevation of plasma catecholamines (via intravenous epinephrine infusion) to the levels observed with dehydration causes immediate reductions in cutaneous blood flow and ensuing hyperthermia (11). Clearly, this indicates that the cutaneous vasculature is responsive to the elevation of plasma catecholamines. Furthermore, the lowering of plasma catecholamines via hyperglycemia from intravenous glucose infusion leads to attenuated hyperthermia, possibly due to the increased heat dissipation produced by the concomitant increases in skin blood flow (11). Therefore, it seems reasonable that the cutaneous vasoconstriction during exercise from dehydration could be due to the pronounced increase in sympathetic nervous system activity, as shown in Figure 3, superimposed on the active vasodilator system. Whether the main avenue of sympathetic response is plasma or locally released catecholamines is not clear. This does not exclude the possibility that reduced cutaneous blood flow might also result from withdrawal of active vasodilation.

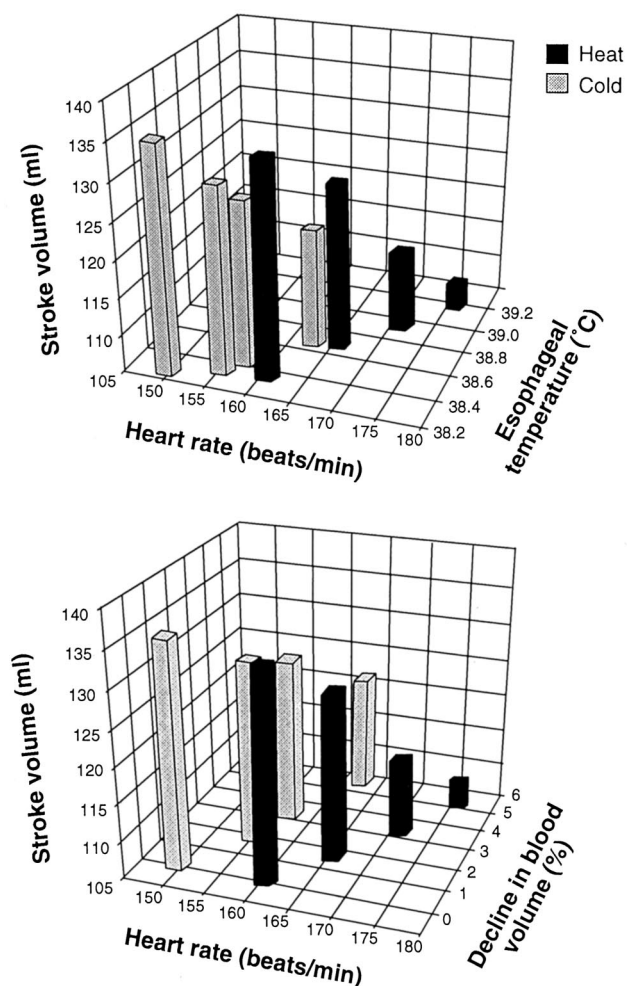


Figure 4. SV responses during exercise in the heat and the cold under various levels of hydration. SV is related to the interaction of HR, blood volume, and esophageal temperature under these varied experimental conditions. (From González-Alonso et al. [7], Figures 7 and 8.)

INTERACTION OF FACTORS

The observation that SV reductions from the combination of dehydration-induced hypovolemia and hyperthermia (i.e., 20–28%) are much larger than the sum of the effects of dehydration or hyperthermia alone (i.e., each 7–8%) (5) indicates an interaction of factors. One interpretation is that the reduced filling pressure from dehydration-induced hypovolemia combined with a shortened diastolic filling time from increased HR and core temperature produces a disproportionately large reduction in end-diastolic volume and thus SV (Figure 3). The interactive effects of increased HR, increased body core temperature, and reduced blood volume on reducing SV during exercise are displayed in Figures 4A and 4B (7).

SUMMARY

The most striking component of “classic” CV drift as well as of “dehydration-induced” CV drift is the reduction in SV. Clearly, reductions in SV with either form of CV drift are not due to increased cutaneous blood flow, and it is unlikely that cutaneous venous volume is increased. Instead, it was recently demonstrated that the reduction in SV with “classic” CV drift can be completely prevented when the normal drift upward in HR is prevented (3). Dehydration-induced reductions in SV are due to the almost inevitable hyperthermia from cutaneous vasoconstriction, as well as reductions in blood volume and increased HR. Hypovolemia accounts for approximately one half of the decline in SV, and hyperthermia is associated with the other half of the decline in SV (3–7,10). It is likely that hyperthermia reduces SV by increasing HR. In agreement with these hypotheses, SV is not reduced during exercise in dehydrated subjects, who have their blood volume expanded to normal levels and their hyperthermia and thus HR acceleration prevented (5).

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