

An Investigation of the Fluid Replacement Effects on Cardiovascular Drift Responses to Strenuous Prolonged Exercise

Luna Rizzo^{1*}, Martin W Thompson²

¹Medical Sciences, Department of Biomedical and Neuromotor, Sciences, University of Bologna, Bologna, Italy

²Exercise, Health & Performance, Faculty of Health Sciences, University of Sydney, Sydney, NSW, Australia

Abstract

The replacement fluid ingested immediately after exercise provides the hydration for the next exercise. Limiting fluid ingested before exercise can cause problems to the thermoregulation body process during the following exercise if that fluid is not adequate. The loss of water from the body due to sweating is a function related to the combined effects of exercise intensity and environmental conditions (temperature, humidity, wind speed). In humans, the sweating can exceed 30 g per minute (1.8 kg per hour). The loss of water through sweating comes from all compartments of body fluids, also including the blood volume. This will cause easily an increase in the concentration of electrolytic in body fluids. Despite, the effect of dehydration on cardiovascular function and thermoregulation, the extent to which exercise performance is impaired by a fluid deficit remains unclear. Although some data indicate that drinking improves athletic ability in executing events of short duration (1 hour) in temperate climates, other data suggest that this might not be the case [1]. The maintenance of glucose concentrations in blood is necessary to optimize the performance of the exercise. In conclusion, the fluids replacement for the body is important to maintain normal hydration, and minimize risk of injury from heat and impairment of exercise performance during exercise. During the exercise lasting more than 1 hour carbohydrates should be added to the solution of fluid replacement to maintain the concentration of glucose in the blood and to increase exercise time to fatigue. The concentration of glucose in the fluid ingested depends on whether there is a primary need for energy or hydration. This review paper is focused on mechanisms of cardiovascular drift (CV_{drift}) to maintenance and replacement fluid therapy during strenuous prolonged exercise. Many aspects are treated in the manuscript. The emerging topics treated in this review paper also include: cardiovascular responses and adaptations to acute exercise, heart rate adaptations during different time of the day in elite athletes, hydration status and improvement of performance, effects of dehydration and hyperthermia on CV_{drift} . The literature on the topic appears to be vast and address many important factors of interest for the performance development occurred in dehydration condition and it is necessary to be known for the preservation of the athletes' health and performance enhancing.

Keywords: Strenuous exercise, Cardiovascular response, Hyperthermia, Glucose tolerance, Performance impairment, Body loses, Fluid deficiency

*Corresponding author: Luna Rizzo, Medical Sciences, Department of Biomedical and Neuromotor, Sciences, University of Bologna, Bologna, Italy; E-mail: luna.rizzo2@unibo.it; luna.rizzo8@gamil.com

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Responses to acute exercise and long-term adaptations to training

Responses to endurance training metabolic adaptations occur in skeletal muscle. Primarily, the oxidative enzymes activity increases and this will increase the number and size of mitochondria. The content of myoglobin in the muscle can be also raised, depending on the oxygen amount accumulated in the fibers of muscle. This kind of adaptation, matched with the increase in muscle blood flow and muscle capillaries, refine the capacity of oxidation of the trained muscle during the endurance activity. Training of endurance also increases the capacity by the muscle to accumulate glycogen [2]. Trained muscles have also the ability to use fat as a source of energy, and this process lead to a higher utilization of glycogen stores. Following endurance training there is an increased capacity by muscles to use fat and mobilize free-fatty acids from depots of fat. In this circumstance, muscles have also an improved capacity to oxidize fat as a consequence of the rise in enzymes of muscles that are responsible for oxidation of fat [2].

In endurance training also occur significant respiratory and cardiovascular changes during stable state of exercise and at rest at submaximal work rate and maximal work rate. In this case, the adaptation mechanisms level depends on different factors. First, the initial personal level of fitness of the individual; the type of intensity, duration, frequency of exercise, and training length in terms of months, weeks, years, etc. In this scenario, long-term cardiovascular adaptations represent one of the major points to which this chapter is focused; it provides an overview of how the cardiovascular system adapts to endurance training, with differences in rate of work.

Following a program of endurance training, cardiac output in both at rest and during exercise performed at submaximal rate, remains unchanged. However, at rates of maximal work, cardiac output is raised substantially, up to 30 percent or more [3]. Important differences are shown in heart rate and stroke volume responses to training exercise. For instance, after exercise, stroke volume is augmented during maximal work, submaximal work, and at rest; on the other hand, heart rate is reduced at rest, during submaximal and post-exercise, and it remains unchanged at maximal rate of exercise. Most changes detected in cardiac output are explained by changes in stroke volume that seems to be the dominant factor responsible for such physiological mechanism. When the volume of plasma is increased also the blood volume obtainable to the right part of heart increases, and later to the left ventricle. This large increase in the end-diastolic volume of left ventricle results in a greater elasticity of the heart walls, with a subsequently stretchy recoil.

Heart rate adaptations during night and day time in athletes

Competitive sports require hard training to improve the results of performance. In order to enhance the performance as much as possible is also important not only focuses the training on the hardness itself, but also on the recovery after training. The recovery after training should be

optimized in order to achieve a most satisfying performance. Usually, recovery after a single exercise bout succeed after 24 hours, but elite athletes may train for three times per day for 6 days a week, with a restrict recovery period of 1 or 2 days in total during the week. If an imbalance between training, competition and recovery state occurs, this can develop an overtraining status in athletes. This imbalance can occur often in professional elite athletes. The early stage of overtraining is called overreaching, and it is use to reflect this kind of situation where a short-term overtraining is occurred. A satisfactory recovery after hard training period should consist of 1 or 2 weeks, while after an overtraining state, the recovery time should be no less than months to 1 year. Autonomic nervous system (ANS) dysfunction or imbalance has been recognized as one of the first signs of the overtraining state in athletes. Heart rate (HR) and heart rate variability (HRV) analyzed from RR-interval (RRI) were used as index for the ANS function, and both have supplied tracks to detect the overtraining state. However, variability rhythm in heart rate is associated with parasympathetic modulation and lower rates are considered to be provoked by a reduced parasympathetic modulation and increased sympathetic modulation of sinus node [4].

Higher values of HRV have been linked to higher levels of physical activity, especially for high-frequency power (HFP) training, despite some study did not find any association between HRV and physical training [5]. A single bout of exercise decreases autonomic nervous system activity for more than 2 days [6]. A successful training period with efficacy recovery leads to increased resources of the ANS (i.e., increased parasympathetic and decreased sympathetic modulation) [7]. Overreaching and overtraining state can lead to a reduction in autonomic resources for responding to stress. Previous studies on overtraining and overreaching have established a decrease in general HRV, as well as shifts toward both sympathetic and parasympathetic predominance [7].

Nocturnal stress hormone is a marker of basal autonomic tone. Its concentration has been used to detect the development of the overtraining state. In the final phase of this development, or in a parasympathetic type of overtraining, the basal urinary catecholamine excretion can be reduced by 50-70%. The reports regarding nocturnal urine catecholamine have been questionable, and thus the stress hormone concentration differences between overtrained and well-trained state are still unclear [8]. A study conducted by Hynynen *et al.* [9] investigated the autonomic imbalance in overtrained athletes during night sleep and after awakening, measuring nocturnal urine stress hormone concentrations and HRV. The hypothesis was that athletes who were overtrained would show weakened autonomic stimulus, linked to a lower HRV and nocturnal hormone concentrations comparing with the control group. For the study were examined 12 severely overtrained subjects, 6 men and 6 women. Overtraining state in athletes was diagnosed by a medical doctor. Night sleep time data such as nocturnal RRI and urine samples was asked to subjects to be collected at home along with HRV records, while for awakening data measurements, subjects were invited to the lab in the early morning.

For cardiac autonomic modulation and urinary stress hormones during night the results observed in the study showed no differences in average nocturnal RRI, HRV indices, and urine stress hormones concentrations between the two groups. For cardiac autonomic modulation after awakening the results observed in the study showed both SDRRI and LFP after awakening was lower in overtrained than in control group. In addition, when responses to awakening (night vs after awakening recordings) were calculated, CVRRI decreased more in OA than in CA. From those results by Hynynen [9], the main finding was that cardiac autonomic modulation after awakening was disturbed in overtrained athletes, whereas the autonomic modulation during night sleep did not differ between the overtrained and control athletes. OA also showed to have lower VO_{2max} than CA. In conclusion to this

study, can be assumed that parasympathetic cardiac modulation after awakening was slightly diminished in the overtraining state [9].

Resting bradycardia is a characteristic of trained athletes. Bradycardia is a slow hear rate, characterized by heart rate of under 60 beats per minute (bpm) in adult people. Generally, bradycardia does not provoke pathologic symptoms until it falls down below 50 bpm. In some cases, bradycardia can be less than 40 beats/min (bpm). When this condition became symptomatic, it may provoke dizziness, fatigue, and weakness. During night sleep bradycardia is common, and the rate can drop under 40 bpm, especially in trained athletes this is considered normal [10].

Bradycardia in athletes is mediated by an increase in parasympathetic and a decrease in sympathetic stimulus at rest. Pronounced bradycardia may result in sinus pauses with junctional escape rhythms, first degree heart block and Wenckebach type second degree block especially at night. Resting bradycardia may predispose to increased atrial or ventricular ectopic activity and, in some cases, atrial fibrillation. As hypertrophy, also bradycardia can be seen as a harmful status and some evidence support this view.

Elite trained athletes can be affected by a syndrome called athletic heart syndrome; in this circumstance they show a very low heart rate at resting that appear as an adaptation to high sport activity and it also help to avoid tachycardia (when heart rate exceeds the normal resting rate) during training. Bradycardia condition can be caused by two different types of disorders: disorders of the Sinoatrial Node (SA node), and disorders of the Atrioventricular Node (AV). In the case of sinus node dysfunction, also called sick sinus syndrome, impulse impaired conduction or disordered automaticity from the sinus node to the atrial tissue occur. In atrioventricular conduction disturbances, impaired conduction in the AV node occur. Individuals who have bradycardia acquire it also congenitally.

The effects of age in individuals can modify some of the aspects previously discussed, in fact it was observed that bradycardia is more common in older athletes who show lower maximum heart rate as well. This might also explain a decreased performance ability associated with the athlete 'age' [10].

A study conducted by Jensen-Urstad *et al.* [11] carried out an investigation on pronounced resting bradycardia in male elite runners associated with high heart rate variability. This study was focused on top-class-endurance-trained athletes. The principal aim investigated and discussed in this study was the occurrence and profundity of bradycardia in elite runners and estimation of cardiac autonomic function by analysis of HRV from 24-hour Holter recordings, especially at night time where most basal conditions can be obtained. For the study 16 male middle- and long-distance runners aged between 25-30 years old, competing at national or international level were examined. Subjects were recruited from fitness club. The main inclusion criteria were to select top athletes with no history of cardiac diseases in the past. Results from the study showed that the athletes had pronounced bradycardia during the night time, with heart rate calculated from four RR intervals < 30 beats/min in five runners. The runners were compared with a control group of 13 sedentary or moderately active subjects, and it was found that the runners had a mean of 14 bpm lower heart rate at night than controls. The runners had also higher heart rate variability in all spectral bands. Also, the time domain (pNN50 and rMSSD) considered to reflect the vagal tone, were considerable higher in the runners compared to the control group. Results from this study stated that an increased parasympathetic tone might partly justify the pronounced resting sinus bradycardias observed in endurance trained runners [11].

To the best state of knowledge there is also another significant study, conducted by Goldsmith *et al.* [12], where it is examined 24-hour HRV in endurance-trained subjects testing a mixed group of

eight runners, swimmers, and bicyclists. In this study was compared 24-h parasympathetic activity in aerobically trained healthy young men versus untrained healthy young men. Parasympathetic activity measurements in this study were assessed from 24-h ECG recordings by calculating high frequency (0.15 to 0.40 Hz) beat to beat heart period variability in eight endurance-trained men and eight untrained men aged 29 yr. Data for sleeping time were analyzed separately when parasympathetic activity is dominant and also for waking hours. Results observed from this study showed that the geometric mean of high frequency power was greater in trained men comparing to untrained men during the day, at night time, and over the entire 24 hours' day. The trained subjects demonstrated longer average NN intervals and greater amplitude of high frequency fluctuations in the endurance-trained subjects. In conclusion, can be stated that parasympathetic activity is greater in trained subjects than untrained, and this response is present in both waking and sleeping time. This research suggests that exercise training increase the activity of parasympathetic system over the entire day and it may be also considered to be a new method to reduce drug therapy often used to treat the autonomic imbalance that occur in many cardiovascular diseases [12].

Exercise and fluid replacement: physiological changes on CVD

During exercise the volume of fluid ingested is often not sufficient to compensate for the sweat loss [13]. Following the exercise, the body fluid deficit may become chronic if attention is not given to re-establishing a euhydrated state [14]. In fact, a complete restoration of fluid deficits cannot occur without replacing of electrolytes by food or beverage (primarily sodium) [15]. During exercise the first electrolytes that are lost through sweating are primarily sodium chloride and less potassium [16].

Water ingested 60 minutes before the race will improve thermoregulation and lower heart rate during exercise [17]. However, the volume of urine will increase 4 times more than when individuals are not ingested fluids before exercise. In short, ingesting 400-600 ml of water before exercise should allow renal mechanisms sufficient time to adjust to the total volume and osmolality of body fluids at optimal levels of preexercise. This serves to prevent or lessen the effects of dehydration during exercise [18].

Without adequate fluid replacement during three two-hour prolonged treadmill running the rectal temperature and heart rate level increases compared to when the individuals are under conditions of full hydration [19]. The most serious effect which is caused by dehydration during prolonged exercise occurs when the heat dissipation of the body is impaired. This effect is very dangerous because it can raise the body temperature over 40 degrees and ultimately precipitate to heat stroke, a condition with a high mortality rate. The exercise-induced dehydration causes hypertonicity in body fluids and it can compromise the flow of blood to the skin [20,21].

Dehydration can also cause a decrease in cardiac output during exercise with a reduction in stroke volume which may be greater than the increase in heart rate [20]. To minimize injury from the heat is stated that the water lost through sweating during exercise should be reinstated at the same rate with which the sweat is lost from the body [22]. However, in prolonged intense exercise (sports such as: cycling or running) this is not possible because the absorption rate is slowed and may lead to gastric distension.

The rate at which fluids and electrolytes will be replaced is also determined by the rate with which these ingested fluids are moving from the stomach to the intestine and later they are absorbed from the intestine to the blood. The rate at which fluid leaves the stomach depends on several complex interactions such as volume, temperature, composition of fluids ingested, and on the intensity of the exercise performed [23].

During prolonged exercise at constant work rate (60-80% maximal oxygen consumption/ $\dot{V}O_{2max}$), Stroke Volume (SV), and Mean Arterial Blood Pressure (MAP) decrease progressively, instead Heart Rate (HR) increases to maintain constant the Cardiac Output (CO) until it reaches its maximum [24]. The reduction in MAP occurs because of the reduction in systemic vascular resistance (SVR) with constant CO [25]. These cardiovascular processes which occur during the exercise are known as "Cardiovascular Drift" and it seems be due to the thermoregulatory need to increase cutaneous blood flow [3].

However, in conditions of dehydration and hyperthermia many physiological changes occur in the body. One of these changes is the decline in CO because there is a larger reduction in SV during prolonged running exercise and this should emphasize the decline in MAP unless the reductions in SVR are prevented [26]. A significant increase also occurs in plasma catecholamines when a significant decrease occurs in CO. It is induced by cardioselective adrenergic beta 1-blockade [27].

Sawka *et al.* [26] have also reported significant reductions in CO with dehydration during prolonged running in humans. Such significant declines in CO due to dehydration occur during prolonged running exercise at constant work load and it provokes the decrease of blood flow to some organs and the decline in blood pressure. Jose' Gonzalez-Alonso *et al.* [28] found that the cardiovascular changes observed during dehydration are solely the result of dehydration and they are not responses to prolonged cycling exercise per se. Given the reductions in CO with dehydration as a result of this the SVR increases so that large reductions in MAP are prevented. The limited reduction in MAP during dehydration shows that the cardiovascular system can minimize the decline in blood pressure and in the blood flow to the skin.

During dehydration, the systolic blood pressure (SBP) was the only component of MAP that declined so markedly. The reduction in SBP with dehydration was the consequence of a decreasing SV and rate of left ventricular systolic emptying. In response to dehydration it was shown by the study of Gonzalez-Alonso *et al.* [28] that increases in systemic vascular resistance (SVR) during the 2nd h of dehydration is due in part to progressive reductions in Cerebral Blood Flow (CBF) despite an increase in core temperature. It is assumed that the reductions in SV and MAP observed by Ekelund and Holmgren [29] are due to the progressive increases in blood flow to skin observed by Johnson and Rowell [30] also.

However, the study by Gonzalez-Alonso *et al.* [28], shows that the reductions in SV during dehydration were not due to increases in CBF. On the other hand, SV was decreased when CBF was also decreased because of cutaneous vasoconstriction. However, during the same study by Jose' Gonzalez-Alonso *et al.* [28], it was shown that SV and CBF were maintained at high levels in subjects when they were euhydrated. Under dehydration conditions Jose' Gonzalez-Alonso *et al.* [28] reported that forearm venous volume is maintained when SV decreases markedly. Therefore, it appears that large reductions in SV associated with dehydration in endurance athlete's men are due to other factors as elevated CBF or shift of the blood to the cutaneous circulation [28].

With dehydration, peripheral pooling might contribute to the decline in SV during prolonged exercise in the heat. Reductions in total blood volume with dehydration have been shown to be responsible for one-half of the reductions in SV during prolonged cycling exercise in the heat [20]. Thus, at the beginning of prolonged exercise the cutaneous vasculature vasodilates in response to increasing core temperature and this vasodilatation, reflected as reductions in CVR or FVR relative to resting values, continues until a core temperature of 38°C is reached [31]. However, when the exercise progresses and the core temperature continue to rise then there is a plateau occurs in the rate of CVR and FVR. This plateau in CVR and FVR has been reported by other researchers [31].

Carbohydrate replacement effects during exercise

Disturbances in body fluids and ionic equilibration may result in muscle fatigue and reduce prolonged exercise performance (as for instance a running lasting longer than 1 hour). Body water lost during exercise through sweating can cause dehydration in both fluid compartments of the body, intracellular and extracellular. Even a small amount of dehydration reached (1% of body weight) can increase cardiovascular strain which is indicated by a disproportionate elevation of heart rate during exercise and limit the body's ability to transfer heat from the muscle contractions to the skin surface where the heat can be dissipated to the external environment [18]. However, consequences of the deficits of body fluids may impair exercise performance and also lead to the development of heat illness [18].

The ingestion of carbohydrate through so-called sports drinks is to maintain the concentration of glucose in the blood and improve the oxidation of carbohydrates during exercise that lasts longer than 1 hour, especially when the glycogen content of exercising muscle is low [32]. To maintain the level of glucose in the blood during continuous exercise at intensities ranging from moderate to high (exercise lasting longer than 1 hour as for instance cycling, running, tennis, race walking), the carbohydrate should be ingested at a rate of 30 - 60 grams per hour [18].

Solutions containing carbohydrate concentrations greater than 10% will cause a net movement of fluid into the intestinal lumen because of their high osmolality when these solutions are ingested during exercise. This can result in an effective loss of water from the vascular compartment and the effects of dehydration may be amplified [33].

Usually the inclusion of glucose such as sucrose and complex carbohydrates in fluid replacement solutions have equal effectiveness in increasing exogenous carbohydrate oxidation, reducing fatigue and improving performance [32]. However, fructose should not be the predominant carbohydrate because it is slowly converted to glucose in the blood and is not immediately oxidized and therefore does not improve performance in the short term [34]. Furthermore, fructose may cause gastrointestinal discomfort with slow absorption from the gut leading to gastric distension [35].

Glucose intravenous infusion and fluid replacement during exercise prevent cardiovascular drift

Hamilton [36] investigated the influence of both hydration and concentration of glucose in the blood on cardiovascular drift during exercise. The first point they determined in this study was that the prevention of dehydration by fluid replacement during exercise prevented the decline in Stroke Volume (SV) and hence cardiac output (CO) remained unchanged [36]. In their experiment water was ingested at a rate that prevented a reduction in body weight after two hours of exercise.

The stroke volume (SV) decreased 15%; cardiac output (CO) decreased 7% during 20 to 120 minutes of exercise with no fluid ingested (NF). While the heart rate (HR) increased 10% and oxygen uptake (VO_2) increased 6%. In contrast, the SV was maintained during 20 to 120 minutes of exercise when fluid replacement (FR) was ingested while the HR increased 5% and so the CO increased 7%. Rectal temperature, SV, and HR were similar during the first hour of exercise with NF and FR. However, after two hours of exercise, the rectal temperature was higher 0.6 C and SV and CO were 11-16% lower during NF than with FR.

In an experiment by Hamilton *et al.* [36], was demonstrated that increases in oxygen uptake (VO_2) and HR and decreases in SV during 2 h of exercise (70% VO_{2max}) can be totally prevented by intravenous infusion of water and sufficient glucose to produce hyperglycemia. Recently Nielsen *et al.* [2], have observed that the whole body VO_2 drift during exercise in the heat is not due to increased VO_2 across the

working legs, which suggests it is due to increased metabolism in organs and/or tissues other the exercising skeletal muscles. It was observed that intravenous infusion of glucose and water, which maintained blood glucose concentration at 10 mM and partially offset dehydration, totally prevented VO_2 and HR from increasing during the 10 to 120 min period of exercise [36].

Intravenous glucose infusion markedly suppresses liver metabolism and glucose production [37]. It should be noted, however, that carbohydrate feedings during exercise that maintain blood glucose concentration at 5 mM do not appear to reduce the upward drift in VO_2 during exercise [38]. Study conducted by Green and Macdonald [39] on the influence of intravenous glucose on body temperature, have showed markedly changes on cardiovascular drift system. The two infusates which were used during the experiment were of equal volume and osmotic concentration. This experiment showed that the hematocrit was decrease by both infusates.

This might occur as a consequence of the infusion of volume and movement of extravascular water towards the vascular compartment because of the osmotic gradient. Intravenous glucose led to marked changes in heat production, heart rate, blood pressure and peripheral blood flow [39]. The cardiovascular and blood flow changes must to increased catecholamine release in some subjects, be due to an increase in plasma volume and osmolality. The influence of hypertonic solution in increasing muscle blood flow is well established [40]. To some extent, the increased muscle blood flow may be due to a reduction in blood viscosity which one would expect to be associated with a fall in hematocrit [39].

In the study by Green and Macdonald [39], the experiment has showed also that skin temperature increased following the infusions of glucose despite an overall greater increase in peripheral blood flow during hyperglycemia. The increase in skin temperature increases heat loss from the body surface [39]. The redistribution of body heat appears therefore to have been the same before glucose infusion and after this. The core temperature decreased after the glucose infusion. This study has demonstrated that glucose infusion loading (500 ml of 20% glucose I.V.) does not disturb the thermoregulation in subjects despite alterations in metabolic heat production and cardiovascular function [39].

A follow-up experiment with the same subjects (n=8) and same exercise conditions (two hours exercise with hydration maintained with the ingestion of water) observed that the continuous intravenous infusion of glucose in water (55ml of 18% glucose) completely prevented the 5-7% increase in VO_{2max} , in CO, and HR. In conclusion, the maintenance of hydration by FR attenuates totally the hyperthermia and prevents the decline in SV and CO observed during 2 hours of exercise when fluid is not ingested (NF). The glucose infusion (GI) consisted of a sterile 18% glucose solution in water that was administered by infusion into a forearm vein starting at 8 minutes and finishing at 118 minutes of exercise [36].

Studies by Hamilton [36] reported changes in plasma and blood volume. The transition from rest to exercise with both NF and FR produced a significant increase in concentration of hemoglobin, and thus the blood volume (BV) decreased. The BV during NF was 4.4 t 0.5%, 5.7 t 0.4%, and 6.7 + 0.6% below resting levels ($P < 0.05$) after 20, 60, 120 minutes exercise, respectively. Compared to NF, FR does not significantly attenuate the increase in rectal temperature during the first 60 minutes of exercise. However, the rectal temperature stabilizes at 38.2°C during 60 to 120 minutes of exercise with FR. The rectal temperature was significantly lower during the second hour of exercise with FR than with NF [36].

An observation by Ekelund⁴¹ was that the progressive decline in SV during prolonged bicycle exercise without FR was associated with progressive decline in venous return to the heart as reflected by

a reduction in the right ventricle and pulmonary artery pressure. The reduction in SV during exercise also occurred when BV was maintained [41]. Rowell [42], hypothesized that the venous return and SV are reduced primarily because of the partitioning of blood volume away from the central circulation to the skin [42]. In support of this hypothesis, Rowell and his colleagues observed that a reduction in skin temperature and in core temperature during exercise prevented the decline in SV during exercise [43].

In conclusion, the present review further extends on the work of these researchers by considering research findings over the past 10-15 years which question the basic premise that cardiovascular drift is a consequence of thermoregulatory adjustments to heat stress. The classic underlying mechanism enunciated by Rowell *et al.* [44] considers the increased skin blood flow observed during given prolonged exercise trial runs of the procedure in hot conditions results in a significant peripheral pooling of blood volume which reduces central blood volume and central venous pressure. The decrease in central venous pressure reduces the rate of cardiac filling and stroke volume declines. Clearly there is a need to review the research findings over the past 10-15 years to provide an overview of the current state of knowledge on the underlying mechanisms governing the phenomenon of cardiovascular drift observed during prolonged intense exercise such as the most common bicycling or running on a treadmill, in hot conditions.

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