Stroke volume during exercise: interaction of environment and hydration

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González-Alonso, J osé, Ricardo Mora-Rodríguez, and Edward F. Coyle. Stroke volume during exercise: interaction of environment and hydration. Am. J. Physiol. Heart Circ. Physiol. 278: H321–H330, 2000.—Euhydrated and dehydrated subjects exercised in a hot and a cold environment with our aim to identify factors that relate to reductions in stroke volume (SV). We hypothesized that reductions in SV with heat stress are related to the interaction of several factors rather than the effect of elevated skin blood flow. Eight male endurance-trained cyclists [maximal O2 consumption (V{sub}O{sub}2{sub}max) 4.5 ± 0.1 l/min; means ± SE] cycled for 30 min (72% V{sub}O{sub}2{sub}max) in the heat (H; 35°C) or the cold (C; 8°C) when euhydrated or dehydrated by 1.5, 3.0, or 4.2% of their body weight. When euhydrated, SV and esophageal temperature (T{sub}es} 38.2–38.3°C) were similar in H and C, whereas skin blood flow was much higher in H vs. C (365 ± 64% higher; P < 0.05). With each 1% body weight loss, SV declined 6.4 ± 1.3 ml (4.8%) in H and 3.4 ± 0.4 ml (2.5%) in C, whereas T{sub}es} increased 0.21 ± 0.02 and 0.10 ± 0.02°C in H and C, respectively (P < 0.05). However, reductions in SV were not associated with increases in skin blood flow. The reduced SV was highly associated with increased heart rate and reduced blood volume in both H (R = 0.96; P < 0.01) and C (R = 0.85; P < 0.01). In conclusion, these results suggest that SV is maintained in trained subjects during exercise in euhydrated conditions despite large differences in skin blood flow. Furthermore, the lowering of SV with dehydration appears largely related to increases in heart rate and reductions in blood volume.

heart rate; skin blood flow; hemodynamics; hyperthermia

It is well established that heat stress in euhydrated subjects can in some situations reduce stroke volume (SV) and increase heart rate during moderately intense exercise such that cardiac output can be compromised. In a classic study, Rowell et al. (27) showed a significantly lower SV (~20 ml), cardiac output (1.0–1.2 l/min), and central blood volume (0.1–0.2 liter) during moderate exercise in a 43°C than in a 26°C environment when core temperature and presumably skin blood flow were higher. Similarly, significant reductions in cardiac output (2.2 l/min) owing to the greater reduction in SV have recently been reported with abrupt elevations in body temperature, heart rate, and skin blood flow (10). This reduced SV with heat stress has generally been attributed to the lower central blood volume and cardiac filling pressure resulting from the increased skin blood flow and volume (13, 23, 24). Evidence showing that the lower SV, right atrial pressure, and central blood volume with whole body heating are reversed by whole body cooling (26) appears to support this hypothesis. However, this manipulation alters not only skin blood flow and skin temperature (T{sub}sk}) but also core temperature, which has been shown to increase heart rate and reduce SV when skin blood flow, blood volume, and cardiac output are constant (8). This notion is consistent with the observation that SV during mild exercise is similar in hot and thermoneutral environments when core temperature is not elevated but skin blood flow is much higher (18). One possibility is that increased core temperature contributes to reductions in SV during moderate intensity exercise primarily by acting on heart rate (5, 10, 28). In this context, it appears possible that the heat stress-mediated lowering in SV in euhydrated subjects is associated with combined elevations in internal temperature acting on heart rate and skin blood flow, rather than independent increases in skin blood flow.

Pronounced dehydration and core hyperthermia during prolonged upright exercise in the heat cause significant reductions in cardiac output, exercising skeletal muscle blood flow, skin blood flow, and mean arterial blood pressure (6–10, 16, 17, 19, 30). In the heat, increases in heart rate and reductions in SV and cardiac output have been shown to be directly related to the magnitude of dehydration, ranging from 1.1 to 4.2% body weight loss (17). With pronounced dehydration in the heat, concomitant reductions in total blood volume are responsible for approximately one-half of the 25- to 40-ml (i.e., 20–27%) decline in SV during exercise (16). The factors related to the remaining reduction in SV are not clear. Because blood volume expansion does not attenuate hyperthermia with dehydration and only attenuates the increase in heart rate by one-third, elevated body temperature and heart rate are also possible factors contributing to the reduction in SV (16). When hyperthermia is fully prevented in dehydrated subjects by exercise in a cold environment, the smaller reduction in SV (i.e., 7%) appears to be solely due to reduced blood volume, because blood volume restoration fully restored SV to euhydrated levels despite the persistence of 3- to 4-liter extravascular dehydration (7). Therefore, dehydration-induced hypovolemia accounts for one-half of the larger lowering in
SV with dehydration in the heat, but it might fully account for the small reductions in SV during exercise in the cold. Hyperthermia therefore reduces SV, yet the mechanism underlying this response and the manner in which internal temperature and related factors, such as heat rate elevations, interact with hypovolemia are not clear.

Therefore, the primary purpose was to determine the effect of large differences in skin blood flow on SV during moderately intense exercise in the heat and cold when core temperature and blood volume were similar in both environments. A second aim was to identify factors related to SV reductions with dehydration during moderate intensity exercise in the heat and cold. We hypothesized that the reductions in SV during moderately intense exercise in euhydrated and dehydrated trained subjects are related to the interaction of body temperature, heart rate, blood volume, and skin blood flow, rather than the independent effect of skin blood flow.

METHODS

Subjects. The eight male endurance-trained cyclists participating in this study had a mean (±SD) age of 22 ± 3 yr, body weight of 70 ± 6 kg, stature of 1.78 ± 0.6 cm, maximal heart rate of 190 ± 5 beats/min, and maximal oxygen consumption (V\textsubscript{O\textsubscript{2max}}) of 4.5 ± 0.3 l/min. The study was approved by the Internal Review Board at the University of Texas at Austin and a written informed consent from each subject was obtained. During the preliminary testing, V\textsubscript{O\textsubscript{2max}} was determined using an incremental protocol on a cycle ergometer. In addition, each subject completed four 2-h practice bouts (60–70% V\textsubscript{O\textsubscript{2max}}) in the heat to acclimate and become familiar with the experimental conditions. On the final practice day, whole body sweating rate was estimated during the 2 h of exercise (60% V\textsubscript{O\textsubscript{2max}}) in the heat. The effectiveness of the heat acclimation procedure was confirmed in this last practice day by the maintenance of stable heart rate and core temperature throughout the exercise when euhydration was maintained.

Experimental design. On four different occasions, subjects first cycled an ergometer (Jaeger ERGOTEST; 60–70% V\textsubscript{O\textsubscript{2max}}) in the heat for 2 h, rested for 45 min in a 23°C environment, and performed two additional 30-min bouts of exercise (72 ± 3% V\textsubscript{O\textsubscript{2max}}), one in the cold and the other in the heat, preceded by 45 min of rest (Fig. 1). During exercise in the cold, the environmental conditions were 8.2 ± 0.3°C dry bulb, 75 ± 2% relative humidity, and 2 m/s wind speed (i.e., wet bulb globe temperature index = 7°C). During exercise in the heat, the environmental conditions were 35.4 ± 0.2°C dry bulb, 47 ± 2% relative humidity, and 2 m/s wind speed (i.e., wet bulb globe temperature index = 29°C).

To manipulate hydration status to four different levels, the subjects received during the initial 2-h exercise period either 1) a large volume of fluid (3.17 ± 0.07 liters; euhydration trial, Euh) or 2) a progressively lower volume of fluid (i.e., 2.15 ± 0.05, 1.09 ± 0.02, or 0.12 ± 0.00 liters; dehydration trials, Deh) (Fig. 1). The fluid replacement solution was made from a commercially available sports drink (Gatorade, Quaker Oats). Different carbohydrate and electrolyte concentrations were mixed to achieve different states of hydration with the same amount of carbohydrate and electrolytes ingestion in each 2-h trial (i.e., 70 g of Gatorade powder containing 41 g of sucrose, 28 g of glucose, 518 mg of Na\textsuperscript{+}, 518 mg of Cl\textsuperscript{−}, and 118 mg of K\textsuperscript{+}). To maintain the obtained hydration status during the subsequent 30-min exercise bouts of each trial, either 0.6 ± 0.1 or 1.0 ± 0.1 liter of 6% Gatorade solution at 5°C was ingested during the rest periods before the subjects exercised in the cold and in the heat, respectively. These volumes of fluid replaced the fluid losses in sweat and urine.

During Euh, body weight after subjects cycled in the cold and in the heat was only 0.2 ± 0.1% (0.18 ± 0.12 kg) below the preexercise weight. During Deh, subjects finished cycling in the cold and the heat with 1.5 ± 0.1% (1.08 ± 0.09 kg), 3.0 ± 0.2% (2.06 ± 0.13 kg), and 4.2 ± 0.2% (3.07 ± 0.09 kg) below preexercise body weight, respectively (i.e., Deh-1.5, Deh-3.0, and Deh-4.2, respectively). All trials were counterbalanced in regard to hydration status (Euh, Deh-1.5, Deh-3.0, and Deh-4.2) and environmental temperature (cold or heat) and separated by at least 72 h.

On the day before the experimental testing, the hydration status of the subjects was standardized by having them adopt the same diet, exercise bout (i.e., ≤1 h of low intensity cycling), and fluid intake. They also ingested 200–300 ml of fluid 2 h before arriving at the laboratory. Upon arrival on the experimental testing day, subjects’ nude body weights were recorded, and a rectal probe was inserted. The subject then put on cycling shorts and shoes, entered the environmental chamber (35°C), put on cycling shorts and shoes, and sat quietly for ≥20 min. Thereafter, a 6-ml blood sample was withdrawn from an antecubital vein using a butterfly catheter, for later determination of baseline hematocrit, hemoglobin concentration, serum osmolality, and electrolytes. The subject then mounted a cycle ergometer and cycled in the upright position for 120 min at ~60% V\textsubscript{O\textsubscript{2max}}.

Upon completion of the first 2 h, the subject removed his clothing and towel dry, and postexercise body weight was recorded. The subject then voided his bladder to determine

![Fig. 1. Sequence of experimental protocol whereby subjects first exercised for 120 min to become dehydrated by 1.5, 3.0, and 4.2% body weight or maintained euhydration by ingesting different volumes of fluid. Thereafter, they performed two additional 30-min bouts of exercise, one in the cold and the other in the heat (35°C), while we determined cardiovascular effects of graded dehydration during the 20- to 30-min period. V\textsubscript{O\textsubscript{2max}}, maximal O\textsubscript{2} consumption.](http://ajpheart.physiology.org/doi/10.1152/ajpheart.00313.2017)
the volume of urine formed during exercise. The subject redressed with dry clothing and then rested for ~45 min in a 23°C environment in front of a fan to return core temperature to baseline level. During the rest period, an esophageal thermistor and a Teflon catheter were inserted (right forearm antecubital vein). After this rest period, a 30-min bout of cycling either in the cold or in the heat was performed (Fig. 1).

Before the subject exercised in either environmental condition, skin thermostats, a mercury-in-Silastic strain gauge, and a laser Doppler probe were attached (left forearm) after the subject mounted a cycle ergometer (Jaeger ERGOTEST in the heat and Monark 818 in the cold). Both the mercury-in-Silastic strain gauge and the laser Doppler probe remained attached to the forearm during the entire exercise and rest periods. While the subject remained seated on the ergometer, attached to the forearm during the entire exercise and rest periods. Where the subject remained seated on the ergometer, resting values of esophageal (Tes), rectal (Tre), and skin temperature (Tsk) as well as forearm and cutaneous blood flow (CBF) were obtained. Thereafter, subjects began the 30-min exercise period (Fig. 1).

During each 30-min exercise bout, VO2, heart rate, Tre, Tsk, and Tsk were determined continuously, and cardiac output and blood pressure were determined in quadruplicate from 20 to 28 min. Forearm blood flow (FFB) was measured eight times from 13 to 15 min of exercise (venous occlusion plethysmography). CBF was measured continuously from 0 to 10, 11 to 13, and 28 to 30 min of exercise (laser Doppler velocimetry). A 10-ml blood sample was withdrawn at 30 min of exercise, and a rating of perceived exertion was recorded (1).

Analytic techniques. A more detailed description of the analytic methods can be found elsewhere (7). VO2 was measured using computerized open-circuit spirometry. Cardiac output was determined using a computerized version of the CO2 rebreathing technique of Collier (3) and adjusted for hemoglobin concentration (14). Heart rate was measured using a heart rate monitor (Uniq CIC Heartwatch). SV was calculated as cardiac output/heart rate. Systolic blood pressure and diastolic blood pressure were measured on the left arm using an automatic blood pressure monitor (STBP-680; Colin Medical Instruments). Mean arterial pressure (MAP) was calculated as [(2 × diastolic blood pressure) + systolic blood pressure]/3 (expressed as mmHg). Systemic vascular conductance was calculated as cardiac output/MAP (expressed in peripheral conductance units of l·min⁻¹·mmHg⁻¹).

FFB and forearm venous volume were measured using venous occlusion plethysmography with a mercury-in-Silastic strain gauge (34) while the left arm was supported by a sling. CBF was determined using a laser Doppler flowmeter (ALF 21, Transonic Systems, Ithaca, NY) with a collecting frequency of 1 Hz. The probe was placed 2 cm from the plethysmographic strain gauge. Exercise time for the onset of skin vasodilation was defined as the minute at which a sustained increase of CBF started. Tes threshold for the onset of vasodilation was defined as the Tsk at the time for the onset of skin vasodilation. CBF sensitivity was defined by the slope of the linear portion of the CBF-Tsk relationship. The linear portion of the data was selected by visual inspection, and slopes were determined by least-squares linear regression analysis. Forearm vascular conductance was calculated as FFB/MAP and expressed in conductance units (i.e., ml⁻¹·100 ml⁻¹·min⁻¹·mmHg⁻¹). Cutaneous vascular conductance was calculated as CBF/MAP (expressed in V/mmHg). Because the laser Doppler probe remained in place during the entire exercise and rest period, this measurement accurately detected changes in CBF and cutaneous vascular conductance between environments on the same day. The laser Doppler CBF values after 10 and 28 min of exercise were similar, suggesting that a plateau in skin blood flow had been reached. Accordingly, the average of the two measures is reported.

Total sweat volume was estimated from the difference in body weight (Acme Scale; CA; FW 150 KAI) before and after exercise while corrected for fluid intake, respiratory water loss, saliva volume, and body weight loss due to the exchange of O2 and CO2 (22). Percent dehydration was estimated from the difference in body weight after each 30-min bout of cycling in the heat and in the cold compared with the basal euhydrated body weight while again correcting for body weight loss due to carbon losses (22).

Tes was measured with a thermistor (YSI 491) inserted through the nasal passage a distance equal to one-quarter of the subject's standing height (29). Tsk was measured with a thermistor (YSI 401) inserted 15 cm past the anal sphincter. Mean Tsk was calculated from the skin temperatures measured at six sites (i.e., upper arm, forearm, chest, back, thigh, and calf) using the weighting method of Hardy and Dubois (11).

The percent change in blood volume and plasma volume was calculated using the equations of Dill and Costill (4).

Serum osmolality was determined by the freezing point depression technique (Advanced Instruments 3MO). Serum electrolyte concentrations were measured with an automated electrolyte analyzer (Nova 5). Serum glucose and plasma lactate concentrations were determined with a YSI 23 glucose-lactate analyzer. Plasma norepinephrine and epinephrine concentrations were determined using a radioenzymatic assay (2).

Statistical analysis. Data were analyzed using a two-way (hydration status by environmental temperature or hydration by time) analysis of variance with repeated measures. When F values were significant, pairwise differences were identified using Tukey's honestly significant difference post hoc procedure. Step-wise forward regression analysis was also used to test the strength of the association of SV as a dependent variable and forearm blood flow, heart rate, blood volume, and esophageal temperature as independent variables. The significance level was set at P = 0.05. Data are presented as means ± SE.

RESULTS

Preexercise body weight and resting hemoglobin concentration were similar during the four trials (mean range 70.9–71.3 kg and 14.5–14.6 g/dl, respectively). VO2 was also similar during the 30-min bouts of exercise in the cold and in the heat under all levels of hydration (in both environments VO2 = 3.2 ± 0.11/min).

Changes in blood volume, plasma volume, serum osmolality, and serum electrolyte concentration. The effects of dehydration on lowering blood volume and plasma volume while elevating osmolality and sodium concentration were not different at a given level of dehydration during exercise in the cold and heat (Table 1).

Serum glucose and plasma lactate concentration. No significant differences in serum glucose concentration were observed with graded dehydration in either environment (Table 1). Plasma lactate did not increase with graded dehydration in the cold, whereas in the heat, it was significantly higher during Deh-3.0 and Deh-4.2 compared with Euh (Table 1).

Core and skin temperature. When euhydrated, Tsk after 30 min of exercise was similar in both environ-
ments (38.3 ± 0.1°C and 38.5 ± 0.2°C in the cold and heat, respectively; not significant). However, the rate of increase in T<sub>es</sub> was faster in the cold than in the heat during the first 10 min of exercise, as reflected by the higher T<sub>es</sub> (38.1 ± 0.1°C vs. 37.7 ± 0.1°C, respectively; P < 0.05). In the heat, the average T<sub>es</sub> during the 20- to 30-min period of exercise was 0.21 ± 0.03°C higher with every 1% increase in dehydration (38.2 ± 0.1, 38.4 ± 0.2, 38.7 ± 0.2, 39.0 ± 0.1°C in Euh, Deh-1.5, Deh-3.0, and Deh-4.2, respectively; Fig. 2A). During exercise in the cold, the rise above rest in T<sub>es</sub> (ΔT<sub>es</sub>) was similar at all levels of hydration (1.1–1.2 ± 0.1°C; Fig. 2B), with 75–85% of the elevation occurring during the first 10 min of exercise. However, the average T<sub>es</sub> during the 20- to 30-min period of exercise in the cold was significantly elevated with Deh-4.2 compared with Euh due to the higher initial T<sub>es</sub> (38.3 ± 0.1, 38.3 ± 0.1, 38.4 ± 0.1, 38.7 ± 0.1°C in Euh, Deh-1.5, Deh-3.0, and Deh-4.2, respectively; Fig. 2B). In either environment, T<sub>sk</sub> during the 10- to 30-min period of exercise was not significantly affected by increasing dehydration level (mean range 22.5–23.1°C and 34.5–35.1°C in the cold and heat, respectively; Table 2).

FBF and CBF. At rest, FBF was similar at all levels of hydration; yet, it was significantly higher in the heat compared with the cold (2.4 ± 0.3 vs. 1.7 ± 0.1 ml·100 ml<sup>-1</sup>·min<sup>-1</sup>; P < 0.05). After 13–15 min of exercise at all levels of hydration, FBF was 1.6- to 2.6-fold (P < 0.05) higher in the heat compared with the cold (Table 2). Only at the greatest level of dehydration in the heat was FBF significantly lower compared with Euh values (8.3 ± 0.8 vs. 10.1 ± 1.1 ml·100 ml<sup>-1</sup>·min<sup>-1</sup>, respectively; P < 0.05). The same pattern of response was observed in CBF (Fig. 3). In the heat, the increase in CBF from resting values during Deh-4.2 was significantly lower than during Euh. As expected, the in-

Table 1. Hematological responses after 30 min of exercise in heat and in cold under four levels of hydration

<table>
<thead>
<tr>
<th>Variables</th>
<th>Environment</th>
<th>Hydration Status</th>
<th>Euh</th>
<th>1.5% Dehydration</th>
<th>3.0% Dehydration</th>
<th>4.2% Dehydration</th>
</tr>
</thead>
<tbody>
<tr>
<td>% Decline in BV</td>
<td>Heat</td>
<td>0.0 ± 0.0</td>
<td>1.2 ± 1.0</td>
<td>2.3 ± 1.7*</td>
<td>5.1 ± 2.7*</td>
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<tr>
<td></td>
<td>Cold</td>
<td>0.0 ± 0.0</td>
<td>1.8 ± 1.2</td>
<td>3.2 ± 1.0*</td>
<td>5.5 ± 2.1*</td>
<td></td>
</tr>
<tr>
<td>% Decline in PV</td>
<td>Heat</td>
<td>0.0 ± 0.0</td>
<td>2.1 ± 2.0</td>
<td>3.3 ± 1.7*</td>
<td>5.3 ± 2.0*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cold</td>
<td>0.0 ± 0.0</td>
<td>3.3 ± 2.7</td>
<td>4.3 ± 1.3*</td>
<td>7.6 ± 3.2*</td>
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</tr>
<tr>
<td>Hemoglobin, g/dl</td>
<td>Heat</td>
<td>15.6 ± 0.5</td>
<td>15.9 ± 0.4*</td>
<td>16.2 ± 0.3*</td>
<td>16.5 ± 0.4*</td>
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<tr>
<td></td>
<td>Cold</td>
<td>15.7 ± 0.4</td>
<td>15.9 ± 0.4</td>
<td>16.1 ± 0.3*</td>
<td>16.5 ± 0.4*</td>
<td></td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>Heat</td>
<td>45.9 ± 1.2</td>
<td>46.5 ± 0.8</td>
<td>47.5 ± 0.9*</td>
<td>47.7 ± 1.2*</td>
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<tr>
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<td>Cold</td>
<td>46.6 ± 1.3</td>
<td>47.3 ± 0.8</td>
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<td>48.5 ± 1.1*</td>
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<td>Osmolality, mosmol/kg</td>
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<td>285 ± 3*</td>
<td>291 ± 2*</td>
<td>297 ± 1*</td>
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<td></td>
<td>Cold</td>
<td>280 ± 2</td>
<td>284 ± 2*</td>
<td>292 ± 2*</td>
<td>297 ± 2*</td>
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<tr>
<td>[Na&lt;sup&gt;+&lt;/sup&gt;], mM</td>
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<td>145 ± 1</td>
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<td>Cold</td>
<td>144 ± 1</td>
<td>145 ± 1</td>
<td>149 ± 1*</td>
<td>151 ± 1*</td>
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<tr>
<td>[Cl&lt;sup&gt;-&lt;/sup&gt;], mM</td>
<td>Heat</td>
<td>105 ± 3</td>
<td>107 ± 2</td>
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<tr>
<td>[K&lt;sup&gt;+&lt;/sup&gt;], mM</td>
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<td>Glucose, mM</td>
<td>Heat</td>
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<td>4.5 ± 0.2</td>
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<tr>
<td></td>
<td>Cold</td>
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<td>4.0 ± 0.4</td>
<td>3.7 ± 0.1</td>
<td>4.3 ± 0.2</td>
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<tr>
<td>Lactate, mM</td>
<td>Heat</td>
<td>2.0 ± 0.2</td>
<td>2.5 ± 0.2</td>
<td>2.8 ± 0.2*</td>
<td>2.7 ± 0.1*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cold</td>
<td>2.0 ± 0.2</td>
<td>2.4 ± 0.2</td>
<td>2.4 ± 0.2</td>
<td>2.3 ± 0.2</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SE for 8 subjects. %BV and %PV, percent decline in blood volume and plasma volume from the euhydrated condition; [Na<sup>+</sup>], serum sodium concentration; [Cl<sup>-</sup>], serum chloride concentration; [K<sup>+</sup>], serum potassium concentration. *Significantly different from euhydrated condition, P < 0.05.
Increase in CBF from resting values was much greater in the heat than in the cold (360–440 vs. 60–70%, respectively; Fig. 3).

Threshold for cutaneous vasodilation and skin blood flow sensitivity. Exercise in the cold was characterized by a delayed onset and elevated $T_{es}$ threshold for vasodilation (290–335 s and 37.7–37.8°C, respectively) compared with the heat (Fig. 3). In the heat, Deh-4.2 vs. Euh resulted in 1) a delayed onset and increased $T_{es}$ threshold for skin vasodilation (205 ± 28 vs. 120 ± 26 s and 37.5 ± 0.1 vs. 37.1 ± 0.1°C during Deh-4.2 and Euh, respectively; both $P < 0.05$), 2) a reduced CBF sensitivity (314 ± 23 vs. 688 ± 31% increased from resting value per°C, respectively; $P < 0.05$; Fig. 3A), and 3) a lower plateau FBF and CBF (see Table 2; Fig. 3A). In the cold, there were no significant alterations in the threshold for onset of vasodilation, CBF sensitivity, or plateau CBF with Deh-4.2 (Fig. 3B).

Hemodynamics. When euhydrated, SV was similar during exercise in the cold and in the heat (Fig. 4). In both environments, however, SV was lowered in proportion to the degree of dehydration (Fig. 4D). SV was lowered by 6.4 ± 1.3 ml for every 1% dehydration in the heat but only by 3.4 ± 0.4 ml per 1% dehydration in the cold ($P < 0.05$). When euhydrated, cardiac output was significantly higher in the heat compared with the cold (21.5 ± 0.7 l vs. 20.3 ± 0.6 l/min, respectively; $P < 0.05$; Fig. 4B). In the heat, cardiac output was lowered in proportion to the degree of dehydration, and it was significantly ($P < 0.05$) lower than Euh during Deh-3.0 and Deh-4.2 (Fig. 4B). In contrast, in the cold, cardiac output was not significantly reduced by dehydration.

At all levels of hydration, heart rate was significantly ($P < 0.05$) higher by 11–14 beats/min during exercise in the heat compared with those in the cold (Fig. 4C). In the heat, heart rate elevation with increasing dehydration was slightly higher than in the cold (3.7 ± 0.4 vs. 3.2 ± 0.4 beats/min per 1% dehydration during exercise, respectively; $P = 0.14$; Fig. 4C).

MAP and diastolic blood pressure were significantly lower during exercise in the heat compared with the cold at all levels of hydration (i.e., delta mean range = 9–11 and 12–14 mmHg, respectively; both $P < 0.05$). In the heat, systolic blood pressure tended to decline with increasing dehydration (~2 mmHg per 1% dehydration), but in the cold it was unaltered. Furthermore,
systemic vascular conductance was significantly higher in the heat versus cold in Euh, Deh-1.5, and Deh-3.0 (Fig. 4E). During exercise in the heat, systemic vascular conductance was significantly lower during Deh-3.0 and Deh-4.2 compared with Euh, becoming close to the values observed during exercise in the cold (P < 0.05; Fig. 4E).

At all levels of hydration, forearm vascular conductance and cutaneous vascular conductance were 1.3- to 2.6-fold higher in the heat compared with cold (Table 2). Only at the greatest level of dehydration in the heat was forearm vascular conductance significantly lower compared with Euh values (0.078 ± 0.006 vs. 0.097 ± 0.012 ml·100 ml⁻¹·min⁻¹·mmHg⁻¹ in Deh-4.2 vs. Euh, respectively; P < 0.05). In contrast in the cold, forearm vascular conductance tended to increase (0.018 ± 0.003 vs. 0.030 ± 0.003 ml·100 ml⁻¹·min⁻¹·mmHg⁻¹ in Euh and Deh-4.2, respectively; not significant).

Plasma catecholamines. At all levels of hydration, plasma norepinephrine was significantly (P < 0.05) higher during exercise in the heat compared with the levels in the cold (3–7 nM or 27–43% higher; Fig. 5A). Furthermore, the significant increase in plasma norepinephrine above the Euh condition in Deh-4.2 was significantly higher in the heat than in the cold (7.2 vs. 3.8 nM or 65 ± 20 vs. 37 ± 13%, respectively; P < 0.05; Fig. 5A). Moreover, plasma epinephrine was only significantly elevated above Euh at the two highest levels of dehydration during exercise in the heat (Fig. 5B). However, plasma epinephrine when Euh was similar in both environments (3.0–3.1 ± 0.7 nM).

Total sweat volume. Total sweat volume during 30 min of exercise was significantly lower in the cold compared with the heat at any given level of hydration (0.40 ± 0.06 vs. 0.74 ± 0.08 liter; P < 0.05). However,
Environmental heat stress and dehydration are known to markedly reduce SV during moderately intense exercise. By having subjects exercise in the heat and cold in euhydrated and dehydrated states, we examined the influence of a three- to fourfold difference in skin blood flow on SV when exercise intensity, amount of dehydration, reductions in blood volume, and increases in serum electrolytes and osmolality were all similar in both environments. We found that when endurance-trained subjects were euhydrated, SV was the same in the heat and in the cold, despite a drastically elevated skin blood flow in the heat. Furthermore, reductions in SV with graded dehydration in the heat and cold were largely associated with lower blood volume and increased core temperature and heart rate.

DISCUSSION

Exercise hemodynamics in euhydrated subjects. The present observation that SV in euhydrated trained men was similar in the heat and cold despite skin blood flow being much higher in the heat agrees with previous findings of Nadel and co-workers (19) during low-intensity exercise. They observed a similar SV but higher cardiac output during mild exercise in a 36°C compared with a 20°C environment when FBF was fourfold higher, but Tm was not different (18). This is also consistent with the observation that SV is maintained and cardiac output increases 3–6 l/min with profound elevations in skin blood flow, core temperature, and heart rate during rest and mild intensity exercise (15, 20, 21, 23). Presently, cardiac output was also elevated by 1.2 l/min in the heat compared with the cold during moderately intense exercise when core temperature was similar in both environments. This higher cardiac output together with probably lower visceral blood flow in response to elevated sympathetic activity (12, 23, 25) appear to totally account for the higher skin blood flow in the heat (15). The present observation, however, is in sharp contrast to the marked reductions in SV and maintained or reduced cardiac output observed with combined increases in core temperature, heart rate, and skin blood flow during moderate and high intensity exercise in hot versus thermoneutral or cold environments (19, 27). Hence, it appears that combined increases in core temperature, heart rate, and skin blood flow do not reduce SV at rest and during low intensity exercise but are generally associated with a lower SV during moderate and high intensity exercise. Addressing the separate effects of skin blood flow and core temperature on SV, we have recently shown that the increase in internal temperature from 38°C to 39°C in euhydrated subjects with constant skin blood flow, blood volume, and cardiac output reduces SV and increases heart rate (8). Presently, we observed that when Tm and blood volume are similar, SV is maintained in the heat compared with the cold despite markedly different skin blood flow. This maintenance of SV when euhydrated in the heat was most likely due to a compensatory increase in left ventricular contractility, secondary to an elevated sympathetic activity as suggested by the 27% higher norepinephrine concentration. Taken together, these results support the notion that reductions in SV with heat stress are not due to the sole effect of elevated skin blood flow but rather to the interaction of several factors, among which body temperature plays a major role.

Exercise hemodynamics in dehydrated subjects. An important finding of this study was that the reductions in SV with dehydration during moderate intensity exercise in the heat were attenuated by one-half in the cold, thus allowing the prevention of the significant reductions in cardiac output with dehydration in the heat. Furthermore, in the cold, high levels of dehydration did not reduce systemic or cutaneous vascular conductance as it did in the heat. However, the increase in heart rate, which paralleled the increase in norepinephrine concentration, was only slightly higher in the heat compared with the cold. The observation that SV still declined up to 11% (i.e., 15 ml) when skin perfusion was very low in the cold indicates that at least one-half of the decline in SV with dehydration during moderate intensity exercise in the heat was clearly not due to an elevated pooling of blood in the skin veins (13, 23, 24). Indeed, the step-wise multiple regression analysis excluded forearm blood flow as a factor related to the SV response in the heat and the cold (partial correlation r = 0.26; not significant; Fig. 6) and instead identified heart rate and blood volume as the best predictors of the SV response in both environments (R = 0.96 in the
heat and \( R = 0.85 \) in the cold, both \( P < 0.01 \). \( T_{es} \) did not increase the predicting power of heart rate and blood volume, because it was significantly correlated with heart rate \( (R = 0.84 \) in the heat and \( R = 0.50 \) in the cold; \( P < 0.01 \); Fig. 7 and Fig. 8). This could be interpreted to mean that part of the observed increase in heart rate in the heat and cold with graded dehydration was associated with the rise in core temperature. Although this type of statistical analysis has limitations, it provides a general description of the factors that exerted the greatest influence on SV decline in both environments. As discussed below, evidence in the literature indeed supports the idea that the decline in SV with dehydration during moderate intensity exercise is associated with the combined influences of dehydration-induced hypovolemia and core hyperthermia, with the latter mainly acting by increasing heart rate \( (5, 10, 28) \).

In this study, reductions in blood volume with graded dehydration (due largely to plasma volume losses) were the same in the heat and cold, suggesting that the effect of reduced intravascular volume on venous return and SV was similar in both environments. It seems therefore that blood volume was not, by itself, the factor responsible for the greater SV reduction in the heat (see Fig. 7; Table 1). In the cold, reduced blood volume accounts for most of the observed reductions in SV (up to 11%), since blood volume restoration has been shown to fully restore the decline in SV \( (7\%) \) to euhydrated control levels during exercise in the cold, despite the persistence of a 3- to 4-liter extravascular dehydration \( (8) \). In contrast in the heat, reduced blood volume only accounts for one-half of the larger \( (22\%) \) reduction in SV with dehydration \( (16) \). The remaining one-half of the SV reduction in the heat is associated with the still-persistent hyperthermia and elevated heart rate.

In the present study, the greater reduction in SV during exercise in the heat was in part associated with higher elevations in core temperature and heart rate \( (Fig. 8) \). This argument is based on the previous observation that a 1°C increase in \( T_{es} \) results in a 9 beat/min \( (5\%) \) elevation in heart rate and 11 ± 3 ml \( (7\%) \) reduction in SV \( (8) \). More recently, it has been shown that heart rate elevations with heat stress are strongly correlated to increases in \( T_{es} \) from 36°C to 40°C \( (r^2 = 0.98; P = 0.001) \) when the initial body temperature is varied from 36°C to 38°C \( (10) \). Interestingly, SV reductions with heat stress during moderate intensity exercise also paralleled the increase in core temperature, yet skin blood flow reached a plateau in all conditions at \( T_{es} \sim 38°C \) \( (10) \). It therefore seems likely that the increases in heart rate associated with elevations in core temperature above 38°C contribute to reductions in SV with dehydration. Supporting the notion of an independent effect of heart rate on SV, Frizstche et al. \( (5) \) recently demonstrated that blunting the 11% increase in heart rate during 60 min of exercise with \( \beta \)-blockade totally prevents the concomitant 13% decline in SV after 15 min of cycling, when skin blood flow, blood volume, cardiac output, and core temperature are unaltered. This also agrees with the observations that increases in heart rate with atrial pacing reduce SV at rest and during exercise in humans \( (21) \) and dogs \( (31–33) \). The alternative possibility that high cardiac temperature reduces SV by altering cardiac contractility appears unlikely based on our recent observation that elevations in central blood volume with supine exercise restores two-thirds of the decline in SV during upright exercise despite equal hyperthermia and dehydration \( (9) \).

The larger reductions in SV with dehydration in the heat could have also been influenced by the elevated
circulatory strain reflected by the 11–13 beats/min higher heart rate, 27–43% higher norepinephrine concentration, and 9–11 mmHg lower mean arterial blood pressure compared with the cold at any level of hydration. It is also possible that right atrial pressure and central blood volume were somewhat lower in the heat compared with the cold possibly in response to the elevated blood flow and volume to the skin (26, 27). Thus it is reasonable to think that during moderate intensity exercise in the heat, the cardiovascular system of dehydrated subjects is at its limit for compensating for a reduced left ventricular end-diastolic volume. Left ventricular end-diastolic volume is largely a function of filling pressure and diastolic time. It is therefore likely that the 200- to 300-ml lower blood volume compared with euvohydriated reduced venule pressure, and the larger increase in heart rate reduced diastolic time in the heat. Furthermore, when combined, hypovolemia and reduced filling time apparently exerted a greater effect on reducing ventricular end-diastolic volume and thus SV in the heat compared with the cold.

In conclusion, we found that when trained men were euhydrated, SV during moderate intensity exercise was similar in the heat and cold despite large differences in skin blood flow. However, cardiac output was higher in the heat, providing most of the increase in skin blood flow compared with the cold. Furthermore, the lower SV with dehydration in both environments was highly associated with reductions in blood volume and increases in core temperature and heart rate. The reduction in SV with dehydration was greater in the heat compared with the cold and appeared to be largely due to the higher core temperature and higher heart rate, with the elevated skin blood flow apparently exerting a minor role. The attenuation of the reductions in SV in the cold allowed cardiac output to be maintained despite dehydration. Therefore, these results suggest that most of the reduction in SV with dehydration during moderate intensity exercise is associated with lower blood volume and increased core temperature and heart rate.

The authors give special thanks to Winnie Taagerup from the August Krogh Institute (University of Copenhagen) for performing the catecholamine analyses.

This study was partially supported by grants from the Gatorade Sports Science Institute and the Spanish Ministry of Education and Science. J. González-Alonso and R. Mora-Rodríguez were supported by scholarships from the Ministry of Education and Science of Spain. Present address of J. González-Alonso: The Copenhagen Muscle Research Center, Rigshospitalet, Section 7652; Blegdamsvej 9, DK-2100 Copenhagen Ø, Denmark (E-mail: jga@mmrc.dk).

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Received 12 May 1999; accepted in final form 1 September 1999.

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