Supine exercise restores arterial blood pressure and skin blood flow despite dehydration and hyperthermia

José González-Alonso, Ricardo Mora-Rodríguez, and Edward F. Coyle.

Supine exercise restores arterial blood pressure and skin blood flow despite dehydration and hyperthermia. Am. J. Physiol. 277 (Heart Circ. Physiol. 46): H576-H583, 1999.—We determined whether the deleterious effects of dehydration and hyperthermia on cardiovascular function during upright exercise were attenuated by elevating central blood volume with supine exercise. Seven trained men [maximal oxygen consumption (VO₂max) 4.7 ± 0.4 l/min (mean ± SE)] cycled for 30 min in the heat (35°C) in the upright and in the supine positions (VO₂ 2.93 ± 0.27 l/min) while maintaining euvohum by fluid ingestion or while being dehydrated by 5% of body weight after 2 h of upright exercise. When subjects were euvohum, esophageal temperature (Tₑ) was 37.8–38.0°C in both body postures. Dehydration caused equal hyperthermia during both upright and supine exercise (Tₑ 38.7–38.8°C). During upright exercise, dehydration lowered stroke volume (SV), cardiac output, mean arterial pressure (MAP), and cutaneous vascular conductance and increased heart rate and plasma catecholamines [30 ± 6 ml, 3.0 ± 0.7 l/min, 6 ± 2 mmHg, 22 ± 8%, 14 ± 2 beats/min, and 50–96%, respectively; all P < 0.05]. In contrast, during supine exercise, dehydration did not cause significant alterations in MAP, cutaneous vascular conductance, or plasma catecholamines. Furthermore, supine versus upright exercise attenuated the increases in heart rate (7 ± 2 vs. 9 ± 1%) and the reductions in SV (13 ± 4 vs. 21 ± 3%) and cardiac output (8 ± 3 vs. 14 ± 3%) (all P < 0.05). These results suggest that the decline in cutaneous vascular conductance and the increase in plasma norepinephrine concentration, independent of hyperthermia, are associated with a reduction in central blood volume and a lower arterial blood pressure.

Therefore, the combined effects of dehydration and hyperthermia during upright exercise cause a severe deterioration in cardiovascular function. The effects of dehydration amounting to 4% of body weight (i.e., ~3,000 ml) on reducing skin blood flow is not caused simply by the characteristic 200- to 300-ml reduction in plasma volume, because Montain and Coyle (21) have shown that plasma volume expansion during dehydration failed to prevent reductions in forearm blood flow or to attenuate hyperthermia. It is likely that this amount of plasma volume expansion did not have a very large effect on central blood volume, even though it prevented hypovolemia and partially restored SV (21).

The present study has used exercise in the supine position as a method to more effectively raise central blood volume in dehydrated subjects. Dehydration and hyperthermia are thought to reduce central blood volume during exercise in the upright position. Alterations in central blood volume can influence both cardiopulmonary and arterial baroreceptors, which have the potential to alter sympathetic response to exercise as well as vascular conductance to skin and other organs (19, 28, 32). Therefore, the main purpose of this study was to determine whether large increases in central blood volume, achieved by supine exercise, prevent the reduction in cutaneous vascular conductance (CVC) and MAP and the increase in plasma norepinephrine that are characteristic of dehydration and hyperthermia during upright exercise.

METHODS

Subjects. The seven endurance-trained male cyclists participating in this study had a mean (± SD) age of 27.6 ± 1.9 yr, weight of 74.0 ± 10.7 kg, height of 178.3 ± 5.4 cm, maximal heart rate of 186 ± 5 beats/min, and maximal oxygen consumption (VO₂max) of 4.7 ± 0.4 l/min. This study was approved by the Institutional Review Board at the University of Texas at Austin, and written informed consent was obtained. During the preliminary testing, VO₂max was first determined during upright cycling using an incremental protocol. The subjects were well acclimated to the heat by bicycling outdoors at ambient temperatures ≥35°C. To adapt to exercise in the supine position, each subject completed at least twelve to fifteen 30-min bouts of supine cycling in a 3-wk period. Before the experimental trials were begun, a sweating rate was determined during 2 h of cycling (~60% VO₂max) in the heat.

Experimental design. On two separate occasions, subjects first cycled for 2 h on an ergometer (Jaeger ERGOTEST), rested for 45 min in a thermoneutral environment (23°C), and then performed two counterbalanced, randomly assigned 30-min bouts of cycling, one in the upright and another in the supine position. These bouts were interspaced by 45 min of rest in a 23°C environment (Fig. 1). All the exercise was performed in the heat (35°C dry bulb; 50% relative humidity, wind speed 2 m/s) and at an intensity of 62 ± 2% of upright VO₂max (220 ± 12 W; 80–90 rpm).
During the first 2 h of exercise, the subjects randomly received either 1) a large volume of fluid (3.60 ± 0.36 liters) to maintain a normal hydration level (i.e., euhydration trial) or 2) a small volume of fluid (0.20 ± 0.01 liters) insufficient to replace the 1.6 l/h of water loss in sweat so that subjects became dehydrated by 4.9 ± 0.2% of their body weight (i.e., dehydration trial). During each rest period, before exercising in the upright or supine positions, subjects ingested 1.3 ± 0.3 liters of fluid (i.e., 6% Gatorade solution, containing 40 g/l sucrose, 20 g/l glucose, 20 mM Na⁺, 20 mM Cl⁻, and 3 mM K⁺) during both euhydration and dehydration trials so that the same hydration status observed after the 2-h bout of exercise was maintained. In the euhydration trials, the subjects' body weight after cycling in the upright and the supine positions was 3.30 ± 0.42 kg (4.7 ± 0.2%) lower than the initial body weight. The trials were separated by 5–7 days.

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. On arrival on the experimental testing day, subjects' nude body weights (i.e., baseline values) were recorded, and rectal and esophageal temperature thermistors were inserted. The subjects then put on shorts, socks, and cycling shoes, entered the environmental chamber (35°C), and sat quietly in an armchair for ≥15 min. During this time, a heart rate monitor was attached and a Teflon catheter was inserted into an antecubital vein for blood sampling. After ≥15 min of seated rest, a 6-ml blood sample was withdrawn for later determination of baseline hematocrit, hemoglobin (Hb) concentration, serum osmolality, and electrolytes. The subject then mounted the cycle ergometer and started cycling in the upright position for 120 min at 62 ± 2% \( \overline{V_{O_2}} \)max. Physiological responses of the same subjects with and without dehydration during this first 2-h exercise bout have been published previously (9).

On completion of the first 2 h, the instrumentation and clothing were rapidly removed from the subject, who then toweled dry, and postexercise body weight was recorded. The subject then voided his bladder to determine the volume of urine formed during exercise. Next, the subject redressed in dry clothing and rested for 45 min in a thermoneutral environment (23°C) in front of a fan to return core temperature to the baseline level. After this rest period, a 30-min bout of cycling in either the upright or supine position was performed. During supine exercise, the subjects lay on a table while secured with straps around the shoulders. The table was set ∼50 cm below the center of the ergometer crank arms. During supine exercise, an additional fan, apart from the frontal one used during exercise in both body positions, was placed 1.5 m above the subjects to offset the diminished heat dissipation from the back so as to maintain a similar core temperature during supine and upright exercise. Before subjects exercised in either body position, skin thermistors, a mercury-in-Silastic strain gauge, and a laser Doppler probe (left forearm) were attached. The mercury-in-Silastic strain gauge and the laser Doppler probe remained in place throughout exercise as well as during the 45-min resting period between exercise bouts.

During each 30-min exercise bout, \( \overline{V_{O_2}} \) was determined from 3 to 18 min of exercise and during each determination of cardiac output. Cardiac output was determined in quadruplicate from 20 to 28 min of exercise, using a \( \text{CO}_2 \) rebreathing technique. Blood pressure was determined during each determination of cardiac output. Heart rate, esophageal temperature (\( T_e \)), rectal temperature, and skin temperature were recorded continuously. Forearm blood flow was measured 8–10 times at rest and after 13–18 min of exercise using venous occlusion plethysmography. Cutaneous blood flow was measured at rest and after 10–12 min and 28–30 min of exercise (laser Doppler flowmetry). In a follow-up study, cutaneous blood flow was measured continuously. A 10-ml blood sample was withdrawn at 30 min of exercise. A rating of perceived exertion was recorded at 30 min of exercise (2).

Analytic techniques. A more detailed description of the analytic methods can be found elsewhere (9). \( \overline{V_{O_2}} \) was measured using computerized open-circuit spirometry. Cardiac output was determined using a computerized version of the \( \text{CO}_2 \) rebreathing technique of Collier (3) and adjusted for Hb concentration (20). Heart rate was measured using a heart rate monitor (Uniq CIC Heartwatch). SV was calculated as cardiac output divided by 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). MAP was calculated as \([ (2 \times \text{diastolic blood pressure}) + \text{systolic blood pressure}] / 3 \). Systemic vascular resistance was calculated as MAP divided by cardiac output and expressed as peripheral resistance (in mmHg·l⁻¹·min⁻¹).

Forearm blood flow was measured using venous occlusion plethysmography with a mercury-in-Silastic strain gauge on the left arm (33). Cutaneous blood flow was determined using a laser Doppler flowmeter (ALF 21; Transonic Systems, Ithaca, NY). The probe was placed 2 cm from the plethysmographic strain gauge. During skin blood flow measurements, a sling supported the entire weight of the left arm. Laser Doppler skin blood flow values obtained after 10 and 28 min of exercise were very similar, suggesting that a plateau in skin blood flow had been reached. Accordingly, the average of these two measures is reported. Forearm vascular conduc-

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**Experimental Protocol**

<table>
<thead>
<tr>
<th>Dehydration</th>
<th>Rest 45 min in 23°C</th>
<th>Upright/Supine</th>
<th>Rest 45 min in 23°C</th>
<th>Upright/Supine</th>
<th>Supine/Upright</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euhydration (fluid replacement) [Control]</td>
<td>120 min upright cycling at 62% ( \overline{V_{O_2}} )max in 35°C</td>
<td>30 min at 62% ( \overline{V_{O_2}} )max; 35°C</td>
<td>30 min at 62% ( \overline{V_{O_2}} )max; 35°C</td>
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</table>

Fig. 1. Sequence of experimental protocol whereby subjects first exercised for 120 min to become dehydrated or remained euhydrated (by fluid ingestion) and then performed 2 additional 30-min bouts of exercise, one in the upright and the other in the supine position, while cardiovascular effects of dehydration and hyperthermia were determined during the 20- to 30-min period. \( \overline{V_{O_2}} \)max, maximal oxygen consumption.
RESULTS

Resting circulatory and thermoregulatory data. Resting heart rate was similar in all conditions (79–81 beats/min), except during dehydration before upright exercise, when it was significantly elevated above all other conditions (90 ± 3 beats/min; P < 0.05). Resting core temperature was similar (36.8–36.9°C) before the 30 min of exercise in all conditions, as was skin temperature (34.7–34.9°C). Resting forearm blood flow (2.8–3.2 ml·100 ml⁻¹·min⁻¹) and FVC before the initial 120-min and subsequent 30-min bouts of upright cycling were all similar. Before supine exercise, resting forearm blood flow was somewhat higher (4.1–4.6 ml·100 ml⁻¹·min⁻¹) than that observed before upright exercise but again was not affected by dehydration. When subjects were either euhydrated or dehydrated, resting laser Doppler skin blood flow was not significantly different before supine compared with upright exercise (0.2–0.3 ± 0.1 V).

Hydration status and oxygen consumption during exercise. After the 30-min bouts of exercise were finished in either body position, body weight was similar to preexercise baseline values during euhydration (i.e., within 0.2 kg) but was reduced by 4.7% during dehydration. Vo₂ was identical during exercise in either body position in the euhydration and dehydration trials (2.93 ± 0.27 l/min).

Blood variables during exercise. As expected, dehydration compared with euhydration resulted in a significantly lower blood volume during exercise in both body positions, largely because of plasma volume losses (i.e., ~200–300 ml lower as Hb concentration was 3.7–4.3% higher). On the other hand, serum osmolality, serum sodium, and chloride concentrations all reflected the hydration status, being equally increased (P < 0.05) with dehydration compared with euhydration during exercise in both body positions (Table 1).

Serum glucose and blood lactate concentrations. No statistical differences in serum glucose concentration were observed during exercise in either body position or hydration status (Table 1). The low glucose concentration values in all conditions (2.8–3.2 mM) were asymptomatic responses to the preexercise ingestion of the

<table>
<thead>
<tr>
<th>Variables</th>
<th>Upright Exercise</th>
<th>Supine Exercise</th>
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<tbody>
<tr>
<td>%Decline in BV</td>
<td>41 ± 0.8</td>
<td>31 ± 1.1</td>
</tr>
<tr>
<td>%Decline in PV</td>
<td>56 ± 1.1</td>
<td>46 ± 1.5</td>
</tr>
<tr>
<td>Hemoglobin, g/dl</td>
<td>16 ± 0.04</td>
<td>16 ± 0.04</td>
</tr>
<tr>
<td>Hct, %</td>
<td>45 ± 1.1</td>
<td>45 ± 1.1</td>
</tr>
<tr>
<td>Osmolality, mosm/kg</td>
<td>280 ± 2</td>
<td>282 ± 1</td>
</tr>
<tr>
<td>Na⁺, mM</td>
<td>144 ± 1</td>
<td>151 ± 1</td>
</tr>
<tr>
<td>K⁺, mM</td>
<td>5.2 ± 0.1</td>
<td>5.3 ± 0.1</td>
</tr>
<tr>
<td>Lactate, mM</td>
<td>1.7 ± 0.1</td>
<td>2.0 ± 0.2</td>
</tr>
<tr>
<td>Glucose, mM</td>
<td>31 ± 0.2</td>
<td>32 ± 0.2</td>
</tr>
<tr>
<td>Lactate, mM</td>
<td>1.7 ± 0.1</td>
<td>2.0 ± 0.2</td>
</tr>
</tbody>
</table>

Values are means ± SE for 7 subjects. BV, blood volume; PV, plasma volume; Hct, hematocrit; Na⁺, serum sodium concentration; K⁺, serum potassium concentration; Cl⁻, serum chloride concentration. *Significantly different from euhydration (P < 0.05). †Significantly different from upright exercise (P < 0.05).
carbohydrate-electrolyte solution, which elevated resting glucose concentration to 7–8 mM. Blood lactate concentration was significantly higher during supine compared with upright exercise in either hydration status (P < 0.05; Table 1).

Body temperature during exercise. With euhydration in either body position, $T_{es}$ increased to 37.8–38.0 ± 0.1°C during exercise. With dehydration, $T_{es}$ increased during both upright and supine exercise to 38.7–38.8 ± 0.1°C, which was significantly higher than that during euhydration (P < 0.05; Fig. 2). The same pattern of response was observed in rectal temperature (Table 2). Dehydration did not affect mean skin temperature in either exercise position. However, during supine exercise, mean skin temperature was significantly higher than during upright exercise (35.2 vs. 34.2°C, respectively; P < 0.05) because of the higher back skin temperature (34.2 vs. 37.8°C; respectively; P < 0.05).

Forearm skin temperature was similar in all conditions during each 30-min bout of exercise (33.8–34.2°C; Table 2).

Table 2. Body temperatures, forearm blood flow, and skin blood flow after 30 min of exercise in the upright and supine positions with euhydration or dehydration

<table>
<thead>
<tr>
<th>Variables</th>
<th>Upright Exercise</th>
<th>Supine Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_{es}$, °C</td>
<td>37.8 ± 0.0</td>
<td>38.7 ± 0.1*</td>
</tr>
<tr>
<td>$T_{re}$, °C</td>
<td>37.9 ± 0.1</td>
<td>38.6 ± 0.1*</td>
</tr>
<tr>
<td>$T_{sk}$, °C</td>
<td>34.1 ± 0.2</td>
<td>34.0 ± 0.1*</td>
</tr>
<tr>
<td>$T_{sk}$ forearm, °C</td>
<td>33.8 ± 0.3</td>
<td>33.9 ± 0.3</td>
</tr>
<tr>
<td>FBF, ml·100 ml⁻¹·min⁻¹</td>
<td>11.2 ± 1.8</td>
<td>8.3 ± 1.6*</td>
</tr>
<tr>
<td>FVC, 10⁻¹ ml·100 ml⁻¹·min⁻¹·mmHg⁻¹</td>
<td>1.16 ± 0.20</td>
<td>0.92 ± 0.20*</td>
</tr>
<tr>
<td>CBF, %upright at same hydration</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>CVC, %upright at same hydration</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

Values are means ± SE for 7 subjects. $T_{es}$, esophageal temperature; $T_{re}$, rectal temperature; $T_{sk}$, mean skin temperature; FBF, forearm blood flow (index of skin blood flow in resting forearm); FVC, forearm vascular conductance; CBF, laser Doppler skin blood flow; CVC, cutaneous vascular conductance. Both CBF and CVC are expressed as %increase from upright exercise in a given hydration condition. *Significantly different from euhydration (P < 0.05). †Significantly different from upright exercise in same hydration status (P < 0.05).

Influence of dehydration and hyperthermia on hemodynamics, skin circulation, and plasma catecholamines during exercise. The effects of dehydration and hyperthermia during the first 120 min of upright exercise in these subjects have been reported in detail previously (9) and in general were similar to the responses during the subsequent 30 min of upright exercise presently reported. In the 30-min bouts, cardiovascular responses were measured during the 20- to 30-min period of exercise when the differences in body temperature between the dehydration and euhydration conditions were the largest (~0.8°C; Fig. 2). Specifically, comparing dehydration with euhydration during upright exercise, SV was 30 ± 6 ml lower and heart rate was 14 ± 2 beats/min higher during dehydration (P < 0.05; Fig. 3). In addition, cardiac output and MAP were 3.0 ± 0.7 l/min and 6 ± 2 mmHg lower, respectively, whereas systemic vascular resistance was 0.5 ± 0.2 mmHg·l⁻¹·min higher during dehydration (all P < 0.05) compared with euhydration values.

Comparing dehydration with euhydration during supine exercise, SV was 20 ± 8 ml lower and heart rate was 7 ± 2 beats/min higher during dehydration (both P < 0.05). In addition, cardiac output was 1.7 ± 0.9 l/min lower (P < 0.05), yet MAP was unaltered, indicating that systemic vascular resistance was 0.5 ± 0.2 mmHg·l⁻¹·min higher during dehydration (P < 0.05) compared with euhydration values (Fig. 3).

During supine exercise, plasma norepinephrine concentration was significantly higher than during upright exercise in either hydration status (P < 0.05). Plasma epinephrine concentration was also significantly higher during supine exercise compared with that during upright exercise in both euhydration and dehydration (both P < 0.05; Fig. 3). In contrast, with dehydration during supine exercise, forearm blood flow and FVC did not decline significantly (Fig. 4).

During upright exercise, dehydration resulted in significantly (P < 0.05) higher plasma norepinephrine (5.4 nM or 50 ± 15%) and epinephrine concentrations (4.3 nM or 96 ± 42%) compared with those during euhydration (Fig. 5). However, during supine exercise plasma norepinephrine and epinephrine concentrations were not different from those during upright exercise (Fig. 5).

Influence of body posture on hemodynamics, skin circulation, and plasma catecholamines during exercise. Compared with the effects of upright exercise in subjects with dehydration, supine exercise attenuated the increases in heart rate and the reductions in SV and cardiac output (7 ± 2 vs. 9 ± 1% higher heart rate, 1% higher heart rate, 1% lower SV, and 1% lower cardiac output, P < 0.05; Table 1).
13 ± 4 vs. 21 ± 3% lower SV, 8 ± 3 vs. 14 ± 3% lower cardiac output during supine vs. upright, respectively; all P < 0.05). Moreover, supine exercise prevented the significant reductions in MAP, forearm blood flow, and FVC as well as the significant increases in plasma catecholamines observed with dehydration during upright exercise.

On the other hand, compared with upright exercise in euhydrated subjects, supine exercise restored two-thirds of the SV decline and prevented one-third of the increase in heart rate observed with upright exercise in dehydrated subjects (Fig. 3 and Table 1). During supine exercise in dehydrated and hyperthermic subjects, SV

![Fig. 3. Cardiovascular responses during 20- to 30-min period of exercise in heat in upright and supine positions with euhydration or dehydration (4.7% body wt): mean arterial pressure (A), cardiac output (B), heart rate (C), stroke volume (D), and systemic vascular resistance (E). Values represent means ± SE for 7 subjects. *Significantly different from euhydration (P < 0.05). †Significantly different from upright exercise (P < 0.05).](image)

and cardiac output were not statistically different from those measured during upright exercise in euhydrated subjects [130 ± 11 vs. 141 ± 9 ml/beat SV and 20.9 ± 1.5 vs. 21.3 ± 1.1 l/min cardiac output, respectively; not significant (NS)]. Furthermore, MAP was significantly higher than that measured during upright exercise in euhydrated subjects (107 ± 2 vs. 93 ± 2 mmHg; P < 0.05; Fig. 3). The higher MAP during supine compared with upright exercise was accompanied by both higher systolic blood pressure (195 ± 7 vs. 172 ± 7 mmHg,
respective; P < 0.05) and diastolic blood pressure (62 ± 1 vs. 54 ± 3 mmHg, respectively; P < 0.05). In addition, during supine exercise in dehydrated subjects, forearm blood flow and FVC tended to be higher than during upright exercise in euhydrated subjects (18 and 7% higher, respectively; NS; Fig. 4, A and B), whereas plasma catecholamines were not different (Fig. 5). Furthermore, whether subjects were euhydrated or dehydrated, skin blood flow and CVC during supine exercise were significantly higher than during upright exercise (Table 2).

Forarm blood flow, skin blood flow, FVC, and CVC during exercise in the follow-up study. During upright exercise, dehydration similarly reduced forearm blood flow and FVC as in the main study (i.e., 9–10%). Furthermore, with dehydration during upright exercise, laser Doppler skin blood flow and CVC were significantly (P < 0.05) lower compared with the same measurements with euhydration (Fig. 4, C and D). In contrast, dehydration did not significantly reduce forearm blood flow, skin blood flow, FVC, or CVC during supine exercise. Additionally, supine exercise resulted in higher laser Doppler skin blood flow and CVC than upright exercise in euhydrated subjects (Fig. 4, C and D). Nevertheless, dehydration compared with euhydration during supine exercise resulted in a delayed onset of vasodilatation (252 ± 21 vs. 203 ± 16 s, respectively; P < 0.05) and increased T_{es} threshold for vasodilatation (37.37 ± 0.11 vs. 37.05 ± 0.11°C, respectively; P < 0.05).

Sweating rate. No differences in total sweat volume during the 30 min of exercise were observed when subjects exercised in either state of hydration or either body position (mean range 0.8 ± 0.1 – 0.9 ± 0.1 l).

Rate of perceived exertion. With euhydration, the rate of perceived exertion was identical during exercise in both body positions (15 ± 1 units). With dehydration, the rate of perceived exertion tended to be higher than with euhydration during both upright (16 ± 1 vs. 15 ± 1 units; P = 0.08) and supine exercise (17 ± 1 vs. 15 ± 1 units; P = 0.06).

**DISCUSSION**

Our purpose was to determine whether the alterations in cardiovascular function caused by dehydration and hyperthermia during upright exercise were attenuated or prevented by augmenting central blood volume with exercise in the supine position. The exercise intensity, the amount of dehydration, and the increases in serum osmolality, serum sodium, and T_{es} during exercise in the upright position were identical to those in the supine position. The most important observations of the present study were that the reductions in MAP and CVC and the increase in plasma norepinephrine concentration that are characteristic of exercise in the upright position when subjects are dehydrated and hyperthermic were totally absent during supine exercise, despite equal dehydration and hyperthermia. Furthermore, supine exercise compared with upright exercise when subjects were dehydrated and hyperthermic restored two-thirds of the reduction in SV and prevented one-third of the increase in heart rate. These results suggest that a reduction in central blood volume and MAP are factors associated with reduced CVC and increased plasma norepinephrine with dehydration and hyperthermia during upright exercise.

The only previous study aimed at addressing whether supine exercise prevents or attenuates the effects of dehydration on SV and heart rate during exercise in the sitting position was performed by Saltin and Stenberg (30). They tested two subjects, first when euhydrated early in exercise and then again when dehydrated after a 195-min exercise period. They found an 18% decline in SV during exercise in both the supine and the sitting positions. However, Saltin and Stenberg (30) observed that cardiac output was maintained during prolonged upright exercise, in contrast to the present study, which elicited a more severe hyperthermic stress. Nevertheless, they observed MAP to be lower when subjects were dehydrated during prolonged upright exercise, as observed presently.

The present study was designed to test subjects who were trained in both modes of cycling exercise while measurements were made at the same V_{O2} and in a counterbalanced order to control for the effect of exercise per se on V_{O2} and heart rate drifts. In addition, subjects experienced an identical rise in core temperature with dehydration (T_{es} ~ 0.8°C higher than with
dehydration) when exercising in the supine and upright positions, allowing the identification of the independent effect of increased central blood volume and venous return. Supine exercise was performed with the legs above the heart level and the trunk lying on a table in the horizontal position. Our main assumption is that this manipulation did indeed cause a significantly higher venous return, central blood volume, and left ventricular end-diastolic volume compared with those measured during upright exercise. Previous reports of SV, central blood volume, central venous pressure, and left ventricular end-diastolic volume during supine versus upright exercise support the present assumption (1, 5, 26). More specifically, the presently observed 10- to 20-ml higher SV during supine versus upright cycling in euhydrated and dehydrated subjects was probably the result of a higher left ventricular end-diastolic volume, because end-systolic volume is already very low during moderately intense exercise (<30 ml end-systolic volume; see Ref. 26).

We have recently shown that the lowering of SV with dehydration during upright exercise is related to the combined influences of hypovolemia, concomitant to dehydration-induced plasma volume losses, and hyperthermia (10). This is based on the observation that, when dehydration-induced hypovolemia and hyperthermia are fully prevented by an intravenous infusion of a plasma volume expander and exercise is performed in the cold, no reductions in SV, cardiac output, or MAP are observed despite the 3- to 4-l extravascular dehydration (10). In addition, these variables are unaltered during prolonged exercise when euhydration is maintained and $T_{es}$ is stabilized at ~38°C (8, 9). Separately, dehydration-induced hypovolemia and hyperthermia (+1°C higher core temperature) exert similar effects because both reduce SV 7–8% and increase heart rate 5% without significantly affecting cardiac output or MAP (10). It is when they are combined during upright exercise, as in the present study, that cardiac output declines because of the synergistic 19–27% reduction in SV (8–11, 21–23, 29, 31).

Our important observation that supine exercise, even in dehydrated and hyperthermic subjects, restored FVC and CVC and lowered plasma norepinephrine to levels observed during exercise in euhydrated subjects emphasizes the important effect of central blood volume on baroreflex control of skin circulation and the sympathetic nervous system. It therefore appears that the reductions in CVC and increase in norepinephrine concentration observed during upright exercise in dehydrated and hyperthermic subjects are caused by reductions in central blood volume. It is known that central blood volume greatly influences the circulation to skin from the observations that elevations in central blood volume with water immersion or supine exercise offset the plateauing of skin blood flow at high core temperatures that are normally seen during exercise in the upright position in euhydrated subjects (24, 25). MAP was also maintained at euhydration levels in the present study during supine exercise in dehydrated subjects but declined significantly during upright exercise in dehydrated subjects. Therefore, the reduced MAP with dehydration during upright exercise was also associated with significant increases in plasma norepinephrine and epinephrine (50–95%) and reductions in forearm skin blood flow and FVC (27 ± 6 and 22 ± 8%, respectively; Fig. 4, A and B). Apparently, the present effect of dehydration (i.e., 3,300 ml) on reducing skin blood flow during upright exercise is not caused only by the concomitant 200- to 300-ml reduction in plasma volume. In this light, Montain and Coyle (21) have previously shown that plasma volume expansion during upright cycling in similarly dehydrated subjects failed to restore forearm blood flow or reduced core temperature, possibly because it did not sufficiently raise central blood volume.

During upright exercise, a lowered central blood volume and a reduced MAP with dehydration and hyperthermia would likely result in the unloading of both low- and high-pressure baroreceptors (19, 28). There are three possibilities by which low- and high-pressure mechanoreflexes (baroreceptors) could mediate the significant reductions in CVC and FVC with dehydration and hyperthermia during upright exercise. High- and low-pressure mechanoreflexes could be inhibiting active vasodilator outflow, superimposing sympathetic vasoconstriction on the cutaneous blood flow response, or influencing both processes (14–18, 22). The elevation of plasma norepinephrine by dehydration during upright exercise and the restoration of plasma norepinephrine by supine exercise despite dehydration support the notion that cutaneous vasoconstriction is involved in dehydration-induced vasoconstriction of the skin (22). However, this does not lessen or exclude the possibility that withdrawal of active vasodilation is also involved. Taken together, these results indicate that factors such as hypernatremia, hyperosmolality, and hyperthermia, which were identical during upright and supine exercise with dehydration, are not the primary signals mediating the reduced CVC and FVC with dehydration during upright exercise. These factors, however, might influence the time and $T_{es}$ at the onset of vasodilation and potentiate the action of other mechanisms (6, 18, 23). A more plausible possibility is that dehydration during upright exercise is sensed through reductions in central blood volume and MAP and that this reduces CVC by sympathetic vasoconstriction of the cutaneous circulation and possibly by withdrawal of active vasodilation. An unresolved question regards the mechanisms responsible for the 11 ml/beat (8%) lower SV with dehydration and hyperthermia during supine exercise compared with that in subjects euhydrated with a lower core temperature. This still-reduced SV was associated with the persistent dehydration-induced hypovolemia, hyperthermia, and elevated heart rate. Indeed recent results support the notion that increases in heart rate, paralleling the rise in core temperature from 36 to 40°C ($r^2$ = 0.98; $P$ = 0.001), contributes to reductions in SV with heat stress, particularly at core temperatures >38°C when cutaneous blood flow reaches a plateau level (11). Providing a more direct support of...
this hypothesis, Fritzsche et al. (7) have recently demonstrated that blunting the progressive rise in heart rate during 60 min of exercise with β-blockade totally prevents the decline in SV from occurring after 15 min of cycling exercise in a thermoneutral environment.

In summary, these results suggest that the decline in cutaneous vascular conductance and the increased plasma norepinephrine concentration with dehydration during upright exercise, independent of hyperthermia, are associated with a reduction in central blood volume and a lower arterial blood pressure. Furthermore, increased central blood volume derived from exercise in the supine position reversed most of the reduction in SV experienced with dehydration, whereas the persistent reduction in SV was associated with an elevated heart rate and hyperthermia.

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