Effects of marked hyperthermia with and without dehydration on VO₂ kinetics during intense exercise

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Nybo, Lars, Thorbjørn Jensen, Bodil Nielsen, and José González-Alonso. Effects of marked hyperthermia with and without dehydration on VO₂ kinetics during intense exercise. J Appl Physiol 90: 1057–1064, 2001.—This study determined whether marked hyperthermia alone or in combination with dehydration reduces the initial rate of rise in O₂ consumption (VO₂ on-kinetics) and the maximal rate of O₂ uptake (VO₂ max) during intense cycling exercise. Six endurance-trained male cyclists completed four maximal cycle ergometer exercise tests (402 ± 4 W) when euhydrated or dehydrated (4% body wt) with normal (starting esophageal temperature, 37.5 ± 0.2°C; mean skin temperature, 31°C) or elevated (+1 and +6°C, respectively) thermal strain. In the euhydrated and normal condition, subjects reached VO₂ max (4.7 ± 0.2 l/min) in 228 ± 34 s, with a mean response time of 42 ± 2 s, and fatigued after 353 ± 39 s. Hyperthermia alone or in combination with dehydration reduced mean response time (17–23%), VO₂ max (16%), and performance time (51–53%) (all P < 0.01) but did not alter the absolute response time (i.e., the time to reach 63% response in the control trial, 3.2 ± 0.1 l/min, 42 s). Reduction in VO₂ max was accompanied by proportional decline in O₂ pulse and significantly elevated maximal heart rate (195 vs. 190 beats/min for hyperthermia vs. normal). Preventing hyperthermia in dehydrated subjects restored VO₂ max and performance time by 65 and 50%, respectively. These results demonstrate that impaired high-intensity exercise performance with marked skin and internal body hyperthermia alone or in combination with dehydration is not associated with a diminished rate of rise in VO₂ but decreased VO₂ max.

Therefore, the purpose of this study was twofold: 1) to determine whether VO₂ on-kinetics would be blunted in conditions of marked dehydration and hyperthermia [i.e., 4% body wt loss; esophageal temperature (Tₑs) = +1°C and mean skin temperature (Tₛk) = +6°C] expected to impair VO₂ max, and 2) to determine to what extent the hampered maximal aerobic capacity is related to the independent effects of dehydration and hyperthermia. We speculated that the VO₂ on-response could be attenuated when hyperthermic and dehydrated subjects initiate an intense exercise bout. Furthermore, we hypothesized that the impairment of high-intensity exercise performance with dehydration and hyperthermia is largely related to effects of hyperthermia on reducing VO₂ max. There are many possible heat strain conditions resulting from different levels of dehydration and hyperthermia. We chose to study...
large differences in hydration status and body temperature, which could represent physiological conditions experienced by athletes performing in hot environments without fluid replacement, compared with cool environments with full fluid replacement.

**METHODS**

**Subjects.** Six endurance-trained male cyclists participated in this study. They had a mean (± SD) age of 24 ± 2 yr, body weight of 73.3 ± 6.9 kg, and height of 182 ± 5 cm. Maximal heart rate (HR$_{\text{max}}$) and VO$_2$$_{\text{max}}$ determined during an incremental protocol were 189 ± 8 beats/min and 4.69 ± 0.46 l/min (64.5 ± 3.0 ml·kg$^{-1}$·min$^{-1}$), respectively. The subjects were fully informed of the risks and discomforts associated with the experiments before they gave their informed written consent to participate. All subjects completed six practice trials to adapt to the maximal tests and the hot environment. On the last practice day, the subjects performed two maximal tests separated by 1 h, revealing the same VO$_2$$_{\text{max}}$, HR$_{\text{max}}$, and performance time when environmental and hydration conditions were unchanged. Furthermore, VO$_2$$_{\text{max}}$ and HR$_{\text{max}}$ attained during the tests with constant workload were similar to the values determined during the tests with incremental protocol.

**Experimental design.** On two occasions separated by 5–7 days, the subjects completed four counterbalanced maximal exercise tests on a cycle ergometer (Monark 829E) (see Fig. 1). Each maximal test was separated from previous exercise by a 1-h interval consisting of 45 min of rest, 12 min of light cycling (~50% VO$_2$$_{\text{max}}$), and 3 min of rest while the subject was seated on the cycle ergometer. The maximal tests were performed with either a normal (starting T$_{\text{sk}}$ = 37.5 ± 0.2°C (± SE) and T$_{\text{sk}}$ = 30.8 ± 0.6°C; normal) or a hyperthermic (T$_{\text{sk}}$ = 38.5 ± 0.2°C and T$_{\text{sk}}$ = 37.0 ± 0.2°C; hyper) condition. Core and skin temperatures were manipulated by changing the temperature of the water perfusing a jacket that was in contact with the skin of the trunk and arms (water temperature: 14°C in normal and 44°C in hyper). The subjects wore the perfused jacket throughout the last 15 min of the resting period, during the 12 min of light cycling, and during the maximal tests. In addition, to lower the elevated body temperature from previous exercise and to ensure a normal starting body temperature in normal trials, subjects were immersed in 15°C water for 15 min. In the hyper trials, heat stress was increased during the resting and light exercise periods by having the subjects wear rain trousers. The exercise tests were performed until exhaustion at a work intensity that exhausted the subjects within 5–8 min (402 W). A maximal exercise test was performed with either a normal (starting Tes = 37.5 ± 0.2°C (± SE) and Tes = 33.5 ± 0.2°C; normal) or an elevated thermal strain (hyperthermia; hyper) condition. The maximal tests were separated from previous exercise by 1-h interval consisting of 45 min of rest, 12 min of light cycling (50% maximal O$_2$ consumption [VO$_2$$_{\text{max}}$]), and 3 min of seated rest on the cycle ergometer.

**Experimental protocol**

<table>
<thead>
<tr>
<th>Trial</th>
<th>Description</th>
<th>Protocol</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DH</strong></td>
<td>Dehydration (4% of body weight)</td>
<td>2 hr exercise at ~50% VO$<em>2$$</em>{\text{max}}$ in 37°C</td>
</tr>
<tr>
<td><strong>EU</strong></td>
<td>Euhydration (fluid replacement)</td>
<td>2 hr exercise at ~50% VO$<em>2$$</em>{\text{max}}$ in 37°C</td>
</tr>
</tbody>
</table>

On a given day, the maximal tests were preceded by a 2-h exercise bout at ~50% of VO$_2$$_{\text{max}}$ in the heat (~37°C, 15–20% relative humidity), in which the subject’s hydration status was altered. On 1 day [dehydration (DH) trial], the subjects only ingested 0.3 liter of a concentrated carbohydrate-electrolyte solution and became dehydrated by 4.0 ± 0.3% of their body weight. On the other day [euhydration (EU) trial], they ingested 3.0 ± 0.2 liters of diluted carbohydrate-electrolyte solution, thereby maintaining their hydration status. The solution, which contained 100 g of carbohydrate on both days, was divided into eight equal aliquots and ingested every 15 min during the 2-h exercise bout.

VO$_2$, O$_2$ kinetics, and heart rate. Pulmonary VO$_2$, CO$_2$ production, and ventilation were measured on-line, breath by breath, with Medgraphics cardipulmonary exercise testing system CPX/D (Saint Paul, MN). To characterize VO$_2$ kinetics, a monoexponential function of time [f(t)] with the formula

$$f(t) = V_{O2, baseline} + ΔV_{O2} \ast [1 - e^{-(t-T_{bas})}]$$

where VO$_2$$_{\text{baseline}}$ is the VO$_2$ during the minute before starting the maximal exercise bouts and ΔVO$_2$ is the increase at time t above that at baseline, was fitted to the individual data, and parameter values (delay constant ($T_d$) and time constant ($τ$) that yielded the lowest sum of squared residuals were determined (36). No attempt was made to fit a mathematical model to the initial phase because of the limited number of data points during the first 10–15 s of exercise (15). Furthermore, the application of a two-exponential model to the raw VO$_2$ data did not improve the mathematical fitting (both mono- and two-exponential models, $r = 0.94 ± 0.01$), thus rendering the basis for selecting the simplest fitting model. In contrast to previous studies examining VO$_2$ kinetics during heavy exercise (2, 19, 22), it was only feasible to perform one maximal trial per exercise condition in the present study. Mean response time ($τ$) was then calculated by solving the above equation with respect to the time necessary to reach 63% (corresponding to time constant of monoeponential response; $τ$) of the difference between baseline (rest) and the maximal response (plateau corresponding to VO$_2$$_{\text{max}}$). Heart rate was recorded every 5 s with a Polar Sports Tester (Polar Electro). O$_2$ pulse (O$_2$$_{\text{pulse}}$) was calculated by dividing VO$_2$ by heart rate.

Core and skin temperature. T$_{sk}$ was measured with a thermocouple (MOV-A, Ellab, Copenhagen, Denmark) inserted through the nasal passage at a distance equal to one-fourth of the subject’s height (33). T$_{sk}$ was calculated from the skin temperatures (thermocouple A-H3, Ellab) measured at six sites (i.e., back, chest, upper arm, forearm, thigh, and calf) using the weighting method of Hardy and DuBois (16). The esophageal and skin thermocouples were connected to a recorder (CTF 9008 Precision Thermometer and Fo-Value...
Computer, Ellab), and temperatures were registered every 30 s with an accuracy of 0.1°C. Muscle temperature was measured in the vastus lateralis in one subject with a needle probe (model MKA-A, Ellab) inserted 3 cm into the muscle.

Blood analysis. All resting blood samples were withdrawn from an antecubital vein after the subject had been seated for at least 15 min. Hemoglobin and hematocrit were determined in duplicate using an ABL 510 analyzer (Radiometer, Copenhagen, Denmark). Percent changes in blood volume and plasma volume were calculated by using the equations of Dill and Costill (7). Lactate and blood glucose concentrations were measured by using a glucose and lactate YSI model 2700 analyzer (Yellow Springs Instruments). Plasma osmolality was determined by the freezing point-depression technique (advanced osmometer model 3D3, Advanced Instruments), whereas plasma norepinephrine and epinephrine concentrations were determined by using a RIA kit (KatCombi RIA, Biotech-IgG, Copenhagen, Denmark).

Statistical analysis. One- and two-way ANOVA with repeated measures were performed to test significance among the different trials. After a significant F test, pairwise differences were identified by using Tukey’s honestly significant difference post hoc procedure. When appropriate, significant differences were also identified by using Student’s paired t-test. The significance level was set at P < 0.05. Data are presented as means ± SE.

RESULTS

Hydration status. Hydration status at the beginning of the 2 testing days (DH vs. EU) was not different, as indicated by similar body weights (73.3 ± 3.0 vs. 73.2 ± 2.9 kg, respectively), plasma osmolality (285 ± 1 vs. 286 ± 1 mosmol/kgH2O, respectively), and hemoglobin concentration (14.8 ± 0.3 g/dl on both days). In DH, body weight declined to 70.7 ± 2.8 kg (4.0 ± 0.3% body wt loss), whereas plasma osmolality rose to 297 ± 3 mosmol/kgH2O, and hemoglobin concentration increased to 153 ± 0.3 g/dl, reflecting a 4.1 ± 1.1% blood volume reduction. In EU, body weight, plasma osmolality, and hemoglobin concentration remained unchanged (Table 1). No differences in hydration were detected between hyper and normal trials on the respective days (Table 1).

Core and skin temperature. During the four maximal trials, Tsk increased progressively throughout exercise, whereas Tmc remained constant (Fig. 2). The rate of increase in Tmc was similar in the four trials (0.18°C/min), thereby maintaining the ~1°C higher Tmc in hyper vs. normal. However, because of the longer exercise times in normal, Tmc rose over a longer period, and at exhaustion the difference in Tmc between EU-normal and EU-hyper, therefore, decreased to 0.3°C (38.7 ± 0.3 vs. 39.0 ± 0.2°C) and was no longer significant, whereas the difference between DH-normal and DH-hyper declined to 0.5°C (38.5 ± 0.2 vs. 39.0 ± 0.2°C; P < 0.05). Tsk was at all time points ~6°C higher (P < 0.01) in the hyper trials compared with the normal trials (see Fig. 2B).

V02 and performance time. V02 before (baseline) and during the first 60 s of exercise was similar in all trials (Fig. 3A and Fig. 4). However, after 60 s of exercise, V02 was significantly lower in EU-hyper and DH-hyper compared with EU-normal (P < 0.05; Fig. 3A). At 156 ± 17 s (exhaustion in the shortest trial), V02 was reduced by 0.44–0.45 l/min in the hyper trials, and the total amount of O2 consumed until this time was 0.6 l/min in EU-hyper and DH-hyper compared with EU-normal (P < 0.01; Table 2). V02max was equally reduced by 16 ± 1% in the two hyper trials, and performance time was shortened by approximately one-half compared with that in EU-normal (51 ± 6% reduction in EU-hyper and 53 ± 6% in DH-hyper; both P < 0.01). Preventing hyperthermia in dehydrated subjects (DH-normal) restored two-thirds of the decline in V02max and almost one-half of the reduction in performance time. However, compared with EU-normal, both V02max and performance time with dehydration alone were significantly reduced by 5 ± 2 and 26 ± 8% (P < 0.05), respectively (Table 2).

V02 on kinetics. V02 τ, defined as the time necessary to reach 63% of the rise in V02 between baseline and the maximal response, was significantly shorter in the two hyper trials compared with EU-normal (Table 3). However, this was due to the reduced V02max and not to a faster rise in V02 in the hyper trials (63% response was reduced from 3.2 ± 0.1 l/min in EU-normal to 2.7 ± 0.1 l/min in the hyper trials, P < 0.05; see Fig. 3A). Therefore, the time to reach the same V02 as the 63% response in EU-normal (3.2 ± 0.1 l/min) was calculated, showing no statistically significant differences among the four trials (absolute response time; Table 3).

Heart rate and O2pulse. Heart rate was 15–25 beats/min higher throughout exercise in the hyper trials compared with EU-normal (P < 0.05; Fig. 3B).

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Table 1. Hematologic parameters at rest and during intense cycle ergometer exercise with euhydration and dehydration under normal or elevated thermal strain

<table>
<thead>
<tr>
<th>Variables</th>
<th>Euhydration (DH-normal)</th>
<th>Hyperthermia (DH-hyper)</th>
<th>Dehydration (EU-hyper)</th>
<th>Normal (EU-normal)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin, g/dl</td>
<td>14.8 ± 0.3</td>
<td>15.3 ± 0.3*</td>
<td>15.3 ± 0.3*</td>
<td></td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>45.2 ± 0.6</td>
<td>46.3 ± 0.6</td>
<td>47.0 ± 0.6*</td>
<td></td>
</tr>
<tr>
<td>Blood volume, %</td>
<td>0.0 ± 0.0</td>
<td>0.1 ± 0.4</td>
<td>-4.5 ± 0.3*</td>
<td>-3.7 ± 0.5*</td>
</tr>
<tr>
<td>Osmolality, mosmol/kgH2O</td>
<td>283 ± 1</td>
<td>297 ± 4*</td>
<td>298 ± 3*</td>
<td></td>
</tr>
<tr>
<td>Lactate, mmol/l</td>
<td>5.9 ± 0.6</td>
<td>5.6 ± 0.6</td>
<td>5.1 ± 0.6</td>
<td></td>
</tr>
<tr>
<td>Glucose, mmol/l</td>
<td>3.5 ± 0.2</td>
<td>4.4 ± 0.6</td>
<td>4.9 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>Epinephrine, nmol/l</td>
<td>8.0 ± 1.3</td>
<td>6.1 ± 1.5</td>
<td>6.0 ± 1.4</td>
<td></td>
</tr>
<tr>
<td>Norepinephrine, nmol/l</td>
<td>27.7 ± 5.6</td>
<td>18.8 ± 1.1</td>
<td>17.8 ± 5.8</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SE for 5 subjects. Blood volume is expressed as percent change from euhydration normal. Hemoglobin, hematocrit, and osmolality were measured before exercise while subjects were resting on the cycle ergometer (values shown) and at exhaustion. Values at exhaustion showed a similar pattern of response as those at rest but were also influenced by different exercising time. Lactate, glucose, epinephrine, and norepinephrine concentrations were only measured at exhaustion. *Significantly different from euhydration normal and euhydration-hyperthermia (P < 0.05).
Furthermore, HR_max was elevated by 5 ± 1 beats/min in the hyper trials compared with the respective trials with normal body temperatures (P < 0.01; Table 2). Dehydration without hyperthermia did not alter HR_max. However, the attainment of HR_max occurred faster in the DH condition compared with EU-normal (Fig. 3B).

O_2 pulse followed the same general pattern of response as V̇O_2. However, O_2 pulse reached a maximum within 100 s of exercise in all trials. O_2 pulse was reduced by 17 ± 2% in the hyper trials compared with EU-normal (P < 0.01, Fig. 3C), which is highly correlated to the reductions in V̇O_2_max (r² = 0.95–0.98). Preventing hyperthermia in the DH condition restored most of the decline in O_2 pulse (see Table 2).

**Blood and plasma variables.** Blood glucose and lactate concentrations both at the beginning of the maximal tests and at exhaustion were similar in all four conditions (Table 1). Furthermore, at exhaustion, no differences were detected in forearm venous plasma concentrations of epinephrine and norepinephrine (Table 1).

![Graphs](image)

**Fig. 2.** Esophageal temperature (A) and mean skin temperature (B) responses during maximal cycle ergometer exercise at constant power output when subjects were euhydrated or dehydrated with normal or elevated thermal strain (EU-normal, EU-hyper, DH-normal, DH-hyper). Values are means ± SE for 6 subjects. *Significantly higher than EU-normal, P < 0.05.

**Fig. 3.** O_2 consumption (V̇O_2; A), heart rate (B), and O_2 pulse (C) responses during maximal cycle ergometer exercise at constant power output when subjects were euhydrated or dehydrated with normal or elevated thermal strain. Values are means ± SE for 6 subjects. In EU-normal, it looks as if a plateau in V̇O_2 is not obtained; however, this is due to the averaging effect, because all subjects maintained a plateau over the last 125 ± 23 s (range 60–230 s) of the exercise period (see Fig. 4). *Significantly different from EU-normal, P < 0.05.
with dehydration led to an augmented HRmax, suggesting that pronounced hyperthermia alone or in combination with dehydration is associated with hyperthermia. We also observed that pronounced hyperthermia alone or combined dehydration and hyperthermia is associated with hyperthermia. We also observed that reduced maximal aerobic capacity was primarily related to a lowering in stroke volume and/or arteriovenous \( \text{O}_2 \) difference.

Despite marked reductions in the maximal response, the initial rate of rise in \( \text{Vo}_2 \) was unaltered with either hyperthermia alone or combined hyperthermia and dehydration. The same interpretation of the data is reached when either a single- or a two-exponential model is used to describe the \( \text{Vo}_2 \) kinetics (both \( r = 0.94 \pm 0.01 \), as both reveal the same absolute response (i.e., time to reach \( 3.2 \pm 0.1 \) l/min, \(-42 s \)). In the face of an unchanged \( \text{Vo}_2 \) during the initial phase of exercise, it appears that the significant reduction in \( \tau \) from 42 to 33–35 s in the hyper trials compared with control only reflected the reduction in maximal response. The unaltered initial rate of rise in pulmonary \( \text{Vo}_2 \) in the hyper trials implies that the rate of muscle mitochondrial respiration was unchanged at the onset of exercise and that any possible reductions in \( \text{O}_2 \) delivery to the exercising leg muscles during the first minute of exercise were adequately counteracted. It is also clear that the higher muscle temperature (~1°C; see Ref. 10) in the hyper trials did not accelerate the oxidative reactions in the active muscles, corroborating previous findings during submaximal exercise (10, 11, 14, 22). However, \( \text{Vo}_2 \) was only similar during the initial 60 s of exercise, being thereafter significantly reduced by hyperthermia alone and combined dehydration and hyperthermia during the plateau phase.

The relative decline in \( \text{Vo}_{2\text{max}} \) (16%) with hyperthermia alone or in combination with dehydration is in agreement with the 27% \( \text{Vo}_{2\text{max}} \) reduction previously observed with hyperthermia alone (25) or combined dehydration and hyperthermia (6), but it is greater than the 3–10% \( \text{Vo}_{2\text{max}} \) decline found by others (21, 26, 32). The present observation, however, is in sharp contrast to four studies reporting that hyperthermia has no effect or increases \( \text{Vo}_{2\text{max}} \) (3, 27, 29, 37). Interestingly, Bergh and Ekblom (3) found a 2% rise in \( \text{Vo}_{2\text{max}} \) with elevated core temperature (~31°C). The main reason for this discrepancy appears to be the difference in thermal stress between the hyper and control conditions, spe-

**DISCUSSION**

The main finding of this study is that marked hyperthermia alone or combined dehydration and hyperthermia do not alter \( \text{Vo}_2 \) on-kinetics, despite the fact that they drastically reduce \( \text{Vo}_{2\text{max}} \). The observations that hyperthermia with or without dehydration resulted in similar reductions in \( \text{Vo}_{2\text{max}} \) and that prevention of hyperthermia in dehydrated subjects restored most of the decline in \( \text{Vo}_{2\text{max}} \) indicate that most of the decline in \( \text{Vo}_{2\text{max}} \) with combined dehydration and hyperthermia is associated with hyperthermia. We also observed that pronounced hyperthermia alone or in combination with dehydration led to an augmented HRmax, suggesting that reduced maximal aerobic capacity was primarily related to a lowering in stroke volume and/or arteriovenous \( \text{O}_2 \) difference.

Despite marked reductions in the maximal response, the initial rate of rise in \( \text{Vo}_2 \) was unaltered with either hyperthermia alone or combined hyperthermia and dehydration. The same interpretation of the data is reached when either a single- or a two-exponential model is used to describe the \( \text{Vo}_2 \) kinetics (both \( r = 0.94 \pm 0.01 \)), as both reveal the same absolute response time (i.e., time to reach \( 3.2 \pm 0.1 \) l/min, \(-42 s \)). In the face of an unchanged \( \text{Vo}_2 \) during the initial phase of exercise, it appears that the significant reduction in \( \tau \) from 42 to 33–35 s in the hyper trials compared with control only reflected the reduction in maximal response. The unaltered initial rate of rise in pulmonary \( \text{Vo}_2 \) in the hyper trials implies that the rate of muscle mitochondrial respiration was unchanged at the onset of exercise and that any possible reductions in \( \text{O}_2 \) delivery to the exercising leg muscles during the first minute of exercise were adequately counteracted. It is also clear that the higher muscle temperature (~1°C; see Ref. 10) in the hyper trials did not accelerate the oxidative reactions in the active muscles, corroborating previous findings during submaximal exercise (10, 11, 14, 22). However, \( \text{Vo}_2 \) was only similar during the initial 60 s of exercise, being thereafter significantly reduced by hyperthermia alone and combined dehydration and hyperthermia during the plateau phase.

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**Table 2. Oxygen consumption, heart rate, and performance during intense cycle ergometer exercise with euhydration and dehydration under normal or elevated thermal strain**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normal</th>
<th>Hyperthermia</th>
<th>Hyperthermia</th>
<th>Dehydration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Performance time, s</td>
<td>353 ± 39</td>
<td>170 ± 25*</td>
<td>156 ± 17*</td>
<td>251 ± 21†</td>
</tr>
<tr>
<td>( \text{Vo}_{2\text{max}} ), l/min</td>
<td>4.74 ± 0.21</td>
<td>3.99 ± 0.18*</td>
<td>3.96 ± 0.12*</td>
<td>4.47 ± 0.12†</td>
</tr>
<tr>
<td>Ventilation, l/min</td>
<td>179.8 ± 6.1</td>
<td>171.2 ± 6.2</td>
<td>167.0 ± 6.9</td>
<td>167.6 ± 3.1</td>
</tr>
<tr>
<td>RER</td>
<td>1.13 ± 0.03</td>
<td>1.22 ± 0.04*</td>
<td>1.23 ± 0.01*</td>
<td>1.14 ± 0.01†</td>
</tr>
<tr>
<td>Maximal HR, beats/min</td>
<td>190 ± 3</td>
<td>185 ± 3*</td>
<td>194 ± 5</td>
<td>189 ± 4†</td>
</tr>
<tr>
<td>( \text{O}_2 ) pulse, ml ( \text{O}_2 )/beat</td>
<td>25.0 ± 1.2</td>
<td>20.5 ± 1.0*</td>
<td>20.8 ± 0.6*</td>
<td>24.0 ± 0.5†</td>
</tr>
<tr>
<td>( \text{Vo}_2 ) at 156 ± 17 s, l/min</td>
<td>4.41 ± 0.14</td>
<td>3.97 ± 0.19*</td>
<td>3.96 ± 0.12*</td>
<td>4.36 ± 0.12†</td>
</tr>
<tr>
<td>Total ( \text{Vo}_2 ) at 156 ± 17 s, liters</td>
<td>8.1 ± 1.2</td>
<td>7.5 ± 1.1*</td>
<td>7.5 ± 1.2*</td>
<td>7.9 ± 1.2†</td>
</tr>
<tr>
<td>HR at 156 ± 17 s, beats/ min</td>
<td>177 ± 3</td>
<td>194 ± 3*</td>
<td>194 ± 5*</td>
<td>179 ± 4†</td>
</tr>
</tbody>
</table>

Values are means ± SE for 6 subjects. \( \text{Vo}_2 \), \( \text{O}_2 \) consumption; \( \text{Vo}_{2\text{max}} \), maximal \( \text{Vo}_2 \); RER, respiratory exchange ratio; HR, heart rate. *Significantly different from euhydration-normal \((P < 0.05)\). †Significantly different from dehydration-hyperthermia \((P < 0.05)\).
cifically whether or not internal body and skin temperatures were simultaneously elevated. The level of thermal stress that the cyclists experienced in this study was high, as core temperature was elevated by 1°C and $T_{es}$ was $\sim$6°C higher throughout the exercise. This is of similar magnitude to that incurred in studies finding the greatest reductions in aerobic capacity (6, 25). In one additional subject, $V_{O2\text{ max}}$ did not decline during intense cycle ergometer exercise, even with a greatly elevated core temperature (39.8°C), as long as skin temperature was below $\sim$32°C, which further supports the notion of an interaction between elevated skin and internal body temperature. The large thermal stress experienced by subjects in this study might also explain the finding that superimposing dehydration on hyperthermia did not lead to additional reductions in $V_{O2\text{ max}}$. Restoration of most of the decline in $V_{O2\text{ max}}$ (65% restoration) when hyperthermia was prevented in the presence of dehydration in the present and other studies (4, 35) argues strongly in favor of this notion. Furthermore, studies during submaximal exercise reveal that cardiac output only declines when both core and skin temperatures are elevated (12, 29), but it is maintained when only core temperature is 1°C higher (10) or is increased when only skin temperature is elevated by $\sim$12°C (13). Together, available evidence suggests that maximal aerobic capacity, while being sensitive to different alterations in core and skin temperature, is only markedly compromised when both skin and internal body temperatures are simultaneously elevated.

The impairment of $V_{O2\text{ max}}$ with hyperthermia alone or in combination with dehydration is tightly coupled with the parallel decline in $O_2\text{ pulse}$ ($r^2 = 0.95–0.98$). Given its dependence on arteriovenous $O_2$ difference and stroke volume, the decline in $O_2\text{ pulse}$ in the present study could potentially be ascribed to alterations in either of these factors. However, previous reports of arteriovenous $O_2$ difference during intense exercise indicate that hyperthermia itself does not reduce $O_2$ extraction (29, 37). Therefore, assuming a maximal arteriovenous $O_2$ difference of 170 ml $O_2$/l blood in the EU trials (29, 37), the observed 16–17% reduction in $O_2\text{ pulse}$ and $V_{O2\text{ max}}$ with hyperthermia could be accounted for by an $\sim$26-ml reduction in stroke volume, leading to an $\sim$4 l/min reduction in cardiac output.

These estimations are consistent with the reductions in stroke volume and cardiac output observed with dehydration and hyperthermia during prolonged exercise in the heat (9–14, 23, 24). Whereas the contribution of reduced stroke volume requires experimental support, it is clear that the lower $V_{O2\text{ max}}$ with hyperthermia and combined dehydration and hyperthermia was not limited by impaired $HR_{\text{max}}$. On the contrary, hyperthermia increased $HR_{\text{max}}$ by $\sim$5 beats/min (195 beats/min). The observation that the $HR_{\text{max}}$ was always close to but never $>$190 beats/min in all the preliminary intense training sessions, during maximal incremental exercise tests, and during the DH alone trial, strongly supports the reproducibility of this finding. The hyperthermia-induced tachycardia is a well-described phenomenon during submaximal exercise (9, 26, 29). However, to our knowledge, this is the first study to report that $HR_{\text{max}}$ is significantly elevated with hyperthermia. The mechanism underlying the small (<3%), although significant, elevation in $HR_{\text{max}}$ with hyperthermia in this study is unclear. One possibility could be a direct effect of temperature on intrinsic heart rate, given the 0.3–0.5°C higher $T_{es}$ at exhaustion in the hyper trials (8, 20, 30). A second possibility could be an increased sinus-atrial node depolarization rate, secondary to reduced stretching of the heart owing to declined stroke volume (Frank-Starling mechanism). A third possibility could be an elevated sympathetic β-receptor activation of the heart in response to high body temperature and unloading of high and low baroreceptors sensing lower blood pressure and central blood volume (13, 29).

The present experimental intervention that resulted in large differences in hydration level and body temperature could represent the best and worst case scenarios (full vs. no fluid replacement) that endurance athletes could encounter during events requiring high-power outputs for several minutes during competition in hot environments. For instance, this could be the case for endurance cyclists having to sprint during or

### Table 3. Maximal oxygen uptake mean response time and absolute response time during intense cycle ergometer exercise with euhydration and dehydration under normal or elevated thermal strain

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Normal</th>
<th>Hyperthermia</th>
<th>Dehydration</th>
<th>Normal</th>
<th>Hyperthermia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\tau$</td>
<td>$\tau'$</td>
<td>$\tau$</td>
<td>$\tau'$</td>
<td>$\tau$</td>
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<tr>
<td>1</td>
<td>41.5</td>
<td>33.1</td>
<td>41.5</td>
<td>26.7</td>
<td>46.5</td>
</tr>
<tr>
<td>2</td>
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<td>38.7</td>
<td>33.6</td>
<td>37.0</td>
</tr>
<tr>
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<td>35.8</td>
<td>37.9</td>
<td>45.6</td>
<td>38.1</td>
<td>44.6</td>
</tr>
<tr>
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<td>33.2</td>
<td>41.7</td>
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</tr>
<tr>
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<td>41.2</td>
<td>38.2</td>
<td>46.2</td>
</tr>
<tr>
<td>6</td>
<td>39.0</td>
<td>33.8</td>
<td>40.2</td>
<td>29.6</td>
<td>34.7</td>
</tr>
</tbody>
</table>

Mean ± SE: $42.2 \pm 2.3 \text{ s, } 35.0 \pm 0.9^c \text{ s, } 41.5 \pm 1.0 \text{ s; } 32.6 \pm 2.0^c \text{ s, } 41.6 \pm 2.0 \text{ s, } 39.4 \pm 0.9 \text{ s.}$

Values are expressed in s. Mean response time ($\tau$) is defined as the time to reach 63% of the increase in $V_{O2}$ from baseline to the maximal response. Absolute response time ($\tau'$) is defined as the time necessary to reach the same $V_{O2}$ as the 63% response in euhydration-normal (i.e., 3.2 ± 0.1 l/min). *Significantly shorter than euhydration-normal ($P < 0.05$).
REFERENCES


