15° Head-down tilt attenuates the postexercise reduction in cutaneous vascular conductance and sweating and decreases esophageal temperature recovery time

Natalie H. McInnis, W. Shane Journeay, Ollie Jay, Emily Leclair, and Glen P. Kenny

Laboratory of Human Bioenergetics and Environmental Physiology, School of Human Kinetics, Faculty of Health Sciences, University of Ottawa, Ottawa, Ontario; and Toxicology Graduate Program and Department of Veterinary Biomedical Sciences, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Canada

Submitted 30 March 2006; accepted in final form 23 May 2006

Studies employing recovery modes have attempted to elucidate the relative roles of nonthermal factors such as central command, mechanoreceptors, and baroreceptors in the modulation of heat loss responses during exercise recovery (3, 19, 33, 39). Collectively, the recovery mode studies have determined that attenuating the baroreceptor unloading effect associated with upright inactive recovery, through active or passive recovery, preserves SkBF. Central command did not influence the SkBF response. Sweating, however, can be influenced by central command and mechanoreceptors, albeit the role for baroreceptors remains unresolved.

During upright inactive recovery, there is a growing body of evidence in support of a possible relationship between hemodynamic changes postexercise and heat loss responses. Journeay et al. (18) specifically examined this issue by manipulating postexercise hemodynamics using lower body pressure. They observed a reflex increase in SkBF, sweating, and heat flux with application of +45 mmHg lower body positive pressure (LBPP) in the upright seated (URS) posture. Furthermore, an increase in the rate of core temperature decay was noted after reversal of postexercise hypotension by application of positive pressure to the lower extremities. This effect was not seen under lower body negative pressure or no pressure conditions. It was postulated that LBPP triggered a nonthermal baroreceptor-mediated increase in heat loss responses secondary to a redistribution of blood from the lower extremities to the central circulation.

LBPP produces its effects via increased barometric pressure around the lower extremities. This leads to enhanced microvascular compression in the tissues and produces a pressure gradient that tends to increase central blood volume. With the application of sufficient barometric pressure to the lower extremities, this technique may activate both cardiopulmonary and arterial baroreceptors (7). For example, a study showed that application of +45 mmHg of LBPP postexercise engaged both the cardiopulmonary and arterial baroreceptors (18). Also, it has been suggested that LBPP may activate mechanoreceptors particularly at pressures >20 mmHg (7), whereas others have ruled out mechanoreceptor activation during LBPP (32). One possible limitation of the LBPP technique in thermoregulatory studies is that possible convective airflow to the lower extremities may increase heat loss from the lower extremities. Furthermore, our laboratory has previously observed an en-
hanced rate of core temperature decay with application of
LBPP (18); however, this may not be a convenient cooling
strategy in the treatment of hyperthermic individuals due to the
time associated with transfer to the pressure box.
In contrast, head-down tilt (HDT) is known to increase
central blood volume by promoting venous return through a
reduction in the hydrostatic forces present in the upright
posture. Specifically, under resting conditions at tilt angles of
<30°, this technique is thought to engage the cardiopulmonary
baroreceptors without changes in systemic mean arterial
pressure (MAP) and therefore reduces the likelihood of engaging
the arterial baroreceptors (7–10, 27, 31). Thus HDT is another
method by which to manipulate postexercise hemodynamic
responses, and it allows for the attenuation of the baroreceptor
unloading effect of upright exercise recovery. Additionally,
HDT enables us to intervene immediately postexercise and
remove the possible effects of mechanoreceptor activation
and/or convective airflow on the lower extremities associated
with LBPP.
Thus the purpose of this experiment was to examine the
effect of HDT, and by extension the role of baroreceptors, on
prolonged postexercise heat loss and hemodynamic responses.
We tested the hypothesis that recovery from dynamic exercise
in the HDT position would augment the heat loss responses of
cutaneous vascular conductance and sweating relative to up-
right recovery in association with an augmented hemodynamic
response and increased rate of esophageal temperature (Tsa)
decay.

METHODS

Subjects

Seven healthy, physically active men volunteered and gave written
consent to participate in this study. The study was approved by the
Research Ethics Board at the University of Ottawa. Five to 7 days
before the experiments, peak oxygen consumption (V̇O₂ peak) was
measured during a progressive cycle ergometer protocol that required
the participant to cycle at a cadence of 80 revolutions/min while the
ergometer resistance was increased at 0.5 kp every 2 min. The
V̇O₂ peak data were used to select the submaximal workload for the
experimental exercise phase of the study. Subjects were 20 ± 0.8 (SE)
yr old and 182 ± 0.4 cm tall, weighed 78.3 ± 4.2 kg, and had a mean
V̇O₂ peak of 56.1 ± 2.6 ml·kg⁻¹·min⁻¹.

Measurements

Heart rate (HR) was monitored using a Polar coded transmitter,
recorded continuously and stored with a Polar Advantage interface
and Polar Precision Performance software (Polar Electro Oy, Kem-
pele, Finland). MAP was estimated from the integration of a nonin-
vasive recording of blood pressure at the middle digit of the left hand
(Finapres 2300, Ohmeda, Madison, WI) fixed at heart level (the third
intercostal space). The arm and hand were maintained in position
using a cotton arm sling. The position of the hand was verified before
all measurements. The Finapres system is based on the Penaz volume
clamp method (dynamic unloaded arterial wall principle). MAP was
verified periodically throughout the protocol by auscultation. Typi-
cally, measurements were verified at 5-min intervals during the first
15 min of recovery and at 10- to 15-min intervals thereafter.

Pulmonary oxygen consumption was estimated using a metabolic
cart (model CPX/D, Medgraphics, St. Paul, MN) during V̇O₂ peak
assessment preceding the experimental trials. Cardiac output (CO)
was estimated using the CPX/D computerized version of the CO₂
rebreathing technique of Defares (5). It has been shown that Doppler-
derived aortic blood flow (CO) measurements correlate well with the
indirect carbon dioxide rebreathing method (11). The Defares method
has also been shown to work well in “un-steady-state” testing (14).
Stroke volume (SV) was calculated as CO/HR. Total peripheral
resistance (TPR) was calculated as MAP/CO.

SkBF was estimated using laser-Doppler velocimetry (PeriFlux
System 5000, main control unit; PF5010 LDPM, function unit;
Perimed, Stockholm, Sweden) at the left midanterior forearm. The
laser-Doppler flow probe (PR 401 angled probe, Perimed) was taped
to cleaned skin, in an area that did not appear by visual inspection to
be overly vascular and from which consistent readings were noted
(28). Cutaneous vascular conductance (CVC) was calculated as the
ratio of laser-Doppler flow to MAP. At the end of the experiment,
local skin temperature was raised to 42°C using a heating element (PF
5020 temperature unit, Perimed) that housed the laser-Doppler flow
probe, until peak CVC (CVCpeak) was measured (~30 min) (35).
CVCpeak was determined as a sustained elevated plateau in local
SkBF. CVC data are presented as a percentage of maximal CVC as
determined by local heating (%CVCpeak). All SkBF measurements were
taken in the period preceding rebreathing to avoid causing fluctuations
in SkBF data at each time point. SkBF measurements were recorded from
the left midanterior forearm such that the arm was level with the heart.

Sweat rate was measured using a 5.0-cm² ventilated capsule placed
over the median inferior aspect of the trapezius muscle. Anhydrous
compressed air was passed through the capsule and over the skin
surface (Brooks 5850, mass flow controller, Emerson Electric, Hot-
field, PA). The vapor density of the effluent air was calculated from
the relative humidity and temperature measured using the Omega
HX93 humidity and temperature sensor (Omega Engineering, Stan-
ford, CT). Sweat rate was defined as the product of the difference in
water content between effluent and influent air and the flow rate. The
flow rate through the capsule was 1.0 l/min. The sweat rate value
was adjusted for skin surface area under the capsule (expressed in
mg·min⁻¹·cm⁻²).

Esophageal temperature (Tsa) was monitored continuously using a
pediatric esophageal temperature probe (Mon-a-therm, Mallinckrodt
Medical, St. Louis, MO) inserted through the nares to a depth
one-fourth of the standing height of the subject, whereby the tip of the
thermocouple is estimated to be at the level of the left atrium (30).
Skin temperature was recorded at 11 sites using 0.3-mm-diameter
T-type thermocouples integrated into heat-flow sensors (model FR-
025-TH44018-6, Concept Engineering, Old Saybrook, CT). The area-
weighted mean skin temperature (Tsk) and heat flux (HFsk) were
obtained by assigning the following regional percentages: head 6%,
upper arm 9%, forearm 6%, finger 2%, chest 19%, upper back 9.5%,
lower back 9.5%, anterior thigh 10%, posterior thigh 10%, anterior
calf 9.5%, and posterior calf 9.5% (13). Temperature data were
collected and digitized (data acquisition module model 3497A,
Hewlett-Packard) at 5-s intervals, displayed graphically in real time
and stored on hard disk (model PC-312, 9000, Hewlett-Packard).

The HDT maneuver was performed on a Teeter Hang Ups FS500
inversion table. The upper body only was supported by a meshed
cotton and nylon net secured to the upper frame of the tilt table. The
15° HDT position was measured using a Unitek Magnetic Polycast
Protractor (model 3310-036, Polytex, Empire Level Manufacturing,
Milwaukee, WI).

Experimental Protocol

Each subject performed a total of three experimental trials carried
out in random order. Experiments were separated by a minimum of
48 h during which subjects were instructed to avoid physical activity
and excessive stressors such as exposure to hot or cold temperatures,
particularly during the period between awakening and experimenta-
tion and during transit from home to the laboratory. Trials were
performed at the same time of day for each subject to avoid circadian
variation in Tsk and Tsa. Subjects were asked to fast at least 3 h before

J Appl Physiol • VOL 101 • SEPTEMBER 2006 • www.jap.org
experimentation, and water ingestion was permitted ad libitum during this time. On arrival at the laboratory, subjects clothed in shorts and athletic shoes were fitted with the appropriate instruments. All experimental trials were performed at an ambient temperature of 24.0 ± 0.5°C and a relative humidity of 45%.

After instrumentation, subjects remained resting for 10 min during which baseline measures were recorded. Subjects were then required to perform one of three experimental protocols. These were 1) 60 min in the URS posture followed by 60 min in the 15° HDT position; 2) 15 min of cycle ergometry at 75% of their predetermined VO2_peak followed by 60 min of recovery in the URS posture; or 3) 15 min of cycle ergometry at 75% of their predetermined VO2_peak followed by 60 min of recovery in the 15° HDT position. The tilt table was placed adjacent to the cycle ergometer to facilitate the transition from the bike to the tilt table after cessation of exercise. The average time to effect this transition was <30 s.

At the end of each experiment, CVCpeak was determined using a local heating protocol as described previously.

Statistical Analyses

A two-way ANOVA with repeated measures was used to analyze the data using the repeated factors of postexercise recovery time (levels: 2, 5, 8, 12, and 15 min and every 5 min until 60 min) and recovery mode (levels: 15° HDT and URS). The dependent variables employed were the changes from preexercise or baseline rest in Tes, Ta, HFsk, CO, HR, %CVCpeak, MAP, TPR, SV, and sweat rate. All values represent the means and standard deviation for seven subjects. The exercise and no-exercise sessions were analyzed separately to isolate the effect of recovery mode. For ANOVA main effects, Huynh-Feldt corrected statistics are reported where the assumption of sphericity was not met. Pairwise comparisons were performed using paired sample t-tests. The level of significance was set at an alpha level of 0.05. All analyses were performed using the statistical software package SPSS 12.0 for Windows (SPSS, Chicago, IL).

RESULTS

Tes Response

Exercise condition. The change in Tes at the end of exercise relative to preexercise rest was not significantly different between the URS and HDT trials (P = 0.235), with Tes elevated by 1.28°C (SD 0.29) preceding URS and by 1.18°C (SD 0.25) preceding HDT. During recovery, change in Tes from preexercise rest became less with postexercise recovery time [F(1,9,9,4) = 37.0, P < 0.001] and was influenced by recovery mode [F(1,5) = 10.1, P = 0.007]. The interaction between recovery mode and postexercise recovery time for change in Tes [F(2.8,14.1) = 4.7, P = 0.019] is demonstrated by the significantly lower elevation from preexercise rest in Tes during URS compared with HDT (P ≤ 0.05) after 15 min of postexercise recovery and for the remainder of the 60-min recovery period (Fig. 1A). Tes remained significantly (P = 0.006) elevated by 0.22°C (SD 0.12) above preexercise rest after 60 min of URS recovery, but it was 0.15°C (SD 0.08) below preexercise rest after 60 min of HDT recovery.

No-exercise condition. Without preceding exercise, change in Tes relative to resting values did significantly change with time [F(2.1,10.7) = 1.9, P = 0.198] or recovery mode [F(1,5) = 4.7, P = 0.082]; however, a significant interaction was apparent between time and recovery mode [F(1.8, 9.1) = 0.041] as demonstrated by a small (~0.1°C) but significantly (P ≤ 0.05) lower Tes during HDT compared with URS between 15 and 30 min.

Skin Temperature, Dry Heat Loss, Sweating, and %CVCpeak

Exercise condition. After exercise, the elevations in Tsk from preexercise rest became lower as postexercise recovery time progressed [F(1,6) = 25.8, P = 0.002], with a trend for the influence of recovery mode [F(1,6) = 5.3, P = 0.061]. The interaction between mode and time [F(2.8,16.8) = 5.8, P = 0.007] is evidenced by a significantly lower Tsk after 12 min of HDT recovery and for the remainder of the 60-min recovery period (P ≤ 0.05). At the end of the postexercise period, Tsk was 0.49°C (SD 0.53) lower after HDT compared with URS. Similarly, the elevations in HFsk from preexercise rest became lower with postexercise recovery time [F(1,6,9,4) = 168.9, P < 0.001]; however, no differences were observed between recovery modes [F(1,6) = 1.2, P = 0.316]. Sweat rate was significantly elevated above preexercise values before HDT and URS recovery trials (P ≤ 0.05) and then became reduced throughout postexercise recovery [F(5,3,31.8) = 554.6, P < 0.001]. Furthermore, sweat rate was influenced by recovery mode [F(1,6) = 20.3, P = 0.004], with a significantly greater sweat rate observed during HDT recovery relative to URS between 8 and 45 min postexercise (P ≤ 0.05; Fig. 1B). The
elevation in %CVC peak above preexercise rest after exercise before both HDT and URS recovery trials became lower with postexercise recovery time \( F(2,6,13.2) = 17.6, P < 0.001 \). Furthermore %CVC peak was influenced by recovery mode \( F(1,5) = 49.4, P = 0.001 \) with significantly greater values observed throughout the 60-min postexercise recovery period in the HDT position compared with URS (\( P \leq 0.05 \); Fig. 1C).

No-exercise condition. Without preceding exercise, changes in \( T_{sk} \), \( HF_{sk} \), %CVC peak, and sweat rate relative to resting values were not influenced by time or recovery mode (\( P > 0.05 \)).

Hemodynamic Responses

All hemodynamics data for all conditions are summarized in Table 1.

Exercise condition. The elevations in CO after exercise of 15.8 l/min (SD 2.9) before HDT and 16.5 l/min (SD 5.1) before URS were not significantly different (\( P = 0.818 \)). These elevations from rest in CO became lower during postexercise recovery \( [F(2,7,13.7) = 151.1, P < 0.001] \), but they were not influenced by recovery mode \( [F(1,5) = 0.005, P = 0.948] \) (Fig. 2A). The elevations in SV after exercise of 46.6 ml (SD 26.0) before HDT and 51.2 ml (SD 34.5) before URS were not significantly different (\( P = 0.852 \)). However, these elevations from rest in SV became lower with postexercise recovery time \( [F(3,2,15.8) = 27.3, P < 0.001] \) and were influenced by recovery mode \( [F(1,5) = 12.4, P = 0.017] \) with a significantly greater SV observed during HDT recovery throughout the 60-min postexercise recovery period (\( P < 0.05 \); Fig. 2B). After exercise, no significant difference was observed between the elevation in HR of 99.1 beats/min (SD 11.3) before HDT and 97.1 beats/min (SD 12.3) before URS recovery (\( P = 0.726 \)). HR elevation above rest became lower with postexercise recovery time \( [F(3,8,23.0) = 297.9, P < 0.001] \), and it was influenced by recovery mode \( [F(1,6) = 34.2, P = 0.001] \) with a significantly lower HR elevation observed during HR relative to URS throughout the 60-min postexercise recovery period (\( P \leq 0.05 \); Fig. 2C). The changes in MAP from preexercise rest after exercise recovery were not significantly different between HDT and URS (\( P = 0.376 \)). However, during postexercise recovery, change in MAP was different between recovery modes \( [F(1,5) = 27.1, P = 0.003] \), and it was influenced by recovery time \( [F(2,8,14.0) = 42.0, P = 0.001] \) with a significantly higher MAP observed during HDT between 2 and 50 min of postexercise recovery (\( P \leq 0.05 \); Fig. 3A). The changes in TPR from rest after exercise of \( -10.4 \text{ mmHg} \cdot \text{l}^{-1} \cdot \text{min}^{-1} \) (SD 1.7) before HDT and \( -10.5 \text{ mmHg} \cdot \text{l}^{-1} \cdot \text{min}^{-1} \) (SD 2.5) before URS were not significantly different (\( P = 0.834 \)). However, change in TPR from preexercise rest became less throughout postexercise recovery \( [F(5,2,25.8) = 18.0, P < 0.001] \), but it was not influenced by recovery mode \( [F(1,5) = 2.2, P = 0.200] \; \text{Fig. 3B}.\)

No-exercise condition. Without preceding exercise, changes in CO, MAP, and TPR relative to resting values were not influenced by time or recovery mode (\( P > 0.05 \)). The change in HR from resting values was significantly different between recovery modes \( [F(1,6) = 21.2, P = 0.004] \). HR was not influenced by recovery time \( [F(2,0, 12.1) = 2.7, P = 0.110] \); however, a significantly lower HR during HDT compared with URS was observed throughout recovery (\( P \leq 0.05 \)). A signifi-

![Table 1. Mean relative changes from baseline in cardiac output, stroke volume, heart rate, mean arterial pressure, and total peripheral resistance during the control trials preceded by no exercise.](http://jap.physiology.org/Downloadedfrom)

J Appl Physiol • VOL 101 • SEPTEMBER 2006 • www.jap.org
significant difference between recovery modes was also seen for changes in SV from resting values \( F(1,5) = 6.0, P = 0.050 \), with SV greater during HDT relative to URS between 2 and 50 min of recovery \( (P \leq 0.05) \).

**Sweat Rate and \%CVC_{peak} as a Function of \( T_{es} \)**

The change of sweat rate (Fig. 4) and CVC (Fig. 5) as a function of \( T_{es} \) after exercise show that after exercise the HDT intervention causes a significantly greater \%CVC peak after 5 min of postexercise recovery \( (P \leq 0.05) \) and a significantly greater sweat rate after 15 min of postexercise recovery. The combination of these increased heat loss responses contribute to a significantly lower \( T_{es} \) after 15 min of recovery in the HDT position \( (P \leq 0.05) \).

**DISCUSSION**

The most important observation from this study is that the manipulation of postexercise hemodynamics using HDT attenuates the fall of MAP, CVC, and sweat rate relative to the URS posture. The augmented MAP and heat loss responses in the HDT condition were also associated with an enhanced rate of \( T_{es} \) decay compared with the URS recovery posture. Thus HDT attenuates the reduction in heat loss responses and MAP in individuals recovering from dynamic exercise. These findings support previous work that employed LBPP to alter postexercise hemodynamics and heat loss (18) and further underscore a baroreceptor-mediated attenuation in heat loss responses during URS exercise recovery.

Our observation of an increased rate of \( T_{es} \) decay in the HDT position is consistent with the elevated heat loss responses of CVC and sweating throughout recovery. In recent studies examining postexercise heat loss responses, it has been shown

---

**Fig. 2.** Effect of upright seated recovery posture (○) and the 15° head-down tilt recovery position (△) after 15-min of cycle ergometry at 75% peak oxygen consumption on cardiac output (CO; A), stroke volume (SV; B), and heart rate (HR; C). *Significant difference between upright seated recovery, \( P \leq 0.05 \).

**Fig. 3.** Effect of upright seated recovery posture (○) and recovery in a 15° head-down tilt recovery position (△) after 15 min of cycle ergometry at 75% peak oxygen consumption on mean arterial pressure (MAP; A) and total peripheral resistance (TPR; B). *Significant difference between upright seated recovery, \( P \leq 0.05 \).

**Fig. 4.** Relationship of esophageal temperature with sweat rate during recovery from 15 min of cycle ergometry at 75% peak oxygen consumption in the upright seated posture (filled symbols) and 15° head-down tilt position (open symbols). *Significant difference between recovery positions at a given time point for sweat rate, \( P \leq 0.05 \). †Significant difference between recovery positions at a given time point for esophageal temperature, \( P \leq 0.05 \).
that there is a significant nonthermal contribution to the control of these responses. Studies employing recovery modes have attempted to elucidate the relative roles of nonthermal factors such as central command, mechanoreceptors, and baroreceptors in the modulation of heat loss responses during exercise recovery (3, 19, 33, 39). In toto these experiments concluded that during upright inactive recovery CVC is influenced predominantly by cardiopulmonary baroreceptors. Two studies exist, however, in men (19) and women (20) where different levels of MAP were observed between recovery modes, and therefore the role of the arterial baroreceptor population in heat loss response modulation cannot be ignored. Additional evidence also exists that attenuating the cardiopulmonary and arterial baroreceptor unloading effect associated with exercise recovery helps preserve the response of CVC (15, 18). Our present study differs from previous work in that we have studied the effect of HDT on prolonged recovery responses, whereas others have employed recovery modes (3, 19, 33, 39) or lower body pressure (18). The present study also supports a cardiopulmonary and arterial baroreceptor-mediated influence on postexercise CVC. This is supported by the fact that the cutaneous circulation is on the efferent arm of the baroreflex (4, 16). In our no-exercise condition, 15° HDT engaged the cardiopulmonary baroreflex as indicated by reflex bradycardia, an increase in SV, and no change in MAP. However, we noted a difference in MAP between HDT and the URS posture postexercise. Thus, whereas 15° HDT may isolate the cardiopulmonary baroreceptors under resting conditions, we observed that during exercise recovery HDT leads to an altered MAP response compared with URS recovery posture. This means that between postexercise recovery positions there are different degrees of arterial baroreceptor loading and thus we cannot rule out a possible role for the arterial baroreceptors in the observed CVC response.

The above-mentioned recovery mode studies demonstrated that sweat rate during postexercise recovery can be modulated by central command, mechanoreceptors, and possibly baroreceptors. Under resting conditions, some researchers did not observe decreases in sweating with baroreceptor unloading (37, 40), whereas others have observed a reduction in the thermosensitivity with baroreceptor unloading at rest (34) and during exercise (17, 29). There remains conflicting information regarding the possible baroreceptor influence on sweating during the postexercise period. Our data support a role for cardiopulmonary and/or arterial baroreceptors in the modulations of sweat rate as inferred by the hemodynamic responses to HDT. Under conditions of LBPP, there may be confounding influences on sweat rate such as air currents in the chamber or mechanoreceptor activation secondary to increased atmospheric pressure around the limbs. In our present study, we employed the HDT technique, thereby removing such possible confounders. We observed that when subjects recovered in the HDT position, sweat rate declined more slowly than during recovery in the URS posture, suggesting that baroreceptors are likely involved in the response. In contrast to previous work whereby applying LBPP stimulated sweat rate after restoring MAP (18), this study commenced the HDT intervention immediately postexercise. A study by Wilson et al. (39) concluded that factors other than thermal or baroreceptor loading status contribute to sweat rate during exercise recovery; however, their observations were limited to 5 min postexercise in the supine posture. We first noted a significant difference after 5 min of recovery. Thus it is possible that a role for baroreceptors in the modulation of postexercise sweat rate might be masked in the initial 5-min period and become more apparent later in recovery. Although speculative, our data support this possibility.

HDT tends to cause a shifting of blood headward by countering the hydrostatic pooling of blood seen in the URS posture (31). URS inactive recovery is thought to aid in the accumulation of blood in the venous system of the lower extremities in the absence of the muscle pump (2, 12, 26). This hemodynamic consequence may also contribute to postexercise hypotension (12). Our data indicate that without an exercise treatment, there were no significant effects on hemodynamic variables other than a decrease in HR and a concomitant increase in SV in the HDT recovery position relative to the URS posture. This hemodynamic response of SV is consistent with previous observations of resting subjects in the HDT position (10, 27, 31). However, these studies did not show a decrease in HR. The difference in our observations may be due to the fact that previous studies compared HDT with the supine posture and not the URS posture. Conversely, after exercise, we observed significant differences in SV, HR, and MAP between HDT and URS subjects. This supports the contention that HDT reduced the baroreceptor unloading effect of exercise recovery, thus under scoring the significant effect of HDT when performed after dynamic exercise.

In the URS posture reductions in CVC and sweat rate were observed despite a sustained elevation in T_{es}. This has been reported previously (15, 18, 23–25, 38) and suggests a possible resetting of the T_{es} relationship with CVC and sweat rate (see Figs. 4 and 5). When plotting the URS data with the responses observed in the HDT posture, the data points shift upward and to the left. This demonstrates that at a given T_{es}, the responses of CVC and sweat rate are greater in the HDT posture.

Our observation of an enhanced rate of T_{es} decay in the HDT condition is significant. Although recovery mode studies have
observed augmented heat loss responses, they have not reported significant changes in core temperature even when performed up to 20 min postexercise (19, 20, 33). Conversely, application of +45 mmHg LBPP was effective in increasing the rate of $T_{es}$ decline in URS subjects postexercise (18). The present study also reported a greater decrement in $T_{es}$ when HDT was applied postexercise. Moreover, HDT may have some practical significance as it is technically easier to perform than transferring subjects to a lower body pressure chamber. A logical extension to this study for future work includes the examination of the responses of severely hyperthermic individuals (core temperature $>39.5^\circ$C) to the HDT intervention. This will elucidate the effectiveness of HDT as potentially a more practical alternative for the active cooling methods currently recommended.

We conclude that during recovery from dynamic exercise, heat loss responses are compromised in the URS posture secondary to nonthermal baroreceptor influences. Specifically, application of HDT attenuates the reduction in CVC, sweat rate, and MAP typically observed postexercise. The augmented heat loss responses observed under the HDT position also resulted in an increased rate of $T_{es}$ decay relative to the URS recovery posture. HDT may therefore promote heat loss in hyperthermic individuals postexercise and provides many technical advantages over LBPP.

ACKNOWLEDGMENTS

This research was supported by a Discovery Grant from the Natural Sciences and Engineering Research Council of Canada (to G. P. Kenny).

REFERENCES


