Heat balance precedes stabilization of body temperatures during cold water immersion

Peter Tikuisis
Defence Research and Development Canada, Human Performance and Protection, Toronto, Ontario, Canada M3M 3B9

Submitted 26 December 2002; accepted in final form 5 March 2003

Tikuisis, Peter. Heat balance precedes stabilization of body temperatures during cold water immersion. J Appl Physiol 95: 89–96, 2003. First published March 14, 2003; 10.1152/japplphysiol.01195.2002.—Certain previous studies suggest, as hypothesized herein, that heat balance (i.e., when heat loss is matched by heat production) is attained before stabilization of body temperatures during cold exposure. This phenomenon is explained through a theoretical analysis of heat distribution in the body applied to an experiment involving cold water immersion. Six healthy and fit men (mean ± SD of age = 37.5 ± 6.5 yr, height = 1.79 ± 0.07 m, mass = 81.8 ± 9.5 kg, body fat = 17.3 ± 4.2%, maximal O2 uptake = 46.9 ± 5.5 l/min) were immersed in water ranging from 16.4 to 24.1°C for up to 10 h. Core temperature (Tco) underwent an insignificant transient rise during the first hour of immersion, then declined steadily for several hours, although no subject’s Tco reached 35°C. Despite the continued decrease in Tco, shivering had reached a steady state of 2 × resting metabolism. Heat debt peaked at 932 ± 334 kJ after 2 h of immersion, indicating the attainment of heat balance, but unexpectedly proceeded to decline at ~48 kJ/h, indicating a recovery of mean body temperature. These observations were rationalized by introducing a third compartment of the body, comprising fat, connective tissue, muscle, and bone, between the core (viscera and vessels) and skin. Temperature change in this “mid region” can account for the incongruity between the body’s heat debt and the changes in only the core and skin temperatures. The mid region temperature decreased by 3.7 ± 1.1°C at maximal heat debt and increased slowly thereafter. The reversal in heat debt might help explain why shivering drive failed to respond to a continued decrease in Tco, as shivering drive might be modulated by changes in body heat content.

Body cooling; shivering; heat debt; prediction; model

ACCIDENTAL IMMERSION IN COLD water is often fatal, increasing with a lowered water temperature and a diminished level of thermal protection. In extremely cold water, the body’s maximal rate of shivering heat production is exceeded by the rate of heat loss, and the body will progressively cool to ambient temperature. However, if the water is not sufficiently cold so that the individual can achieve a steady state of thermogenesis that balances the rate of heat loss, then survival time can be markedly extended (29). The development of heat balance and the evolution of body temperatures characteristic of this state is the subject of this paper. It is often assumed that heat balance is accompanied by the stabilization of body temperatures, as implied by the application of the “core-shell” concept (5). This concept suggests that changes in body heat content (∆BHC) can be deduced from changes in only the core (deep body) and skin temperatures. Previous investigators have already demonstrated that the thermometric determination of ∆BHC is not valid during non-steady-state changes in body temperatures (17, 23, 34). If, additionally, heat balance precedes the stabilization of body temperatures, then the thermometric determination of ∆BHC might be exaggerated, because further lowering of core temperature would suggest a contradictory continuance of heat debt.

This latter intriguing possibility presented itself during a previous investigations involving the immersion of women in 18°C stirred water (31). In that study, the subjects were immersed to the neck level in a bath calorimeter (to be described later), and complete thermometric and calorimetric data were obtained for seven of the subjects that lasted 90 min of immersion (mean ± SD of age = 24.0 ± 7.0 yr, mass = 65.3 ± 7.9 kg, height = 1.69 ± 0.06 m, and body fat = 24.1 ± 6.5%). Their rate of change in rectal temperature near the end of the immersion (about −0.7°C/h) was not synchronous with the diminishing rate of heat debt, which was within 2% of its estimated asymptotic value. As the skin temperature reached a mean steady-state value of −20.8°C after 15 min of immersion, these results suggest that the calorimetric and “core-shell” thermometric determinations of ∆BHC are incompatible and that the stabilization of body temperatures, specifically the core, lags heat balance.

The study described herein was undertaken to examine ∆BHC and changes in body temperatures during immersion in cold water of sufficiently long duration so that heat balance would be attained. It is hypothesized that heat balance (i.e., the convergence of heat production and heat loss) would precede the stabilization of body temperatures, and by implication ∆BHC cannot be ascertained thermometrically by assuming fixed weighting factors of only the core and...
skin temperatures during non-steady state, as previously surmised (17, 23, 34). Indeed, it is proposed that a third compartment of the body not fully represented by the core (deep body) and skin undergoes a compensatory change in temperature (i.e., an increase when deep body temperature is decreasing and skin temperature is nearly constant) to satisfy the constraint of heat balance. Herein we deduce the temperature change in this “mid region” through the principle of heat conservatism. Furthermore, it is understood that the temperatures of the mid region and core are not implied to be uniform or gradient free, but that they represent average values.

MATERIALS AND METHODS

Subjects and laboratory visits. Six fit, nonsmoking male subjects were recruited to participate in the study (protocol L-240), which was approved by the DRDC Toronto Human Ethics Committee. Subjects were fully informed of the procedures and risks according to the experimentation ethics guidelines and signed a volunteer consent form. Subjects reported to the laboratory on three occasions separated 1 wk apart for 1) medical clearance (with special attention given to health limitations for long-term cold water immersion including a 12-point ECG assessment) and baseline measurements, 2) a familiarization trial, and 3) the long-term immersion trial. The baseline measurements included the physical characteristics of mass, height, surface area (32), percent body fat (21), and maximum aerobic power (maximal oxygen uptake via treadmill running starting level at a moderate pace (−10 km/h) followed by 1% grade increases per minute until exhaustion), as summarized in Table 1.

During the second visit, the subjects underwent a familiarization trial with the laboratory setting and test procedures. Subjects were fully instrumented for immersion in cold water, and data were collected during the immersion. The period of immersion did not exceed 2 h subject to the constraints of the termination criteria (outlined below). This period was deemed sufficiently long to test the adequacy of the water temperature selected to ensure the attainment of heat balance. During the third visit, the subject underwent the same procedure as during the familiarization trial except that the water temperature might have been adjusted (explained below) and the duration would have exceeded 2 h.

Before each trial, subjects were required to abstain from alcohol and exercise for 48 h, follow an otherwise normal routine the night before, and consume a prescribed breakfast with an additional nutritional supplement (250 kcal Ensure beverage provided by the laboratory) during the morning of each trial. Normal caffeine consumption was not restricted before arrival to minimize the possibility of headaches due to caffeine withdrawal during the lengthy immersion period.

Strategy. Long-term immersion in cold water was accomplished by establishing a condition of cold stress that each subject could normally defend via the heat produced by shivering so that heat balance could be achieved. Hence, subjects were immersed head-out in a water-filled calormeter that was stirred and initially set at a predetermined temperature. This temperature was targeted to generate a high, but not maximal, level of shivering by using a model prediction that accounted for the subject’s anthropometric characteristics (26) and adjusted according to the subject’s response during the familiarization immersion. On the basis of the author’s previous work and the experience of others (14), it was hypothesized that core temperature would stabilize above 35°C provided that the subject’s shivering intensity was maintained.

Instrumentation. Before immersion, the subject dressed down to a bathing suit and self-inserted a rectal probe (Pharmaseal 400 Series, Baxter Healthcare, Valencia, CA) 15 cm past the anal sphincter for core temperature measurement. This was followed by the placement of 12 recalibrated (8) heat flux (HF) sensors (model FM-060, Concept Engineering, Old Saybrook, CT) for skin temperature and heat flux measurements and 3 polar ECG skin electrodes (ProPaq 100 Series, Protocol Systems, Beaverton, OR) for heart rate monitoring. One HF sensor was placed on the forehead, and its values of temperature and heat flux will be reported separately. The overall mean skin temperature and heat flux (including the head) were determined by using the Hardy-DuBois weighting formula (18). Respective mean-weighted values of the immersed portion of the body will be referred to as $T_{sk\text{-imm}}$ and $HF_{imm}$ (i.e., $T_{sk\text{-imm}} = \sum x_i T_{sk} / 0.93$, where $x_i$ is the Hardy-DuBois weight associated with the $i$th immersed site and 0.93 is the sum of all weights excluding the head). All temperatures and heat fluxes were recorded every minute and are reported in degrees centigrade and watts per meter squared, respectively.

Total body heat loss was determined, in part, by using the calorimetrically determined body heat loss (HI-cal, in W), which is deemed more accurate than using $HF_{imm}$, although both will be compared for methodological purposes. The water calometer has a capacity of 1,200 liters and was used in a passive mode to measure the amount of heat deposited to the water every 5 s, which was averaged every minute. The heat deposited was deducted from the temperature of the water, averaged from simultaneous readings at 10 evenly spaced locations within the calormeter (YSI Thermistors, Yellow Springs, OH), and the tare of the calormeter that was...

<table>
<thead>
<tr>
<th>Table 1. Subject characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject</td>
</tr>
<tr>
<td>---------</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td>5</td>
</tr>
<tr>
<td>6</td>
</tr>
<tr>
<td>Mean ± SD (G6)</td>
</tr>
<tr>
<td>Mean ± SD (G4)</td>
</tr>
</tbody>
</table>

HT, height; WT, mass; SA, body surface area; BF, fatness; $V_{O2\text{max}}$, maximal aerobic power; $T_w$ and $T_m$, average water and air temperatures during immersion, respectively; $T_{imm}$, immersion time.
measured for 30 min before and 30 min after the immersion. The air temperature was adjusted to ~2°C below the water temperature to minimize any increase in water temperature.

Open-circuit indirect calorimetry was used to determine oxygen uptake and carbon dioxide production during the preimmersion resting and immersion periods. During monitoring, the subject was continuously connected to a mouthpiece except for 1 min at the end of each hour when drinking water was provided. The expired gases were directed through a breathing valve and hose assembly to a 5-liter mixing box connected in series to a ventilation module that measured the expired ventilation rate (VMM ventilation measurement module, Interface Associates, Irvine, CA). A sample line directed gases from the mixing box to oxygen and carbon dioxide analyzers (respective AMETEK models S-3A11 and CD-3A, Applied Electrochemistry, Paoli, PA). The oxygen consumption and the respiratory exchange ratio were determined every minute by using nonprotein oxidation formulas to calculate energy expenditure (19) and averaged every 15 min (the average during the first 15 min of each hour excluded the initial 3 min to allow for sufficient system flushing after the mouthpiece was reconnected).

Immersion protocol. After instrumentation, the subject lay down covered with a blanket on the calorimeter bed support for 30 min under a thermoneutral condition. A resting metabolic rate was determined during the last 10 min, and a finger prick blood sample was taken for blood glucose monitoring (One Touch II blood glucose monitoring system, Lifescan, Mitpitas, CA). While still on the bed support, the subject was then transferred into the water calorimeter in a semisupine position to the neck level until one of the termination criteria was met. The subject was provided with 3 ml water/kg lean body mass at 36°C during the last 10 min of each hour during cooling (1 min in this case). All heat loss variables and the metabolic rate are reported in watts, and the heat debt is reported in kilojoules (3.6 kJ = 1 W·h).

Determination of \( \Delta T_{\text{mid}} \). The concept of heat implies that any heat debt incurred by the body must equal the sum of products of the negative temperature changes (\( \Delta T \), in °C) in all body components and their corresponding heat capacities and masses, as follows

\[
\text{debt} = -WT \cdot \sum f_i \cdot c_i \cdot \Delta T_i
\]  

where WT is the body mass (in kg), and \( f \) and \( c \) are the whole body fraction and heat capacity (kJ·kg\(^{-1}·\)°C\(^{-1} \)) of the \( i \)th component, respectively (note that the negative sign coupled with a decrease in component temperature yields a loss of heat, i.e., heat debt). In the present application, it is assumed that the mid region comprises all components of the body not fully represented by the core and skin, that is, fat, connective tissue, muscle, and bone. The latter three components are collectively abbreviated as "cmb." Thus the change (\( \Delta \)) in the temperature of the mid region of the body (\( T_{\text{mid}} \)) is predicted by

\[
\Delta T_{\text{mid}} = \frac{-\text{debt}/WT - f_{sk} \cdot c_{sk} \cdot \Delta T_{sk} - f_{co} \cdot c_{co} \cdot \Delta T_{co} - f_{cmb} \cdot c_{cmb}}{f_{fat} \cdot c_{fat} + f_{cmb} \cdot c_{cmb}}
\]  

where the skin (including the head) is denoted by "sk" and the core ("co") comprises the blood vessels and viscera. Equations 3 stipulates that the temperature change in the mid region is negative (i.e., becomes cooler) if the heat debt exceeds the amount accountable by the temperature decreases in the core and skin alone, and vice versa. Table 2 shows the tissue fractions and heat capacities of the various body components, as adopted from Werner and Buse (36).

The component fractions of Eq. 2 are thus calculated as

\[
f_{fat} = \frac{\%BF}{100}
\]

\[
f_{sk} = 0.062 \cdot (1 - f_{fat})
\]

\[
f_{co} = 0.159 \cdot (1 - f_{fat})
\]

\[
f_{cmb} = 0.779 \cdot (1 - f_{fat})
\]

and the heat capacity of the nonfat mid region (3.26 kJ·kg\(^{-1}·\)°C\(^{-1} \)) was calculated from the fractional contributions of its components

\[
c_{cmb} = \frac{0.080 \cdot c_{connective \ tissue} + 0.521 \cdot c_{muscle} + 0.178 \cdot c_{bone}}{0.779}
\]

*J Appl Physiol* • VOL 95 • JULY 2003 • www.jap.org
Table 2. Component fractions and heat capacities of the nonfat portion of the body, and the heat capacity of fat

<table>
<thead>
<tr>
<th>Component</th>
<th>Nonfat Fraction, %</th>
<th>Heat Capacity, kJ·kg⁻¹·°C⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>6.2</td>
<td>3.7</td>
</tr>
<tr>
<td>Connective tissue</td>
<td>8.0</td>
<td>3.2</td>
</tr>
<tr>
<td>Muscle</td>
<td>52.1</td>
<td>3.8</td>
</tr>
<tr>
<td>Bone</td>
<td>17.8</td>
<td>1.7</td>
</tr>
<tr>
<td>Vessels</td>
<td>1.8</td>
<td>3.6</td>
</tr>
<tr>
<td>Viscera</td>
<td>14.1</td>
<td>3.6</td>
</tr>
<tr>
<td>Mid region</td>
<td>[77.9]</td>
<td>[3.3]</td>
</tr>
<tr>
<td>Core region</td>
<td>[15.9]</td>
<td>[3.6]</td>
</tr>
<tr>
<td>Fat</td>
<td>2.3</td>
<td></td>
</tr>
</tbody>
</table>

The fractions of the mid and core regions shown in square parenthesis are the sums of fractions of the connective tissue, muscle, and bone, and of the vessels and viscera, respectively. The heat capacity of the nonfat mid region is determined by using Eq. 8; that of the core is common to the vessels and viscera.

It is also noted that \( c_f \), decreases with increasing body fatness of the subject because of the relatively low heat capacity of fat (Table 2).

Data analysis. The data were averaged every 15 min and analyzed by using a one-way analysis of variance for repeated measures (Statistica, StatSoft, Tulsa, OK) to determine a main effect of time in body temperatures, sensations, reasoning, metabolic rate, heat loss, and heat debt with acceptance at \( P < 0.05 \). A Newman-Keuls means-comparison test criterion of \( P < 0.05 \) was applied for post hoc analyses where main differences were found. Regression analysis was also conducted on the mean results and on certain individual subject responses to verify significant trends over time. Specifically, slope tests with acceptance at \( P < 0.05 \) were conducted on data leading to an obvious inflection point or on data that were isolated to determine at what point the slope was no longer significant. Where indicated, means are reported with ±SD.

RESULTS

Table 1 summarizes the characteristics and test conditions of all subjects (G6), who lasted a minimum of 4 h of immersion, and of the four subjects (subject 3–6; G4) who lasted at least 5.5 h. The water temperature \( (T_w) \) increased by 0.9 ± 0.6°C over the entire duration of immersion for all subjects. Average values of water and air temperature ranged from 16.4 to 24.1°C (mean of 19.4°C for all subjects) and from 15.8 to 22.3°C (mean of 17.6°C), respectively. Immersion times ranged from 4 to 10 h and averaged 6.3 ± 2.1 h for all subjects. Hypoglycemia was not a factor in the shivering response (12) of the subjects because no individual blood glucose value was <2 mmol/l. All other measured physiological variables underwent significant changes over the period of immersion analyzed and presented (4 and 5.5 h for G6 and G4, respectively).

As expected, the subjects’ rating of tiredness and cold sensation increased over time; following are the results for G6 only. Tiredness increased from 0.7 ± 0.8 (between not and barely tired) at the start of immersion to 3.7 ± 2.7 (between somewhat and moderately tired) after 4 h of immersion. Similarly, cold sensation increased from 3.3 ± 0.8 (between cool, but comfortable and uncomfortably cool) to 6.3 ± 1.5 (between cold and very cold). Reasoning, on the other hand, did not change over 4 h of immersion. Neither the number of true/false questions answered (average of 32.5 ± 0.4 in 2 min) nor the percentage of correct responses (81.2 ± 2.2%) was affected.

Figure 1 shows the mean changes in the subjects’ core temperature and immersed skin-water temperature difference. Core temperature displayed a small, although insignificant, initial transient rise over the first hour followed by continuous declines of 0.32 and 0.34°C/h for G6 and G4 (up to 195 min), respectively. The change for G4 after 195 min of immersion, although still negative (−0.06°C/h), was not significant. Core temperature did not drop below 35°C in any of the subjects, including subjects 3 and 5 who tolerated the immersion for 10 and 7.5 h, respectively.

The mean skin-water temperature difference of the immersed body reached a near steady state after 15 min of immersion. It decreased modestly, but significantly, from 1.80 ± 0.43°C at 30 min to 1.32 ± 0.35°C at 240 min for G6, and from 1.95 ± 0.47°C at 30 min to 1.31 ± 0.24°C at 330 min for G4 (the decrease after 60 min of immersion was −0.074 and −0.079°C/h for G6 and G4, respectively, based on regression). Given that the water temperature increased by 0.49 ± 0.35°C and 0.96 ± 0.38°C for G6 and G4, respectively, it turns out that skin temperature rose slightly (0.32°C in absolute terms) during the latter portion of the immersion for G4 only. Although this change is accounted for in the analysis of heat balance, it would not have measurably affected the thermoregulatory response. Calculation of
with the development of a steady level of water disturbance, and averaged $320 \pm 15$ and $347 \pm 22$ W for G6 and G4, respectively. Heat loss decreased rapidly from $778 \pm 209$ and $857 \pm 217$ W during the first 15 min of immersion to $317 \pm 69$ and $357 \pm 62$ W between 45 and 60 min for G6 and G4, respectively. Thereafter, heat loss continued to decrease more slowly, but significantly at respective reductions of 18.7 and 15.0 W/h. The contributions of heat loss from the immersed portion of the body (HL\_cal), from the head (HL\_head), and from respiration (Q\_resp) were $87.0 \pm 2.5$, $4.6 \pm 0.7$, and $8.4 \pm 1.8\%$, respectively, of the total body heat loss.

Heat loss peaked after 120 min of immersion, reaching $932 \pm 334$ and $1,017 \pm 397$ kJ for G6 and G4, respectively. Thereafter, heat debt proceeded to decline moderately, but significantly, at respective reductions of 48.2 and 33.8 kJ/h, indicating a recovery of body heat. This unexpected reversal is the result of a significant decline in body heat loss whereas the metabolic rate remained unchanged.

Figure 3 shows the predicted mean temperature change in the mid region of the body according to Eq. 3. The initial rapid decreases of $3.69 \pm 1.14$ and $3.98 \pm 1.30\degree C$ after 120 min of immersion for G6 and G4, respectively, are followed by reversals and small but significant increases thereafter. Not surprisingly, this pattern largely mirrors the change in heat debt. Essentially, the mid region temperature initially decreased because of an increasing heat debt that could not be accounted for by the decreases in core and skin temperatures alone when a fixed value of “a” was assumed.

Fig. 3. Mean ± SD predicted temperature change in the mid region of the body (\(\Delta T\_mid\)) and the value of “a” (see text) pertaining to the core-shell representation of the body corresponding to the change in body heat content.
However, the mid region temperature was predicted to recover with the reversal of heat debt, and this recovery was further enhanced by the continued decrease in core temperature. Also shown in Fig. 3 is the predicted value of “a” according to Eq. 11 if the change in body heat content was to be explained by changes in only the core and skin temperatures. The “a” decreases initially to a minimum value of 0.61 after 75 min of immersion and increases thereafter returning close to its assumed initial value of 0.7.

Following is a detailed analysis of the results of the two subjects (subjects 3 and 5) that lasted the longest periods of immersion (10 and 7.5 h, respectively). Although both subjects indicated significant increases in cold sensation, only subject 3 expressed an increase in tiredness, and, interestingly, neither subject’s reasoning ability was compromised throughout their immersions. These results are presented, in part, to illustrate the wide variation in thermoregulatory response and body cooling that help to explain the variability seen in Figs. 1–3. For example, subject 3 had a lower metabolic rate due to shivering (even when normalized for body mass or surface area) despite having a lower core temperature than subject 5 and an increasing heat debt (reaching 1,160 kJ after 10 h of immersion). In contrast, the heat debt of subject 5 peaked at 900 kJ after 75 min of immersion and subsequently decreased to 370 kJ at 7.5 h.

Although both subjects had similar skin-water temperature differences, their actual immersed skin temperatures were quite different because of the different water immersion temperatures (Table 1). Mean weighted skin temperatures (including the head) were 25.8 and 21.1°C for subjects 3 and 5, respectively, after the first 15 min of immersion. Respective core temperatures were 35.93 and 36.97°C, and, according to Tikuisis and Giesbrecht (30), the predicted metabolic rates due to shivering (i.e., above resting values) are 134.6 and 73.0 W/m². The subjects’ respective metabolic rates at rest were 50.3 and 61.4 W/m². When coupled with the metabolic rates of 136.9 and 186.1 W/m² measured during the immersion, subjects 3 and 5 respectively shivered 48% less and 52% more than expected. This analysis helps explain why subject 3 incurred a continuous heat debt during the immersion that led to a decreasing mid region temperature whereas subject 5 recovered heat. This variability in individual responses to cold exposure is not unique (24, 28) but illustrates the complexity and challenge involved in the development of prediction models of survival for cold exposure (25, 26).

DISCUSSION

The present study is consistent with certain findings of Hayward and Keatinge (14), who reported on the lowest water temperatures in which immersed individuals’ core temperatures were stabilized. They reported a mean critical water temperature of 20.8 ± 6.1°C for seven male subjects [body fatness = 17.1 ± 2.5%, estimated from body density (9)]. The mean critical water temperature estimated for the subjects of the present study, on the basis of the regression of the Hayward and Keatinge results, is 20.6 ± 4.9°C. That this latter value is higher than the actual mean water temperature used (19.4 ± 2.6°C) can be explained by the absence of additional cold stress that Hayward and Keatinge imposed on their subjects by spraying their heads with water to simulate a more complete immersion. These investigators also reported a mean rectal temperature of 35.9 ± 0.4°C at stabilization and that no individual value dropped below 35°C, as in the present study. However, without any confirmatory measurement of heat loss, they assumed that heat balance was attained when the rectal temperature stabilized (immersion times were not provided), which is contrary to our findings that indicate heat balance precedes temperature stabilization.

It is evident that heat balance for the entire group of subjects was attained at ~120 min of immersion whereas rectal temperature continued to decline (Fig. 2). Although it is known (23) that rectal temperature usually lags other core temperature sites (e.g., esophageal), the lag is far less than the period of time that rectal temperature continued to decrease after heat balance was attained. Furthermore, the correspondence of rectal temperature to the change in body heat content is acceptable when that change is relatively slow (23), as was the case after heat balance. Thus the hypothesis that heat balance would occur before the stabilization of body temperatures is supported, and it concurs with the trend noted in the earlier study involving the immersion of women in 18°C water (31). Yet heat balance was only temporary as heat debt began to decrease, which is all the more remarkable and seemingly contradictory with the continued decrease in core temperature. This, however, underscores the presence of an evolving compensatory temperature change throughout the mid region and, as demonstrated below, its temperature (taken as an average) remained below that of the core.

In Fig. 4, we compare the core and mid region temperatures of G6, assuming that the latter value started at 36°C. This illustration suggests that these temperatures would asymptotically approach respective values of ~36 and 33°C, which concurs with the expectation that core temperature should remain above mid

![Fig. 4. Mean ± SD of the Tₐₚ (solid line) and predicted Tₘₐ₈ (dashed line) for G6.](http://jap.physiology.org/DownloadedFrom)
region temperature. It is instructive to verify this further by examining the consequence of under- or over-estimating the core temperature. Reanalysis of the data assuming that the core temperature was 1°C higher or lower than the measured rectal temperature led to respective $T_{\text{mid}}$ values of 0.19°C lower or higher than shown in Fig. 4. The smallness of this correction is due to the considerably larger mass of the mid region compared with the core over which heat is distributed. That is, the ratio of the net heat capacities (in kJ/°C) of the core to the mid region is 0.19 for both G6 and G4, identical to the ratio of a temperature correction, if required. Thus, given the low sensitivity of the mid region temperature to a correction in core temperature, the qualitative aspects of this study are not affected even if the core temperature was misrepresented by the rectal measurement.

Webb (33) measured various body temperatures of six seminude men exposed to 15°C air for 2–3 h and reported a mean decrease of 3.9°C in the subcutaneous tissue temperature at six different body locations and of 2.5°C in muscle tissue at two sites (back and thigh) at 2 and 4 cm depth. Despite the differences in exposure conditions, these temperature changes are similar to the decrease in the mid region deduced in the present study (Fig. 4). This similarity, however, is not unreasonable given that $T_{\text{mid}}$ represents the average value over the entire mid region, from cold subcutaneous tissue to the warmer deep bone and musculature. The extrapolation of the core and mid region temperatures shown in Fig. 4 to their asymptotes corresponds to a steady-state heat debt of $\sim$860 kJ. Of course, the continuing evolution of heat distribution and body temperatures well beyond 4 h of immersion, as described earlier for subjects 3 and 5, dispel any certainty of predicting individual thermoregulatory responses.

The redistribution of heat in the body and consequent evolution of regional body temperatures is apparently a dynamic process that characterizes the entire duration of the immersion. Thus the concept of a core-shell representation of the body’s heat content is inadequate for immersion in cold water lasting at least 10 h. Interestingly though, the value of “a” (Eq. 11) deduced at the end of the immersion for G6 and G4 (Fig. 3) agrees closely with the value of 0.67 suggested by Bittel (2). However, this must be viewed as coincidental because the end of immersion did not represent a steady-state condition, in which a value closer to 0.75 might be expected (34).

Among others, Livingstone (17) pointed out the inadequacy of the core-shell concept to account for the non-steady-state distribution of heat within the body. Livingstone concluded that the parameter “a” (Eq. 11) must be continually adjusted to reflect the true change in mean body temperature, as confirmed in Fig. 3. Webb (34) also concluded that the thermometric determination of $\Delta BHC$ using a fixed value of “a” is only valid with properly timed measurements of core and skin temperatures. Using cooling data of 1 h duration, Webb calculated an average value of 0.65 for “a” with large variability, which agrees with the present finding. Examining data of cold air exposure, Tikuisis (27) suggested the addition of a mid region to align the thermometric and partitional calorimetric determinations of heat debt. The results of the present study involving lengthy cold water immersion concur with these earlier conclusions.

Finally, the shivering response of the subjects in this study was consistent with another study (29) that examined shivering endurance during cold water immersion. That study found that subjects tended to shiver at a steady (albeit irregular) rate despite a continued decrease in core temperature. It was postulated that this exhibited a desensitization and fatigue of the shivering drive to cold. The present finding of steady-state shivering after 60 min of immersion in cold water with a continued decrease in core temperature supports this postulate.

However, there is another possible interpretation, that shivering did not increase because of a decrease in heat debt. Perhaps it is not coincidental that, as heat debt approached its peak value (Fig. 2), shivering had already achieved a high steady-state value. It is conceivable that any drive to increase shivering further because of a continued decrease in core temperature might have been modulated by a decreasing heat debt. This suggests that thermoregulation might be governed by more than just changes in core and skin temperatures, specifically that changes in body heat content are involved. This concept was previously proposed by Glaser and Newling (11), who stated that “essential mechanisms of temperature regulation are concerned with a constant thermal balance, not a constant deep body temperature.” In their study involving short-term mild cooling, they concluded that maintenance of thermal balance is a fundamental property of temperature control in humans, whereas the deep and superficial temperatures may vary considerably. Webb (35) substantiated this view on the basis of numerous calorimetric studies and concluded that “changes in body heat content drive deep body temperatures.” The hypothesis that heat balance precedes the stabilization of body temperatures is consistent with these views.

In an early review paper, Hammel (13) postulated that specialized temperature transducers are located at many sites on the surface of the body and within it, which can be viewed as a variant of the concept of heat content regulation. Hammel also stated that uncertainty arises over which body temperatures are being transduced and acted on to generate a thermoregulatory response. Additional support of this concept was expressed by Snellen (22), who proposed that mean body temperature is regulated, which is essentially equivalent to heat content regulation, as decreases in mean body temperature are proportional to heat debt. Yet Cabanac (6) refuted this concept because of a lack of experimental verification involving cold stress. Indeed, the regulation of heat content alone cannot explain why shivering intensity plateaued after 1 h of immersion in the present study whereas heat debt continued to increase for another hour and then decrease thereafter. Therefore, whether desensitization...
of shivering actually occurs within a few hours of cold water immersion, or shivering drive is modulated by changes in body heat content, or whether both are factored concurrently remains an open question that requires further investigation.

The author gratefully acknowledges the expert assistance of Allan Keefe, who was primarily responsible for conducting the experiments, from subject recruitment to data acquisition. I am also grateful for the kind assistance of several members of the Human Protections and Performance group including Dr. Michel Ducharme, Robert Limmer, and Jan Pope, and for the medical supervision of Dr. Cyd Courchesne. Finally, I especially thank all the subjects for their willingness to endure the rather stressful cold condition that made this study possible.

This study was supported by DND trust 2c101.

REFERENCES