

**Thea Pretorius, Gerald K. Bristow, Alan M. Steinman and Gordon G. Giesbrecht**  
*J Appl Physiol* 101:669-675, 2006. First published Apr 13, 2006; doi:10.1152/jappphysiol.01241.2005

**You might find this additional information useful...**

---

This article cites 24 articles, 10 of which you can access free at:

<http://jap.physiology.org/cgi/content/full/101/2/669#BIBL>

Updated information and services including high-resolution figures, can be found at:

<http://jap.physiology.org/cgi/content/full/101/2/669>

Additional material and information about *Journal of Applied Physiology* can be found at:

<http://www.the-aps.org/publications/jappl>

---

This information is current as of October 29, 2008 .

## HIGHLIGHTED TOPIC | *A Physiological Systems Approach to Human and Mammalian Thermoregulation*

### Thermal effects of whole head submersion in cold water on nonshivering humans

Thea Pretorius, Gerald K. Bristow, Alan M. Steinman, and Gordon G. Giesbrecht

Laboratory for Exercise and Environmental Medicine, Health, Leisure and Human Performance Research Institute, University of Manitoba, Winnipeg, Canada

Submitted 29 September 2005; accepted in final form 24 March 2006

**Pretorius, Thea, Gerald K. Bristow, Alan M. Steinman, and Gordon G. Giesbrecht.** Thermal effects of whole head submersion in cold water on nonshivering humans. *J Appl Physiol* 101: 669–675, 2006. First published April 13, 2006; doi:10.1152/jappphysiol.01241.2005.— This study isolated the effect of whole head submersion in cold water, on surface heat loss and body core cooling, when the confounding effect of shivering heat production was pharmacologically eliminated. Eight healthy male subjects were studied in 17°C water under four conditions: the body was either insulated or uninsulated, with the head either above the water or completely submersed in each body-insulation subcondition. Shivering was abolished with buspirone (30 mg) and meperidine (2.5 mg/kg), and subjects breathed compressed air throughout all trials. Over the first 30 min of immersion, exposure of the head increased core cooling both in the body-insulated conditions (head out:  $0.47 \pm 0.2^\circ\text{C}$ , head in:  $0.77 \pm 0.2^\circ\text{C}$ ;  $P < 0.05$ ) and the body-exposed conditions (head out:  $0.84 \pm 0.2^\circ\text{C}$  and head in:  $1.17 \pm 0.5^\circ\text{C}$ ;  $P < 0.02$ ). Submersion of the head (7% of the body surface area) in the body-exposed conditions increased total heat loss by only 10%. In both body-exposed and body-insulated conditions, head submersion increased core cooling rate much more (average of 42%) than it increased total heat loss. This may be explained by a redistribution of blood flow in response to stimulation of thermosensitive and/or trigeminal receptors in the scalp, neck and face, where a given amount of heat loss would have a greater cooling effect on a smaller perfused body mass. In 17°C water, the head does not contribute relatively more than the rest of the body to surface heat loss; however, a cold-induced reduction of perfused body mass may allow this small increase in heat loss to cause a relatively larger cooling of the body core.

hypothermia; heat loss; submersion; perfused body mass; thermal model; symptomless hypothermia; thermal core; cold-water near drowning

MANY RECREATIONAL, COMMERCIAL, and military activities involve cold water and the possible development of accidental hypothermia. Several studies (6, 11, 14, 21) have addressed the effect of cold-water immersion on the rate of body core cooling. The initiation and degree of hypothermia are related to many variables, including water temperature, insulation, duration of exposure, and the amount of body surface area (BSA) exposed to the water. The effect of whole head cold-water submersion on core cooling is unknown.

One hypothesis predicts a substantial heat loss through the head due to the great amount of surface blood flow in the scalp and because scalp blood vessels do not vasoconstrict in response to cold as do surface vessels in other body areas (8). An alternative hypothesis predicts minimal heat loss from the head because submersion of the head and neck would only involve 7–9% more of the body surface area (20). As well, mathematical modeling predicts minimal conductive heat loss directly through the scalp and skull (27). This topic has important practical implications for conditions where the whole head is completely submersed in cold water, such as self-contained underwater breathing apparatus (SCUBA) diving, and would also be relevant to cerebral protection during cold-water near drowning (3).

A few studies on core cooling have addressed whole head cooling in animals exposed to cold water (6) and in humans exposed to cold air (8, 24). Human studies involving cold-water immersion, however, have only included partial exposure of the dorsal head. In general, dorsal head immersion in cold water has little effect on core temperature when the remainder of the body is not cold stressed. However, when the body is also exposed to cold water, additional immersion of the dorsal head has increased core cooling by 250%<sup>1</sup> in 1–2°C water (1), 87% in 10°C water (21), and 39% in 12°C water (9).

The latter study (9) was the first head-immersion protocol to measure surface heat loss while eliminating the confounding effect of shivering heat production with meperidine (Demerol). Surprisingly, heat loss from the immersed dorsal head was not proportionately greater than for other body areas. Nevertheless, the 39% increase in core cooling rate during dorsal head immersion was proportionately much greater than the 10% increase in surface of heat loss from the dorsal head and upper chest. This exaggerated core cooling rate was proposed to result from the increased heat loss affecting a smaller thermal core because of intense thermal stimulation of the dorsal head

<sup>1</sup> These studies on prisoners of war in Dachau during World War II were grossly unethical, and the results are often considered invalid and unusable because of the emaciated condition of the prisoners as well as questions regarding the protocol and accuracy of the results.

Address for reprint requests and other correspondence: G. Giesbrecht, 211 Max Bell Centre, Univ. of Manitoba, Winnipeg, Canada R3T 2N2 (e-mail: giesbrec@cc.umanitoba.ca).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

with resultant peripheral vasoconstriction and reduced perfused body mass (i.e., thermal core) (26).

In toto, results from dorsal head immersion studies support this explanation because an increased thermal stimulus (i.e., water temperature decreasing from 12 to 1°C) would further reduce the thermal core, thus increasing core cooling when the dorsal head was immersed. The comparative effect of whole head submersion is unknown. Additional submersion of the face would not only increase the thermal stimulus for vasoconstriction but, through trigeminal nerve afferents, would also stimulate the human dive response, a mechanism that conserves oxygen by inducing bradycardia and peripheral vasoconstriction (5, 16).

The purpose of this study was to expand on previous work (9, 21) by isolating the thermal effects of whole head cooling where shivering heat production was eliminated by meperidine and buspirone (23). Specifically, we wished to determine whether whole head submersion would affect core cooling only when the rest of the body was cold exposed and to confirm the disparity between the relative increase in heat loss (with head submersion) and a disproportionately greater increase in core cooling.

As in our laboratory's previous study (9), increased heat loss from the submersed head was not expected to be disproportionately greater than its relative surface area. It was also hypothesized that, because of the extra facial stimuli during cold exposure, whole head submersion would increase the core cooling rate in both the body-exposed as well as body-insulated conditions. Finally, in the body-exposed conditions, the relative increase in core cooling caused by head submersion was expected to be greater than seen previously with dorsal head cooling.

## METHODS

### Subjects

The experimental protocol was approved by the University of Manitoba Education/Nursing Research Ethics Board. Eight volunteer male subjects, each of whom provided written, informed consent and proof of SCUBA diving certification, were selected for the study. These subjects were both mentally and physically healthy, they had no significant medical history, and none had male-pattern baldness. They completed a medical (Physical Activity Readiness Questionnaire) questionnaire to screen for cardiorespiratory disease and other conditions that could be exacerbated by exposure to cold water. They were studied on four separate occasions, at least 48 h apart and at the same time each day to control for circadian effects. Abstinence from alcohol, tobacco, and strenuous exercise for 12 h before the study was requested. They were also asked to only consume a light meal before coming to the laboratory to minimize any potential nausea caused by meperidine infusion. Compliance to these requests was confirmed before each trial.

Height, weight, age, and skinfold thickness at four sites were measured; percent body fat was calculated based on body density, estimated from the sum of four skinfolds (2). Anthropometric data for the subjects are shown in Table 1.

### Instrumentation

For each trial, subjects wore a swimsuit while being instrumented in a room at an ambient temperature of 22°C. Core temperature was measured by a thermocouple in the esophagus ( $T_{es}$ ) at the level of the cardiac atria. This site has previously been shown to provide the closest correlation to intracardiac temperature (15). Single-channel

Table 1. Descriptive data for eight subjects

Subject No.	Age, yr	Height, cm	BSA, m <sup>2</sup>	Mass, kg	Sum of Four, Skinfolds, mm	Body Fat, %
1	46	183	2.08	86.0	316	18.0
2	35	184	2.13	90.0	311	22.6
3	22	182	2.00	78.5	286	17.0
4	34	173	1.92	78.0	289	25.0
5	31	176	2.25	110.0	322	30.8
6	33	182	2.39	120.0	341	30.2
7	42	178	2.29	113.0	340	26.3
8	31	173	1.92	78.0	290	12.8
Mean	34.3	178.8	2.12	94.2	311.8	22.8
SD	7.3	4.6	0.2	17.4	22.3	6.5

BSA, body surface area.

electrocardiogram and heart rate were also monitored for the duration of each trial and recorded at 30-s intervals with the metabolic information. An intravenous line was introduced into a hand vein for drug and/or saline administration. A pulse oximeter probe was affixed to a finger to monitor arterial oxygen saturation.

Cutaneous heat flux (W/m<sup>2</sup>) and skin temperature (°C) were measured from 12 sites (listed below) using thermal flux transducers (Concept Engineering, Old Saybrook, CT) according to our laboratory's standard procedures (11). BSA was calculated as follows: area (m<sup>2</sup>) = weight<sup>0.425</sup> (kg) · height<sup>0.725</sup> (cm) · 0.007184. The following regional percentages were assigned based on Layton et al. (20): forehead 4%, dorsum of the head 3%, chest 8.75%, abdomen 8.75%, back 17.5%, forearm 9%, back of the upper arm 7%, front of the thigh 9.5%, back of the thigh 9.5%, front of the lower leg 6.5%, back of the lower leg 6.5%, and top of the foot 7%. A light mesh hood was used to hold the dorsal head transducer snugly against the hair on the back of the head. Flux was defined as positive when heat traversed the skin toward the environment (i.e., heat loss) and values for each transducer (W/m<sup>2</sup>) were converted into watts per region as follows:

$$\text{Flux}_{\text{region}} (\text{W}) = \text{transducer flux (W/m}^2) \cdot$$

$$\text{BSA (m}^2) \cdot \text{regional percent} \cdot 0.01$$

Oxygen consumption ( $\dot{V}O_2$ ) was measured with an open circuit from expired minute volume and inspired and mixed expired gas concentrations sampled from a mixing box ( $V_{\text{max}}$  229 by Sensormedics). Because subjects were completely submersed in half of the trials, they breathed dry compressed air through baseline and immersion/submersion in all trials; the compressed-air tank remained outside the tank and was therefore not cooled. To facilitate metabolic measurements, a standard SCUBA regulator (Blizzard, Sherwood, Lockport, NY) was modified and connected to corrugated tubing so that all expiratory gas could be collected by the metabolic system. Metabolic measurements of respiratory activity conducted in hyperbaric conditions but analyzed at normobaric conditions will overestimate ventilatory parameters but will accurately measure metabolic variables (i.e.,  $\dot{V}O_2$ ).

$\dot{V}O_2$  and respiratory exchange rate (RER) were measured to calculate metabolic rate ( $M$ ) (in W) as follows:

$$M (\text{W}) = \dot{V}O_2 (\text{l/min}) \cdot 69.7 \{4.686 + [(RER - 0.707) \cdot 1.232]\} \quad (26)$$

Respiratory heat loss (RHL) was calculated independently from metabolism (7):

$$\text{RHL (W)} = 0.09M$$

Total energy production for the immersion/submersion was calculated by converting  $M$  to kilojoules. Total energy loss was calculated as the sum of total body cutaneous heat flux and RHL. The net energy balance was determined by integrating the difference between total energy loss and production over the first 30 min of immersion. This

method correlates well with direct measurements of tissue heat (19, 22).

### Immersion Conditions

Subjects were immersed four times in 17°C water. This water temperature was used, rather than the 12°C water temperature used in our laboratory's previous study (21), because it was found that meperidine, at the maximum cumulative dose allowed (2.5 mg/kg iv), could only successfully inhibit shivering at the higher water temperature. In all trials, buspirone (oral 30 mg) was also used because these two agents synergistically decrease the shivering threshold without increasing sedation and respiratory suppression (23). The increased suppression of cold-induced shivering was necessary because of the increased cold stress caused by the submersion of the whole head compared with the dorsal head in the previous studies.

For each condition, subjects were lowered with an electronically isolated hoist into the water. Two of the conditions involved complete submersion and required the breathing of compressed air. Thus, for all trials (baseline and immersion/submersion), subjects wore a nose clip and breathed compressed air as previously described. The left arm was completely immersed while the right hand was held out of the water for measurements of arterial oxygen saturation and protection of the intravenous site.

*Body exposed, head out.* The subjects wore only a bathing suit and were immersed to the neck with the head positioned above the water. Subjects wore a light fleece cap, which helped keep the mesh hood and posterior head heat flux disk in place.

*Body exposed, head in.* The subjects wore only a bathing suit and were lowered until the entire head was completely submersed. Subjects wore a diving mask, which helped keep the mesh hood and dorsal head heat flux disk tight against the scalp. The straps, however, were positioned so that they did not cover the heat flux disk.

*Body insulated, head out.* Subjects wore a 1.5-mm-thick vulcanized rubber dry suit worn over thermal underwear (jacket and pants), a fleece suit, two pairs of socks, and a wool glove on the left (immersed) hand. The dry suit hood was not worn; however, a light fleece cap helped keep the mesh hood and posterior head heat flux disk in place. They were immersed to the neck with the head positioned out of the water.

*Body insulated, head in.* The subjects wore the same dry suit/insulation combination and diving mask as previously described. They were lowered until the entire head was completely submersed.

### Protocol

During instrumentation, subjects ingested buspirone in three 10 mg doses, at 45, 30, and 15 min before the start of baseline. After instrumentation, subjects prepared for baseline measurements. In the body-insulated conditions, they donned the insulative clothing and dry suit, but the fleece suit and dry suit were only pulled up to the knees to prevent overheating before immersion/submersion. Twenty minutes of baseline measurements were then started. After 10 minutes, subjects completed the donning of the fleece suit and dry suit (if applicable) and were given 1.25 mg/kg of meperidine intravenously (diluted in 10 ml of saline) injected in five 2-ml aliquots in successive 2-min intervals. They were then lowered by an electronically isolated hoist into the water.

Initially, meperidine injections during the baseline were administered while the subjects were sitting. Some of the subjects experienced nausea due to a meperidine-induced hypotension. One subject became nauseous after the second baseline dose of meperidine and was lowered into the water to allow hydrostatic pressure to support his blood pressure and reverse the nausea. To prevent further incidents of nausea and hypotension in the remaining trials, subjects lay on a stretcher for the second half of baseline.

Normally, a higher dose of meperidine would be required to suppress shivering in the body-exposed conditions compared with the

body-insulated conditions. However, to control for pharmacological effects, the same (maximum) drug doses were given to each subject for all conditions. For five of the subjects (including the first three tested), a body-exposed trial was completed first. During submersion, shivering heat production, as indicated by increased  $\dot{V}_{O_2}$  and subjective evaluation, was inhibited as required with supplemental injections of meperidine to the maximal cumulative dose of 2.5 mg/kg. During their subsequent body-insulated trials, meperidine was administered according to the same dosing schedule as used in their body-exposed trial(s).

To achieve a balanced design, the first trial for the other three subjects was a body-insulated condition. For these subjects, the meperidine dosing schedule followed the average schedule determined from the two body-exposed trials of the first three subjects tested. In their remaining body-exposed trials, meperidine was given according to the predetermined schedule unless shivering occurred; in these cases the next doses were given as required to suppress shivering, to the maximum cumulative dose.

In all trials, arterial oxygen saturation was monitored to screen for respiratory depression. Pulse amplitude on the oximeter was monitored to confirm that saturation values were reliable; if pulse amplitude was inadequate, alternate fingers were used. If saturation decreased below ~95%, subjects were roused and encouraged to breathe more vigorously.

The subjects remained immersed until one of four removal criteria was met: 1) immersion time of 45 min, 2) voluntary request by a subject for removal, 3)  $T_{es}$  reached 34°C, or 4) termination of immersion by investigator for safety reasons. On removal from the cold water, subjects were placed in a 40°C stirred water bath until  $T_{es}$  was >36°C and they felt comfortably warm.

### Data Analysis

Some subjects cooled quickly in the body-exposed conditions and reached a  $T_{es}$  of 34°C in as little as 32.5 min. Therefore, most of the analysis was done for 30 min, the longest time during which all subjects was immersed in all conditions.

The following calculations were made for each condition: 1) decrease in  $T_{es}$  from baseline to 30 min and to the end of immersion, 2) rate of core cooling (calculated by linear regression for  $T_{es}$  data from 10 to 30 min of immersion), 3) area-weighted mean skin temperature; 4) cutaneous heat loss from various body segments, 5) metabolic heat production (kJ) over the first 30 min of immersion, and 6) net energy balance in the different conditions. Group results were calculated for each condition and were compared using repeated-measures analysis of variance. Results are reported as means  $\pm$  SD.  $P < 0.05$  identified statistically significant differences. The Holm-Sidak test was used for post hoc analysis of significant differences. The  $\chi^2$  test was used to compare the observed heat loss from various body regions to the expected values (based on the assumption that regional heat loss is proportional to that region's relative surface area).

### RESULTS

Subjects were closely monitored for adverse affects during the serial meperidine injections. On immersion, there was a transient increase in metabolism. Subsequent injections of meperidine alleviated cold discomfort as well as metabolic and visual evidence of shivering.

All subjects remained immersed/submersed for 45 min in both body-insulated conditions. In the body-exposed, head-out condition, one subject reached the cutoff  $T_{es}$  of 34°C within 36 min and was removed. In the body-exposed, head-in condition, three subjects exited the water before 45 min because they reached the cutoff  $T_{es}$  of 34°C within 32.5–39 min. Because the longest time at which all eight subjects were immersed/



submersed in all four conditions was 32.5 min, data are presented and analyzed for the first 30 min of immersion/submersion.

### Core Temperature Responses

$T_{es}$  decreased significantly after 15 min in all conditions (Fig. 1). The only two conditions in which all subjects remained in the cold water for 45 min were both body-insulated conditions. After 30 min of immersion in these conditions,  $T_{es}$  decreased from baseline values by  $0.47 \pm 0.2$  and  $0.77 \pm 0.2^\circ\text{C}$  in the head-out and head-in conditions, respectively ( $P < 0.02$ ) with the two conditions being significantly different from each other ( $P < 0.05$ ). In the body-exposed conditions,  $T_{es}$  was lower in the head-in condition than in the head-out condition from 15 min of immersion/submersion onward ( $P < 0.02$ ). After 30 min,  $T_{es}$  decreased by  $0.84 \pm 0.2^\circ\text{C}$  in the head-out condition and by  $1.17 \pm 0.5^\circ\text{C}$  in the head-in condition ( $P < 0.02$ ). Core cooling rates, between 10 and 30 min of immersion, were  $1.1 \pm 0.4$  and  $1.6 \pm 0.5^\circ\text{C/h}$  for body-insulated, head-out and body-insulated, head-in conditions, respectively, and  $1.8 \pm 0.6$  and  $2.5 \pm 0.9^\circ\text{C/h}$  for body-exposed, head-out and body-exposed, head-in conditions, respectively. There was no difference between the body-insulated, head-in and body-exposed, head-out conditions throughout the experiment.

### Mean Skin Temperature Responses

During the meperidine baseline period, mean skin temperature was slightly, but significantly, higher during body-insulated conditions ( $33.6 \pm 0.6^\circ\text{C}$ ) than without insulation ( $31.1 \pm 0.6^\circ\text{C}$ ;  $P < 0.1$ ). Area-weighted mean skin temperature decreased slightly from baseline in the body-insulated, head-out condition (to  $30.9 \pm 0.9^\circ\text{C}$ ) after 30 min of immersion. Head submersion in this subcondition resulted in a significantly lower area-weighted mean skin temperature of  $29.3 \pm 0.5^\circ\text{C}$  ( $P < 0.001$ ), area-weighted mean skin temper-

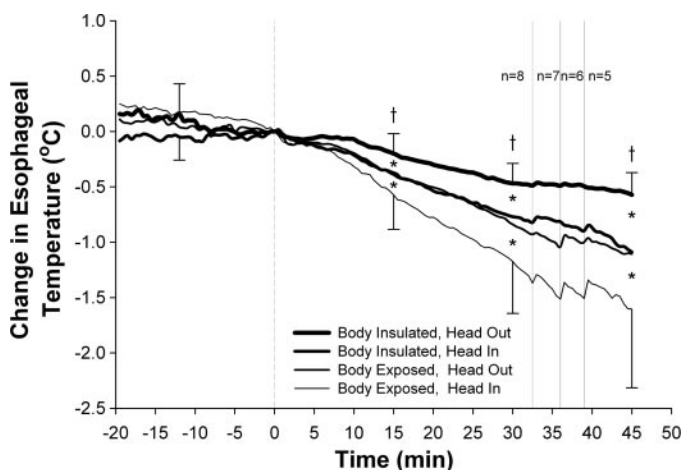


Fig. 1. Esophageal temperature in nonshivering subjects. Twenty minutes of baseline include 5 meperidine injections (0.25 mg/kg each) at 2-min intervals from minutes -10 to -2. Time 0 indicates immersion.  $n$ , Number of subjects. After 32.5 min, some subjects were removed from the cold water at different times in some of the body-exposed conditions. Therefore, data after this point are presented only for the number of subjects still participating in all 4 conditions. \*Separates all conditions that are significantly different,  $P < 0.05$ . †Lower than baseline,  $P < 0.02$ .

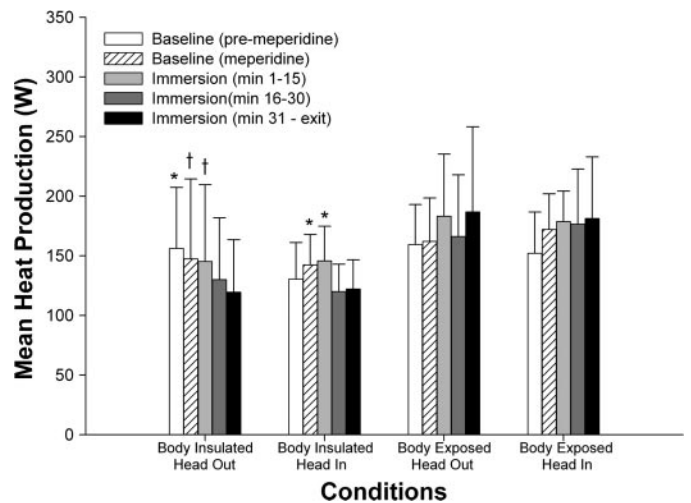


Fig. 2. Metabolic heat production for baseline (two 10-min periods) and the three 15-min periods of immersion. \*Greater than immersion (minutes 16–30) and immersion (minute 31 to exit),  $P < 0.02$ . †Greater than immersion (minute 31 to exit),  $P = 0.01$ .

ature in these two conditions were significantly higher than both the body-exposed conditions after 30 min of cooling where area-weighted mean skin temperature decreased more in the head submersion condition (to  $18.5 \pm 0.5^\circ\text{C}$ ) than in the head-out condition (to  $19.4 \pm 0.3^\circ\text{C}$ ;  $P = 0.002$ ).

### Metabolic Responses

Baseline values for  $\dot{V}O_2$  were similar for all conditions and there were no differences between the initial 10 min of baseline and the 10 min of meperidine injections (Fig. 2).  $\dot{V}O_2$  remained at, or below, baseline levels throughout immersion in both body-insulated conditions due to the suppressing effect of meperidine. In the two body-exposed conditions,  $\dot{V}O_2$  remained the same throughout the experiments; thus no evidence of shivering heat production was detected.

There were no intercondition differences in heart rate. Baseline heart rate was  $79 \pm 3$  beats/min. After a transient increase to  $98 \pm 17$  beats/min (body-exposed conditions) and  $94 \pm 22$  beats/min (body-insulated conditions) during entry into the water, heart rate decreased gradually in all conditions from  $82 \pm 4$  to  $78 \pm 6$  beats/min from 10 to 30 min of immersion respectively.

### Cutaneous Heat Loss

Whole body cutaneous heat loss during baseline and cooling was greater in the body-exposed conditions than in the body-insulated conditions (Fig. 3). Heat loss increased markedly immediately on immersion, with the effect in exposed skin gradually decreasing as skin cooled and the temperature gradient between skin and water decreased. Head submersion resulted in a significantly higher rate of heat loss in both the body-insulated and body-exposed subconditions.

Heat loss and energy production during 30 min of cooling are presented in absolute terms in Fig. 4; values are for total energy production, total heat loss (including whole body cutaneous and RHL), and loss from the body excluding the head, as well as each main body region. Total heat loss was  $\sim 2.6$  times greater in the body-exposed conditions ( $914 \pm 96$  and  $988 \pm$

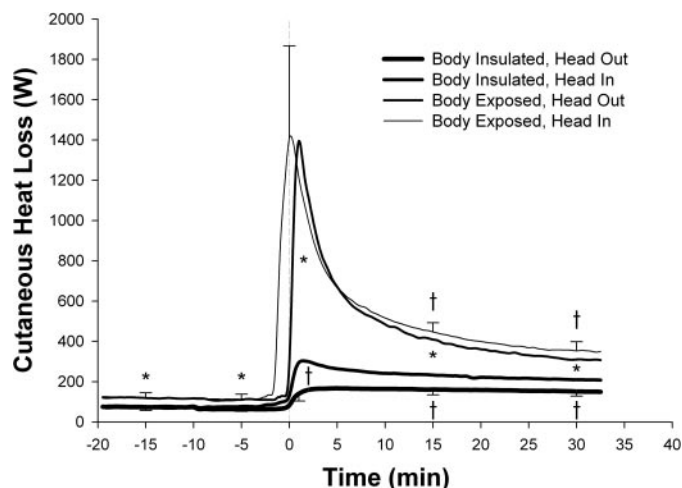


Fig. 3. Whole body cutaneous heat loss ( $n = 8$ ) values for baseline (20 min) and immersion (30 min) periods. \*Greater than body-insulated conditions,  $P < 0.001$ . †Greater than head-out in the same subconditions,  $P < 0.05$ .

88 kJ with head-out and head-in respectively;  $P = 0.003$ ) than in the body-insulated conditions ( $298 \pm 54$  and  $440 \pm 48$  kJ for head-out and head-in, respectively;  $P < 0.001$ ). Similarly, head heat loss increased considerably with submersion both in the body-exposed conditions ( $17 \pm 5$  and  $98 \pm 11$  kJ with head-out and head-in, respectively;  $P < 0.001$ ) and in the body-insulated conditions ( $17 \pm 2$  and  $102 \pm 16$  kJ for head-out and head-in, respectively;  $P < 0.001$ ). In the body-insulated subcondition, heat loss from the body was also greater in the head-in condition ( $322 \pm 38$  kJ) than in the head-out condition ( $269 \pm 51$  kJ;  $P = 0.024$ ). Compared with the body-insulated conditions, heat loss was greater with the body-exposed in all areas except the head ( $P < 0.001$ ).

Except for the face, most of the head was covered with hair. The skin was not shaved in any area for comparison of heat loss from shaved and hair-covered areas of the immersed scalp. Heat loss was  $\sim 28\%$  less from hair covered skin ( $234 \text{ W/m}^2$ ) than from the bare forehead ( $323 \text{ W/m}^2$ ). A light mesh hood was used to hold the heat flux transducer snugly against the hair of the back of the head. This eliminated a layer of water between the hair and transducer, thus ensuring that heat loss from the skin, and through the hair, was channeled through the transducer. The mesh was light and provided negligible insulation.

A  $\chi^2$  analysis indicated that there were no significant differences between observed and expected regional heat losses when the body and head were under similar thermal conditions (i.e., body-insulated, head-out and body-exposed, head-in). Head heat loss was proportionally greater than its surface area in the body-insulated, head-in condition and proportionally lower in the body-exposed, head-out condition.

The negative energy balance during immersion was significantly higher in the body-exposed conditions ( $-717$  and  $-645$  kJ with head-in and head-out, respectively) than body-insulated conditions ( $-220$  and  $-63$  kJ with head-in and head-out, respectively) ( $P < 0.001$ ).

## DISCUSSION

This was the first ethically approved study to evaluate the isolated contribution of whole head cooling to lowering of core

temperature (as low as  $34^\circ\text{C}$ ) when the confounding factor of shivering heat production was inhibited. As expected, head submersion in  $17^\circ\text{C}$  did not cause a disproportionately large increase in surface heat loss. Rather, the 7% increase in our laboratory's submersed surface area elicited a comparable 10% increase in heat loss. Similar to our laboratory's previous study (9), head submersion increased the rate of core cooling by 39% when the body was also cold stressed (i.e., head out  $1.8^\circ\text{C/h}$  vs. head in  $2.5^\circ\text{C/h}$ ). Surprisingly, head exposure also increased core cooling (by 45%) when the body itself was not cold stressed (i.e., head out  $1.1^\circ\text{C/h}$  vs. head in  $1.6^\circ\text{C/h}$ ). Although a greater disparity was expected between the relative increase in core cooling rate compared with the relative increase in total heat loss during head submersion, the results in the present study (head submersion in body-exposed and body-insulated conditions resulted in an average of 42% increase in core cooling with 7% increase in exposed surface area) were not different from previous work with dorsal head immersion (9). It was also noteworthy that the core cooling rate was similar whether only the head ( $1.6^\circ\text{C/h}$ ), or only the body ( $1.8^\circ\text{C/h}$ ), was exposed to cold water, despite a large difference in total heat loss (446 vs. 926 kJ, respectively).

The present results for body-exposed conditions are comparable to three previous studies using dorsal head cooling. With the body exposed to cold water and shivering intact, additional dorsal head immersion increased core cooling from  $3.8$  to  $9.4^\circ\text{C/h}$  (250%) in  $1\text{--}2^\circ\text{C}$  water (1), and from  $1.5$  to  $2.8^\circ\text{C/h}$  (87%) in  $10^\circ\text{C}$  water (21). When the latter study was repeated in  $12^\circ\text{C}$  with shivering suppressed with meperidine, dorsal head cooling increased the cooling rate by 39% (from  $3.6$  to  $5.0^\circ\text{C/h}$ ). Hayward et al. (14) reported similar relative differences when uninsulated subjects were physically active in  $10^\circ\text{C}$  water. They demonstrated that drown proofing (which intermittently submersed the whole head) increased core cooling by 36% to  $4.6^\circ\text{C/h}$ , compared with  $3.4^\circ\text{C/h}$  while treading water with the head above water.

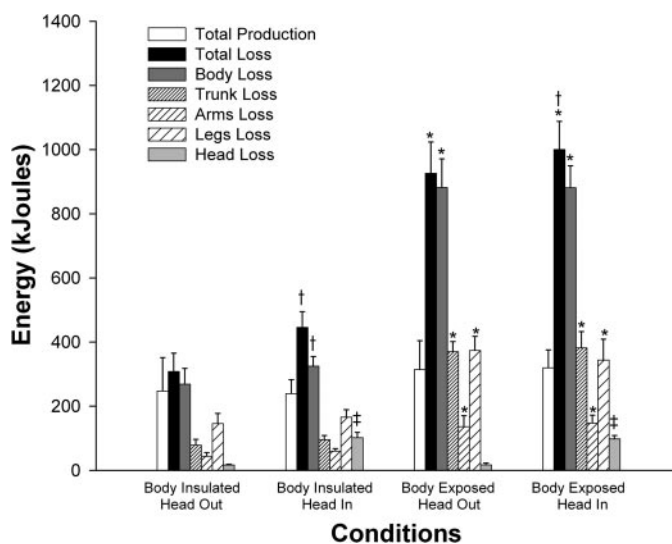


Fig. 4. Energy production and loss during 30 min. of immersion in  $17^\circ\text{C}$  water ( $n = 8$ ). Total loss includes whole body cutaneous and respiratory heat loss. Body loss includes trunk, legs, and arms. \*Greater than body-insulated conditions,  $P < 0.001$ . †Greater than head out in the same subconditions,  $P < 0.005$ . ‡Greater than head-out conditions,  $P < 0.001$ .

The novel finding in the present study was that, even when the body was insulated, whole head submersion further decreased core temperature (i.e., by 0.47°C with head-out and 0.77°C with head-in after 30 min).

#### *Possible Mechanisms for the Results*

The contribution of head submersion to surface heat loss and core temperature cooling was of major interest in this study. Blood flow in the face and scalp remains relatively high and constant compared with the rest of the body. Hertzman (17) found that the ratio of head blood flow to surface area is 4–10 times greater than seen in the trunk and proximal limbs. Froese and Burton (8) found little or no head skin vasoconstriction in response to cold, whether the cold stimuli came from the head alone or even during cooling of the whole body surface.

The present results are consistent with previous data (9) in that the supposition of proportionately greater heat loss from the head was not supported. The measured heat loss from the head in both head-in conditions was only ~100 kJ (compared with ~17 kJ in the two head-out conditions). In contrast, total heat loss in the body-exposed configurations was ~914 and 988 kJ, for head-out and head-in conditions, respectively. In the latter case, the head accounted for only ~10% of the total body heat loss when both the head and body were submersed. The surface area of the submersed head is ~7% of the total surface area of the body. These results thus indicate that heat loss from the head is not disproportionately increased over what would be expected from the head's contribution to total body surface area. This confirms the prediction of Xu et al. (27), who hypothesized minimal heat loss from the whole head based on modeling and experimental findings.

The present study confirms results from previous body-exposed trials (1, 9, 21); additional head cooling (in this case by total submersion) increased core cooling proportionately more (39%) than the increase in total heat loss (10%). This exaggerated core cooling likely results from the extra heat loss affecting a smaller thermal core due to peripheral vasoconstriction secondary to intense cold stimulation of the body and head (26). This phenomenon also provides a likely explanation for similar responses to the body-exposed head-out, and body-insulated head-in conditions. Even though heat loss, when only the body was exposed (925 kJ), was over twofold more than when only the head was exposed (446 kJ), cooling rates were virtually the same.

This was the first demonstration of a core cooling effect of head exposure when the body was not cold stressed. Whereas dorsal head cooling had no core cooling effect under similar conditions previously (1, 9, 21), total head submersion increased core cooling in this case. This could be predicted because facial submersion was expected to have a synergistic effect of inducing peripheral vasoconstriction and reducing the thermal core. With facial submersion, vasoconstriction would result from thermoregulatory control mechanisms. However, the human dive reflex, mediated by the trigeminal nerve, is known to also induce peripheral vasoconstriction, in this case as an oxygen-sparing mechanism (5, 16, 18). This oxygen-sparing mechanism occurs in thermoneutral water (i.e., 34°C) (5) and increases at cooler water temperatures (i.e., 20–25°C) (16, 18). The dive reflex likely plays a significant role in the core cooling response even though the usual bradycardia (25)

was not seen. In the present study, the bradycardia reflex may have been attenuated or overridden as the act of breathing diminishes the usual heart rate slowing response (18). Also, the high dose of intravenous meperidine could also cause sinus tachycardia through a vagolytic mechanism which may have attenuated bradycardia (13).

Finally, the augmentation of core cooling by head submersion in body-exposed conditions was expected to be greater than demonstrated previously with dorsal head immersion (9). In that study, it was hypothesized that, when the body was exposed, additional intense cooling of the scalp would increase vasoconstriction and further decrease the thermal core. Thus a relatively small increase in heat loss through the dorsal head would have an exaggerated core cooling effect. In the present study, the synergistic effect of facial and scalp cooling was expected to augment all of these effects, resulting in a greater relative effect on core cooling. However, core cooling increased similarly (~39%) whether the dorsal head was immersed in 12°C water or the whole head was submersed in 17°C water. It is likely that the effect of increased blood flow redistribution from facial cooling was attenuated as 17°C water provided less thermal stimuli.

Further work is required in which tissue temperature and blood flow are measured to confirm the mechanisms for these results.

#### *Practical Implications of Results*

This study has practical implications regarding the gradual onset of symptomless hypothermia in SCUBA divers and may be relevant to protective brain cooling in cold-water near-drowning victims.

Submersion of the scalp and face during diving will tend to enhance core (and brain) cooling even in cool water that only increases the integrated thermal signal by a moderate amount. The moderate thermal vasoconstriction may be synergistically augmented by the facially stimulated dive response. Thus a moderate increase in heat loss may result in significant core cooling without the discomfort and shivering normally experienced during submersion in colder water. SCUBA divers may be at risk if they experience symptomless hypothermia to the point of altered mental capacity (10, 21).

According to Gooden et al. (12), the dive response is attenuated with a normal breathing pattern and when a face mask is worn with the head insulated. Thus maintaining a slow deep breathing pattern (as is recommended for SCUBA diving) and insulating the head prophylactically should help prevent the development of symptomless hypothermia.

The core cooling effect of head submersion in this study was relatively small compared with the 3–5°C decrease in brain temperature required to provide brain protection from anoxia in cold-water drowning (4). The present study only tested the convective and conductive heat loss from the surface of the head but, for obvious reasons, did not include heat loss through the surface of the upper airways or lungs as seen when water is repeatedly breathed in and out (6, 27).

In conclusion, whole head submersion in 17°C water did not contribute relatively more than the rest of the body to total surface heat loss. Consequent to submersion and cooling of the scalp and face, thermal stimulation and the human dive response likely reduced the perfused body mass, thus a small



increase in total heat loss caused a relatively large cooling of the body core. Further work is warranted to quantify the effects of scalp and/or face immersion in cold water on regional blood flow and alterations in the mass of the thermal core.

#### ACKNOWLEDGMENTS

We thank Doug Evans, of One Stop Diving, Winnipeg, Canada, for supplying the Sherwood regulator used in this study. We thank Perry Schwark for technical assistance. Also, we thank our subjects for participating in this challenging study.

#### GRANTS

This study was supported by the Natural Science and Engineering Research Council of Canada and the Randy Chipperfield Research Fund.

#### REFERENCES

1. **Alexander L.** *The Treatment of Shock From Prolonged Exposure to Cold, Especially in Water.* Washington, DC: Office of Publ. Bd., Dept. of Commerce, 1946, p. 1–228 (Combined Intelligence Objectives Subcommittee. Target 24. Rep. 250).
2. **Brozek J.** Densitometric analysis of body composition: revision of some quantitative assumption. *Ann NY Acad Sci* 110: 113–140, 1963.
3. **Busto R, Dietrich WD, Globus MY, and Ginsberg MD.** The importance of brain temperature in cerebral ischemic injury. *Stroke* 20: 1113–1114, 1989.
4. **Busto R, Dietrich WD, Globus MYT, Valdes I, Scheinberg P, and Ginsberg MD.** Small differences in intrasemic brain temperature critically determine the extent of ischemic neuronal injury. *J Cereb Blood Flow Metab* 7: 729–738, 1987.
5. **Campbell L.** Simultaneous calf and forearm bloodflow during immersion in man. *Aust J Exp Biol Med Sci* 47: 747–754, 1969.
6. **Conn AW, Miyasaka K, Katayama M, Fujita M, Orima H, Barker G, and Bohn D.** A canine study of cold water drowning in fresh versus salt water. *Crit Care Med* 23: 2029–2037, 1995.
7. **Fanger PO.** *Thermal Comfort: Analysis and Applications in Environmental Engineering.* New York: McGraw-Hill, 1972.
8. **Froese G and Burton AC.** Heat losses from the human head. *J Appl Physiol* 10: 235–241, 1957.
9. **Giesbrecht G, Lockhart T, Bristow G, and Steinman A.** Thermal effects of dorsal head immersion in cold water on nonshivering humans. *J Appl Physiol* 99: 1958–1964, 2005.
10. **Giesbrecht GG, Arnett JL, Vela E, and Bristow GK.** Effect of task complexity on mental performance during immersion hypothermia. *Aviat Space Environ Med* 64: 206–211, 1993.
11. **Giesbrecht GG, Goheen MSL, Johnston CE, Kenny GP, Bristow GK, and Hayward JS.** Inhibition of shivering increases core temperature afterdrop and attenuates rewarming in hypothermic humans. *J Appl Physiol* 83: 1630–1634, 1997.
12. **Gooden B, Lehman R, and Pym J.** Role of the face in the cardiovascular responses to total immersion. *Austr J Exp Biol Med Sci* 48: 687–690, 1970.
13. **Gutstein B and Akil H.** Opioid analgesics. In: *Goodman & Gilman's The Pharmacological Basics of Therapeutics* (10th ed.), edited by Hardman J and Limbird L. New York: McGraw-Hill, 2001, p. 569–619.
14. **Hayward JS, Eckerson JD, and Collis ML.** Effect of behavioral variables on cooling rate of man in cold water. *J Appl Physiol* 38: 1073–1077, 1975.
15. **Hayward JS, Eckerson JD, and Kemna D.** Thermal and cardiovascular changes during three methods of resuscitation from mild hypothermia. *Resuscitation* 11: 21–33, 1984.
16. **Heistad DD, Abboud F, and Eckstein J.** Vasoconstrictor response to simulated diving in man. *J Appl Physiol* 25: 542–549, 1968.
17. **Hertzman AB.** Regional differences in the basal and maximal rates of blood flow in the skin. *J Appl Physiol* 1: 234–241, 1948.
18. **Hurwitz B and Furedy J.** The human dive reflex: an experimental, topographical and physiological analysis. *Physiol Behav* 36: 287–294, 1986.
19. **Kurz A, Sessler DI, Christensen R, and Dechert M.** Heat balance and distribution during the core-temperature plateau in anesthetized humans. *Anesthesiology* 83: 491–499, 1995.
20. **Layton R, Mints WJ, Annis J, Rack M, and Webb P.** Calorimetry with heat flux transducers: comparison with a suit calorimeter. *J Appl Physiol* 54: 1361–1367, 1983.
21. **Lockhart T, Jamieson C, Steinman A, and Giesbrecht G.** Life jacket design affects dorsal head and chest exposure, body core cooling and mental performance in 10°C water. *Aviat Space Environ Med* 76: 954–962, 2005.
22. **Matsukawa T, Sessler DI, Sessler AM, Schroeder M, Ozaki M, Kurz A, and Cheng C.** Heat flow and distribution during induction of general anesthesia. *Anesthesiology* 82: 674–681, 1995.
23. **Mokhtarani M, Mahgoub A, Morioka N, Doufas A, Dae M, Shaughnessy T, Bjorksten A, and Sessler D.** Buspirone and meperidine synergistically reduce the shivering threshold. *Anesth Analg* 93: 1233–1239, 2001.
24. **Rasch W, Samson P, Cote J, and Cabanac M.** Heat loss from the human head during exercise. *J Appl Physiol* 71: 590–595, 1991.
25. **Schagatay E and Holm B.** Effects of water and ambient air temperatures on human diving bradycardia. *Eur J Appl Physiol* 73: 1–6, 1996.
26. **Vanggaard L, Eyolfson D, Xu X, Weseen G, and Giesbrecht GG.** Immersion of distal arms and legs in warm water (AVA rewarming) effectively rewarms hypothermic humans. *Aviat Space Environ Med* 70: 1081–1088, 1999.
27. **Xu X, Tikuisis P, and Giesbrecht G.** A mathematical model for human brain cooling during cold-water near-drowning. *J Appl Physiol* 86: 265–272, 1999.