

The effect of drysuit diving in warm water on body temperature and post immersion orthostatic hypotension

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Keywords

Body temperature regulation; Cardiovascular physiology; Hyperthermia; Immersion; Orthostatic intolerance; Protective clothing

Abstract

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Introduction: Warm-water diving can limit heat dissipation, particularly when performed in fully encapsulating protective gear, leading to substantial thermal and cardiovascular strain that may impair diver safety. Following immersion, removal of hydrostatic support combined with heat-induced vasodilation may reduce central blood volume and increase susceptibility to orthostatic intolerance during egress and recovery. The extent to which this thermal strain impairs post-immersion orthostatic tolerance remains unknown.

Methods: Four randomised, crossover immersion trials were conducted at 28°C, 33°C, 38°C without precooling (38°C), and 38°C with precooling (38°C + Cool), with subjects wearing fully encapsulating dive gear. Subjects walked for up to 60 minutes at approximately 50% of $\dot{V}O_{2max}$ heart rate (HR) or until core temperature (Tc) reached 38.5°C, or they voluntarily stopped. Tc, HR, and perceptual measures were recorded every 10 minutes. Orthostatic tolerance was assessed after immersion via a 70° head-up tilt test.

Results: Eight healthy adults completed all aspects of the study. Tc and HR were higher during both 38°C conditions compared with 28°C and 33°C (all $P < 0.01$) with no differences between 38°C and 38°C + Cool. Sweat loss exceeded 1.2 (SD 0.67) L·h⁻¹ in both 38°C conditions compared with ≤ 0.3 (0.32) L·h⁻¹ at 28°C and 33°C ($P < 0.01$). Survival analysis showed orthostatic tolerance decreased with increasing thermal stress (log-rank $P = 0.027$; trend $P = 0.003$). Precooling did not reduce peak Tc or HR, nor did it improve tolerance time in 38°C water.

Conclusions: Encapsulated warm-water diving causes heat stress and cardiovascular strain that persists after immersion, impairing orthostatic tolerance. Precooling does not significantly reduce these outcomes.

Introduction

Diving in contaminated water requires military and occupational divers to operate fully encapsulated in impermeable protective ensembles that impede heat dissipation. Although these systems provide essential protection against toxic exposures, they may promote body heat storage in warm water, increasing the risk of heat-related illness and limiting the duration of safe operations. Previous work identifies thermal strain as a primary limiting factor for endurance during warm-water diving.^{1,2} This ultimately constrains performance and dive profiles. The water temperatures selected for the present study were intended to represent a range of operationally relevant warm-water exposures, from temperate or thermoneutral conditions to extreme warm water environments. The 28°C and 33°C

conditions approximate water temperatures that may be encountered during prolonged operations in warm coastal, harbor, or industrial settings, whereas 38°C represents a high thermal load scenario relevant to enclosed, shallow, sun-heated, industrial, or contaminated-water environments where evaporative heat loss is unavailable and environmental heat gain may be substantial.

Increases in core body temperature during warm-water immersion reflect the combined effects of the environment and metabolic heat. Physical activities commonly performed during diving operations elevate metabolic heat production and further exacerbate heat storage. Increases in core body temperature during warm-water immersion reflect the combined effects of environmental heat load and metabolic heat production. During warm water drysuit diving,

heat exchange remains governed largely by conductive heat flux between the diver and the surrounding water, while encapsulation limits evaporative heat loss and may exacerbate internal heat storage during physical work. Laboratory and field studies in terrestrial environments show that precooling increases heat-storage capacity and delays the onset of life-threatening hyperthermia during exercise,^{3–5} suggesting that similar strategies may mitigate thermal burden during warm-water diving.

In addition to thermoregulatory stress, warm-water diving imposes substantial cardio- and cerebrovascular challenges. Post-immersion weakness and pre-syncope symptoms have been described following passive hot water immersion and exercise in the heat.^{6–8} These responses are largely attributed to sustained peripheral vasodilation, venous pooling, and reduced central blood volume, all of which impair arterial pressure regulation.^{7–9} During immersion, hydrostatic pressure promotes centralisation of blood volume and may transiently stabilise cardiovascular function. However, this protection is rapidly lost after exiting the water. Experimental evidence indicates that the interval following exercise and hot water immersion is characterised by persistent vasodilation and impaired orthostatic tolerance,^{6,7} mechanisms that may be exacerbated by residual hyperthermia.

Diving in warm, contaminated water represents a complex physiological stress model characterised by the simultaneous exposure to heat stress, immersion-induced hydrostatic pressure, physical exertion, and respiratory loading from diving equipment. In addition to increasing work of breathing, helmet and breathing apparatus dead space may contribute to CO₂ retention, which can influence ventilatory drive, cerebral blood flow, and symptoms such as dizziness or lightheadedness. Each of these stressors independently alters systemic and cerebral regulation. Moderate-intensity exercise increases arterial pressure during activity, but is followed by post-exercise hypotension during recovery.⁸ Similarly, hot-water immersion induces peripheral haemodynamic responses including vasodilation, reduced central blood volume, and altered arterial pressure profiles comparable to those observed during post exercise recovery.⁹ Thermal strain during warm-water immersion induced marked peripheral vasodilation and reductions in systemic vascular resistance,¹⁰ while cerebrovascular responses appear region and vessel specific. Increases in internal carotid artery blood flow and shear have been demonstrated during hot water immersion,¹¹ and similar elevations in vertebral artery blood flow have been reported,¹² suggesting that global cerebral perfusion is maintained or elevated during immersion despite systemic vasodilation. However, following immersion, the removal of hydrostatic support combined with persistent peripheral vasodilation may reduce arterial pressure and compromise cerebral perfusion. The convergence of hyperthermia, exercise cessation, and removal of hydrostatic support during egress may predispose divers to hypotension and cerebral hypoperfusion, increasing susceptibility to presyncope or syncope.

Despite the operational relevance of these mechanisms, data describing integrated thermoregulatory and cardiovascular responses during and after encapsulated warm-water diving remain limited. Much of the foundational work informing exposure guidance dates to investigations conducted more than two decades ago.^{1,2} While precooling strategies are well established in terrestrial and occupational settings,^{3–5} their efficacy in encapsulated warm-water diving has not been rigorously evaluated. The purpose of this study was to examine the effects of warm water immersion across a range of water temperatures on thermoregulatory and cardiovascular responses, and to determine whether a precooling intervention improves physiological stability and operational safety. We hypothesised that warm-water diving would increase internal heat storage and impair orthostatic tolerance compared with thermoneutral conditions, and that precooling would reduce heat storage, extend dive tolerance, and attenuate thermal and cardiovascular strain.

Methods

All subjects were recruited from the local community. All subjects completed six laboratory visits, including screening. The study was approved by the University at Buffalo Institutional Review Board (IRB# STUDY00007863). All subjects provided written informed consent prior to participation.

This study employed a randomised, repeated-measures crossover design. Each subject completed four immersion conditions: 28°C, 33°C, 38°C without precooling (38°C), and 38°C with precooling (38°C + Cool). Conditions were separated by a minimum of 72 hours to minimise carryover effects. The order of exposure was randomised and counterbalanced across subjects.

Subjects completed a health history questionnaire and a physical activity readiness questionnaire (PAR-Q+) and were screened for contraindications to ingestion of the telemetric temperature capsule used for continuous core temperature monitoring during experimental visits. Height was measured using a stadiometer (Health-o-meter Professional 402KL, Pelstar LLC, McCook, IL) and body mass was measured to the nearest 0.01 kg using a calibrated digital scale/platform scale (Midrics 2, Minebea Intec USA Inc., Holbrook, NY). The scale was calibrated according to manufacturer specifications before data collection. Body morphology and body composition were assessed using three-dimensional infrared scanning (SS20 Scanner, Size Stream, Cary, NC). Subjects underwent a physician-administered physical examination evaluating musculoskeletal, neurological, cardiovascular, and pulmonary systems. Individuals with a history of neurological, metabolic, pulmonary, or cardiovascular disease or illness were excluded. Maximal aerobic capacity ($\dot{V}O_{2\max}$) was determined using an incremental treadmill protocol with open-circuit spirometry (TrueOne, PARVO Medics, Provo, UT, USA). Subjects with a $\dot{V}O_{2\max}$ less than 30 mL·kg⁻¹·min⁻¹ were excluded.

Subjects reported to the laboratory for all experimental sessions. Experimental procedures were standardised across visits, with the exception of water temperature and the addition of a precooling intervention during one 38°C condition. Subjects were provided standardised pre-visit instructions to arrive normally hydrated, including guidance on fluid intake in the evening before and morning of each experimental visit. Upon arrival, subjects consumed a standardised meal of granola bars providing 4 kcal·kg⁻¹ of body mass and then voided their bladder. This initial urine sample was used to measure urine specific gravity as a pre-exposure screening measure. Nude body mass was measured using the same calibrated scale. Urine specific gravity was measured upon arrival using a handheld refractometer (MASTER-SUR/Na, Atago USA Inc., Bellevue, WA, USA), and subjects were required to have a value < 1.020 before testing. Subjects ingested a telemetric temperature capsule (eCelsius Performance, Hérouville Saint-Clair, France) 6–8 hours prior to immersion or inserted the capsule rectally immediately before testing. Mean skin temperature¹³ (T_{SK}) was recorded every minute throughout the protocol. iButton thermistors (Maxim Integrated, San Jose, CA) were secured to the skin over the triceps, pectoral region, thigh, and calf on the right side of the body to estimate regional skin temperature. An additional sensor was placed on the right temple to measure the temperature inside the dive helmet, and the data were analysed separately. Subjects were then fitted with a heart rate monitor (H10, Polar Electro, Kemple, FI) and donned lightweight base layers (32°Degrees Heat, David Peyser Sportswear Inc., Bay Shore, NY, USA), drysuit, and gloves (Viking HD, Ansell, Iselin, NJ, USA), and a commercial dive helmet (KM 37, Kirby Morgan Dive Systems, Inc., Santa Maria, California, USA). The drysuit was a fully encapsulating textile garment with vulcanised seams (material density 1550 ± 100 g·m⁻²). The helmet was equipped with a 350 SuperFlow regulator and represents the standard configuration for U.S. Navy operations. External weights were added as necessary to offset suit and helmet buoyancy and maintain an upright posture while immersed without impeding movement or unnecessarily increasing underwater workload.

Sweat loss was estimated from changes in nude body mass across each trial. Nude body mass was measured before and after immersion using the same calibrated scale. Any urine produced after the pre-immersion body mass measurement was collected and weighed. Because subjects did not consume fluid during the immersion protocol, precooling, or post-testing, estimated sweat loss was calculated as the reduction in nude body mass corrected for urine loss.

Subjects entered a temperature-controlled annular pool set to 27.9 (SD 0.6)°C, 32.7 (0.3)°C, 37.7 (0.3)°C, or 37.7 (0.5)°C and walked for a maximum of 60 minutes at an exercise intensity of approximately 50% of $\dot{V}O_{2max}$, heart rate (HR) until core temperature (T_c) reached 38.5°C, or until the subject requested to stop. The order of exposure

was randomised and counterbalanced across subjects. Subjects were fully submerged throughout the experimental protocol. During immersion, HR and T_c were recorded every five minutes, and rate of perceived exertion (RPE), thermal comfort, thermal sensation, and sweating sensation were recorded every 10 minutes.

During the 38°C + Cool condition, prior to immersion, subjects donned a commercially available precooling vest (SICHNUL Summer Cooling Vest) that contained ice packs for 30-minutes. Heart rate and T_c were recorded every five minutes throughout the precooling period. Immediately following the 30-minute precooling period, subjects removed the cooling vest, donned dive gear and entered the water to begin the immersion protocol. For the protocols without a precooling intervention, subjects moved directly from the environmental chamber into dive gear and directly into the water.

Post-immersion testing was performed in a similar fashion in an environmental chamber set to 31.8 (0.1)°C and 71 (1.0)% relative humidity to preserve any heat gain from the water immersion during post-testing.

Following post-immersion measurements, subjects underwent a head-up tilt test (HUTT). Subjects were instrumented with equipment to measure HR (3-lead ECG), brachial blood pressure (Propaq CS, Welch Allyn, Skaneateles Falls, NY, USA), respiratory rate (RR), and partial pressure of end-tidal carbon dioxide ($P_{ET}CO_2$, nasal cannula; Reprise, Nonin Medical, Inc., Plymouth, MN, USA). Mean arterial pressure (MAP) was calculated. Following a two-minute supine baseline, subjects were tilted to 70° for up to 20 minutes, until subjects showed physiological or subjective signs of pre-syncope (e.g., nausea, tunnel vision, sweating, dizziness), or the subject requested to stop. Pre-syncope was identified by the onset of rapid, progressive decreases in blood pressure, resulting in sustained systolic blood pressure < 80 mmHg and/or relative bradycardia. Subjects were then returned to a supine position for recovery. The main outcome measure of the HUTT was total time.

STATISTICAL ANALYSIS

Data are presented as mean (SD). Changes in physiological variables were analysed using repeated-measures analysis of variance with factors for condition and time. Data were examined for outliers using a robust nonlinear regression. Linear mixed-effects models were used when data were missing intermittently to preserve within-subject comparisons. Model residuals were assessed using Q-Q plots to confirm normalcy, and Greenhouse–Geisser corrections were applied when sphericity was violated. Post hoc comparisons were performed using Tukey-adjusted multiple comparisons. Paired Student's *t*-tests were used for selected pairwise comparisons where appropriate. Orthostatic

tolerance during HUTT was analysed using Kaplan–Meier survival analysis with log-rank and Gehan–Breslow–Wilcoxon tests. Statistical significance was set at $P \leq 0.05$.

Results

Characteristics for all subjects ($n = 8$, four men, four women) are presented in Table 1. Core temperature increased over time in both 38°C conditions ($P < 0.001$). Mean Tc differed by condition, with higher values in 38°C + Cool compared with 28°C and 33°C ($P = 0.009$ and $P = 0.003$, respectively) and in 38°C compared with 28°C and 33°C ($P = 0.04$ and $P = 0.03$, respectively). No difference was observed between 38°C and 38°C + Cool. Maximum Tc was higher in both 38°C conditions compared with 28°C and 33°C ($P < 0.01$), with no differences between 28°C and 33°C or between 38°C and 38°C + Cool (Figure 1). One subject exceeded the Tc safety threshold (38.5°C) during 38°C, and four subjects terminated early due to heat-related symptoms. During 38°C + Cool, three subjects requested to stop early without exceeding the Tc threshold of 38.5°C. Mean time to termination was 47 min 56 s (38°C) and 40 min 02 s (38°C + Cool) (Figure 1).

Heart rate increased over time in the 38°C and 38°C + Cool conditions (time, $P < 0.001$). Mean HR differed by condition ($P < 0.01$) with higher values in 38°C and 38°C + Cool compared to 28°C (both $P < 0.001$). Heart rate was higher in 38°C compared with 38°C + Cool ($P < 0.001$). Heart rate during 33°C was also higher than 28°C ($P < 0.001$). (Figure 1)

Mean T_{sk} increased over time in 33°C, 38°C, and 38°C + Cool ($P < 0.01$). Mean T_{sk} was higher in 38°C than 38°C + Cool ($P = 0.02$). Maximum T_{sk} was greater in both 38°C conditions compared with 28°C and 33°C ($P < 0.001$), with no difference between 38°C and 38°C + Cool. Helmet temperature increased over time in all conditions ($P < 0.001$). Mean helmet temperature differed across conditions ($P < 0.001$). Maximum helmet temperature was higher in 38°C and 38°C + Cool than 28°C and 33°C ($P < 0.001$), with no difference between 38°C conditions. (Figure 2)

Rate of perceived exertion increased over time ($P = 0.02$). Mean rate of perceived exertion was higher in 38°C + Cool than 33°C ($P = 0.04$). Thermal comfort decreased over time in 33°C, 38°C, and 38°C + Cool ($P < 0.001$). Comfort ratings were higher in 38°C and 38°C + Cool than in 28°C and 33°C ($P < 0.01$), with no difference between the 38°C conditions (Figure 3). Thermal sensation increased over time in all conditions ($P < 0.001$) and was higher in 38°C and 38°C + Cool compared with 28°C and 33°C ($P < 0.01$). Thermal sensation was also higher in 33°C versus 28°C ($P = 0.01$). Sweating sensation increased over time in 38°C and 38°C + Cool ($P < 0.001$) (Figure 3). Across conditions, mean sweating sensation was higher in 33°C, 38°C, and 38°C + Cool compared to 28°C ($P = 0.04$), with the greatest differences observed in 38°C ($P = 0.03$) and 38°C + Cool ($P = 0.04$) (Figure 4).

Table 1

Subject characteristics; BMI – body mass index; bpm – beats per minute; HR – heart rate; SD – standard deviation

Characteristic $n = 8$	Mean (SD)	Range
Age (y)	25 (4)	20–31
Height (cm)	176 (8)	165–188.5
Mass (kg)	79.9 (14.4)	59.2–97.7
BMI (kg·m ⁻²)	25.6 (3.3)	21.8–30.9
Body Fat (%)	24 (10)	14–38
Resting HR (bpm)	66 (10)	53–82
Resting respiratory rate (bpm)	10 (2)	9–16
Resting diastolic blood pressure (mmHg)	112 (8)	100–122
Resting systolic blood pressure (mmHg)	70 (7)	60–76
VO ₂ max (mL·kg ⁻¹ ·min ⁻¹)	46.6 (9.2)	31.8–60.5

Figure 1

Heart rate (HR) in beats per minute (bpm) and core temperature (Tc) over time (A1 and B1, respectively); mean heart rate and core temperature between conditions (A2 and B2, respectively) (see text for explanation of conditions); data presented as mean with whiskers for SD

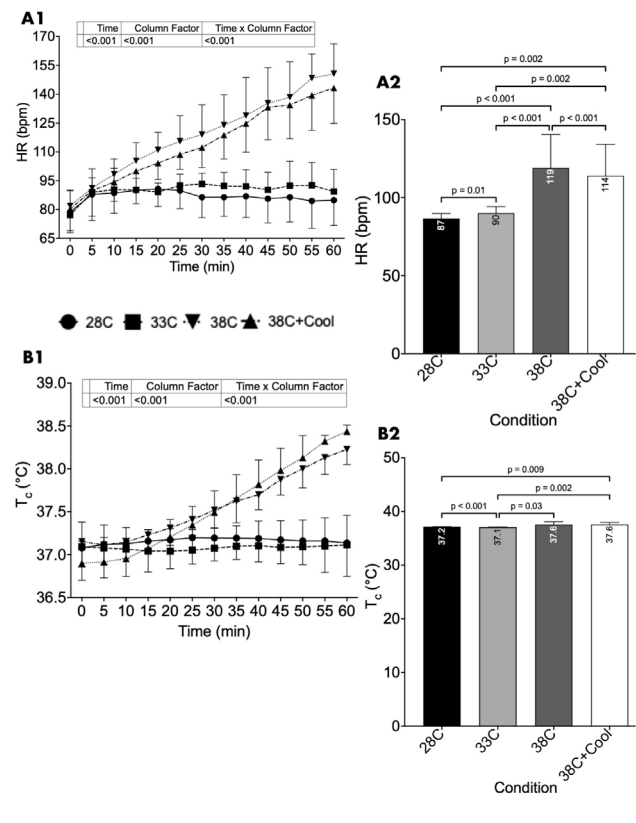
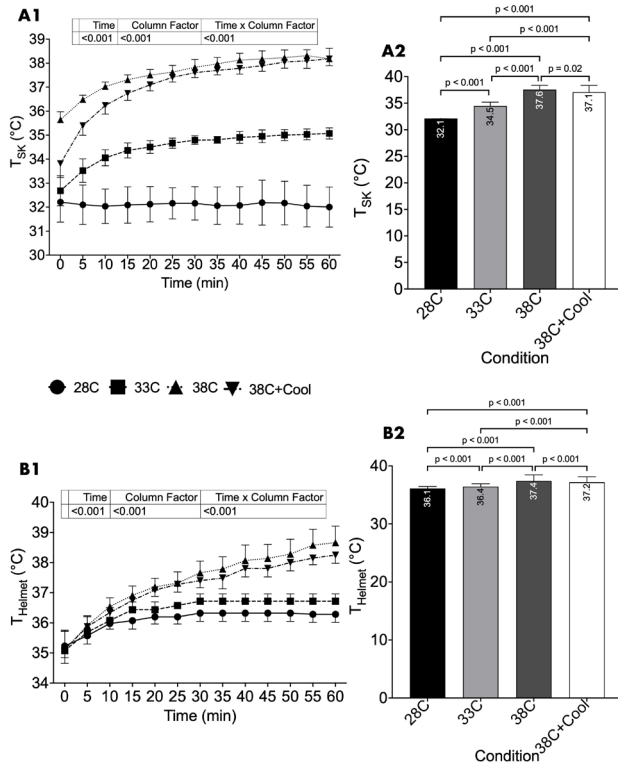


Figure 2

Mean skin temperature (T_{SK}) and helmet temperature (T_{Helmet}) over time (A1 and B1, respectively) and by condition (A2 and B2, respectively) (see text for explanation of conditions); data presented as mean with whiskers for SD

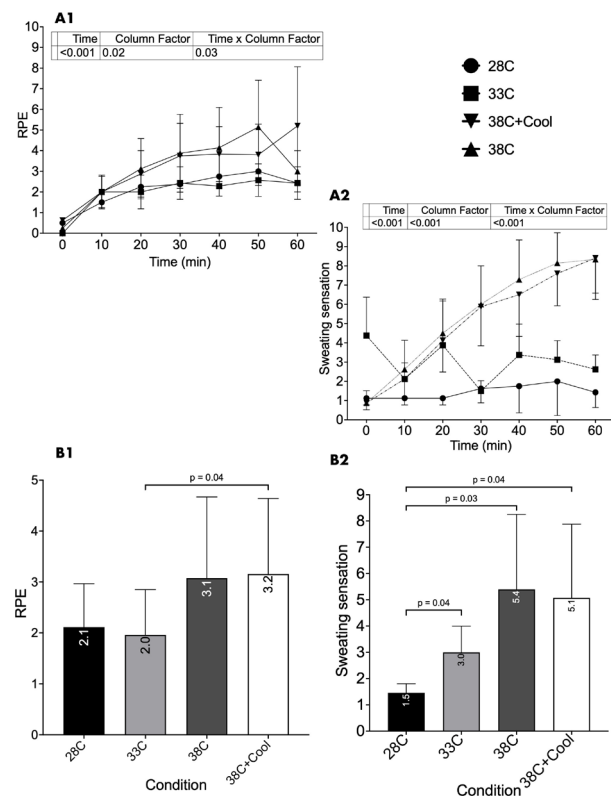


Core temperature differed across conditions during HUTT ($P < 0.001$), remaining higher in both 38°C conditions compared with 28°C and 33°C. T_c did not change over time during HUTT. Skin temperature decreased over time during HUTT in 33°C, 38°C, and 38°C + Cool ($P < 0.001$) and remained stable in 28°C. Mean T_{SK} during HUTT was higher in both 38°C conditions compared with 28°C and 33°C ($P < 0.001$), and higher in 33°C versus 28°C ($P = 0.005$), with no difference between 38°C conditions. Ambient temperature during HUTT did not differ between conditions. (22.1 (1.6)°C, $P = 0.98$). Heart rate increased over time in all conditions ($P < 0.001$). Mean HR was higher in 38°C and 38°C + Cool compared with 28°C and 33°C ($P < 0.001$), and higher in 33°C versus 28°C ($P < 0.001$), with no difference between 38°C conditions. Respiratory rate changed over time ($P < 0.001$). Mean RR was higher in 38°C + Cool and 28°C compared with 38°C and 33°C (all $P \leq 0.02$). End-tidal CO_2 did not change over time. Mean $EtCO_2$ was higher in 28°C compared with all other conditions ($P < 0.001$) and higher in 38°C + Cool compared with 38°C ($P < 0.001$) (Figure 5). Mean arterial pressure did not differ over time during the HUTT. However, MAP was lower during 38°C + Cool compared to 28°C ($P = 0.008$) and 33°C ($P = 0.02$). No other differences in MAP were found.

Survival analysis demonstrated differences in orthostatic tolerance across conditions (log-rank $P = 0.027$), with

Figure 3

Sweating sensation, and rate of perceived exertion (RPE) over time (A1 and A2, respectively) and by condition (B1 and B2, respectively); (see text for explanation of conditions); data presented as mean with whiskers for SD



a significant monotonic trend across increasing thermal stress ($P = 0.003$). Early-phase differences between conditions were also observed (Gehan–Breslow–Wilcoxon $P = 0.035$), indicating reduced tolerance with increasing water temperature (Figure 5). Estimated sweat loss differed across conditions and was higher in 38°C and 38°C + Cool compared with 28°C and 33°C ($P < 0.01$). Body mass decreased from pre- to post-exposure in all conditions ($P < 0.001$), with no differences between conditions (Figure 6).

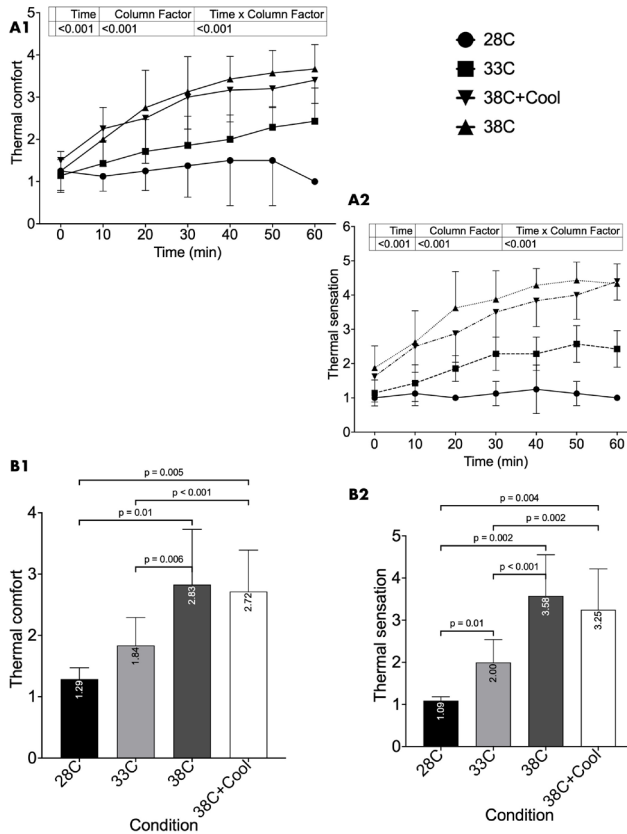
Discussion

Diving in warm water elicited substantial thermal and cardiovascular strain, characterised by progressive increases in core and skin temperature, elevated heart rate, and reduced orthostatic tolerance following immersion. These responses developed rapidly during 38°C exposures, were not meaningfully attenuated by 30 min of precooling, and persisted into the post-immersion period. Together, these findings indicate that encapsulated warm-water diving can produce uncompensable heat stress that compromises physiological stability during egress and recovery.¹⁴

The progressive rise in core temperature observed during 38°C conditions is consistent with uncompensable heat stress, in which environmental heat gain exceeds the capacity

Figure 4

Thermal comfort and thermal sensation over time (A1 and B1, respectively) and by condition (A2 and B2, respectively); (see text for explanation of conditions); data presented as mean with whiskers for SD

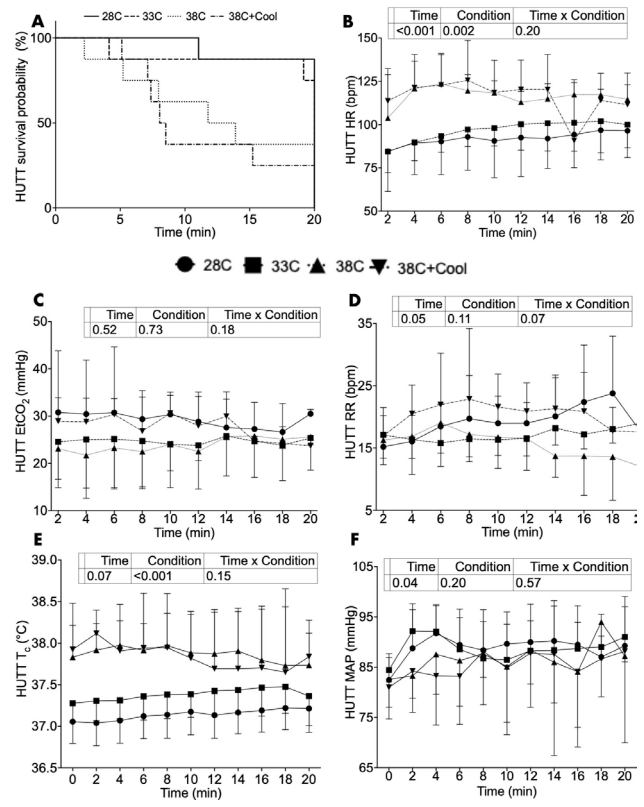


for heat dissipation. In encapsulated systems, evaporative heat loss is unavailable as a thermoregulatory pathway, leaving conductive heat exchange as the primary avenue for heat transfer.^{15,16} The effectiveness of this pathway depends on whether the surrounding water provides a gradient for heat loss or instead imposes an external heat load. These findings extend established terrestrial models of heat stress to immersed encapsulated conditions, demonstrating that even moderate-intensity work is sufficient to produce clinically relevant hyperthermia when environmental heat flux is sustained.^{3,4} Comparable core temperature elevations during warm-water immersion have been reported in both military and occupational diving contexts, consistent with known physiological responses to immersion in which combined environmental and equipment-related stressors alter thermoregulation and cardiovascular function.¹⁷

Heat exchange during immersion depends on the direction and magnitude of the temperature gradient between the body, protective ensemble, and surrounding water. In cooler water, conductive heat transfer favors heat loss from the body to the environment. As water temperature approaches skin temperature, this gradient narrows and conductive heat loss becomes progressively limited.¹⁰ When water temperature

Figure 5

Survival analysis (A), heart rate (HR, B), end-tidal carbon dioxide (EtCO₂, C), respiratory rate (RR, D), core temperature (T_c, E), and mean brachial arterial pressure (MAP, F) during the head-up tilt test (HUTT) over time; data presented as mean with whiskers for SD

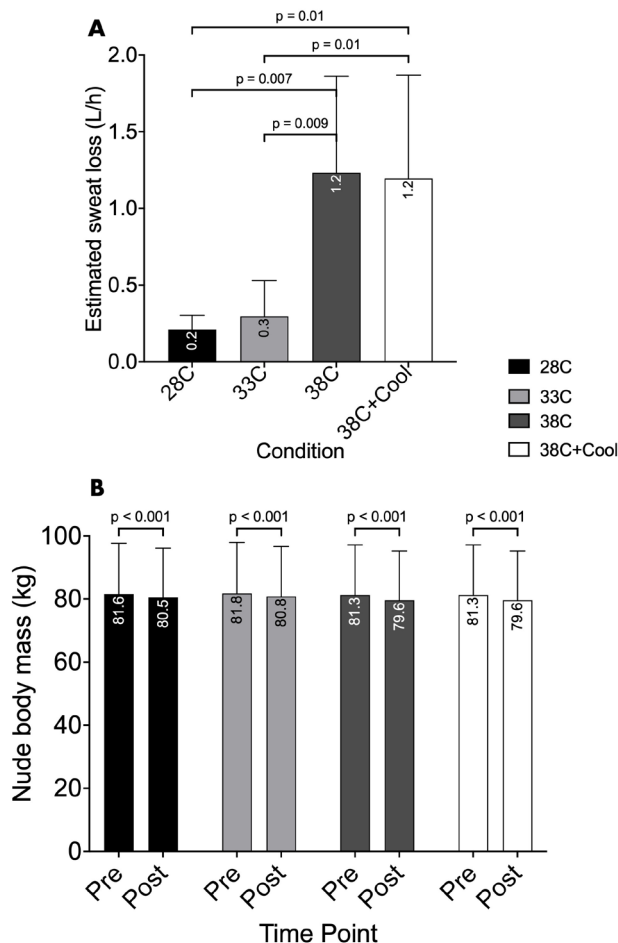


exceeds that of skin or peripheral tissue, the temperature gradient may reverse, promoting environmental heat gain rather than heat dissipation. Under these conditions, increases in cutaneous blood flow will not improve heat loss and may instead facilitate heat transfer from the periphery toward the core.¹⁸ In the present study, the 38°C water conditions likely approached or exceeded effective skin/peripheral temperatures, while encapsulation limited evaporative cooling, producing a thermal environment favoring progressive internal heat storage.^{19,20}

A key finding of this study was the reduction in orthostatic tolerance following warm-water immersion, which worsened with increasing thermal strain, indicating a direct relationship between heat load and cardiovascular instability. This response is consistent with established mechanisms of heat-induced orthostatic intolerance, including sustained vasodilation, venous pooling, and reduced central blood volume.²¹ The transition from immersion to upright posture represents a critical physiological stressor: hydrostatic pressure support for venous return is abruptly removed while peripheral vasodilation persists. Similar impairments in orthostatic tolerance have been observed following both passive heat exposure and exercise in the heat.^{7,22} This combination likely exacerbates reductions in arterial pressure regulation and increases susceptibility to presyncope.¹⁴ From an operational perspective, these

Figure 6

Estimated sweat loss during submersion (A) and change in body mass over the entire experimental visit (B) by condition; (see text for explanation of conditions); data presented as mean with whiskers for SD



findings identify the post-immersion period is a high-risk window for cardiovascular instability.

The head-up tilt test was included as a standardised post-immersion orthostatic challenge to assess cardiovascular stability during recovery. Total tilt tolerance time provides an integrated index of the ability to maintain arterial pressure under upright stress. This outcome is operationally relevant because divers must transition rapidly from hydrostatic support during immersion to unsupported upright posture during egress and post-dive recovery. In heat-stressed individuals, persistent cutaneous vasodilation, reduced central blood volume, and impaired cardiac filling can compromise arterial pressure regulation during orthostatic stress. The observed reduction in tilt-test tolerance following warmer water exposures supports the interpretation that residual thermal strain increases vulnerability to post-immersion cardiovascular instability.

Estimated sweat loss increased during warm-water exposure, exceeding $1.2 \text{ L}\cdot\text{h}^{-1}$ during the 38°C conditions compared with $< 0.4 \text{ L}\cdot\text{h}^{-1}$ at cooler temperatures. Because sweat loss

was estimated from changes in nude body mass, these values should be interpreted as estimates of whole-body fluid loss rather than direct measurements of sweat production. Despite this sustained increase in fluid loss, sweating likely contributed minimally to effective heat dissipation because the encapsulating suit created a high-humidity microenvironment at the skin surface, limiting evaporation and allowing sweat to accumulate within the suit rather than facilitating latent heat loss.²³ As a result, evaporative heat loss was effectively minimised, decoupling sweat production from heat dissipation.^{24,25} Consistent with this interpretation, core temperature increased progressively throughout the 38°C conditions despite greater estimated sweat loss. Under these conditions, sweating represents a physiologically costly and functionally ineffective thermoregulatory response. Although plasma volume was not directly measured, greater fluid loss may have contributed to cardiovascular strain when combined with peripheral vasodilation during egress and recovery.

Perceptual responses paralleled the physiological changes observed during immersion. Ratings of perceived exertion, thermal sensation, and thermal discomfort increased with thermal strain and tracked closely with rising core temperature. Subjective measures of thermal stress are often closely associated with physiological heat storage and may provide important operational indicators of diver tolerance.²⁶ Several subjects approached the predetermined safety cutoff of 38.5°C under 38°C conditions. One subject exceeded the cutoff during the 38°C exposure, and four additional subjects terminated exercise early due to nausea, lightheadedness, or general discomfort. Similar symptoms were reported during the precooling condition, in which three subjects voluntarily terminated the exercise early despite T_{c} remaining below the cutoff. These findings emphasise the operational relevance of heat strain during submersion, which may limit performance prior to critical hyperthermia and may serve as practical indicators of declining physiological tolerance and increase the chances of syncope upon egress.

Precooling with the cooling vest used in the present study did not meaningfully attenuate thermal strain during 38°C immersion. Although precooling can increase heat-storage capacity in terrestrial environments, the magnitude and duration of cooling provided by this intervention may have been insufficient for the uncompensable heat load imposed by encapsulated warm-water diving. These findings should not be interpreted as evidence that all precooling strategies are ineffective. More aggressive, longer-duration, or combined cooling strategies may provide greater benefit and should be evaluated in future studies.

Collectively, these findings support an integrated model of physiological stress during encapsulated warm-water diving. Progressive hyperthermia and vasodilation increase cardiovascular strain during immersion, while hydrostatic pressure partially preserves central blood volume. Upon egress, the loss of hydrostatic support, combined with

persistent vasodilation and post-immersion hypotension, reduces orthostatic tolerance and elevates the risk of syncope.^{27,28} This sequence provides a mechanistic explanation for operational observations of post-dive weakness and collapse, highlighting the transition from immersion to upright posture as a critical vulnerability. Operationally, these findings have direct implications for diver safety and exposure management. Thermal strain developed rapidly in warm-water conditions and is not fully mitigated by protective equipment or precooling. Importantly, risk extends beyond immersion into the immediate recovery period. Exposure guidelines should incorporate both in-water thermal limits and post-immersion recovery strategies. Interventions aimed at reducing heat storage, enhancing post-dive cooling, or mitigating venous pooling may be necessary to improve safety during warm-water operations.

LIMITATIONS

Several limitations should be acknowledged. The laboratory environment may not fully replicate operational diving conditions, including variable workloads, mission duration, and environmental exposures such as current, equipment variability, and task complexity. Additionally, direct measurements of stroke volume, central blood volume, and cerebral perfusion were not obtained, limiting mechanistic resolution. As a result, interpretations of cardiovascular strain and post-immersion orthostatic tolerance are based on indirect indices of haemodynamic function rather than direct quantification of cardiac preload, cerebral blood flow, or regional perfusion. The absence of these measures precludes definitive identification of the relative contributions of reduced central blood volume, altered cardiac function, and cerebrovascular regulation to the observed responses.

Conditions were separated by at least 72 h to reduce carryover effects. However, this interval may not have fully eliminated residual physiological or perceptual effects from prior exposures. Although trial order was randomised and counterbalanced, the possibility of carryover effects cannot be excluded. Hydration status was addressed through standardised pre-visit instructions and pre-exposure urine specific gravity screening but was not directly controlled through prescribed fluid intake or plasma volume assessment. Because hydration status can influence thermoregulatory strain, cardiovascular responses, blood volume, and orthostatic tolerance, residual differences in hydration status may have contributed to interindividual variability in the observed responses.

Mean skin temperature was estimated using a four-site model and iButton thermistors, which may not fully capture regional variation in skin temperature under an encapsulating drysuit. In addition, iButtons are larger than thin surface thermistors and may have locally altered conductive heat exchange between the skin, base layer, and suit. Therefore, skin temperature data should be interpreted as estimates

of regional thermal responses rather than direct measures of skin heat flux. Core temperature was measured using telemetric capsules that were either ingested before testing or inserted rectally immediately before exposure. Although this approach allowed continuous temperature monitoring during immersion, differences in capsule location may have introduced variability in the measured core temperature response.

Conclusions

Diving in warm water induces uncompensable heat stress characterised by progressive hyperthermia, peripheral vasodilation, and increased cardiovascular strain that are not mitigated by precooling. These responses persist beyond immersion and reduce orthostatic tolerance, indicating impaired central haemodynamic stability. The transition to upright represents a period of increased physiological vulnerability and risk of syncope. Thermal load is a primary limitation to diver safety and performance in warm-water environments. Mitigation strategies should address both in-water heat strain and post-immersion orthostatic tolerance.

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Data availability statement

Deidentified data are available upon reasonable request to the corresponding author and after approval by the Institutional Review Board.

Conflicts of interest and funding

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