Immersion Treatment for Exertional Hyperthermia: Cold or Temperate Water?

PREVAILING VIEW
Our viewpoint in this Contrasting Perspective stems from the primary consideration that: Cold water cools faster than other cooling modalities. This minimizes the amount of time an exertional heat stroke (EHS) victim is severely hyperthermic, which dictates outcome. Current evidence strongly indicates that rapid cooling via cold/ice water optimizes survival of persons with EHS.

Physiological and Biophysical Considerations
Water immersion produces high rectal temperature cooling rates: the greatest recorded are in 2°C ice water (0.35°C·min⁻¹), with rates approximately two times greater than that of warmer water (34). Taylor et al. (36) reported rectal temperature cooling of 0.18°C·min⁻¹ and 0.10°C·min⁻¹ for immersion in 14°C and 26°C water. Despite lower cooling rates, they concluded that water with a temperature of 26°C is sufficient for cooling hyperthermic individuals because cooling esophageal temperature to 37.5°C in water with a temperature of 26°C took <3 min (36). However, despite decreasing esophageal temperature to 37.5°C, significant residual heat storage likely remains with shorter immersion, and rectal temperature remains ≥39°C (34). In EHS, risk of adverse outcomes is related to the duration of visceral organ hyperthermia (4,11).

Heat loss provides a more objective evaluation of core-cooling effectiveness than core temperature (Tco) response. Proulx et al. (33) reported that heat gained during exercise in the heat (rectal temperature of 40°C) was eliminated after 5.4 min of immersion in ice water with a temperature of 2°C compared with 7.9, 10.4, and 13.1 min of immersion in ice water with temperatures of 8°C, 14°C, and 20°C, respectively. Thus, although ice water immersion may induce skin vasosconstriction, heat dissipation by conduction/convective heat transfer is unimpeded (33). The temperature gradient between skin and water after immersion in 2°C water was 1.8, 2.5, and 2.6 times greater than the gradient between skin and water in 26°C.

CHALLENGING VIEW
Cold-water immersion is recommended as the gold standard for treating exertional hyperthermia (11). However, the universal application of this treatment is challenged based on differences between meaningful and apparent core cooling, and how water temperature may affect convective heat delivery to the skin. First principles are not debated, nor is the urgency with which cooling should be invoked. It will be recommended that, if your patient has a viable cutaneous circulation, cooling in temperate water (24°C–26°C) will be equally fast, less hazardous, and more comfortable. The logic that underpins this challenge is founded within the physiological significance of several elementary facts, and the analogies that follow summarize these first principles.

For simplicity, consider a bowling ball of homogeneous composition and thermal conductivity. Seal temperature sensors within its structure, and immerse it in hot water until its central temperature stabilizes. Now place the ball into cold water. The resulting changes in sensor temperature are dictated by thermal gradients between the water and each sensor. Temperature differences among sensors are a function of the distance each sensor is from the ball surface.

Now, take a human and place temperature sensors within the esophagus and rectum. Heat this person to an esophageal temperature of 39.5°C, and hold that temperature until all tissues approximate a thermal equilibrium. Place this person into cold water. Notwithstanding variations associated with body shape and size differences, the precision with which esophageal and rectal temperatures track ball temperature is a reflection of the extent to which first-principles thermodynamics dictate human heat transfer. However, temperature deviations are associated with the existence of physiological regulatory systems, and in particular, mechanisms that regulate body temperature.

For bowling balls, central heat loss is determined by conduction. For humans, central heat loss occurs via conduction and convective (mass) flow. Thus, variations in heat transfer that cannot be explained on the basis of first-principles thermodynamics must be functions of modified vascular responses, either at the periphery or the core. When there are no changes in central blood flow during cold-water immersion, then changes in core cooling...
greater than during immersion in 8°C, 14°C, and 20°C water, respectively, allowing for heat to be dissipated faster (33). The higher temperature gradient between skin and water (7.7°C) decreases the importance for skin perfusion. Taylor et al. (36) argued that elevated skin–water gradients are not required to maintain elevated heat transfer. They suggest that immersion in 26°C water reduces skin vasoconstriction, sufficiently maintaining heat extraction and reducing $T_{co}$ to safe levels. However, rectal cooling rate of 0.10°C min$^{-1}$ during immersion in 26°C water is more than three times less than the 0.35°C min$^{-1}$ during immersion in 2°C water. Further, shivering thermogenesis is minimized by shorter exposure in 2°C water (24, 34). Taylor et al. reported immersion times of <3 min on the basis of an exit esophageal temperature of 37.5°C. Although shivering thermogenesis was probably negligible for this short period, a significant residual heat load likely remained (33).

Although Taylor et al. concluded that water temperature >14°C provides rapid heat removal to safe $T_{co}$ levels, this may not be true for big individuals experiencing EHS. Individuals with a greater body mass must lose more heat than smaller individuals do to achieve equivalent reduction in $T_{co}$. In fact, individuals with greater body mass typically demonstrate smaller decreases in $T_{co}$ during cold exposures (40). Fat mass and fat-free mass have different thermal conductivities (3), and high levels of adipose tissue and vasoconstricted muscle tissue contribute to greater total body insulation and reduced $T_{co}$ decay (20, 38). However, differences of low to moderate adiposity (10%–29%) do not affect core cooling during immersion in 8°C cold water after exercise-induced hyperthermia (23). Further, differences in lean mass between individuals are more important in determining rectal temperature cooling rates after exercise-induced hyperthermia than surface area–to-mass ratio and adiposity (24). Taken together, cold-water immersion (CWI; <10°C) can minimize variations in cooling rate caused by large differences in physical characteristics compared with warmer water temperatures.

Medical Considerations—Cooling Efficiency and Survival Rates

Immersion in ice water within a few minutes after collapse or presentation of symptoms has provided 100% survival for the ~400 EHS victims in the 37-yr history of the Falmouth Road Race in Falmouth, MA (32). This race routinely has 8–10 EHS cases per year, with starting temperatures between 106°F and 112°F (41.1°C–44.4°C). In the medical tent, 12 Rubbermaid tubs are filled with water and ice (water temperature < 50°F (10°C)) (32). On arrival, the suspected EHS victim has a rectal thermometer placed and cooling begins within 2–3 min. Patients are typically immersed for 10–20 min ($T_{co}$ drops primarily reflect modulations in skin blood flow. If skin blood flow remains stable between successive immersions of the same individual, then variations in core cooling reflect fluctuations in blood flow at the site of the temperature sensor or local metabolic heat production. If one can accept that these simplifications are based in fact, then these contrasting perspectives can be reduced to differences in data interpretation and the application of this knowledge.

Clearly, the volume-specific heat capacity of water demands that it be advocated as the cooling medium of choice, but which immersion temperature should be used: cold or temperate? At first glance, the notion of using temperate water may seem absurd. However, we have demonstrated that immersion of individuals with rectal temperatures >40°C in temperate water (26°C) will elicit core cooling in <3 min (36). Thus, the challenge is to consider using temperate water to treat exertional hyperthermia, and the answers to two questions may prove helpful in reaching this decision. Should water temperature efficacy be evaluated on the basis of rectal or esophageal tissue cooling rates? A reliance on the former index is central to the prevailing view. Does skin blood flow influence core cooling? The prevailing position seems to dismiss this possibility.

Esophageal versus Rectal Temperature

Local tissue temperatures, and their rates of change, are dictated by thermal energy transfers and local heat production. For deep-body structures, heat is transferred through conductive and convective pathways. Because convective heat loss is a function of local blood flow and the thermal gradients that exist downstream from the core tissues, then variations in esophageal or rectal tissue blood flows can have profound influences on these temperatures.

There is no reason to suspect that esophageal or rectal tissues receive a blood supply other than that necessary to support metabolism, because these are conduit structures. Both sites presumably have similar mean metabolic rates. Nevertheless, between the fifth and tenth thoracic vertebrae, the esophagus is near the left atrium and several intrathoracic blood vessels, including the aorta, whereas the rectum has no close association with similarly large blood vessels (25). These anatomical differences dictate that esophageal temperature rapidly tracks changes in the thermal energy content of the central blood volume (26, 39), whereas rectal temperature responds only when its blood supply changes temperature or when conductive heat transfer affects local heat storage. Thus, the time constants for esophageal and rectal temperature changes vary systematically, with the latter invariably being much slower (29, 39).

Protagonists of the prevailing view advocate rectal temperature as the criterion core temperature (16), suggesting...
Nearly all are discharged by a physician after monitoring without being sent to a hospital (22). The Marine Corps Marathon also uses ice water to cool EHS patients. Helpers (4–6) continuously douse the patient’s body with cold water, while major muscle groups are simultaneously massaged with large ice bags (28). Again, 100% survival is observed when treatment begins immediately after collapse (28). Reported cooling is approximately 70% as effective as CWI, and cooling is usually completed in 15–20 min (28). Similar efficacy has been reported when CWI or dousing was used at the Peachtree Road Race (Cooper 2009, personal communication), US military instillations at Quantico (1), Parris Island (14), Fort Benning, and others. Medical professionals at these races believe that to maximize survival, the time that the individual remains hyperthermic (Tc >105.5°F/40.8°C) must be minimized.

The evidence of Hubbard et al. (18), using an animal model for obvious reasons, showed that the time above a critical temperature was a key factor that dictated survival rate. When they increased the amount of time the rats remained above 104.7°F (40.4°C), survival rate plummeted for both passive heat stroke and EHS animals (18). Figure 1 clearly shows that survival is dictated by the duration of severe hyperthermia (18). Fortunately, the literature for humans indicates an even better survival rate. When they increased the amount of time the rats remained above 104.7°F, survival rate plummeted for both passive heat stroke and EHS animals (18). Again, 100% survival is observed when treatment was started immediately after collapse (28). Reported cooling was approximately 70% as effective as CWI, and cooling was usually completed in 15–20 min (28). Similar efficacy has been reported when CWI or dousing was used at the Peachtree Road Race (Cooper 2009, personal communication), US military instillations at Quantico (1), Parris Island (14), Fort Benning, and others. Medical professionals at these races believe that to maximize survival, the time that the individual remains hyperthermic (Tc >105.5°F/40.8°C) must be minimized.

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Because the length of time an individual is severely hyperthermic dictates survival after EHS, it is imperative to use cooling modalities that promote the most rapid cooling.

![Survival as it Relates to Severity and Duration of Hyperthermia in a Rat Model of Heat Stroke](image)

FIGURE 1—Relationship between severity of hyperthermia measured in degree-minutes and rat survivability. These data are redrawn from Figure 1 of reference 18. Thermal area (degrees × minutes) = ∑ time interval (min) × ½°C above 40.4°C at start of interval + °C above 40.4°C at end of interval.

Core cooling rates cited to support ice-cold water immersion were derived from rectal temperatures (11,27). Curiously disregarded is the fact that, across water immersion temperatures from 2°C to 20°C, esophageal cooling rates reported by Proulx et al. (33) (their Table 2) did not differ significantly from one another, whereas faster rectal cooling in the coldest conditions was selected to support the prevailing position.

Our rectal and esophageal data from cold-water immersion (14°C) are consistent with those of Proulx et al. (33) at this temperature, yet our interpretation differs. In addition, we have demonstrated that, although rectal cooling remains slow, esophageal cooling from 39°C to 37.5°C takes just 2.91 min (36), and from 38.5°C to 37.5°C, it can be achieved in 2.56 min (9), when subjects are immersed in temperate water (26°C). These cooling times were just 45–55 s slower than observed during cold-water immersion (14°C). Neither the absolute cooling times nor the time differences are likely to have adverse clinical implications. However, how can one explain the paradoxical similarity of core cooling during temperate and cold-water immersion?

**Cutaneous Circulation**

Cold-water immersion elicits cutaneous vasoconstriction and venoconstriction in normothermic individuals (21,35), with sympathetic drive being very powerful in water <10°F (22). Presumably, a water temperature exists at which whole-body cutaneous constriction becomes maximal, with heat loss now being dictated by conduction. However, it has recently been shown that water and core temperatures influence constrictor strength (9), with significantly greater constriction evident in cold (14°C) than in temperate water (26°C), although constriction was less powerful in hyperthermic individuals. Thus, convective heat delivery to the periphery was reduced during the colder immersion, resulting in clinically insignificant differences in central blood volume cooling for these profoundly hyperthermic individuals.

Of course, if a viable cutaneous circulation does not exist before immersion (31), then this convective path is ineffective, and colder water will extract heat faster. However, not all heat stroke patients experience circulatory collapse (2,17).

**REPLY TO THE PREVAILING VIEW**

It is well established that rapid cooling enhances the survival of profoundly hyperthermic individuals. Equally...
cooling. The evidence from the literature shows that 14°C water immersion provides a rapid drop in $T_{co}$ (~1°C every 5 min and 1°F every 3 min). However, a more substantial drop occurs with immersion in 2°C ice water (~1°C every 3 min and 1°F every 2 min) (10–12,14,32–34,36,37). Generally, cold- or ice-water immersion will require EHS patients to be cooled for about 10–17 min to reduce $T_{co}$ from a starting ~42.2°C (108°F) to 38.9°C (102°F). Most importantly, patients would only be above a theoretical critical threshold for cell damage of ~40.8°C (105.5°F) for 5–10 min after the onset of cooling (18). Given that no known adverse effects exist from CWI (8) for an otherwise healthy individual who is experiencing an EHS, it seems prudent to use an inexpensive, readily accessible modality proven to guarantee survival (in the EHS cases treated to date) when used within 5 min after collapse. Although we recognize that cold- or ice-water immersion may not be feasible in all field settings because of logistics in military training and battle situations as well as some athletic and manual labor circumstances, we do feel that, for most events, such as athletic (mass medical, sidelines of high school, college, and professional sports), military basic training, and other events, the medical staff should be prepared to use the optimal cooling modality (4). Rubbermaid tubs filled with ice and water can be easily made available with minimal effort and cost (11). In circumstances where CWI cannot be used, a suitable alternative should be considered (milder water temperature baths, cold showers, and rotating ice/wet towels) (11). If possible, the cooling should be performed while transporting the patient to a more optimal cooling facility where CWI can be performed within 15–30 min of collapse. If this is not possible, Herculean efforts should be made on site to use all available means to rapidly lower $T_{co}$ of the EHS patient.

**REPLY TO THE CHALLENGING VIEW**

The fact that survival from EHS is critically dependent on how quickly core temperature can be reduced by the treatment intervention is a point of agreement by both sides. Moreover, there is a clear consensus that water immersion is the most effective way with which to extract heat. However, in question is whether temperate water (~26°C) immersion provides an equally effective condition to achieve a rapid reduction in core temperature to that observed using cold water (~2°C). Current evidence strongly supports the use of CWI as the most potent method for rapidly reducing body temperature. This is because CWI (2°C) provides some of the highest core cooling rates (0.35°C/min$^{-1}$) especially for the second-degree drop in core temperature (0.50°C/min$^{-1}$) (34). These rates are ~3.5–5 times greater than those rates reported by Taylor et al. (36) using temperate water.

Clear is the fact that whole-body immersion provides the most practical and effective cooling. However, although ice water is clearly effective, it is not correct to suggest that it is the only means through which rapid cooling can be achieved. It is also incorrect to claim that there are no known adverse cold-water immersion. The literature favors the prevailing perspective because most groups did not consider the possibility that temperate water could provide an effective treatment, and because cooling was evaluated using inappropriate temperature measurements.

Esophageal temperature measures neither brain nor rectal temperatures. Similarly, carotid artery temperature reflects blood and not brain temperature. However, the heat content of this blood results from heat exchanges that occurred during its last transit through the body, and this influences cranial heat exchange. Because the esophagus lies close to the left atrium and aorta, then its temperature is influenced by that of the central blood volume, and this temperature may provide significant prognostic information. However, the hypothesis that rectal temperature provides a superior quantification of the thermal states of the central nervous system, central blood volume, or visceral organs awaits verification.

In the meantime, it is advocated that cooling recommendations be based on core temperature indices for which this relationship has long been established (6,13,26,30,39). In recent experiments involving temperate water immersion and esophageal temperature measurement (9,36), attempts were made to provide a balance that might permit others to undertake an evidence-based evaluation of water immersion cooling. Immersion in 26°C water cooled profoundly hyperthermic people from 39°C to 37.5°C in <3 min. Reducing water temperature by 12°C cooled subjects only 45–55 s faster. Is the cooling rate in temperate water not fast enough to save lives?

**Heat Loss**

Calorimetric methods can provide significant physiological insight. However, tissue damage results when local temperatures move outside a critical range. Therefore, heat storage and tissue temperatures determine pathological outcomes. Nevertheless, Proulx et al. (33) derived heat storage from 3 core and 11 skin temperatures (partitional calorimetry), and heat loss using heat flux transducers. The implicit assumption of the former method is that thermal equilibrium is approximated among tissues. Because rectal tissues exchange heat primarily through conduction, whereas convective delivery (mass flow) dominates thermal exchanges for tissues close to the heart and large blood vessels, then satisfying this assumption requires time, as described by Jay and Kenny (19) and the accompanying commentaries. However, rectal temperatures continued to rise after exercise and during the first minutes of immersion (33). This phenomenon is
The notion that heat loss is restricted during immersion in CWI as a result of intense vasoconstriction serves as the premise for Taylor’s argument from which he advocates the use of temperate water immersion (26°C) as the preferred method to enhance cooling efficiency. It is argued that rapid and effective heat loss in the absence of an elevated skin–water gradient can still be achieved using temperate water immersion. However, as reported by Proulx et al. (34), the temperature gradient between the skin and the ice water is so great (7.7°C) that an elevated skin perfusion is not essential for the body to cool. Although Taylor et al. (36) noted that immersion in temperature water resulted in esophageal cooling to 37.5°C of less than 3 min, they failed to consider that a significant residual heat storage likely remains, as does a corresponding elevated rectal temperature of ~39°C–39.5°C (33).

EHS typically includes a precipitous drop in blood pressure accompanied by a rise in both HR and respiratory rate, which is exacerbated by the exercise-induced state of hypovolemia (15,31). This is associated with 1) marked reductions in renal and splanchnic blood flow that can prolong the duration of vital visceral organ exposure to hyperthermia and hypoxia and 2) reductions in skin blood flow at a time when delivery of blood to the skin is essential for heat transfer. Under these conditions, the need to alleviate visceral organ hyperthermia is paramount and is best achieved using CWI (34). Moreover, whether the hyperthermic individual is under a state of circulatory collapse or not, CWI will extract heat faster (33) regardless of the level of attenuation in skin perfusion before the start of the treatment.

In addition to the physiological adjustments in response to cold stress (i.e., shivering and skin vasoconstriction), there are important biophysical factors that can influence this response. Increases in body mass and body adiposity can significantly impede heat extraction and therefore reduce core cooling rates during CWI. Studies by both Proulx et al. (34) and Taylor et al. (36) derived core cooling rates on the basis of data obtained from a highly physically active population and do not consider possible biophysical variations among individuals. Bowling ball analogies aside, the principles of thermodynamics dictate that a colder water bath is required to increase heat transfer in larger individuals. Indeed, recent evidence demonstrates that CWI (≤58°C) can minimize variations in cooling rate caused by large differences in physical characteristics compared with warmer water temperatures (23).

The key to maximize the chances of surviving EHS is rapidly decreasing the elevated core body temperature. There is increasing clinical evidence that supports the notion that CWI will maximize the likelihood that an individual will survive EHS. For those who successfully a characteristic of bowling balls and rectums, and shows that, although a positive thermal gradient existed for the superficial and well-perfused tissues, there was a negative gradient for some core sites. These opposing gradients violate steady-state assumptions. Furthermore, the esophageal cooled rapidly during immersion, whereas the rectum cooled five times slower. During the post–immersion phase, the rectal temperature afterdrop was 2.4–5.8 times greater than that for the esophagus. Indeed, when heat losses required to return subjects to normothermia were derived, the difference between rectal (always higher) and esophageal temperatures increased as the water temperature decreased, implying a longer conductive pathway. Thus, at 2°C, the rectum was still 3.3°C warmer after 5.4 min, when subjects were supposedly normothermic. These thermal gradient variations during dynamic phases act to question the objective merit of these heat loss data. However, how then does one dissipate this latent rectal heat? Be patient, for hypothermia will eventually accompany temperate water immersion.

**Body Mass**

Body composition variations are important, but larger people must lose the same thermal energy per unit mass to attain normothermia. Fortunately, such individuals have larger surface areas, which, for the same stature, increase ~100 cm²kg⁻¹. Lemire et al. (23) found no differences in cooling rate due to adiposity, but only evaluated water at 8°C. However, esophageal cooling to 37.5°C occurred within ~3 min. Moreover, esophageal cooling in water at 14°C (36) (0.86°Cmin⁻¹) was slightly faster for subjects 9 kg heavier than those of Proulx et al. (33) (0.77°Cmin⁻¹). Finally, this author counsels against applying rectal cooling observations from thermoneutral individuals (40) to profoundly hyperthermic subjects. Indeed, if one lengthens the conductive pathway to the rectum by using heavier, vasoconstricted subjects, then one must not express surprise when discovering slower rectal cooling. The gold standard must be esophageal temperature.

**Adverse Effects**

The physiological significance of cold shock cannot be dismissed. As previously noted (36), sudden cold immersion can result in significant and adverse physiological changes. These are a function of the rate and magnitude of the skin temperature change, and because hyperthermia elevates skin temperature, then the risk of cold shock in susceptible individuals is elevated. Of course, some individuals are broadly resilient to such stress, but why advocate such extreme cooling when less stressful cooling is equally effective, infinitely more comfortable, and less likely to elicit undesirable adverse effects.
treat EHS on a regular basis, we are encouraged by three facts that are ever-present during our clinical experience:

1. The determination of hyperthermia (as identified with an immediate rectal temperature) before treatment is a foolproof manner for identifying EHS victims (along with the presence of CNS changes). In addition, cooling a hyperthermic individual without EHS using CWI will not adversely affect their health (if rectal temperature is not available and degree of hyperthermia cannot be measured).

2. CWI provides a rapid drop in rectal temperature (remember to aggressively stir water during cooling). The time taken to achieve reductions in core temperature from a typical starting temperature (41°C–43°C) to termination temperature (38.5°C–39.5°C) is very rapid using CWI (~15–20 min).

3. When CWI treatment is begun within 5 min of collapse, survival rate has been reported to be 100% thus far (11). When CWI is not available (or until it becomes available), cool via the next best available cooling modality.

**CONCLUDING STATEMENT**

Although not all cases of EHS can be prevented, the likelihood of morbidity and mortality associated with the condition can be prevented by rapidly implementing appropriate recognition and treatment strategies. Military, industrial, and athletic activities performed under a supervised setting can implement multiple strategies to mitigate the risk of EHS. In the unforeseen chance that an individual is a victim of EHS, CWI is the preferred treatment modality.

**Douglas J. Casa**

*The Korey Stringer Institute*

*Department of Kinesiology*

*University of Connecticut*

*Storrs, CT*

**Glen P. Kenny**

*Human and Environmental Physiology Research Unit*

*School of Human Kinetics*

*University of Ottawa*

*Ottawa, Ontario, Canada*

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