

Chapter 17

COLD WATER IMMERSION

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INTRODUCTION

The body core temperature is maintained daily within a narrow range, despite wide variations in environmental (ambient) conditions and activity levels.¹ In fact, the body core temperature is so consistent that for centuries physicians have used deviation from this normal temperature as an indication of a departure from the healthy state.² Maintenance of a normal temperature and the normal variations of the circadian and lunar rhythms are achieved by changes in all physiological systems, one of the most important of which is alteration in skin blood flow.³ In response to the perception of skin temperature, behavioral adjustments are made in clothing, environment, or both, to avoid sweat-

ing or shivering (ie, to maintain the core level). When exposed to more extreme environmental conditions, powerful physiological effector mechanisms are called into play to defend against hyperthermia and hypothermia. Active vasodilation of blood vessels of the skin and secretion of sweat keep body temperature within a few degrees of normal even in severe conditions of increased ambient temperature, such as marathon running on a hot day.⁴ On the other hand, the defenses against hypothermia are less effective. Individuals exposed to severe cold are unable to maintain thermal steady states for extended periods despite maximum vasoconstriction and high rates of shivering.

REGULATION OF BODY HEAT CONTENT

The physics of heat loss following cold water immersion dictate a more rapid drop in core temperature than from exposure to a cold air environment.⁵ Figure 17-1 illustrates the dramatic difference in body (core) temperature drop when the same individual was exposed alternately to 0°C air and immersed in 10°C water. Magnitude of heat loss and eventual decrease in core temperature depends to a great extent on how much of the body is actually immersed.⁶ In the following discussion, *immersion* is defined as submerged to the neck.

When a body is immersed in water it is obvious that evaporative and radiant heat exchange can no longer occur between the subject and the environment. The major heat exchange in water occurs by means of conduction with the surrounding water. The exceptions to this are the nonimmersed body parts, in most cases the head. The head can represent a significant site of heat loss to the environment owing to its minimal insulation and lack of vasoconstriction.⁷ For example, when the ambient temperature is 4°C, the uncovered head could be responsible for heat loss representing 50% of resting heat production, increasing to 75% at an ambient temperature of -15°C.⁸

The heat dissipation capacity of water is considerably greater than that of air, as the ratio of heat conductivity of water to air is approximately 24:1.⁹ A body immersed in calm water is surrounded by a relatively stable "boundary layer" of fluid. Heat is conducted from the skin into this boundary layer as a function of the temperature gradient, heat transfer coefficient, and surface area of contact,¹⁰ as Equation 1 demonstrates:

$$(1) \quad H = K A (T_w - T_{sk})$$

where H represents heat transported, K the heat transfer coefficient, A the surface area of contact between skin and water, T_w the temperature of water, and T_{sk} the temperature of the skin.

Conservation of body heat and maintenance of core temperature during immersion depends on the temperature of the water. If there is no net heat loss or gain by the body during immersion, the water temperature is within the thermoneutral zone (also called the neutral zone or the vasomotor zone). The

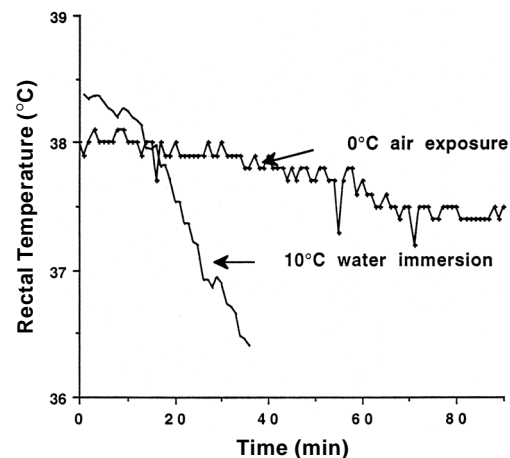


Fig. 17-1. Rectal temperature versus time relationship in one human volunteer under two types of cold stress: 0°C air and 10°C water. The subject is a young white man of average build, fat content, and physical fitness. He is dressed the same for both sessions. Note that even though the water temperature is 10°C higher than the air temperature, the decrease in core temperature is much more rapid in the water immersion, owing to the increased conductivity of the water compared with that of air.

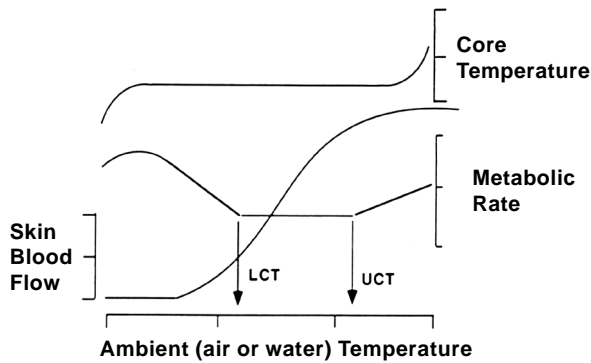


Fig. 17-2. The relationships between core temperature, metabolic rate, and skin blood flow as a function of ambient temperature. The thermoneutral zone is represented by the horizontal segment of the metabolic rate curve and is bounded by the upper critical temperature (UCT) and the lower critical temperature (LCT). In the thermoneutral zone the core temperature is maintained constant by alterations in heat exchange at the surface, represented here by changes in skin blood flow.

Adapted with permission from Eckert R, Randall D, Augustine G. *Animal Physiology: Mechanisms and Adaptations*. New York, NY: WH Freeman; 1978: 580.

ambient temperature defining the thermoneutral zone is a very narrow range around 35°C.^{11,12} Within this limited range of ambient temperatures, nude subjects maintained their core temperature nearly constant for at least 1 hour without shivering or sweating. This ability to maintain a steady state in core temperature was attributed to changes in skin blood flow; that is, vasomotor control.^{12,13} In the thermoneutral zone, skin blood flow is under the reflex influence of skin temperature. An increase in skin temperature results in a rapid increase in skin blood flow and vice versa. It is from this thermoneutral zone that the body moves when cold water immersion occurs, calling into play powerful compensatory mechanisms. The concept of the thermoneutral zone and temperature regulation is summarized in Figure 17-2.

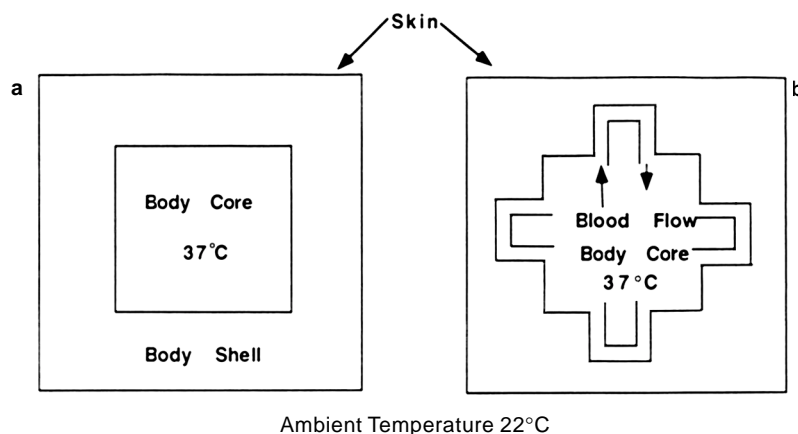


Fig. 17-3. A summary of the Shell-Core model of heat flow in the body. (a) If the blood flow to the shell is minimal, it will provide the largest insulating capacity and therefore retain heat and maintain core temperature. (b) When vasodilation in the shell occurs, the insulating capacity of the shell drops and heat is lost from the core.

Adapted with permission from Vander A, Sherman J, Luciano D. *Human Physiology: The Mechanisms of Body Function*. 7th ed. Boston, Mass: WCB McGraw-Hill; 1997: 628.

As the water temperature falls below the lower critical temperature, peripheral vasoconstriction can no longer effectively prevent heat loss. Therefore, heat loss exceeds the heat produced by the basal metabolic processes, and heat production must be increased to maintain the core temperature. The lower critical temperature is a function of subcutaneous fat thickness and ranges between 30°C and 34°C.¹⁴ Increased heat production can be accomplished by an increase in metabolic rate,⁵ voluntary muscle work (exercise), or shivering, in any combination. Although these three mechanisms will contribute to the maintenance of core temperature, in a cold water environment, the added physical activity associated with exercise will tend to disrupt (ie, to mix) the boundary layer and, to some extent, facilitate heat loss. These mechanisms (increased heat production and maximal vasoconstriction) may be able to stabilize core temperature for some time. However, if the cold stress is of sufficient magnitude and is continued for a long enough time, heat loss will eventually exceed heat production, leading to a decrease in core temperature and eventually to hypothermia. As the core temperature declines past a critical level, the rate of both tissue metabolism and shivering will be depressed, resulting in an accentuated drop in core temperature due to this depression of heat-production mechanisms.

The problem of maintaining core temperature during cold water immersion can be modeled as a simple two-compartment system in which the body core is separated from the surrounding environment by an insulating shell (Figure 17-3). Heat can move from the core through the shell by conduction, essentially a diffusive process, but this mechanism is a very slow transport process, owing to the distances involved. The major pathway for heat movement out of, or heat exchange with, the core and into the shell and subsequently facilitating transfer

EXHIBIT 17-1

RANGER TRAINING INCIDENT REPORT

On 15–16 February 1995, three students in training to become US Army Rangers, ranging in age from 23 to 27 years, died of hypothermia during a routine patrol at the Florida Ranger camp. Failure in supervision and judgment, lack of situational awareness, and lack of control placed Ranger students in water too cold, too deep, and too long for safe operations.

The Ranger Training Brigade trains about 3,000 Ranger students each year. This training takes place in Fort Benning, Georgia, the mountains of northern Georgia, and in the swamps of Florida. The tragedy that occurred to class 3-1995 was a result of unfortunate human errors. However, it does bring to light the need to validate Army water exposure guidance tables.

These Ranger students died during the swamp phase of their training. This is the third and final phase of Ranger School, conducted at Camp Rudder, Florida. By this point in the ranger training process, the philosophy is to allow the student chain of command to make their own decisions involving navigation, judgment, and leadership. The Ranger Instructors (RIs) will only intervene when student errors have made the mission impossible, or make the training unsafe.

According to Ranger training standard operating procedures in preparation for the swamp field training exercise (FTX), water levels are checked 10 days and then again 2 days prior to the FTX. On the initial check, water levels were noted to be 12 to 18 inches lower than those encountered during the last FTX. However, during the 2-day prior check the water level had risen 18 to 24 inches. Data from the National River Forecasting Agency indicated that the river was rising and would reach flood stage around 15 February. This information was not used by the Florida Ranger Training Brigade. The typical way water depth was measured was by visually checking various landmarks in the area rather than through a quantified, repeatable process.

The general forecast for the FTX was partly cloudy with temperatures ranging from 60°F to 70°F. Winds were from the south at 10 to 15 mph, with a 30% chance of rain. From the site recon on 14 February, water depth was noted as knee to mid-thigh deep. Water temperature on 15 February was 52°F, but rose to around 54°F to 59°F during the day.

The general mission for Class 3-1995 was to move 8 to 10 km downriver via a Zodiac boat, move a short distance (700–800 m) through the swamp, and move the remaining few kilometers over ground to their objective. On 15 February the FTX was given the go ahead. The Ranger students embarked downriver in their Zodiac boats.

At approximately 1400 hours, C Company's student platoon leader missed his drop site. Because of the Ranger Training philosophy described above, the RI allowed him to continue downriver. B Company followed C Company downriver. A Company found their drop site and debarked their boats onto dry land. The completed their mission as planned. The decision to allow the student chain of command to continue downriver placed the RIs of B and C companies in an unfamiliar area of the swamp. This is one of the critical factors of this FTX.

Current Ranger standards allow students to be in chest-deep water of 50°F to 55°F for a maximum of 3 hours. If the water temperature is from 56°C to 60°F, the time limit increases to 5 hours. If the whole body is submerged, the time limit is no more than 5 minutes for either condition.

B and C companies found a drop site and reported that the water levels were much higher than briefed. Because of the current it was impossible to move back upriver to a different drop site. The Ranger students decided to move through the swamp away from the river, the general idea being that the water level would have to drop as they approached high ground. The first man entered the water at 1600 hours. Movement through the swamp was much more difficult than anticipated. After the students had been in the water for 1.5 hours, early signs of hypothermia were becoming evident. At this point the RIs took administrative control of the patrol and called for the first of several medical evacuation (medevac) requests.

(Exhibit 17-1 continues)

Exhibit 17-1 *continued*

The RIs decided the best way to reach high ground and get out of the water was by constructing rope bridges to cross the deep water. Because this contingency had not been planned for, it took about 30 to 45 minutes to complete the rope bridges and move all of the students across the water obstacle. During this time the students waited in the chest- to neck-deep water, causing their bodies to continue to cool. After crossing these water obstacles the companies were forced to continue their movement through the swamp.

The medevac helicopter was over the Ranger students by 1740 hours. It was not until 1840 hours that the medevac helicopter departed with the hypothermic students. This delay was caused by the confusion of the current situation as well as problems with extracting the three students from the swamp. All of these students survived.

Once the helicopter departed, the RIs attempted to get the students moving in an organized fashion toward higher ground. The students were reluctant to respond. Their legs were cramping and most were beginning to feel better by staying in one place. During the medevac, the rotor wash of the helicopter combined with the current of the river had caused a group of students to drift away from the main element. These students attempted to push on to high ground on their own. One RI organized a small group to move ahead of the main element. This group was to build a fire to help the students in the main element to rewarm, as well as to guide the lost group to their location.

By approximately 2100 hours, most of the students had reached high ground. However, several students were still missing in the swamp. RIs began searching for the missing Ranger students as individuals at the fire ignited flares to help guide the search. At this point another medevac helicopter arrived. The helicopter was called for seven students in critical need of medical attention. However, because of limited room aboard the helicopter, only five of these students were able to be evacuated. This medevac attempt was greatly hampered by all the confusion on the ground. In panic, the "lost" Ranger students had removed the red lenses from their flashlights. This caused problems for the pilot and crew because the signal for the medevac pickup point is a flashing white light. As the medevac attempt continued, RIs resumed their search. At 2200 hours, two students remained missing in the swamp. All other medevacs were called off because of the weather and the lack of logistical planning. The search was called off at 0340 hours to prevent any further casualties.

Of the soldiers who were successfully medevaced, one died in the emergency room. The two soldiers left behind were carried to the road and picked up by a forward line ambulance and transported to the emergency room. Both of these men were pronounced dead at approximately 0200 hours. At 0530 hours the search resumed for the last missing Ranger student. He was found at approximately 0730 hours lying on a log in the swamp less than 75 feet from high ground. This soldier was pronounced dead at 0853 hours.

The subsequent investigation of this incident prompted the Ranger Training Brigade to task the US Army Research Institute of Environmental Medicine to validate their water safety tables.

Exhibit compiled from material provided by the Public Affairs Office, US Army Infantry Center, Fort Benning, Georgia; 27 March 1995.

into the environment, is by way of blood flow between the two compartments. The size or thickness of the shell is in fact determined by (a) the blood flow to it and (b) the effective area available for heat transport. An increase in blood flow to a body region effectively decreases the size of the shell, resulting in an increase in heat loss. Peripheral vasoconstriction, on the other hand, increases the shell by increasing its thickness and decreasing the effective area for heat transport. This results in an increased insulating capacity, restricting heat loss

and maintaining core temperature.

Hypothermia can be insidious, as exemplified by the deaths of four soldiers (15-16 Feb 1995) who were undergoing the rigorous training to become US Army Rangers (Exhibit 17-1). From a physiological perspective, the students suffered from secondary hypothermia. They were in the ninth day of the final phase of their training at the Ranger School, Camp Rudder, Florida. The training was intense, and as part of that training the men were allowed three meals over 2 days. More than likely, the dif-

ference between the environmental air temperature (60°F–70°F) and the water temperature (52°F) played a role in their deaths. The warm air temperature may have given the false impression that the water was also warm. The men were moving in water at 52°F for a minimum of 1.5 hours. Thus, insufficient calories and sleep coupled with intense, prolonged exercise in cold water led to the deaths of four soldiers. As a consequence, the US Army Research Institute of Environmental Medicine (USARIEM), Natick, Massachusetts, developed new water exposure tables for the Rangers, the particulars of which are privileged information.¹⁵

All body regions do not respond with the same magnitude of peripheral vasoconstriction when placed under a cold stress. The trunk and head

show minimal vasoconstriction, with the areas of maximal heat loss being the neck, lateral thorax, upper chest, and groin.¹⁶ Vasoconstriction of the fingers and toes may cause discomfort significant enough to jeopardize military operations, even though the core temperature is normal. Insulating capability of the shell is a function not only of blood flow but also of fat content. The more body fat contained in the shell, the less the reduction in core temperature for a given immersion water temperature,¹⁷ or the greater likelihood that the core temperature will stabilize at a given water temperature.¹⁸ The importance of the subcutaneous fat layer is illustrated by the successful English Channel swimmers, all of whom are well endowed with this insulation layer.⁹

PHYSIOLOGICAL RESPONSES TO COLD WATER IMMERSION

The body's response to cold water immersion progresses from an initial stress situation to eventual hypothermia and death, depending on time and intervention. During the initial phases of immersion and the stress of cold water exposure, hypothermia is *not* the major concern; other stresses on the body's function are more life threatening. Tipton, Golden, and Hervey^{19,20} have approached the various physiological changes associated with water immersion in a stepwise fashion (Table 17-1). Based on their four stages—initial immersion, short-term immersion, long-term immersion, and postimmersion—and depending on the temperature of the water, it is unlikely that hypothermia would become a problem until some point in stage 3, long-term immersion. The magnitude (severity) of the physiological changes in each of the stages will depend

on a number of factors: water temperature, circulation (ie, water current) or body movement or both, body size, body fat content, and the presence or absence of protective gear (clothing). Stage 4, postimmersion, encompasses changes in core temperature (afterdrop) that may occur following the immersion incident. Note that treatment for hypothermia (rewarming methodology and postimmersion collapse) will not be covered in this chapter, but interested readers can find the topic discussed in Chapter 16, Treatment of Accidental Hypothermia, and in publications by researchers in Great Britain.^{21,22}

The initial response to immersion (stage 1) has a respiratory and cardiovascular component. Respirations may become uncontrolled, with reflex gasping and hyperventilation. This panic response will decrease the breath-holding time and may lead to aspiration with subsequent drowning.²³ Hyperventilation, possibly reaching five times resting values, is associated with an increase in both tidal volume and breathing frequency. The increased ventilation will result in a decrease in the partial pressure of carbon dioxide in the alveolar gas (PACO₂) and subsequently in arterial blood (PaCO₂), an acute respiratory alkalosis leading to cerebral vasoconstriction (with its effects on mental function), and possibly tetanic convulsions.²⁴ As the immersion time progresses, the minute ventilation decreases toward normal, the tidal volume remains increased, and the PACO₂ remains somewhat depressed.²⁵

Heart rate response to cold water exposure depends on the length of exposure time and whether or not complete submersion has occurred. If the

TABLE 17-1

STAGES OF WATER IMMERSION

Stage	Description	Immersion Time (min)
1	Initial immersion	0–3
2	Short-term immersion	3–15
3	Long-term immersion	≥ 30
4	Postimmersion	Core temperature may fall, but after the incident (ie < afterdrop)

Source: Tipton MJ. The concept of an "integrated survival system" for protection against the responses associated with immersion in cold water. *J R Nav Med Serv.* 1993;79:11–14.

victim's face is immersed while he is breath-holding, then heart rate may decrease (bradycardia), an example of one component of the "diving response" or "diving reflex."^{26,27} The complete diving reflex consists of the following components:

1. decrease in heart rate,
2. increase in total peripheral resistance,
3. decrease in cardiac output (ie, stroke volume), and
4. increase in mean arterial pressure.

In humans this cardiovascular reflex will occur during face-only immersion²⁸ as well as in total body immersion.²⁹ The predominant neural drive input to the myocardium associated with the diving reflex is parasympathetic in origin. However, the general cold stimulus also activates the sympathetic neural pathway to the myocardium and drives the systemic changes in total peripheral resistance, resulting in the redistribution of organ (tissue) blood flow.³⁰ This competition between augmented sympathetic and parasympathetic activity may be responsible for arrhythmias that occur at the end of—or just following—face immersion.³¹

If total submersion or face immersion is absent or if breath-holding face immersion has ended, the remaining cardiovascular adjustments are a result of the initial cold stress. There is a dramatic increase in heart rate, which subsides after a few minutes but remains above preimmersion levels; the colder the water, the higher the heart rate. Other cardiovascular sequelae of cold-induced sympathetic activation are also evident: increased systemic arterial pressure, increased total peripheral resistance, and increased cardiac output. During this early sympathetic discharge phase, myocardial conduction abnormalities may be observed, such as atrial and ventricular extrasystoles and sinus arrhythmias.³²

The initial cardiovascular responses to sympathetic activity will eventually subside as the core temperature falls. Heart rate, cardiac output, and systemic arterial pressure will fall as a function of decreasing core temperature.³³ Individual organ (heart, liver, kidney, and brain) blood flow will also progressively decrease at the rate of approximately 5% per degree Centigrade in core temperature drop (estimated from data in Blair³⁴).

The kinetics and conductance of ion channels in the myocardium are temperature dependent. Hypothermia will result in alterations in electrophysiological mechanisms, prolonged action potentials, and refractory period and progressive bradycardia. At temperatures between 28°C and 30°C, the

myocardium becomes susceptible to ventricular tachyarrhythmias.³⁵

The initial cold immersion insult may result in death from drowning, cardiovascular collapse, or both, but *not* from hypothermia. If the initial entry into the cold water is gradual (staged) or is a common occurrence for the individual (eg, cold water swimmers and divers), then the cardiovascular and respiratory changes may be significantly attenuated.²³ If the victim survives the initial insult, the hyperventilation will subside, reducing the ventilation to match the metabolic needs. Heart rate will decrease toward preimmersion levels, with the final value depending on the water temperature. Peripheral vasoconstriction will persist during stages 2 and 3 as a means of preventing heat loss and maintaining core temperature.

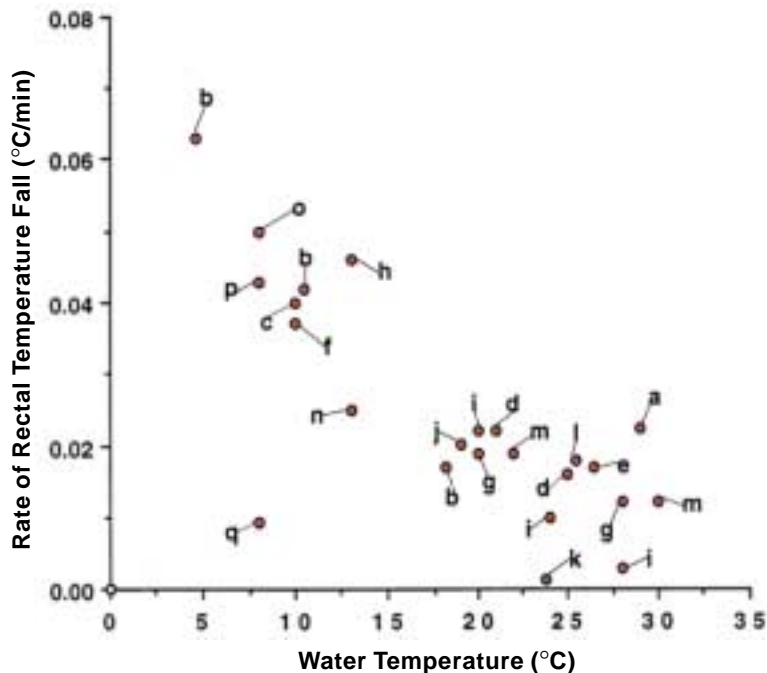
The peripheral circulatory beds, such as fingers, toes, and ears, demonstrate a paradoxical response to cold exposure.^{36,37} The initial vasoconstriction induced by the cold exposure is followed in 5 to 15 minutes by a vasodilation of variable duration and magnitude (cold-induced vasodilation, CIVD). This is followed by a cyclic vasoconstriction-dilation phenomenon with a frequency of 3 to 5 per hour.^{38,39} This CIVD will result in an increase in skin temperature and may contribute to an increase in body heat loss. The magnitude, and therefore the importance, of the CIVD decreases as cold exposure time is prolonged and body heat content and core temperature decrease (CIVD is also discussed in Chapter 15, Nonfreezing Cold Injury).

In stages 2 and 3, shivering will begin in an attempt to increase heat production. The onset of shivering occurs at a skin temperature of 26°C.⁴⁰ As the core temperature continues to fall, shivering will increase, reaching a maximum at a core temperature of approximately 35°C.⁴¹ A continued decrease in core temperature will be accompanied by suppression of shivering, with final cessation at a core temperature around 30°C.⁴²

The further physiological changes associated with cold water immersion in stage 3 will depend on both the water temperature and the exposure time. For the purposes of this discussion it is assumed that the victim is minimally clothed and in a negative heat balance; that is, losing more heat to the environment than can be produced by metabolic processes, shivering, and muscular activity. The rate of core temperature drop will be a function of the water temperature. Figure 17-4 is a compilation of rates of core temperature cooling from 17 sets of published data.^{40,43-59} The subjects in these experiments cannot be considered matched for all con-

Fig. 17-4. The rate of core cooling data as a function of water temperature was extracted from 17 separate published reports (sources a–q, below) on experiments employing human volunteers. For the most part, the subjects were young white men of average build, fat content, and physical fitness. The subjects were not wearing any clothing or employing any devices that were designed to protect against cold exposure.

Data sources: (a) Hayward MG, Keatinge WR. Progressive symptomless hypothermia in water: Possible cause of diving accidents. *Br Med J.* 1979;1:1182. (b) Hayward JS, Eckerson JD, Collis ML. Thermal balance and survival time prediction of man in cold water. *Can J Physiol Pharmacol.* 1975;53:21–32. (c) Hayward JS, Eckerson JD, Collis ML. Thermoregulatory heat production in man: Prediction equations based on skin and core temperature. *J Appl Physiol.* 1977;43(2):377–384. (d) Israel DJ, Heydn KM, Edlich RF, Pozos RS, Wittmers LE. Core temperature response to immersed bicycle ergometer exercise and water temperatures of 21°, 25°, and 29°. *J Burn Care Rehabil.* 1989;10(4):336–345. (e) Veicsteinas A, Rennie DW. Thermal insulation and shivering threshold in Greek sponge divers. *J Appl Physiol.* 1982;52(4):845–850. (f) Wittmers LE, Pozos RS. In situ hypothermia testing. In: Laursen GA, Pozos RS, Hemple FG, eds. *Human Performance in the Cold.* Bethesda, Md: Undersea Medical Society Inc; 1983: 153–168. (g) McArdle WD, Toner MM, Magel JR, Spina RJ, Pandolf KB. Thermal responses of men and women during cold-water immersion: Influence of exercise intensity. *Eur J Appl Physiol.* 1992;65:265–270. (h) Martin S, Diewold RJ, Cooper KE. Alcohol, respirations, skin and body temperature during cold water immersion. *J Appl Physiol.* 1977;43(2):211–215. (i) McArdle WD, Magel JR, Gergley TJ, Spina RJ, Toner MM. Thermal adjustments to cold-water exposure in resting men and women. *J Appl Physiol.* 1984;56:1565–1571. (j) Rochelle RD, Horvath SM. Thermoregulation in surfers and nonsurfers immersed in cold water. *Undersea Biomed Res.* 1978;5(4):377–390. (k) Beckman EL, Reves E. Physiological implications as a survival during immersion in water at 75°F. *Aerospace Med.* 1966;37(11):1136–1142. (l) Weihl AC, Langworthy HC, Manalaysay AR, Layton RP. Metabolic responses of resting man immersed in 25.5°C and 33°C water. *Aviat Space Environ Med.* 1981;52(2):88–91. (m) Martin S, Cooper KE. Alcohol and respiratory and body temperature changes during tepid water immersion. *J Appl Physiol.* 1978;44(5):683–689. (n) Graham T, Baulk K. Effect of alcohol ingestion on man's thermoregulatory responses during cold water immersion. *Aviat Space Environ Med.* 1980;51(2):155–159. (o) Giesbrecht GG, Sessler DI, Mekjavic IB, Schroeder M, Bristow GK. Treatment of mild immersion hypothermia by direct body-to-body contact. *J Appl Physiol.* 1994;76(6):2373–2379. (p) Bristow GK, Sessler DI, Giesbrecht GG. Leg temperature and heat content in humans during immersion hypothermia and rewarming. *Aviat Space Environ Med.* 1994;65:220–226. (q) Bourdon L, Jacobs I, Bell D, Ducharme MB. Effect of triazolam on responses to a cold-water immersion in humans. *Aviat Space Environ Med.* 1995;66(7):651–655.



tributing variables. They were, however, for the most part, young, fit men of average body type not participating in any type of exercise or wearing any special clothing to protect against heat loss during the time of immersion. The data demonstrate the magnitude of core temperature drop as a function of water temperature. These data illustrate the variability from experiment to experiment when evaluating core temperature changes as a function of cold stress.

In emergencies, physiological compensatory mechanisms such as shivering, pulse, and loss of reflexes can be used to obtain a rough estimate of core temperature. Table 17-2 summarizes the relationship between core temperature and signs and

symptoms of hypothermia. The data are divided into three ranges of core temperature: mild hypothermia, 37°C to 33°C; moderate hypothermia, 32°C to 27°C; and severe hypothermia, 26°C to 18°C.

Military research into human performance in extreme environments is conducted by many nations. In 1996, Commander Wolf J. Haenert, MD, PhD, Medical Corps, German Navy, visited the Naval Health Research Center, San Diego, California, and subsequently submitted to Robert S. Pozos, PhD, then head of the Department of Applied Physiology there, a translated report on the German experience with survivors of shipwrecks during World War II. The report, written by Dr. V. Hartmann, a

TABLE 17-2

SIGNS, SYMPTOMS, AND PHYSIOLOGICAL CHANGES ASSOCIATED WITH PROGRESSIVE HYPOTHERMIA

Stage of Hypothermia	Core Temperature Rectal, °C (°F)	Signs and Symptoms
	37 (98.6)	Normal rectal temperature
Mild	36	Increased metabolic rate due to exercise and shivering
	35	Maximum shivering thermogenesis
	34	Amnesia, dysarthria, and judgment problems
	33	Ataxia and apathy
Moderate	32	Stupor; oxygen consumption 75% of normal
	31	Shivering stops
	30	Possible cardiac arrhythmias; pulse and cardiac output 66% of normal
	29	Decreasing consciousness, pulse, and respirations; dilated pupils
	28	Increased sensitivity to ventricular fibrillation; pulse and oxygen consumption 50% of normal
	27 (80.6)	Loss of voluntary motion and reflexes
Severe	26	Major acid-base problems; no response to pain
	25	Cerebral blood flow 30% of normal; possible pulmonary edema
	23	Loss of corneal and oculocephalic reflexes
	22	Maximum risk of ventricular fibrillation; oxygen consumption 25% of normal
	20	Pulse 20% of normal
	19 (66.2)	Electroencephalogram flat
	18	Asystole

Adapted with permission from Danzl DF, Pozos RS. Accidental hypothermia. *N Engl J Med.* 1994;331(26):1757.

medical officer in the German Navy, and Dr. Haenert (who also translated the report), is presented in toto in the Attachment at the end of this chapter. It contains a key observation about afterdrop and documents the rewarming of victims of hypothermia with hot water. In addition, it mentions rewarming victims of hypothermia with a fire. In field situations, many different methods are used

to rewarm victims of hypothermia. However, rewarming with hot water is not possible except aboard ship, and even then, caution should be exercised by monitoring heart rate and blood pH. Warming with fire has disadvantages in that it promotes peripheral vasodilation, which can promote afterdrop. Even in 1944, the advantages of protective clothing were noted.

GENDER DIFFERENCES IN THERMOREGULATION

Differences in thermal regulation between men and women were reported in the early 1940s.⁶⁰ However, only since the late 1970s have the scientific community and funding agencies turned their efforts to address these gender differences.⁶¹ The data are at some points confusing and, to say the least, incomplete.

Body Characteristics

Some of the differences in temperature regulation observed between men and women may be attributed to anthropomorphic characteristics. Com-

pared to men, women tend to be of smaller stature, with resultant larger surface area-to-body mass ratio and lower total thermal mass; these contribute to a more rapid heat loss and decrease in core temperature when exposed to cold stress.⁶² The lower muscle mass of women (vs men) will produce less metabolic heat from both exercise and shivering, leading to a decreased ability to supply heat to the core. The thicker subcutaneous fat layer in women will provide more insulating capacity in the shell and to some extent retard heat loss. However, the net result for women is a faster cooling rate when exposed to an environmental cold stress.⁶³

Temperature Regulation and the Menstrual Cycle

The reproductive system has an important effect on female thermoregulation.⁶³ In the follicular phase of the menstrual cycle (the first half of the cycle, beginning with the onset of menses and ending with ovulation), women thermoregulate similarly to men if both genders are at comparable fitness levels.⁶⁴ However, during the luteal phase of the menstrual cycle (the second half, from ovulation to the onset of menses), hormonal and physiological changes associated with ovulation significantly affect thermoregulation. Resting core temperature is elevated,⁶⁵ and onset of sweating occurs at a higher temperature, suggesting that the set point for temperature regulation has been elevated.⁶⁶

During the luteal phase, finger blood flow shows a greater cold-induced vasoconstriction and a slower recovery when compared with the follicular phase of the menstrual cycle.⁶⁷ It has also been observed that there are menstrual cycle changes in the blood flow of the forearm, leg, and calf.⁶⁸ Regulation of cutaneous blood flow in response to cold exposure is primarily under sympathetic control, and studies have demonstrated the influence of estrogen on the sympathetic nervous activity. Evidence from animal studies indicates that estrogen induces an up-regulation (vasoconstriction) of α_2 -adrenoreceptors.^{69,70} Although much less investigated, the same effects seem to be true for progesterone.⁷¹

Cold-induced vasospastic disorders such as Raynaud's phenomenon are more common in women, with the female-to-male ratio ranging from 2:1 to 9:1.⁷² The onset of these vasospastic attacks occurs with menarche⁷³; the attacks subside after menopause.⁷⁴ The frequency and severity of cold-induced vasospastic episodes also vary with the phase of the menstrual cycle and subside during pregnancy.^{75,76}

Behavioral differences are also associated with the menstrual cycle. In the luteal phase, women sense changes in skin temperature more quickly than during the follicular phase.⁷⁷ In addition, women have a higher skin temperature preference in the luteal phase.⁷⁸ Both these modifications are consistent with an increase in the temperature set point.

Differences in Response to Cold Water Immersion

Data suggest that women exhibit a more rapid drop in core temperature than men when immersed in cold water.^{43,79} This increased rate of core temperature fall is present if both genders are matched for body fat content. Increased body fat in women does provide insulation during water immersion; however, the larger surface area-to-mass ratio and the lower thermal mass contributing to heat production will result in women's faster initial cooling rate during water immersion, compared with that of men.⁶¹

As mentioned earlier, exercise during cold water immersion may produce sufficient heat to retard the drop in core temperature. For men and women of the same body fat composition, decreases in core temperature are greater for women in the resting state or when performing light exercise; however, with more intense exercise, women maintain a higher core temperature than men.⁴⁴ If men and women exercise at the same absolute work intensity, there is no difference in cooling rate.^{43,79} Under these conditions the women are exercising at a higher percentage of their maximal aerobic power and are therefore producing a greater amount of heat per unit thermal mass. Women exercising at the same percentage of their maximal aerobic power as men will cool at a faster rate, illustrating the imbalance between heat loss and production.⁴⁵

PROTECTIVE CLOTHING

Two types of protection can be provided for survival in the case of cold water immersion: flotation devices and hypothermia protection. Therefore, it is beneficial during stage 1 of immersion to be equipped with some type of personal flotation device. The minimal function of this device is to float the victim in such a manner as to keep the face out of the water. The victim will not have to expend energy to stay afloat and will be protected from

drowning if or when hypothermia progresses to the point that voluntary efforts to maintain flotation are no longer possible.

Numerous garments have been designed for hypothermia protection during accidental cold water immersion. One such garment is constructed to function as workable clothing in air, and will provide both flotation and hypothermia protection if the individual falls into cold water (Figure 17-5).

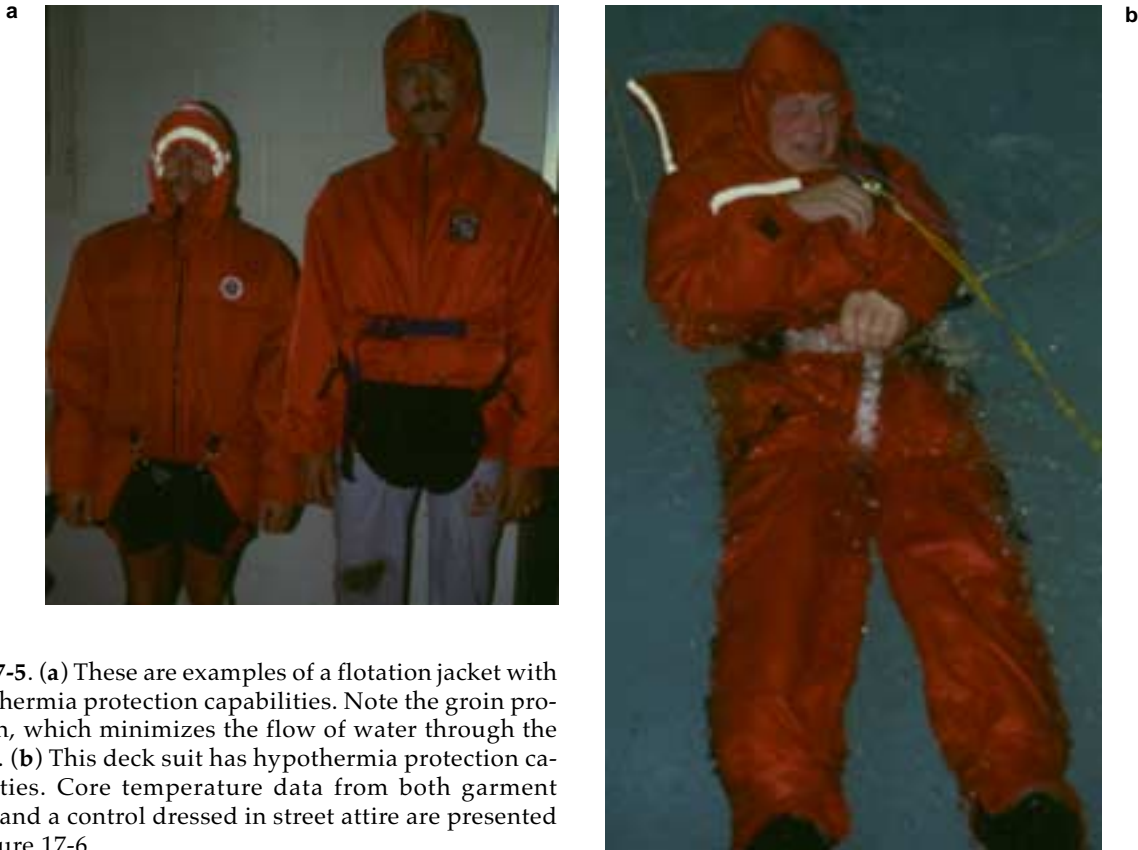


Fig. 17-5. (a) These are examples of a flotation jacket with hypothermia protection capabilities. Note the groin protection, which minimizes the flow of water through the jacket. (b) This deck suit has hypothermia protection capabilities. Core temperature data from both garment types and a control dressed in street attire are presented in Figure 17-6.

These suits and jackets do not keep the victim dry; water enters the garment and is trapped between the victim's body and the suit. Body heat, provided in part by shivering, warms the trapped water, which in this case represents the "boundary layer," and the suit's insulating capacity minimizes the heat loss to the surrounding environment. Figure 17-6 plots data from a subject who was immersed in 10°C water on three different occasions, dressed as follows⁴⁶:

1. As a control, the subject wore street clothes (cotton underwear, shirt, jeans, socks, and tennis shoes) with flotation provided only by a neck collar.
2. The subject wore a jacket equipped with a groin flap to minimize water movement through the jacket.
3. Later, the subject also wore a flotation full-body suit.

The subject's core (rectal) temperature reached 35.6°C by 30 minutes in the control condition, by 60 minutes when clothed in the jacket, and remained above this level for 3 hours when clothed in the full body suit.

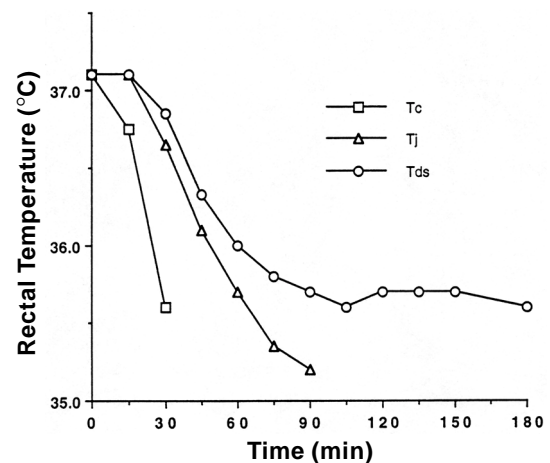


Fig. 17-6. Data representing the rectal temperature versus time plots of a single volunteer undergoing three testing conditions. The subject is a young white man of average build, fat content, and physical fitness. The three water immersions took place at least one week apart, and the water temperature each time was 10°C. In the control condition (T_c) the subject was clothed in regular street attire; in the other two conditions the subject wore either a jacket (T_j) or a deck suit (T_{ds}) designed for hypothermia protection.



Fig. 17-7. This flotation survival suit has hypothermia protection capabilities. Note that the gloves, feet (not shown), and hood are part of the suit, and are intended to keep water entry down to a few cubic centimeters. The subject is entering a tank with the water temperature set at 0°C, in a 2-phase system. Core temperature data from this garment are presented in Figure 17-8.

The efficiency of long-term survival suits (Figure 17-7), which are designed to keep the victim dry and provide hypothermia protection, is illustrated in the data plotted in Figure 17-8. These one-piece, full-body suits allow little or no water into the garment when the subject falls into the water. In this test, the subject was immersed in ice water for 6 hours, and his core temperature remained above 37°C for the entire exposure.

In general, as these data show (see Figures 17-6 and 17-8), some type of flotation device and protective clothing are essential for survival when the victim is in danger of long-term exposure to cold water immersion.

NEAR-DROWNING: HYPOTHERMIA AND THE DIVING RESPONSE

The preceding discussion has for the most part been limited to immersion cold stress under conditions in which the head of the victim remains out of the water. If, however, submersion results in hypoxia due to aspiration or laryngospasm, the accident is classified as a drowning. On the other hand, if the victim is recovered, successfully resuscitated, and lives for “some time” (usually 24 h), the event is classified as a near-drowning.^{80,81}

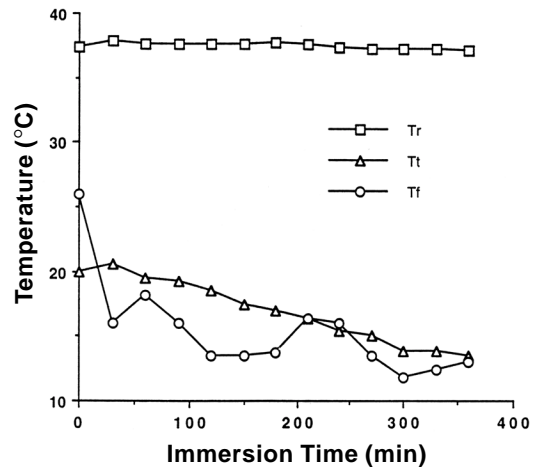


Fig. 17-8. A summary of a volunteer’s 6-hour immersion in ice water (temperature range 1°C–3°C). The protective device worn was a survival suit of the dry type, as depicted in Figure 17-7. T_r represents rectal temperature, T_f represents skin temperature on the ventral aspect of index finger, and T_t represents skin temperature on the ventral aspect of the large toe. Adapted with permission from Wittmers LE, Pozos RS. In situ hypothermia testing. In: Laursen GA, Pozos RS, Hemple FG, eds. *Human Performance in the Cold*. Bethesda, Md: Undersea Medical Society Inc; 1983: 166.

Many military operations require clandestine operations involving Special Forces or US Navy SEALs, who travel to their targets in open minisubmarines and subsequently swim to shore. These operations require scuba diving gear and, in cold water, various kinds of insulating suits. In most operations of this nature, the personnel experience severely cold feet or hands rather than hypothermia. Although the personnel have adequate oxygen and insulation for their truncal regions, pronounced vasoconstriction of the digits—even though they are wearing special gloves and footwear—remains a major challenge for the execution of cold water submersible operations.

The cases of near-drowning most often reported in the popular press involve victims who are young,⁸² have fallen into cold water ($\leq 10^\circ\text{C}$), at the time of recovery are profoundly hypothermic, and have been submerged for a relatively long time. The maximum reported immersion time of a survivor is 66 minutes.⁸³ The question most often asked is *why* do these individuals survive when others do not. Which scientific explanation best explains the

why remains controversial and the reason for this is best expressed by Gooden,⁸⁴ who believes that a major obstacle in the rigorous scientific study of survival from near-drowning is the paucity of objective data from the time the victim enters the water to arrival at the hospital.

Theories for the survival from near-drowning include the following:

1. externally induced hypothermia, resulting in brain and body cooling,⁴⁷ or internal and external hypothermia induced by drowning;
2. the diving response, resulting in oxygen conservation; and
3. a combination of the first two.

Published approaches to near-drowning survival either support mechanisms that combine both the diving response and hypothermia or discount the diving response as irrelevant and attribute survival to a rapid-cooling phenomenon.

If we assume that the diving response is of minimal or no importance with respect to preservation of life in the near-drowning scenario, and that the entire beneficial effect resides in the decrease in body temperature, then the water must be extremely cold and heat must be lost at a very rapid rate. Observations in subjects undergoing head-out

water immersion (see Figure 17-4) show that even in very cold water (eg, 5°C), the rate of core temperature drop is only 0.06°C per minute. It must be pointed out that the body has very effective measures to counter the onset of early hypothermia (ie, peripheral vascular constriction to conserve heat and shivering thermogenesis to produce more heat). The decrease in core temperature may not be rapid enough to provide sole protection for the central nervous tissue. Hypothermia can also be induced by aspirating cold water, which if combined with external cooling, would cool the core very fast.

The diving response has been described in some detail earlier in this chapter. It is present in children, is more pronounced if stimulated by cold water, and is increased under conditions of excessive stress. Face immersion and apnea are accompanied by bradycardia, a decreased limb (ie, peripheral) blood flow, decreased kidney and splanchnic blood flow, and a decreased heat loss. The decrease in organ blood flow would tend to minimize oxygen utilization by those tissues. Also, the decreased heart rate and cardiac output will further reduce cardiac work and therefore oxygen consumption by the myocardium. The overall result is a decrease in metabolism, providing an oxygen-conserving function.⁸² In real life, possibly, drowning and the diving response work together to cause a drop in core temperature.

ACCLIMATIZATION

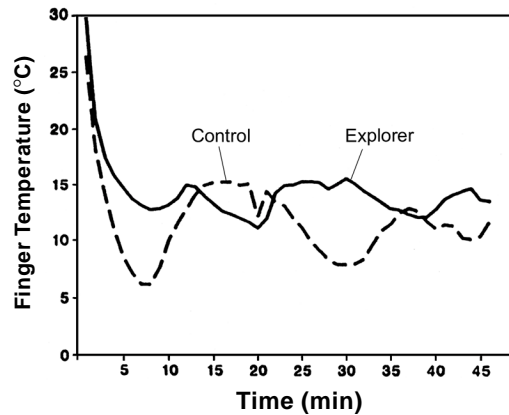
To be consistent with the definitions given by Young,⁸⁵ cold *acclimatization* (or acclimation) means in this chapter “physiological adjustments in response to chronic cold stress.”^{85(p419)} On the other hand, cold *adaptation* refers to “genetic effects manifested as a result of natural selection.”^{85(p419)} The problem in the evaluation of cold acclimatization arises from the wide range in exposure intensity. The magnitude of the intensity is determined by surface area of exposure, ambient temperature, and environmental media (air or water). Because this chapter discusses immersion situations, acclimatization will be limited to cold water exposure.

Depending on the severity of the cold exposure, acclimatization can occur with or without an associated decrease in core temperature. For example, commercial fishermen are required to use their hands in extremely cold water for extended periods while the remainder of their body is relatively well protected from the elements.^{86,87} Under these conditions, the subjects show a reduced vasoconstrictor response to cold stress. An example of this

reduced vasoconstriction on exposure to cold is seen in arctic explorers (Figure 17-9). This alteration of the physiological response to cold will supply warm blood to the extremities, preventing frostbite and freezing injuries as well as prolonging dexterity. However, owing to the fact that the total area of exposure is small and the remainder of the body is protected, there will be no decrease in core temperature.

The next level of exposure would consist of repeated total body immersion for short periods of time. Under these conditions there is little or no change in core temperature; however, there is a delayed onset of shivering.⁸⁸ Because even a short immersion in cold water is a major stress, longer exposures magnify the problem and make it very difficult to measure physiological changes that may reflect acclimatization. To minimize these difficulties and evaluate acclimatization, the subject’s response to cold air exposure, a much lesser stress, is evaluated following repeated cold water immersion. This approach was applied by Young and colleagues⁸⁹ and Bittel.⁹⁰ Both studies showed a de-

Fig. 17-9. The data represent the skin temperature of the subjects' right index finger immersed in ice water (0°C) as a function of time. The group of arctic explorers is compared with a group of controls matched for gender, age, and body type. Skin temperature is a function of skin blood flow. The explorers show much less rapid vasoconstriction when exposed to the ice water, and the magnitude of the drop is less than that seen in the control group. Adapted with permission from Hoffman RG, Wittmers LE. Cold vasodilatation, pain and acclimatization in arctic explorers. *J Wilderness Med.* 1990;1:230.



layed onset of shivering and a lower mean skin temperature in cold water-acclimatized subjects. The rectal temperature results did not match; Bittel's data showed little difference between acclimatized and nonacclimatized subjects, whereas Young's showed a lower rectal temperature at the start and a larger drop in rectal temperature in the acclimatized subjects.

The decreased skin temperature in the acclima-

tized subjects reduces the driving force (ie, the temperature gradient) for movement of heat from the skin into the environment (see Equation 1). This decrease in skin temperature results in a larger gradient between core and shell; therefore, a redistribution of heat internally with a decrease in shell thickness. However, the end result is an increase in effective insulation and thus, conservation of heat.

SUMMARY

The information presented in this chapter has focused on the physiological and behavioral responses to cold water immersion. When compared with cold air exposure, cold water is a much greater stress, drawing heat away from the body approximately 20-fold faster than air of the same temperature. Protection against heat loss in cold water environments is possible, as long as personnel wear protective garments (ie, jackets and suits) as discussed. However, the suddenness of cold water accidents may preclude the opportunity to wear cold water protective gear. In such cases, the onset of hypothermia and drowning is swift and lethal.

Initially, cold stress results in peripheral vasoconstriction. This vascular response provides a method of conserving heat, essentially limiting heat deliv-

ery from the body core and increasing the external insulating layer. As the cold exposure continues, heat loss is compensated for by increased heat production, either from shivering or from muscle contraction. However, when heat loss exceeds heat production, core temperature starts to drop and the subjects proceed toward hypothermia. Changes in the individual's physiology and behavior will signal the magnitude of the core temperature drop and indicate the precautions to be taken or the treatment to be given.

It should be emphasized that cold water immersion, even when the water temperature is relatively mild (but below core temperature) has a great potential for producing the life-threatening condition of hypothermia. In addition, cold water may cause cardiovascular collapse, or drowning, or both.

REFERENCES

1. Hardy JD, DuBois EF. Basal metabolism, radiation, convection and vaporization at temperatures of 22 to 35°C. *J Nutr.* 1938;15:477-497.
2. Wunderlich CE. Medical thermometry: Fundamental principles. In: Woodman, WB, ed. *On the Temperature in Disease.* London, England: New Sydenham; 1871: 1-18.
3. Savage MV. *Control of Skin Blood Flow in the Neutral Zone of Human Temperature Regulation.* Seattle, Wash: University of Washington; 1994. Thesis.

4. Adams WC, Fox RH, Fry AJ, MacDonald IC. Thermoregulation during marathon running in cool, moderate and hot environments. *J Appl Physiol.* 1975;38:1030–1037.
5. Smith RM, Hanna JM. Skinfolds and resting heat loss in cold air and water. *J Appl Physiol.* 1975;39:93–102.
6. Lee DT, Toner MM, McArdle WD, Vrabas IS, Pandolf KB. Thermal and metabolic responses to cold-water immersion at knee, hip and shoulder levels. *J Appl Physiol.* 1997;82(5):1523–1530.
7. Rasch W, Samson P, Cote J, Cabanac M. Heat loss from the human head during exercise. *J Appl Physiol.* 1991;71(2):590–595.
8. Froese G, Burton AC. Heat losses from the head. *J Appl Physiol.* 1957;10(2):235–241.
9. Clark RP, Edholm OG. *Man and His Thermal Environment.* London, England: Edward Arnold Ltd; 1985.
10. Bullard RW, Rapp GM. Problems of body heat loss in water immersion. *Aerospace Med.* 1970;41(11):1269–1277.
11. Craig AB, Dvorak M. Thermal regulation during water immersion. *J Appl Physiol.* 1968;25:28–35.
12. Brengelmann GL, Savage MV. Temperature regulation in the neutral zone. In: Blatteis CM, ed. *The Annals of the New York Academy of Science—Thermoregulation.* New York, NY: New York Academy of Science; 1996: 39–50.
13. Burton AC, Bazett HC. A study of the average temperature of tissues, of the exchanges of heat and vasomotor responses in man by means of a bath calorimeter. *Am J Physiol.* 1936;117:36–54.
14. Toner MM, Sawka MM, Holden WL, Pandolf KB. Effects of body mass and morphology on thermal response in water. *J Appl Physiol.* 1986;60:521–525.
15. Pandolf KB. Senior Research Scientist, US Army Research Institute of Environmental Medicine, Natick, Mass. Personal communication, May 2000.
16. Hayward JS, Collis M, Eckerson JD. Thermographic evaluation of relative heat loss areas of man during cold water immersion. *Aerospace Med.* 1973;44:708–711.
17. Nadel ER, Holmer I, Bergh U, Astrand PO, Stolwijk AAJ. Energy exchange in swimming men. *J Appl Physiol.* 1974;36:465–471.
18. Hayward MG, Keating WR. Role of subcutaneous fat and thermoregulatory reflexes in determining ability to stabilize body temperature in water. *J Physiol (Lond).* 1981;320:229–251.
19. Tipton MJ. The concept of an “integrated survival system” for protection against the responses associated with immersion in cold water. *J R Nav Med Serv.* 1993;79:11–14.
20. Golden FSC, Hervey GR. The “after-drop” and death after rescue from immersion in cold water. In: Adam JA, ed. *Hypothermia Ashore & Afloat.* Aberdeen, Scotland: Aberdeen University Press; 1981.
21. Golden FSC, Hervey GR, Tipton MJ. Circum-rescue collapse: Collapse, sometimes fatal associated with rescue of immersion victims. *J R Nav Med Serv.* 1991;77:139–149.
22. Stoneham MD. Accidental hypothermia. *Lancet.* 1995;345:1048.
23. Hayward JS, French CD. Hypoventilation response to cold water immersion: Reduction by staged entry. *Aviat Space Environ Med.* 1989;60:1163–1165.
24. Cooper EK, Martin S, Simper P. Factors causing hyperventilation in man during cold water immersion. In: Laursen GA, Pozos RS, Hemple FG, eds. *Human Performance in the Cold.* Bethesda, Md: Undersea Medical Society Inc; 1983.

25. Cooper KE, Martin S, Riben P. Respiratory and other responses in subjects immersed in cold water. *J Appl Physiol*. 1976;40:903–910.
26. Elsner R, Gooden B. *Diving and Asphyxia: A Comparative Study of Animals and Man*. Cambridge, England: Cambridge University Press; 1983.
27. Butler PJ, Jones DR. Physiology of diving birds and mammals. *Physiol Rev*. 1997;77(3):837–899.
28. Wittmers LE, Pozos RS, Fall G, Beck L. Cardiovascular response to face immersion (the diving reflex) in human beings after alcohol consumption. *Ann Emerg Med*. 1987;16:1031–1036.
29. Anderson HT. Physiological adaptations in diving vertebrates. *Physiol Rev*. 1966;46:212–243.
30. Kawakami Y, Netelson BN, Buboia A. Cardiovascular effects of face immersion and factors affecting diving reflex in man. *J Appl Physiol*. 1967;23:964–970.
31. Tipton MJ, Kelleher PC, Golden FSC. Supraventricular arrhythmias following breath-hold submersions in cold water. *Undersea Hyperbaric Med*. 1994;21(3):305–303.
32. Keatinge WR. *Survival in Cold Water*. Oxford, England: Blackwell Scientific Publishing; 1969.
33. Wong KC. Physiology and pharmacology of hypothermia. *West J Med*. 1983;138(2):227–232.
34. Blair E. *Clinical Hypothermia*. New York, NY: Blakiston Division, McGraw Hill; 1969.
35. Bjornstad H, Tande PM, Refsum H. Cardiac electrophysiology during hypothermia: Implications for medical treatment. *Arctic Med Res*. 1991;50(Suppl 6):71–75.
36. Lewis T. Observations upon the reactions of vessels of the human skin to cold. *Heart*. 1930;15:177–208.
37. Fox RH, Wyatt HT. Cold-induced vasodilation in various areas of the body surface of man. *J Physiol*. 1962;162:289–297.
38. McCutcheon R, Hoffman RG, Wittmers LE, Pozos RS. Synchronized cold-induced vasodilatation responses in man. *Physiologist*. 1987;30:194.
39. Hoffman RG, Wittmers LE. Cold vasodilatation, pain and acclimatization in arctic explorers. *J Wilderness Med*. 1990;1:225–234.
40. Veicsteinas A, Rennie DW. Thermal insulation and shivering threshold in Greek sponge divers. *J Appl Physiol Respir Environ Exerc Physiol*. 1982;52(4):845–850.
41. Dill DB, Forbes WH. Respiratory and metabolic effects of hypothermia. *Am J Physiol*. 1941;132:685–697.
42. Danzl DF, Pozos RS, Hamlet MP. Accidental hypothermia. In: Auerbach PS, Geehr EC, eds. *Management of Wilderness and Environmental Emergencies*. 2nd ed. St Louis, Mo: Mosby; 1988: 35–76.
43. McArdle WD, Magel JR, Gergley TJ, Spina RJ, Toner MM. Thermal adjustments to cold-water exposure in resting men and women. *J Appl Physiol*. 1984;56:1565–1571.
44. McArdle WD, Toner MM, Magel JR, Spina RJ, Pandolf KB. Thermal responses of men and women during cold-water immersion: Influence of exercise intensity. *Eur J Appl Physiol*. 1992;65:265–270.
45. Graham TE. Alcohol ingestion and sex differences on the thermal responses to mild exercise in a cold environment. *Hum Biol*. 1983;55:463–476.
46. Wittmers LE, Pozos RS. In situ hypothermia testing. In: Laursen GA, Pozos RS, Hemple FG, eds. *Human Performance in the Cold*. Bethesda, Md: Undersea Medical Society Inc; 1983: 153–168.

47. Giesbrecht GG, Sessler DI, Mekjavic IB, Schroeder M, Bristow GK. Treatment of mild immersion hypothermia by direct body-to-body contact. *J Appl Physiol*. 1994;76(6):2373–2379.
48. Hayward MG, Keatinge WR. Progressive symptomless hypothermia in water: Possible cause of diving accidents. *Br Med J*. 1979;1:1182.
49. Hayward JS, Eckerson JD, Collis ML. Thermal balance and survival time prediction of man in cold water. *Can J Physiol Pharmacol*. 1975;53:21–32.
50. Hayward JS, Eckerson JD, Collis ML. Thermoregulatory heat production in man: Prediction equations based on skin and core temperature. *J Appl Physiol Respir Environ Exerc Physiol*. 1977;43(2):377–384.
51. Israel DJ, Heydon KM, Edlich RF, Pozos RS, Wittmers LE. Core temperature response to immersed bicycle ergometer exercise and water temperatures of 21°, 25°, and 29°C. *J Burn Care Rehabil*. 1989;10(4):336–345.
52. Martin S, Diewold RJ, Cooper KE. Alcohol, respirations, skin and body temperature during cold water immersion. *J Appl Physiol*. 1977;43(2):211–215.
53. Rochelle RD, Horvath SM. Thermoregulation in surfers and nonsurfers immersed in cold water. *Undersea Biomed Res*. 1978;5(4):377–390.
54. Beckman EL, Reves E. Physiological implications as a survival during immersion in water at 75°F. *Aerospace Med*. 1966;37(11):1136–1142.
55. Wehl AC, Langworthy HC, Manalaysay AR, Layton RP. Metabolic responses of resting man immersed in 25.5°C and 33°C water. *Aviat Space Environ Med*. 1981;52(2):88–91.
56. Martin S, Cooper KE. Alcohol and respiratory and body temperature changes during tepid water immersion. *J Appl Physiol*. 1978;44(5):683–689.
57. Graham T, Baulk K. Effect of alcohol ingestion on man's thermoregulatory responses during cold water immersion. *Aviat Space Environ Med*. 1980;51(2):155–159.
58. Bristow GK, Sessler DI, Giesbrecht GG. Leg temperature and heat content in humans during immersion hypothermia and rewarming. *Aviat Space Environ Med*. 1994;65:220–226.
59. Bourdon L, Jacobs I, Bell D, Ducharme MB. Effect of triazolam on responses to a cold-water immersion in humans. *Aviat Space Environ Med*. 1995;66(7):651–655.
60. Hardy JD, Du Bois EF. Differences in men and women in their response to heat and cold. *Proc Natl Acad Sci U S A*. 1940;26:389–398.
61. Graham TE. Thermal, metabolic, and cardiovascular changes in men and women during cold stress. *Med Sci Sports Exerc*. 1988;20:185–192.
62. Nunneley SH. Physiological response of women to thermal stress: A review. *Med Sci Sports*. 1978;10:250–255.
63. Stephenson LA, Kolka MA. Thermoregulation in women. *Exerc Sports Sci Rev*. 1993;14:231–262.
64. Kolka MA, Stephenson LA, Rock PB, Gonzales RR. Local sweating and cutaneous blood flow during exercise in hypoxic environments. *J Appl Physiol*. 1987;62:2224–2229.
65. Kleitman N, Ramsaroop A. Periodicity in body temperature and heart rate. *Endocrinology*. 1948;43:1–20.
66. Haslag SWM, Hertzman AB. Temperature regulation in young women. *J Appl Physiol*. 1965;20:1283–1288.
67. Bartelink ML, Wollersheim H, Theeuwes A, van Duren D, Thien T. Changes in skin blood flow during the menstrual cycle: The influence of the menstrual cycle on the peripheral circulation of the healthy female volunteers. *Clin Sci*. 1990;78(5):527–532.

68. Keates JS, Fitzgerald DE. Limb volume and blood flow changes during the menstrual cycle. *Angiology*. 1969;20:624–627.
69. Colucci WS, Gimborne MA Jr, McLaughlin MK, Halpern W, Alexander RW. Increased vascular catecholamine sensitivity and α -adrenergic receptor affinity in female and estrogen-treated male rats. *Circ Res*. 1982;50:805–811.
70. Kondo K, Okuno T, Eguchi T. Vascular action of high dose estrogen in rats. *Endocrinol Jpn*. 1980;27:307–313.
71. Altura BM, Altura BT. Influence of sex hormones, oral contraceptives and pregnancy on vascular muscle and its reactivity. In: Carries O, Shikata S, eds. *Factors Influencing Vascular Reactivity*. Tokyo, Japan: Igaku-Shoin; 1977: 221–254.
72. de Trafford JC, Lafferty K, Potter CE, Roberts VC, Cotton LT. An epidemiological survey of Raynaud's phenomenon. *Eur J Vasc Surg*. 1988;2:167–170.
73. Spittell JA, Raynaud's phenomenon and allied vasospastic disorders. In: Jurgens JL, Spittell JA, Fairbairn JF, eds. *Peripheral Vascular Disease*. Philadelphia, Pa: WB Saunders; 1980: 588.
74. Heslop J, Coggon D, Acheson ED. The prevalence of intermittent digital ischemia (Raynaud's phenomenon) in a general practice. *J R Coll Gen Pract*. 1983;33:85–89.
75. Lafferty K, de Trafford JC, Pottew VC, Cotton TL. Reflex vascular response in the finger to contralateral stimuli during the normal menstrual cycle: A hormonal basis of Raynaud's phenomenon. *Clin Sci*. 1985;68:639–645.
76. Terregino CA, Siebold VACA. Influences of the menstrual cycle on Raynaud's phenomenon and on cold tolerance in normal women. *Angiology*. 1985;36:88–95.
77. Kenshalo DR. Changes in the cool threshold associated with phases of the menstrual cycle. *J Appl Physiol*. 1966;21:1031–1039.
78. Cunningham DJ, Cabanac M. Evidence from behavioral thermoregulatory responses of a shift in set point temperature related to the menstrual cycle. *J Physiol (Paris)*. 1971;63:236–238.
79. Kollias J, Bartlett L, Bergsteinova B, Skinner JS, Buskirk ER, Nicholas WL. Metabolic and thermal response of women during cooling in cold water. *J Appl Physiol*. 1974;36:577–580.
80. Modell JH. *Pathophysiology and Treatment of Drowning and Near-Drowning*. Springfield, Ill: Charles C Thomas; 1971.
81. Tabling JH. Near-drowning and its treatment. In: Pozos RS, Wittmers LE, eds. *The Nature and Treatment of Hypothermia*. Minneapolis, Minn: University of Minnesota Press; 1983: 221–231.
82. Levin DL. Near-drowning. *Crit Care Med*. 1980;8(10):590–595.
83. Bolte RG, Black PG, Bowers RS, Thorne JK, Corneli HM. The use of extracorporeal rewarming in a child submerged for 66 minutes. *JAMA*. 1988;260:377–379.
84. Gooden BA. Why some people do not drown: Hypothermia versus the diving response. *Med J Australia*. 1992;157(2):629–632.
85. Young AJ. Homeostatic responses to prolonged cold exposure: Human cold acclimatization. In: Fregly JM, Blatteis CM, eds. *Handbook of Physiology*. Section 4, Vol 1. *Environmental Physiology*. New York, NY: Oxford University Press; 1996: 419–438.
86. Nelms DJ, Soper DGJ. Cold vasodilation and cold acclimatization in the hands of British fish filleters. *J Appl Physiol*. 1962;17:444–448.

87. Eagan CJ. Local vascular adaption to cold in man. *Fed Proc.* 1963;22:947–952.
88. Lapp MC, Gee GK. Human acclimatization to cold water immersion. *Arch Environ Health.* 1967;15:568–579.
89. Young AJ, Muza SR, Sawka M, Gonzales RG, Pandolf KB. Human thermoregulatory responses to cold air are altered by repeated cold water immersion. *J Appl Physiol.* 1986;60(5):1542–1548.
90. Bittel JH. Heat debt as an index for cold adaptation in men. *J Appl Physiol.* 1987;62(4):1627–1634.

Chapter 17: ATTACHMENT

SHIPWRECK AND HYPOTHERMIA: EXPERIENCES GAINED BY THE GERMAN NAVY, 1939–1945

As a result of the loss of documents at the end of World War II, the German Naval Medical Service has not been able as yet to prepare a comprehensive medical report, but only managed to present an overview obtained by individual existing reports. Thus, it is also no longer possible to determine the number of naval personnel who died as a result of hypothermia or fatigue or the number of civilians killed during the great ship foundering in the course of the evacuation of the East in 1945. According to estimates, however, tens of thousands of people died in the icy floods.

Like the states of the western allies, the German Naval Medical Service was inadequately prepared for hypothermia problems of shipwrecked persons at the beginning of the war. Although many members of the Navy had been shipwrecked during World War I, the rescue capabilities at the beginning of the century were limited to such an extent that most persons had already died when they were recovered, and the cause of death was simply considered "drowning." Thus, an original clinical picture of hypothermia was neither assumed nor scientifically examined until the outbreak of World War II.

The 1940 publications of *The Medical Advisor for Submarines* and the *Bulletin on Medical Measures to be Taken in the Case of Submarine Accidents* did not yet deal with issues concerning hypothermia or cold water rescue. Only the improved rescue capabilities, and in particular, the events during the occupation of Norway in spring 1940, with a large number of shipwrecked persons in the cold waters of the Skagerrak and North Sea as well as the now frequent sea engagements in the northern area, forced those in command to intensively work on medical issues concerning distress at sea. The Army and Air Force only started to deal with similar problems after the mass occurrence of cold injuries during the winter battle near Moscow in 1941, and because of the large number of ditched aircraft crews suffering from cold injuries. At this point, it should be specifically pointed out that the Naval Medical Service was in no way involved in the hypothermia experiments in concentration camps, although it profited passively by the results of these experiments.

For the invasion operation, almost the entire German fleet was standing in Scandinavian waters in the morning hours of 9 April 1940. There were various sea engagements and several thousands of shipwrecked German forces. The available reports give evidence of the amazingly inadequate preparation and of the ignorance of the crews and embarked personnel, as well as of the poor medical facilities in view of the risks imposed by shipwreck and hypothermia. What alarmed the helpers again and again was the frequently observed phenomenon of the "afterdrop." After the foundering of a troop carrier, 79 members of the Army were drifting in the Skagerrak at a water temperature of 1°C. Thirty of the crew had taken off their clothes to enable them to swim better; the rest wore protective clothing. None of those who remained clothed died; after they were rescued and had changed into dry clothes they were fit for duty again. Those who had removed their clothes lay shivering in the berths of the destroyer crew after their rescue. Subjectively, they felt well and still comprehensively discussed the individual phases of the torpedoing. Suddenly, one after the other fell silent and lifelessly sank back onto the berth. They all died within 6 hours. The ship's doctor, who was overtaxed with the treatment of the casualties, immediately administered Lobelin and cardiovascular drugs to the collapsed patients, but without success (Tidow, 1960, p38).

At the same time, the cruiser *Blücher*, with a crew of 1,400 men and approximately 650 embarked Army soldiers, was torpedoed in the Oslo Fjord. The water temperature in this area was 2°C, the air temperature 0°C. The distance from the foundering ship to the dry land was only 300 to 400 meters, or about 20 minutes' swimming. Still, hundreds of soldiers died from fatigue or hypothermia. The surviving third ship's doctor, naval medical assistant Dr Pietsch, reported:

On the smaller islands many numb persons were lying who refused to jump into the water despite shouts from the shore to swim the last meters to the shore. Upon that, I jumped into the water, reached some islands and was able ... to take 6 men ashore. In one case resuscitative measures were unsuccessful. All others were recovered in front of lighted fires some faster, others slower (Medical Action Report, pp116–117).

Only after June 1941 did the Naval Medical Office in the German Naval Supreme Command for ships and boats without doctors issue preliminary directives for the treatment of rescued shipwrecked persons. The directives demanded the *slow* warming of patients suffering from severe hypothermia. The patients were initially to be kept in cool rooms, rubbed, and packed in dry clothes. These directives, however, taking individual circumstances into account, were already slightly modified to the following:

The decision as to which rescued persons are to be treated as patients suffering from hypothermia cannot alone be made on the basis of duration of stay in the water and the prevailing water temperature. The degree of hypothermia varies from individual to individual. Therefore, the objective impression must be also taken into consideration.

This directive attached particular importance to the careful transport of patients suffering from hypothermia on the receiving ship.

By 1942, the principles of treatment had changed. Now the *rapid* warming therapy was propagated as the best treatment method for patients suffering from severe hypothermia. This opinion was retained until the end of the war. Thus, the *Medical Advisor for Submarines* formulated in 1944:

- a. Shipwrecked persons suffering from hypothermia must in any case be subjected to an immediate intensive warming. This rewarming is accomplished by means of
 - hot baths at about 45°C, or
 - hot showers at about 45°C, or
 - repeated pouring over with fairly hot water.
- b. As an additional measure, cold compresses are applied to the forehead.
- c. Cardiac stimulants are to be slowly injected after the body has warmed internally, otherwise there is a risk of shock.
- d. The administration of alcohol has to be avoided until circulation, respirations and consciousness are completely normal again (*Medical Advisor for Submarines*, 1944, p32).

In addition, the regulation pointed out for the first time that in cases of immersions, unnecessary swimming movements should be avoided and only should rely on the floating capacity of the life jacket.

In the course of time, the positive effect of an appropriate and adequate clothing of the shipwrecked person in the water became more and more evident and led to changes in training. The crew members who during the first years of the war had taken off their warming clothing before jumping into the water in order to be able to swim better were now “repeatedly reminded to keep all clothes on the body, whenever possible” (Missler, 1981, p19). The success of such measures became evident during the foundering of a destroyer in the polar sea in May 1942. Thirty-eight crew members, most of the personnel from the upper deck with life jackets underneath their protective clothing reached—after a longer period of swimming at a water temperature of 1°C and an air temperature of -10°C and a wind force of 5—floating parts of the wreck that were constantly flooded by the sea. Six hours after the sinking, a submarine took in the shipwrecked persons. During the examination, the rescue personnel noticed that the upper bodies were in parts still dry while the outer clothing had turned into icy armor, which had to be cut open with knives and saws. Therapeutically, the rapid rewarming was performed; however, three boiler men wearing lighter clothing died after a short time.

Various reports also point out the important role of a stable psychical constitution and a strict discipline of shipwrecked persons suffering from hypothermia. For example, in autumn 1942, a lifeboat with 4 survivors and 20 corpses reached the coast of northern Norway after a cruise of almost 9 days. At wind force 7, a sea disturbance of 5–6, with only a small supply of food and inadequate clothing, the passengers were completely soaked and suffering from hypothermia. The chief coxswain had the command in the boat and held the control fin in his arms with his last ounce of strength, because his hands were frostbitten and he could no longer use them. During the cruise many of the passengers suffered from states of delusion and confusion and force had to be used to prevent them from jumping overboard. Discipline was good until the end.

In another case, a rescued shipwrecked person in the wintry Baltic Sea in 1945 reported on a person who was shipwrecked for the second time and who, in an almost hopeless situation, motivated a group of swimmers to hold out until their rescue (compare Missler, 1981).

The extraordinarily different circumstances in the area of submarine warfare in relation to shipwreck and hypothermia is illustrated by the following report on the last mission of the submarine U 550 at the East Coast of the United States on 16 April 1944 (compare Haenert, 1994). It shows in an exemplary manner how shipwrecked submarine crews were not always taken in by enemy vessels but were sometimes abandoned to their fates:

During a convoy battle, the type IX C submarine with a crew of 57 men was pursued with depth charges by superior US screen units and forced to surface. When the upper hatch was opened, three destroyers lying in a circle around the submarine fired at it with their internal guns. Resistance was no longer possible amidst the hail of shells; rather, the U 550 quickly foundered so that the crew left the submarine in two groups and swam away from the wreck. The only rescue facilities on board the submarine were divers and several one-man inflatable lifeboats. As protective clothing, parts of the crew wore a leather combination; others had not found the time to put on warmer clothing. The embarked naval medical assistant, Dr Torge, did not have the opportunity to take any additional lifesaving measures. The larger group of about 40 persons under the command of the first officer on watch swam toward the nearest US destroyer, as ordered. The destroyer, however, suddenly pulled away and did not make any rescue attempt. All of the soldiers died of hypothermia. The second group, 13 members of the crew, swam in the 5°C cold water for about 15 minutes to the USS *Joyce* drifting the vicinity, which took them aboard. On board, medical treatment was provided to those who suffered from moderate hypothermia or gunshot injuries.

With the activation of the small combat elements of the German Navy late in 1944, hypothermia problems gained a new significance. The crew members of these very different surface and subsurface vessels were particularly exposed to the cold. Therefore, the Navy tested cold protection measures in September 1944 for the first time. The first tests were performed on the foam suits of the Air Force, during which the soldiers noticed not only the significant insulation provided but also the good buoyancy and horizontal position in the water that the foam suits allowed. Later, the wool-in-plush underwear of the frogmen was also improved (compare Hartmann and Noeldeke, 1994).

Irrespective of all efforts aimed at improving hypothermia therapy and prophylaxis, the effects of hypothermia reached a sad climax during the disastrous foundering in the wintry Baltic Sea in the course of the evacuation of the eastern German territories. Expert prophylaxis and treatment were impossible from the very beginning, in the view of the large number of refugees. As examples of the terrible death toll at the end of the war stand two overcrowded refugee ships, the *Wilhelm Gustloff* and the *Goya*, which were torpedoed on 30 January and 17 April 1945, respectively, carrying about 6,000 people each, mainly women and children. Both ships sank and their passengers died of exposure to the cold.

SOURCES

1. Binder F, Schluenz HH. *Schwerer Kreuzer Bluecher*. Herford; 1990.
2. Haenert W. *Ueberleben auf See. U-bootrettung vor fuenfzig Jahren und heute*. Unveroeffentlichtes Manuskript; 1994.
3. Hartmann GV, Noeldeke H. *Der Sanitaetsdienst bei den Kleinkampf Verbaenden der Kriegsmarine*. Unveroeffentlichtes Manuskript; 1994.
4. Matschke RG. *Ueberleben auf See. Medizinische Aspekte des Schiffbruechigen in historischen Darstellungen*. Duesseldorf; 1975.
5. *Medical Action Report. Cruiser Bluecher*. Bundesarchiv-Militaerarchiv Freiberg; RM 92/5088; 116–119.
6. *Medical Advisor for Submarines*. Published from the German Submarine Command. Berlin; 1940.
7. *Medical Advisor for Submarines*. Published from the German Naval Supreme Command. Berlin; 1944. Bundesarchiv Militaerarchiv Freiberg; RMD 8/276.
8. Missler H. Erfahrungsbericht eines Schiffbruechigen. In: *Unterkuehlung im Seenotfall. Bericht ueber das Symposium vom 25. Bis 27.4.1980*. Cuxhaven, Muenchen; 1981: 18–20.
9. Sanitaetsdienst der Kriegsmarine. II. Kriegsbericht abgeschlossen am 31.08.1941. Bundesarchiv-Militaerarchiv Freiberg; RM 7/95.
10. Schadowaldt H. Schiffbruechige im kalten Wasser. *Leserbrief Deutsches Aerzteblatt-Aerztliche Mitteilungen*. 1966;63:1072–1075.
11. Schoen H. *Die Gustloff-Katastrophe*. Stuttgart; 1984.
12. Tidow R. Aerztliche Fragen bei Seenot. *Wehrmedizinische Mitteilungen*. 1960;Heft 2:17–20, Heft 3:37–41.
13. Vorlaeufige Richtlinien fuer die Behandlung geretteter Schiffbruechiger. Hrsg. Vom Oberkommando der Kriegsmarine. Marinemedizinalamt; Juni 1941.

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